Advanced Life Support algorithm

Chapter 6

Contents

Learning outcomes

To enable you to:

- Manage a cardiac arrest using the ALS algorithm
- Understand the importance of minimising interruptions to chest compressions during CPR
- Appropriately and promptly treat shockable and non-shockable rhythms
- Know when and how to give drugs during cardiac arrest
- Treat the reversible causes of cardiac arrest
- Understand the role of waveform capnography during CPR

Introduction

Heart rhythms associated with cardiac arrest are divided into two groups:

- shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)
- non-shockable rhythms (asystole and pulseless electrical activity (PEA)).

The main difference in the treatment of these two groups of arrhythmias is the need for attempted defibrillation in patients with VF/pVT. Other 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 and 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 in special circumstances (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/pVT. Although drugs and advanced airways are still included among ALS interventions, they are of secondary importance to high quality, uninterrupted chest compressions and when appropriate early defibrillation. In the most recent ALS guidelines there is an increase in emphasis on monitoring during CPR, especially the use of waveform capnography. Also the identification and treatment of reversible causes of cardiac arrest, including when able the use of focused cardiac and lung ultrasound assessment.

Basic Life Support

D — **Dangers?**

R — **Responsive?**

S — **Send** for help

A — Open **Airway**

B — Normal **Breathing?**

C — Start **CPR**
30 compressions : 2 breaths

D — Attach **Defibrillator (AED)**
as soon as available, follow prompts

Continue CPR until responsiveness or normal breathing return

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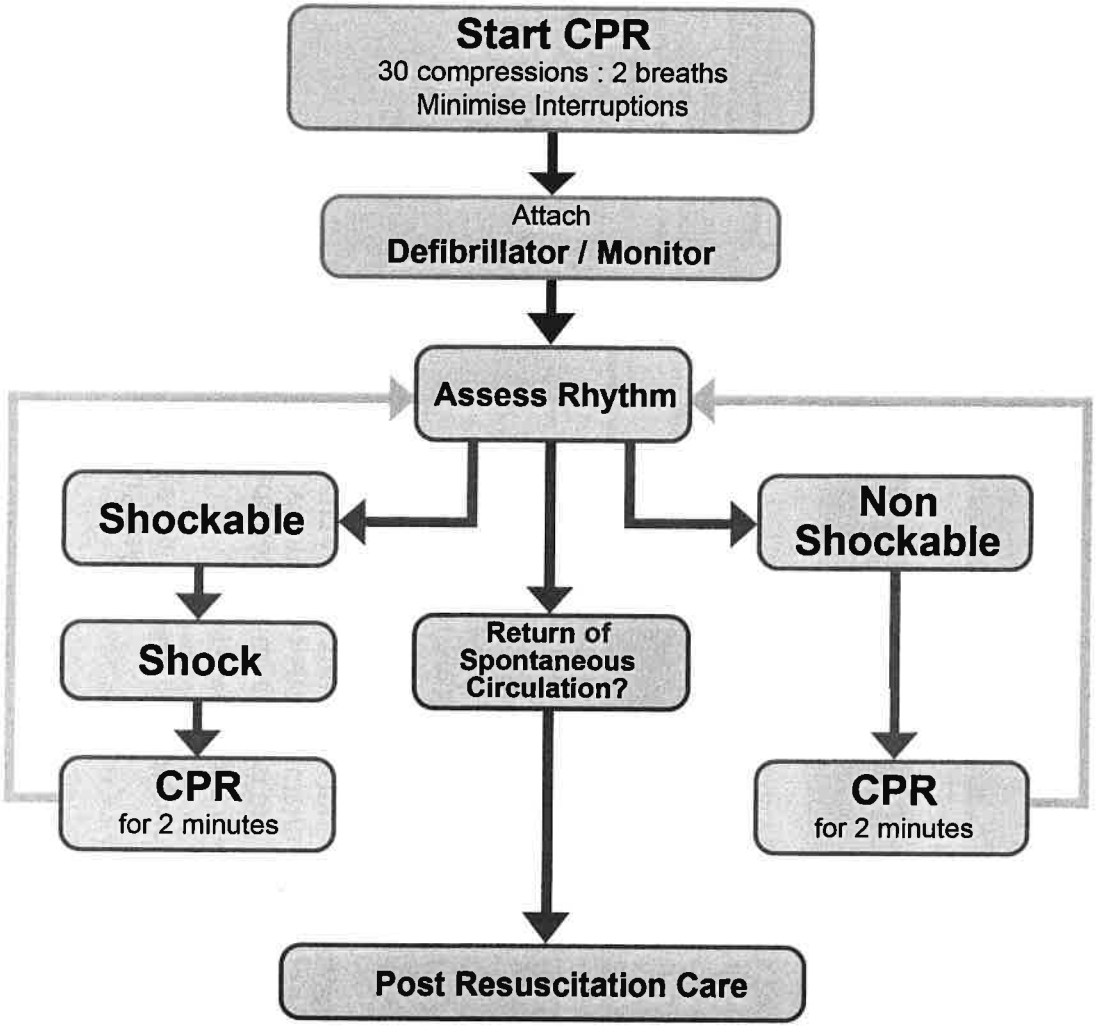
ANZCOR Basic Life Support chart

Advanced Life Support for Adults



During CPR

Airway adjuncts (LMA / ETT)
Oxygen
Waveform capnography
IV / IO access
Plan actions before interrupting compressions
(e.g. charge manual defibrillator)

Drugs

Shockable

* Adrenaline 1 mg after 2nd shock
(then every 2nd loop)
* Amiodarone 300mg after 3 shocks

Non Shockable

* Adrenaline 1 mg immediately
(then every 2nd loop)

Consider and Correct

Hypoxia
Hypovolaemia
Hyper / hypokalaemia / metabolic disorders
Hypothermia / hyperthermia
Tension pneumothorax
Tamponade
Toxins
Thrombosis (pulmonary / coronary)

Post Resuscitation Care

Re-evaluate ABCDE
12 lead ECG
Treat precipitating causes
Aim for: SpO2 94-98%, normocapnia and normoglycaemia
Targeted temperature management

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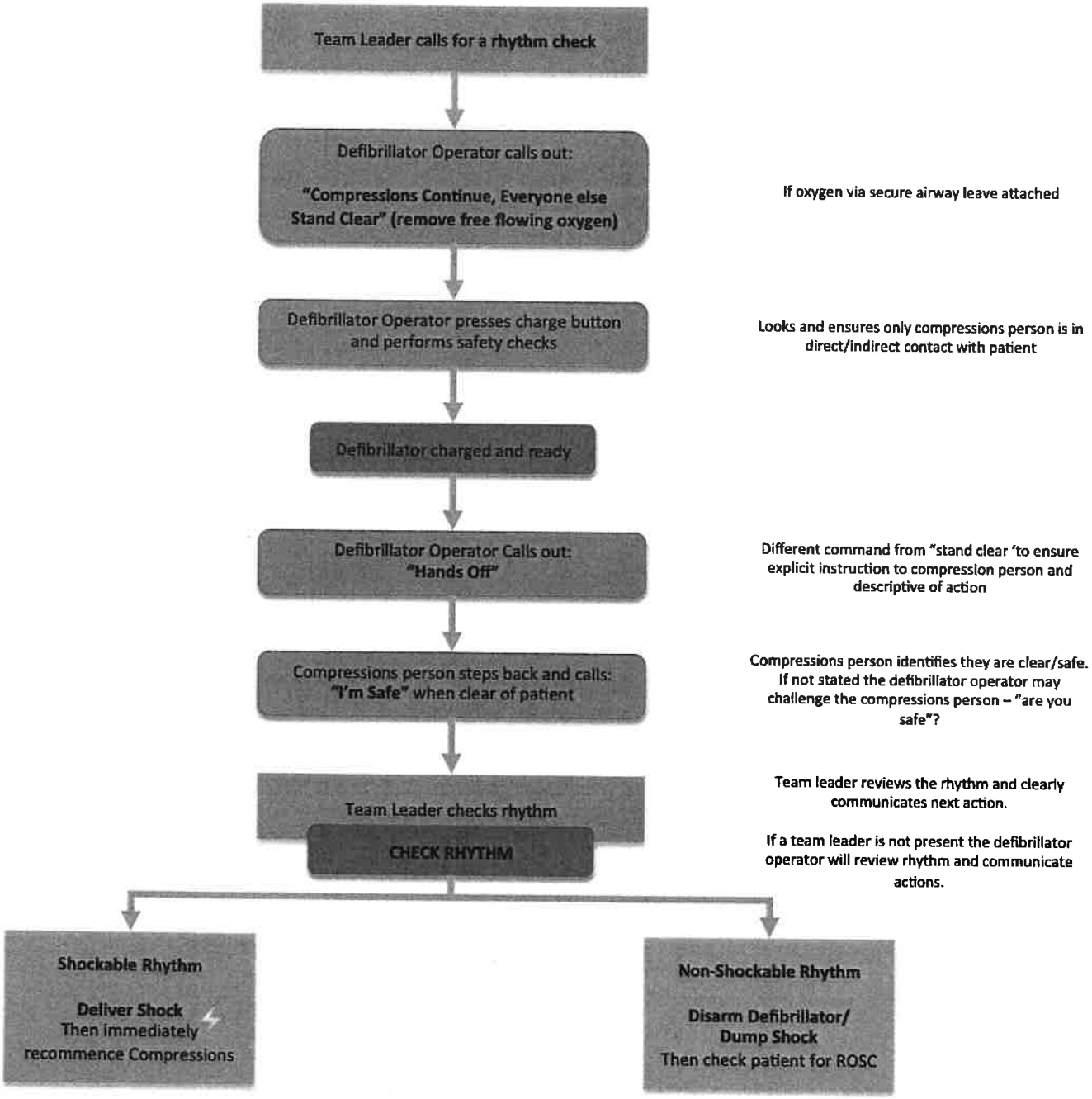
Figure 6.1 Adult Advanced Life Support algorithm

Manual Mode Defibrillation Sequence

At no time should rescuer safety be compromised



This is not intended as a script and wording may change to suit local needs. The exact wording is not as important as the clear unambiguous communication of the principles and actions of the skill. The aim is to minimise interruptions to compressions.

Safety is the overriding concern at all times. In the absence of a Team Leader - Defibrillator Operator analyses rhythm

If a patient is tolerating effective chest compressions without displaying ROSC and VT is found when compressions are paused, the shock should be immediately delivered. There is no need to check for a pulse prior to delivering a shock to a patient tolerating effective chest compressions found to be in a shockable rhythm.

Figure 6.1a Manual mode defibrillation sequence

Shockable rhythms (VF/pVT)

The first monitored rhythm is VF/pVT in approximately 20 to 30% of cardiac arrests, in-or out-of-hospital. VF/pVT 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/pVT)

A manual defibrillator is used in the sequence described below. Further information about defibrillation can be found in Chapter 7.

1. Confirm cardiac arrest – check for signs of life or if trained to do so, check breathing and pulse simultaneously.
2. Call emergency response/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 mid-axillary line, (usually shown on pad placement diagram on the pads).
4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team. Pauses in chest compressions should be brief (aiming for less than 5 s).
   - A pause is the ideal time to swap the person performing chest compressions to reduce rescuer fatigue so needs to be considered and planned.
5. Without stopping chest compressions, plan to charge defibrillator. Team leader indicates the need to perform a rhythm check.
6. The designated person selects the appropriate energy on the defibrillator (200 J biphasic for first shock and may increase to maximum (360 J) for second and subsequent shocks) - then calling "COMPRESSIONS CONTINUE-EVERYONE ELSE STAND CLEAR" and to remove (any free flowing) oxygen delivery device as appropriate while pressing the charge button (Figure 4.3), simultaneously.
7. Ensure that the rescuer giving the compressions is the only person touching the patient (directly or indirectly). While the defibrillator is charging, confirm all rescuers other than the individual performing the chest compressions is "standing clear" by performing visual safety check. Including, ensuring that free flowing oxygen is removed.

Figure 6.2 Continuous chest compressions during charging with a manual defibrillator. Warn all rescuers other than the individual performing the chest compressions to "stand clear". This patient has a tracheal tube and remains connected to the oxygen supply. A CPR quality and feedback device is used by the rescuer giving chest compressions.

Figure 6.3 Shock delivery. No one is touching the patient during shock delivery

8. Once the defibrillator is charged and the safety check is complete, stop chest compressions. Call out/tell the individual who is performing the chest compressions "HANDS OFF" to pause compressions.
9. When person performing compressions confirms they are clear by stating "I'M SAFE" e.g. by placing both hands in the air, making eye contact and confirming they are clear and it is safe to act.
   - If the compressions person does not clearly indicate they are safely away from the patient, the defibrillator operator may ask/challenge them with "ARE YOU SAFE." This is to confirm safety before reviewing the rhythm.
10. Confirm/Check rhythm (Team Leader confirms VF/VT), if shockable deliver the shock safely (Figure 4.4). (If the patient has had no response to effective compressions and VT is displayed - treat as pulseless VT).
11. Then without reassessing the rhythm or feeling for a pulse, immediately restart CPR (using a ratio of 30:2), starting with chest compressions (Figure 4.5).
    - Do not reassess the rhythm or feel for a pulse. This pause in chest compressions should be brief and no longer than 5 s.
12. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR. Do not interrupt compressions until rhythm check, (unless signs of circulation.
13. If VF/pVT, repeat steps 4 – 12 above and deliver a second shock. Resume chest compressions immediately. Give adrenaline 1 mg IV/IO following the second shock during CPR.
14. If VF/VT persists, repeat steps 4 – 12 above and deliver a third shock. Resume chest compressions immediately. Give amiodarone 300 mg IV while performing a further 2 min CPR.
15. Repeat this 2 min loop CPR - defibrillation sequence if VF/VT persist

16. Give further adrenaline 1 mg IV/IO after alternate shocks (i.e., alternate 2 minute loops of CPR).
17. If organised electrical activity compatible with a cardiac output is seen during a rhythm check, disarm defibrillator/dump charge into machine then, seek evidence of ROSC: (check for signs of life, a central pulse and end-tidal CO_2 if available).
    a. If there is ROSC, start post-resuscitation care.
    b. If there are no signs of ROSC, continue CPR and switch to the non-shockable algorithm.
18. If asystole is seen, disarm defibrillator/dump charge into machine then continue CPR and switch to the non-shockable algorithm. The interval between stopping compressions and delivering a shock must be minimised. Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation

Chest compressions are resumed immediately after delivering a shock (without checking the rhythm or a pulse) because:

- Even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation. The time until ROSC can be longer than two minutes in as many as 25% of successful shocks.
- 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.

Current evidence is insufficient to support or refute the routine Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs during CPR. The use of both adrenaline and amiodarone is currently recommended, based largely on an increased short-term survival in humans.

- The first dose of adrenaline is given during the 2–min period of CPR after delivery of the second shock.
- Give amiodarone 300 mg after three defibrillation attempts (three shock loops in an arrest/the shocks do not need to be in sequential loops).

Subsequent doses of adrenaline are given after alternate 2-min loops of CPR for the aduration of the cardiac arrest. If VF/VT persists, or recurs, a second dose of 150 mg amiodarone may be considered after a total of five defibrillation attempts. Lignocaine 1 mg kg^{-1}, may be used as an alternative if amiodarone is not available, but do not give lignocaine if amiodarone has been given already.

It is important in shock-refractory VF/pVT 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 shockable rhythm - VF/pVT.

When the rhythm is checked 2 min after giving a shock, if a non-shockable rhythm is identified, and the rhythm is one that could be compatible with a pulse, palpate a central pulse. Also look for other evidence of ROSC (e.g. sudden increase in end-tidal CO_2 or evidence of cardiac output on any invasive monitoring equipment). Any rhythm check must be brief, and pulse checks undertaken only if a rhythm that may be compatible with a pulse is observed. If a rhythm compatible with a pulse 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 ROSC. If there is any doubt about the presence of a palpable pulse, 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.

If there is any doubt about whether the rhythm is asystole or extremely fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation as continuing high quality CPR may improve the amplitude and frequency of the VF and improve the chance of subsequent successful defibrillation to a perfusing rhythm. If the rhythm is clearly identified as VF, attempt defibrillation.

Precordial thump

A precordial thump has a very low success rate for cardioversion of a shockable rhythm. Its routine use is not recommended. Consider a precordial thump only when it can be used without delay whilst awaiting the arrival of a defibrillator in a monitored pVT arrest (less chance of success in VF). 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.

Witnessed and monitored VF/pVT cardiac arrest

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

- Confirm cardiac arrest and shout for help
- If the initial rhythm is confirmed as VF/pVT, give up to three quick successive (stacked) shocks if the first shock can be delivered within 20 seconds of onset of arrest.
- Rapidly check for a rhythm change and, if appropriate check for a pulse and other signs of ROSC after each defibrillation attempt
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

The patient should be well perfused and oxygenated immediately pre-arrest for best chance of successful defibrillation.

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

If this initial three-(stacked) shock strategy is unsuccessful for a monitored VF/pVT cardiac arrest, follow the ALS algorithm. Treat these three (stacked) shocks as if only the first single shock has been given. These initial three stacked shocks are considered as giving the first shock in the ALS algorithm. The first dose of adrenaline should be given after another shock attempt if VF/pVT persists. Amiodarone is given after three shock attempts in different loops, irrespective of when they are given during the cardiac arrest (i.e. give amiodarone following two more loops with a shock being delivered, with the three stacked shocks counting as the first shock).

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity (other than ventricular tachyarrhythmia) that would normally be associated with a palpable pulse. These patients may 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. 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 rarely successful outside certain settings (e.g. recent onset, witnessed, post-operative, failed pacemaker - chapter 10).

Treatment for PEA and asystole

- Start CPR 30:2.
- Give adrenaline 1 mg IV/IO as soon as intravascular access is achieved, (ideally within first loop of CPR).
- Continue CPR 30:2 until the airway is secured - then continue chest compressions without pausing during ventilation.
- Recheck the rhythm after 2 min:
  - If electrical activity compatible with a pulse 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 or asystole):
  - continue CPR
  - recheck the rhythm after 2 min and proceed accordingly
  - give further adrenaline 1 mg IV after alternate 2-min loops of CPR
  - if VF/pVT at rhythm check, change to shockable side of algorithm.

During CPR

During the treatment of persistent VF/pVT or PEA/asystole, emphasis is placed on high 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, do not attempt defibrillation at this stage; instead, continue with CPR until the 2-min 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-min 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 (1/3 Anterior-Posterior Diameter or > 5 cm) and rate (100–120 min^{-1}), and ensure there is a recoil of the chest at the end of each compression.

As soon as the airway is secured, continue chest compressions without pausing during ventilation. Change the individual undertaking compressions every 2 min or earlier if necessary.

Airway and ventilation

A bag-mask, or preferably, a supraglottic airway (SGA) should be used without the availibility of personnel skilled in tracheal intubation (Chapter 7). Once a SGA 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, interrupt chest compressions 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 5 s. After intubation, confirm correct tube position with waveform capnography, and secure the tube adequately. Once the patient's trachea has been intubated, continue chest compressions, at a rate of 100–120 min^{-1} without pausing during ventilation. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until after ROSC.

Monitoring during CPR

Several methods can be used to monitor the patient during CPR and potentially help guide ALS interventions. These include:

- Clinical signs such as breathing efforts, movements and eye opening can occur during CPR. These can indicate ROSC and require verification by a rhythm and pulse check, but can also occur because high quality CPR can generate a sufficient circulation to restore signs of life including consciousness.
- Pulse checks can be used to identify ROSC when there is an ECG rhythm compatible with a pulse, but may not present in those with low cardiac output states and a low blood pressure. The value of attempting to feel arterial pulses during chest compressions to assess the effectiveness of chest compressions is unclear. A pulse that is felt in the femoral triangle can indicate venous rather than arterial blood flow. Carotid pulsation during CPR does not necessarily indicate adequate myocardial or cerebral perfusion.
- Monitoring the heart rhythm through pads, paddles or ECG electrodes is a standard part of ALS. Motion artefact prevent reliable heart rhythm assessment during chest compressions.
- End-tidal carbon dioxide measured with waveform capnography. The use of waveform capnography during CPR is addressed in more detail below.
- Feedback or prompt devices can monitor CPR quality data such as compression rate and depth during CPR, and provide real-time feedback to rescuers (Figure 6.2). Be aware that some devices fail to compensate for compression of the underlying mattress during CPR on a bed when providing feedback. The use of these devices may be considered as part of a broader system of care that includes CPR quality improvement initiatives such as debriefing based on the data collected.
- Blood sampling and analysis during CPR is used to identify potentially reversible causes of cardiac arrest. Avoid finger prick samples because they may not be reliable; instead, use samples from veins or arteries. Blood gas values are difficult to interpret during CPR. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state. Analysis of central venous blood may provide a better estimation of tissue pH.
- Invasive cardiovascular monitoring in critical care settings (e.g. continuous arterial blood pressure and central venous pressure monitoring). Invasive arterial pressure monitoring will enable the detection of even very low blood pressure values when ROSC is achieved.
- The use of focused echocardiography/ultrasound to identify and treat reversible causes of cardiac arrest, and identify low cardiac output states ('pseudo-PEA') is discussed below.

Waveform capnography during advanced life support

Carbon dioxide is a waste product of metabolism with approximately 400 L produced each day by an individual. It is carried in the blood to the lungs where it is exhaled. End-tidal CO_2 is the partial pressure of CO_2 at the end of an exhaled breath. It reflects cardiac output and lung blood flow. During CPR, end-tidal CO_2 values are low, reflecting the low cardiac output generated by chest compression. An increase during CPR in the monitored value of CO_2 to normal (35 to 45 $mmHg^{-1}$) or near normal may indicate ROSC. Waveform capnography enables continuous real time end-tidal CO_2 to be monitored during CPR. It works most reliably in patients who have a tracheal tube, but can also be used with a supraglottic airway or bag-mask.

End-tidal carbon dioxide is the partial pressure of CO_2 at the end of an exhaled breath. It reflects cardiac output and pulmonary blood flow (CO_2 is transported by the venous system to the right side of the heart and then pumped to the lungs by the right ventricle), as well as the ventilation minute volume. During CPR, end-tidal CO_2 values are low, reflecting the low cardiac output generated by chest compression. Waveform capnography enables continuous real time end-tidal CO_2 to be monitored during CPR. It works most reliably in patients who have a tracheal tube, but can also be used with a supraglottic airway device or bag mask.

The role of waveform capnography during CPR

- Ensuring tracheal tube placement in the trachea. Correct tube placement relies first on observation and auscultation to ensure both lungs are ventilated.
- Monitoring of the ventilation rate during CPR and avoiding hyperventilation.
- Monitoring the quality of chest compressions during CPR. End-tidal CO_2 values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value.
- Identifying ROSC during CPR. An increase in end-tidal CO_2 during CPR may indicate ROSC, and prevent unnecessary and potentially harmful administration of adrenaline in a patient with ROSC. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- Prognostication during CPR. Precise values of end-tidal CO_2 depend on several factors including the cause of cardiac arrest, bystander CPR, chest compression quality, ventilation rate and volume, time from cardiac arrest and the use of adrenaline. Values are higher after an initial asphyxial arrest and with bystander CPR, and decline over time after cardiac arrest. Low end-tidal CO_2 values during CPR have been associated with lower ROSC rates and increased mortality, and high values with better ROSC and survival. End-tidal CO_2 values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

Practical aspects of waveform capnography

A capnograph is a device that displays a waveform of the concentration of CO_2 as it varies during expiration and a numerical value. This is usually termed waveform capnography and is the most useful display for clinical use.

Equipment

Portable monitors that measure the end-tidal CO_2 and display the waveform are now readily available (Figure 6.4).
Most capnographs use side-stream sampling. A connector

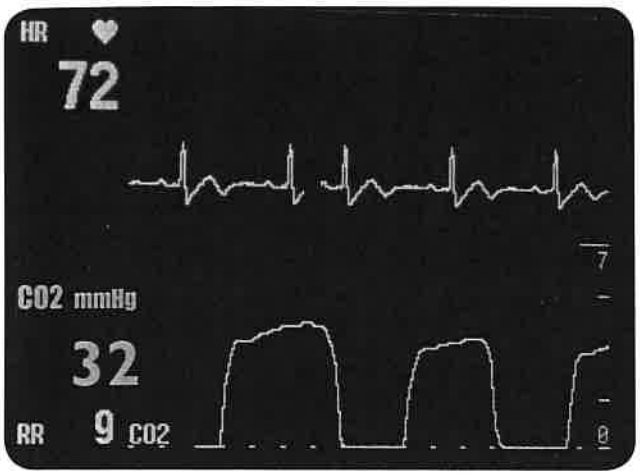


Figure 6.4 Example of a monitor screen showing end-tidal CO_2 waveform (capnography). This patient had a return of spontaneous circulation, and chest compressions have stopped. The ECG shows sinus rhythm with a rate of 72 min^{-1}. The end-tidal CO_2 waveform shows an end-tidal CO_2 value of 32mmHg, and a ventilation rate (RR) of 9 min^{-1}. On clinical assessment the patient had a palpable carotid pulse.

(T-piece) is placed in the breathing system, usually on the end of the tracheal tube or supraglottic airway device. This has a small port on the side to which is attached a fine bore sampling tube. A continuous sample of gas is aspirated (about 50 mL min^{-1}) and analysed by using the property of absorption of infrared light.

An alternative system is mainstream sampling in which the infrared source and detector are contained within a cell or cuvette which is placed directly in the breathing system, usually between the tracheal tube or supraglottic airway device and circuit. Gas is analysed as it passes through the sensor and none is removed from the system.

The capnography waveform

The shape of the waveform that is displayed will depend on the time scale and the patient's condition. (Figure 6.5). A capnogram consists of 4 phases and plots CO_2 concentration over time.

Phase I, respiratory baseline, is shown as A-B: This is the baseline and indicates the end of inspiration. In effect the concentration of CO_2 is being measured in air (or whatever gas is being delivered) which of course is virtually zero.

Phase II—also known as the expiratory upstroke—shown as B-C. The expiratory upstroke should be steep. B-C: This represents the start of expiration, with a rapid rise in the concentration of expired CO_2. The expired gas initially contains no CO_2 as it has come from the anatomical dead space (i.e. the volume of the respiratory tract that does not take part in gas exchange (larynx,

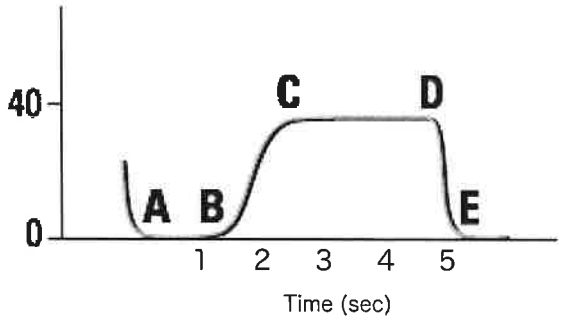


Figure 6.5 Typical capnography waveform from a single breath (note the time scale)

trachea, main bronchi)). As gas from alveoli that contains CO_2 starts to be expired, it mixes with this gas and the concentration rises.

Phase III, the expiratory plateau, represents exhalation of mostly alveolar gas; this is shown as C-D.

C-D: Eventually the alveolar plateau is reached. This represents gas from alveoli taking part in gas exchange. The slight gradual increase during this phase is due to the fact that not all alveoli empty at the same rate.

Point D is the EtCO2 level at the end of a normal exhaled breath; D: At the end of expiration the concentration of CO_2 is maximal, this is the end-tidal CO_2. In healthy patients, this is normally about 38 mmHg (35-45 mmHg), slightly lower than would be obtained if the arterial $PaCO_2$ was measured at the same point in time.

Finally, the inspiratory downstroke or Phase IV, shown as D-E.

D-E: As inspiration starts, air containing no CO_2 is mixed with a small amount of residual expired gas in the breathing circuit. This is rapidly diluted until gas containing no CO_2 is being inspired.

End-tidal CO_2 during CPR

Reliable end-tidal CO_2 monitoring during CPR will usually require a tracheal tube or, when there is a good seal, a supraglottic airway device. Figure 6.6 shows how end-tidal CO_2 values can change during CPR.

- The presence of an end-tidal CO_2 waveform indicates the tracheal tube is positioned in the airway. Check that both lungs are being ventilated by looking and by listening with a stethoscope as the tube may have passed down into a bronchus and only be ventilating one lung.
- The rate of ventilation is 10 min^{-1} (Figure 6.6).
- Soon after the second defibrillation attempt there is a significant increase in the end-tidal CO_2 value during CPR. This is often the first indicator of ROSC and often precedes other indicators such as the presence of a palpable pulse. It is a result of the improved circulation transporting accumulated CO_2 from the tissues to the lungs. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

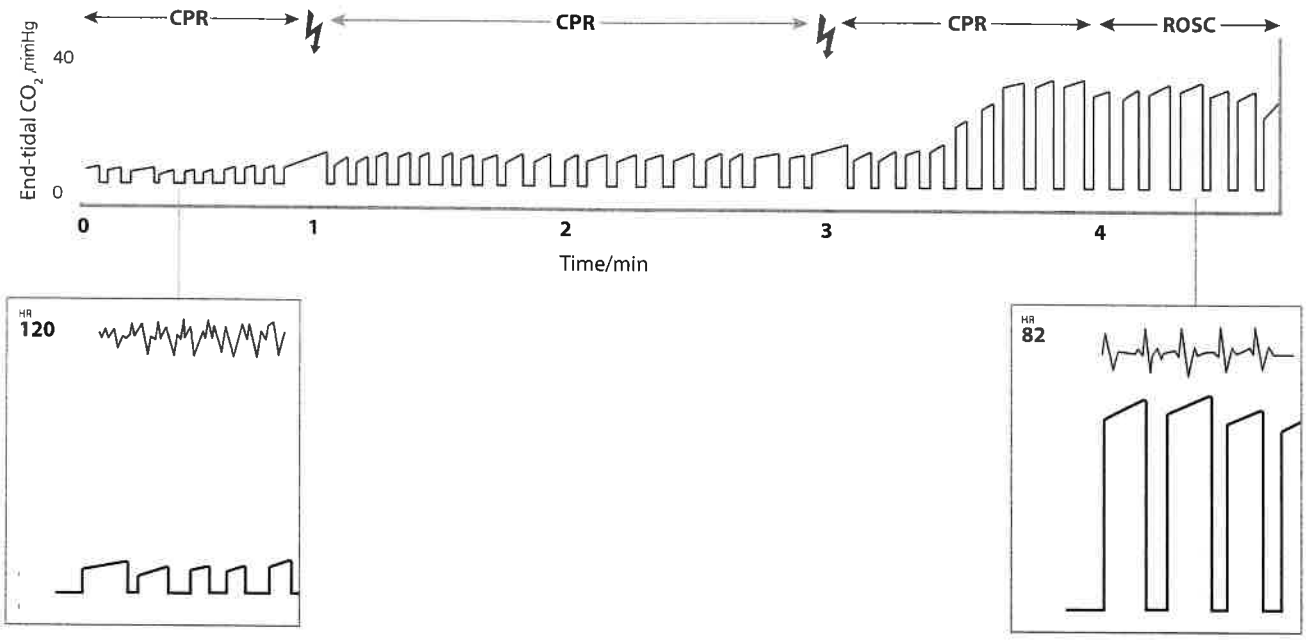


Figure 6.6 Waveform capnography showing changes in the end-tidal CO_2 during CPR and after ROSC. The boxes show the monitor displays at the times indicated. In this example the patient's trachea is intubated at zero minutes. The patient is ventilated at 10 min^{-1} and given chest compressions (indicated by CPR) at about two per second. A minute after tracheal intubation, there is pause in chest compressions and ventilation followed by a defibrillation attempt, and chest compressions and ventilation then continue. Higher-quality chest compressions lead to an increased end-tidal CO_2 value. There is a further defibrillation attempt after 2 min of chest compressions. There are then further chest compressions and ventilation. There is a significant increase in the end-tidal CO_2 value during chest compressions and the patient starts moving and eye opening. Chest compressions are stopped briefly, the monitor shows sinus rhythm and there is a pulse indicating ROSC. Ventilation continues at 10 min^{-1}.
CPR – cardiopulmonary resuscitation; ROSC – return of spontaneous circulation; HR – heart rate

- Failure to achieve an end-tidal CO_2 value > 10 mmHg (1.33 kPa) after 20 min of CPR is associated with a poor outcome in observational studies. A specific end-tidal CO_2 value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO_2 values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

Signs of life during CPR

If signs of life (such as regular respiratory effort, movement) or readings from patient monitors compatible with ROSC (e.g. increase in end-tidal CO_2 or arterial blood pressure waveform) appear during CPR, stop CPR briefly and check the monitor. If a rhythm compatible with a pulse 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.

Vascular access

The role of drugs during cardiac arrest is uncertain. Some patients will already have intravenous access at the time they have a cardiac arrest. If this is not the case ensure CPR had started and defibrillation, if appropriate, attempted before considering vascular access. Although peak drug concentrations are higher and circulation times 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 to facilitate drug delivery to the central circulation. This can be achieved with the use of a continuously running IV line that may in some cases be easier and more practical than intermittent bolus flushing.

If IV access is difficult or impossible, consider the IO route (Figure 6.7). Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a vein. Fluid and drug administration via IO route will need to be under pressure to achieve good flow rates. This may be achieved using a pressure bag or syringe. The proximal tibia and distal tibia may be the most practical in resuscitation. These are sites of IO access that minimally interfere with compressions or defibrillation and reduce potential crowding towards the head of the patient.

Intraosseous (IO) infusion as a means of vascular access has been recognised for close to a century and has seen resurgence in the last decade particularly for use in resuscitation in adults. A number of studies suggest it is a viable alternative to intravenous (IV) access. Intraosseous access is also quicker than central venous access in patients in whom peripheral venous access is not possible. Insertion of central venous catheters (CVC) during resuscitation requires considerable skill and may lead to prolonged interruptions to chest compressions. Current recommendations are to establish IO access if IV access is not possible or associated with a delay in obtaining vascular access.

Use of intraosseous access during resuscitation

The marrow of long bones has a rich network of vessels that drain into a central venous canal, emissary veins, and, ultimately, the central circulation. Therefore, the bone marrow functions may be viewed as a non-collapsible venous access route when peripheral perfusion is poor.

- The three main insertion sites for IO access recommended for use in adults are the proximal tibia or distal tibia and the proximal humerus.
  - Alternative sites include distal femur and sternum
  - Consideration on the site of access will need to include positioning of team members performing other tasks and roles.
  - A common site recommended for intraosseous (IO) insertion is the proximal tibia because it provides a flat surface with a thin layer of overlying tissue and ease of identifying landmarks. Also, it is distant from the airway and chest, where resuscitation attempts are in progress.
  - Locate a flat area of bone 2 cm medial to tibial tuberosity, or approximately 2 - 3 cm below the patella and 2 cm medial. Identifying these landmarks helps avoid hitting the growth plate.
  - Ideally perform insertion using sterile gloves and technique
  - Cleanse the area with a recommended solution and drape it.
  - Use local anaesthetic if required.
- Contraindications to IO access include:
  - trauma/fracture, infection or a prosthesis (e.g. joint replacement) at the target site
  - recent IO access (previous 48 h) in the same limb including a failed attempt
  - failure to identify the anatomical landmarks.
- Training in the specific device to be used is essential. Site of insertion, identification of landmarks and technique for insertion will differ depending on the device being used.
  - Errors in identification of landmarks or in insertion technique increase the risk of failure and complications.
- Once inserted, confirm correct placement before delivery of drugs or infusion of fluids. Attempt to aspirate from the needle; presence of IO blood indicates correct placement, absence of aspirate does not necessarily imply a failed attempt.
- There are reports of IO blood being used for laboratory analysis including glucose, haemoglobin and electrolytes. Samples must be labeled as bone marrow aspirate before being sent to the laboratory.
- Flush the needle to ensure patency and observe for leakage or extravasation. This is best achieved using an extension set flushed with 0.9% saline attached to the hub of the needle before use.
- Once IO access has been confirmed, resuscitation drugs including adrenaline and amiodarone can be infused. Fluids and blood products can also be delivered but pressure will be needed to achieve reasonable flow rates using either a pressure bag or a syringe.
- Follow the manufacturer's guidance both for securing the needle and the maximum length of time it can be left in place.
- Complications associated with IO access include:
  - extravasation into the soft tissues surrounding the insertion site
    - extravasation of hypertonic or caustic medications, such as sodium bicarbonate calcium chloride can result in necrosis of the muscle.
  - dislodgement of the needle
  - local heamatoma
  - compartment syndrome due to extravasation
  - fracture or chipping of the bone during insertion
  - pain related to the infusion of drugs/fluid
  - fat emboli
  - infection/osteomyelitis

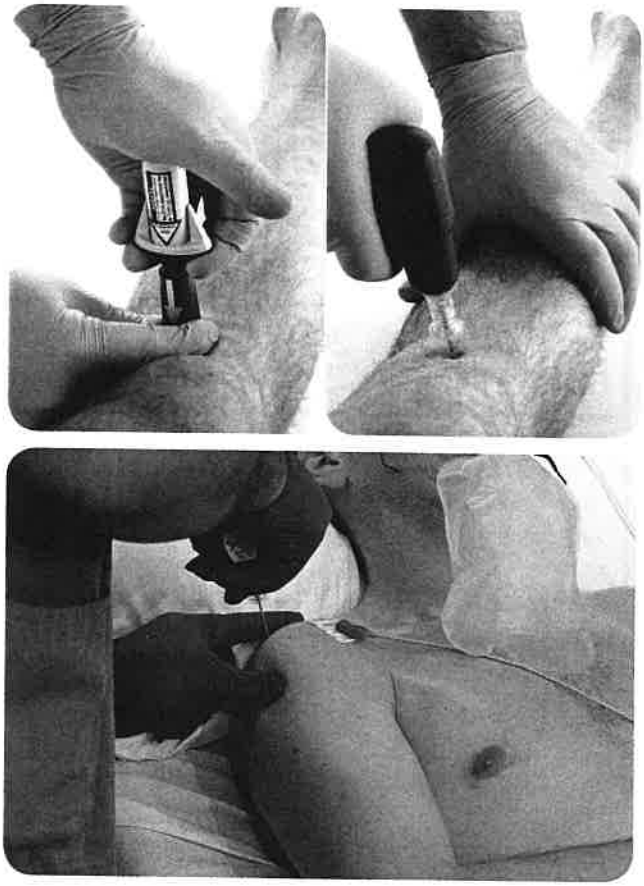


Figure 6.7 Intraosseous device insertion into proximal tibia and humerus using three different insertion methods

An IO line is as efficient as an intravenous route and can be inserted quickly, even in the most poorly perfused patients. Once alternative vascular access is established the IO access should be removed.

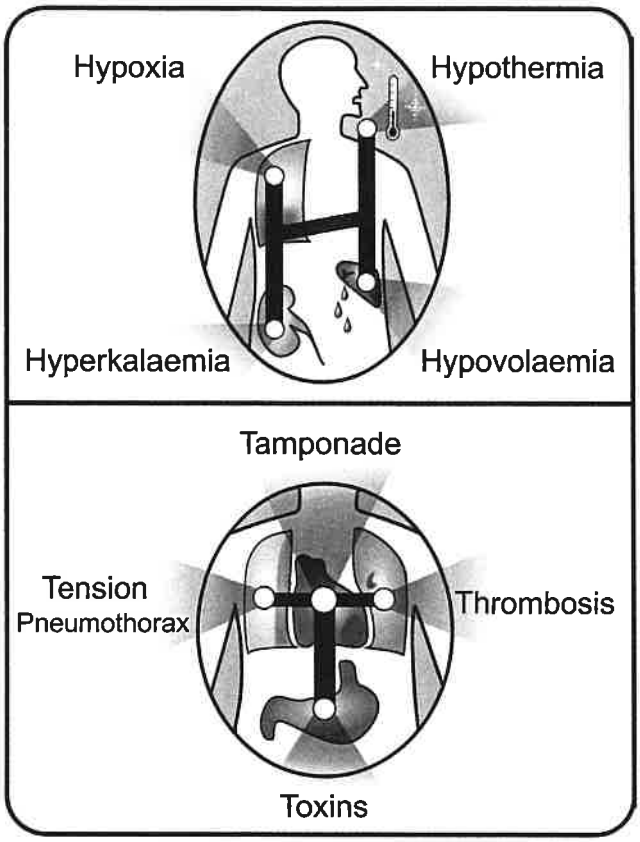


Figure 6.8 The four Hs and four Ts

Identification and treatment of reversible causes

Potential causes or aggravating factors for which specific treatment exists must be considered during any cardiac arrest. In seeking to identify the reversible causes in an arrested patient, consideration of the immediate pre-arrest history, examination and investigation need may to be included in the assessment. The identification and treatment of one cause of arrest does not preclude the presence of other reversible causes.

For ease of memory, these are divided into two groups of four based upon their initial letter - either H or T (Figure 6.8). More details on many of these conditions are covered in Chapter 12.

Hypoxia

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

The four Hs

Hypoxia

Minimise the risk of hypoxia by ensuring that the patient's lungs are ventilated adequately with 100% oxygen during CPR. 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. Where possible, seek evidence of pre-arrest state and observations, respiratory rate, SpO2 and effort of breathing.

If ROSC is achieved adjust the inspired oxygen to target an oxygen saturation of 94–98% (Chapter 13).

Hypovolaemia

Many hypovolaemic arrests present with pulseless electrical activity. However the possibility of shockable rhythms developing as the initial rhythm or during the arrest phase remains. Hypovolaemia may be due to severe haemorrhage. Evidence of haemorrhage may be obvious, (e.g. trauma), or occult (e.g. gastrointestinal bleeding, or rupture of an aortic aneurysm). Examination of the patient during CPR for obvious bleeding or bleeding internally may be required. If the patient is post operative a simple check of the wound or wound drains and observation/fluid charts may give some clues. Intravascular volume should be restored rapidly with fluid and blood transfusion, coupled with urgent interventions to stop the haemorrhage. Full cross matched blood may not be immediately available and alternative fluids and compatible blood transfusion may need consideration initially. The choice of the fluid used for resuscitation will be limited on the type of fluid available.

Other causes of hypovolaemia may not be obvious and include distributive shock (e.g. anaphylaxis and sepsis). In these cases the intravascular volume and filling are interrupted. Again fluid replacement is the initial strategy in these cases of hypovolaemia. For all hypovolaemic arrests more than one point of vascular access may be required.

Further supportive measures may be required once ROSC is achieved. This may include surgical intervention, other interventional procedures, inotropes and invasive monitoring.

Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders

These are detected by direct measurement/biochemical tests or suggested by the patient's medical history (e.g. renal failure (Chapter 12)). Where possible bedside or proximity testing should occur. A venous sample may be analysed by a blood gas analysis machine or bedside/point of care testing devices including the glucometer. ECG changes, (chapther 12) can be subtle, and do not always occur.

Hyperkalaemia - Severe elevation is ≥ 6.5 mmol L^{-1}

Intravenous calcium is indicated in the presence of hyperkalaemia. Calcium does not act to lower serum potassium levels, calcium directly antagonizes the myocardial effects of hyperkalemia by restoring cardiomyocyte resting membrane potential, thereby stabilising the cell membrane. Protective measures including administration of calcium chloride 10% (10 mL) and use of shifting agents (glucose 25 g + 10 units short acting insulin) and consider sodium bicarbonate 50 mmol IV if

severe acidosis or renal failure are present. On achieving ROSC the need for dialysis to remove potassium needs consideration.

Calcium gluconate may be used in place of calcium chloride when the chloride solution is not available. Calcium is often provided intravenously in two different forms: chloride and gluconate. An ampule of calcium gluconate – most commonly 10mL of the 10% formulation – contains 8.9 mg/mL of elemental calcium. An ampule of10 mL 10% calcium chloride provides a threefold higher concentration of elemental calcium (27.2 mg/mL). Another potential complicating factor is that calcium gluconate must be hepatically metabolised before its associated calcium becomes bioavailable. In the setting of cardiac arrest or patients with hemodynamic instability or poor liver function, it may be preferable to use calcium chloride.

Intravenous calcium chloride may be indicated in the presence of hyperkalaemia, hypocalcaemia, and calcium channel-blocker overdose. Increasing extracellular calcium concentrations creates a high calcium gradient that may cause calcium influx, leading to improvement in conduction disturbances and contractility.

Causes of acute hypocalcaemia include shock, sepsis, pancreatitis, and drug toxicities.

Hypokalaemia

Hypokalaemia is defined as a serum potassium level < 3.5 mmol L^{-1}.

Severe hypokalaemia is defined as a serum potassium level $<$ 2.5 mmol L^{-1} and may be associated with symptoms including arrhythmias. The assessment is direct measurement (ideally point of care), and evaluation of possible causes usually directly related to loss or intake including drugs, gastrointestinal and renal/dialysis loss or poor intake. To treat hypokalaemia during cardiac arrest the administration of potassium 5 mmol IV as a bolus with consideration for administration of magnesium suflate 2 g IV. Repletion of magnesium stores will facilitate a more rapid correction of hypokalaemia.

Hypothermia/Hyperthermia

In hypothermia specific modifications may be needed in cardiac arrest depending on the core temperature, (chapter 12). Suspect hypothermia in any drowning incident (Chapter 12); use a low reading thermometer to measure core temperature. Hypothermia will be suspected if the patient feels abnormally cold to the touch which may inidicate a need for measurement of the temperature without interrupting chest compressions. Hypothermic patients will need rewarming and if the case being is in the pre-hospital environment, this should not delay transfer to hospital. Rewarming pre-hospital with warm intravenous fluids is no longer recommended so other active and passive measures must be considered. When in the hospital environment options for active rewarming may be increased with the resources available.

Hyperthermia is an abnormally elevated body core temperature (>40.6 °C,) that may result when the rate of heat gained by the body is greater than the rate of heat loss, such that the net heat balance of the body is positive. There are no reliable published data sources on the occurrence of hyperthermia, heat-related illness, exertional heat stroke or episodes with complications that result in death in Australia. Causes of hyperthermia induced cardiac arrest may include hot environmental conditions and malignant hyperthermia.

Heat exhaustion is a condition of fatigue that is caused by prolonged exposure to high temperatures, particularly when combined with high humidity and strenuous activity. Symptoms including headache, nausea, vomiting and malaise may be present. People who suffer from heat exhaustion usually recover rapidly with assistance. A person suffering from heat exhaustion does not typically involve excessive hyperthermia and will have a core body temperature of <40°C.

Heat stroke is a systemic inflammatory response with a core temperature > 40.6 °C, accompanied by altered mental state or collapse and varying levels of organ dysfunction. Sweating has often ceased, and hot dry skin is evident, though in some victims profuse sweating may be present. Cooling techniques for heat stroke commence with simple methods and are guided by the cooperation level of the patient (chapter 12).

During exercise, metabolic energy is converted into mechanical and thermal energy to produce movement. Up to three quarters of this energy may be liberated as heat, making humans heavily reliant on heat loss through evaporative means (e.g. sweating, breathing). As air/environmental temperature rises, the gradient for heat exchange between the skin and the environment is lowered and body temperature rises, making it more difficult to liberate the thermal energy. With the thermal energy building the core body temperature will rise. Sweating increases as core body temperature rises until 39°C, when maximal sweat rates are achieved. If the fluids are not replaced and dehydration is a result, sweating ceases and the body's core temperature may rise at a faster rate. Progression towards a critically high body temperature results in increasing symptom severity; from cramps to exhaustion and heat stroke.

To treat hyperthermia in cardiac arrest active cooling needs to be commenced, using techniques similar to those used for targeted temperature management. These may include the use of cooling mats, wrapped ice packs, intravascular cooling techniques, IV fluids (with consideration to normalising electrolyte levels), and extracorporeal methods.

No medications have been demonstrated as effective in the generic treatment of hyperthermia. In malignant hyperthermia as a reaction to anaesthetic agents the triggering agents must be ceased immediately. In these cases start active cooling and dantrolene should be used. Other drugs such as 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines also cause a condition similar to malignant hyperthermia and the use of dantrolene may be beneficial.

On achievement of ROSC targeted temperature management and standard post resuscitation care should be initiated. This will commence with an ABCDE assessment and involve a team approach to ongoing care.

The four Ts

Thrombosis

Coronary thrombosis is a common cause of cardiac arrest. While it is not a mechanical circulatory obstruction, the thrombus may induce arrhythmias or 'primary pump' failure. If an acute coronary syndrome is suspected as the cause of a refractory cardiac arrest, it may be feasible to perform percutaneous coronary angiography and percutaneous coronary intervention during ongoing CPR. This may require an automated mechanical chest compression device and/or extracorporeal CPR to maintain a circulation during the procedure. A decision to transport a patient with ongoing CPR should consider a realistic chance of survival (e.g. witnessed cardiac arrest with initial shockable rhythm (VF/pVT) and bystander CPR). Intermittent ROSC also strongly favours a decision to transport. Routine administration of fibrinolytic for the treatment of in-hospital and out-of hospital cardiac arrest is not recommended.

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause of cardiac arrest consider giving a fibrinolytic drug immediately. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported, even in cases requiring in excess of 60 min of CPR. However there is also an increased risk of severe bleeding. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 30 minutes and up to 60–90 min before termination of resuscitation attempts. In some settings extracorporeal CPR, and/or percutaneous/ surgical or mechanical thrombectomy can also be used to treat pulmonary embolism.

Tension Pneumothorax

Tension pneumothorax is the progressive build-up of air within the pleural space, usually due to a lung laceration which allows air to escape into the pleural space but not to return. Positive pressure ventilation may exacerbate this 'one-way-valve' effect. Progressive build-up of pressure in the pleural space pushes the mediastinum towards the opposite hemithorax. This build-up of pressure in the pleural obstructs venous return to the heart once intra-thoracic pressure exceeds central venous pressure. This leads to circulatory instability and may result in cardiac arrest. The diagnosis is made clinically, and may be suspected in particular in cases such as:

- Trauma
  - thoracic trauma (penetrating or blunt)
- Iatrogenic
  - thoracic surgery
  - recent thoracic procedures
    - insertion of temporary pacing wire
    - existing chest drain - misplaced/blocked
    - permanant pacemaker/internal defibrillator implant
    - transthoracic needle aspiration or biopsy
    - subclavian or jugular vein catherterisation
    - thoracentesis
    - closed pleural biopsy
- Asthma
- Chronic obstructive pulmonary disease
- Pulmonary barotrauma

In the peri-arrest phase the patient may experience chest pain, respiratory distress, increasing then decreasing (pre-terminal) respiratory rate, air hunger, tachycardia, low/falling SpO_2, hypotension and altered consciousness. In ventilated patients the onset may be more rapid with high ventilation pressures. If the patient has been intubated the position of the tube must be assessed (particularly in suspected cases of left sided tension pneumothorax). In the absence of haemodynamic compromise, it is prudent to wait for the results of an emergent chest X-ray prior to intervention.

Ultrasound will assist in the diagnosis if available. Clinical signs can be difficult to appreciate may include:

- difficulty to ventilate possible – high back pressure
- abnormal chest rise/fall on the affected side
- decreased breath sounds on affected side
- hyper-expanded chest - an increased percussion note
- tracheal deviation away from affected side (possibly a late sign)

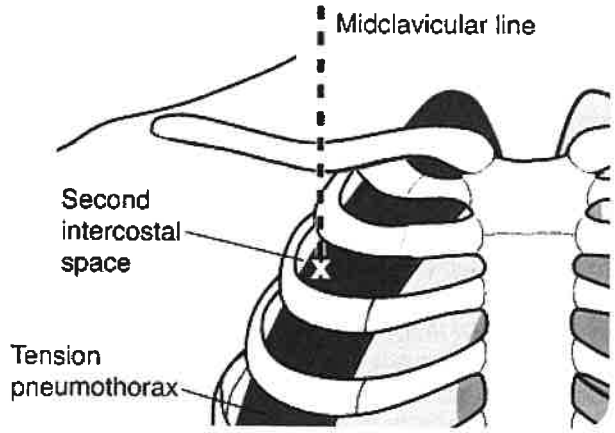


Figure 6.9 Position for needle thoracocentesis

Treatment is to decompress rapidly by thoracostomy or needle thoracocentesis of the affected side and then followed by insertion of a chest drain when appropriate. Decompression by needle thoracocentesis is a rapid method using a 14 gauge (or larger) long cannula/needle and should be performed by an appropriately skilled operator. The response to needle chest decompression may not be rapid and CPR may need to continue. Potential issues/failure for the needle decompression to be successful include:

- Obstruction by:
  - blood
  - tissue
  - cannula kinking/bending/compressed
- Missing a localised tension pneumothorax (e.g. cannula too short)
- Inability to drain a large air leak
- Moving, dislodging and falling out
- Requirement for repeated needle decompression

The advantage of needle decompression is that it can be fast to achieve. Longer needles, cannula, and catheters are available to penetrate the pleura to decompress the tension pneumothorax and with adequate thickness to minimise the risks of kinking.

Once the needle decompression is performed, the patient requires constant monitoring and reassessment, as it is a temporising method only. A relieved tension may re-accumulate undetected if not monitored.

If an operator is sufficiently skilled and can perform the ("blunt/finger") thoracostomy rapidly, ideally without interrupting compressions, this may be the treatment of choice, (particularly if the patient is mechanically ventilated on positive pressure ventilation). It allows maximal pleural cavity evacuation and lung re-expansion with the advantage of a ready made access for the chest drain. It has been shown to be safe and effective in trained physician led trauma care. This may take longer than the needle decompression to perform and difficulty will increase with movement from compressions and proximity of others team members. Some advanced trauma protocols advocate ceasing compressions in order to perform the decompression. Where possible in ALS interruptions to compressions should be avoided.

Some papers and clinicians advocate a third method of decompression with a needle or catheter inserted in the fifth intercostal space on the affected side. This is performed in fifth intercostal space in anterior axillary line to prevent life threatening hemorrhage. This procedure should only be attempted by appropriately skilled personnel. Needle aspiration or small catheter insertion are effective, comfortable and, safe alternatives to thorcostomy in selected patients.

On achieving ROSC follow the standard post resuscitation care. Irrespective of which method of decompression is used, the patient will need follow up with radiological investigations including a chest X-ray and the establishment of a secure chest drain.

Tamponade

Cardiac tamponade is life-threatening, slow or rapid compression of the heart due to the pericardial accumulation of fluid, pus, blood, clots, or gas This can be the consequence of effusion, trauma, or rupture of the heart, resulting in reduced ventricular filling and subsequent haemodynamic compromise. Cardiac tamponade is difficult to diagnose because the typical signs of distended neck veins and hypotension cannot be assessed during cardiac arrest. Focused cardiac ultrasound performed during CPR can be used to diagnose a pericardial effusion during CPR (see below). Tamponade may be more likely to be a reversible cause of cardiac arrest in some particular cases including:

- Thoracic trauma (blunt/penetrating)
- Recent thoracic/cardiac surgery
- Insertion of central access/lines
  - temporary pacing wire
  - central line insertion
- Recent angiography/PCI
- Recent myocardial infarct
- Recent pacemaker/defibrillator implant
- Thoracic neoplasm or mediastinal radiation therapy
- Known pericardial effusion
- Renal Failure (uremia)
- Pericarditis (bacterial/viral)
- Infectious diseases e.g. tuberculous

In the pre-arrest phase the patient may report signs and symptoms including tachypnoea, dyspnoea and on examination may have pulsus paradoxus, low-voltage QRS or electrical alternans on ECG, and Kussmaul's sign. The classic presentation of patients with pericardial tamponade includes Beck's triad of jugular venous distention, muffled/distant heart sounds, and hypotension often with narrow pulse pressure. However these signs may only be briefly present if at all prior to cardiac arrest.

Cardiac arrest after penetrating chest trauma or after cardiac surgery should raise strong suspicion of tamponade and the need for resuscitative thoracotomy should be considered in this setting (Chapter 12). Ongoing bleeding can cause a rapid re-accumulation of blood within the pericardium resulting in the pericardiocentesis being a temporary measure at best. In the setting of post cardiac surgery, re-sternotomy for patients with cardiac arrest should be considered in an appropriately staffed and equipped intensive care unit. Re-sternotomy performed outside these specialised environments has poor results. Chest compressions should not be withheld while preparing for emergency re-sternotomy. Transthoracic or trans-oesophageal echocardiography are very useful when they are readily available to help elucidate the cause of the cardiac arrest (including haemoperitoneum, haemothorax, tension pneumothorax and cardiac tamponade).

In other cases where tamponade is the reversible cause resuscitative thoracotomy may also be considered as the definitive treatment. This option may not always be possible or practical. Pericardiocentesis guided by ultrasound should be considered for treatment of cardiac arrest associated with suspected cardiac tamponade while non-image guided pericardiocentesis is an acceptable alternative only if echocardiography is not available. Fluid resuscitation should be continued as indicated while awaiting definitive management.

Performing a pericardiocentesis during chest compressions is very difficult. As a planned procedure pericardiocentesis is performed with the aid of clear real time imaging, in a controlled environment. Performing the same procedure during resuscitation is extremely difficult. Similar to tension pneumothorax in advanced trauma protocols compressions are withheld, but in ALS this is not optimal. In cases when cardiac tamponade has been definitely identified as the reversible cause, the subxiphoid or apical approach pericardiocentesis have been reportedly used during cardiopulmonary resuscitation. This requires highly skilled personnel and a coordinated team effort. When cardiac ultrasound is not available, electrocardiographic monitoring has been used in cases to indicate when the needle makes contact with the myocardium. A blind approach (without imaging or ECG) is associated with high morbidity and mortality. Complications most often associated with

pericardiocentesis include cardiac dysrhythmias, cardiac puncture, pneumothorax, coronary-vessel injury, diaphragmatic injury and death. Ultrasound guidance is one way to reduce the risks of the potential complications of this procedure.

In many cases the procedure may be futile particularly with myocardial rupture and ceasing resuscitation may need consideration. If ROSC is achieved after completing the procedure, the patient will need standard post resuscitation care and continued monitoring for signs or symptoms of recurrent tamponade until definitive care can be provided.

Toxins

Without a specific history of accidental or deliberate ingestion, poisoning by therapeutic or toxic substances may be difficult to detect. In some cases may be revealed by laboratory investigations often after resuscitation efforts. (Chapter 12). Where available, the appropriate antidotes should be used but most often treatment is supportive. Specific antidotes such as naloxone, flumazenil, digoxin specific antibody fragments, phentolamine, and glucagon are not proven to improve mortality when administered during cardiac arrest.

There is very little data regarding the success or otherwise of specific therapies during the management of cardiac arrest due to toxic substances. However there is a larger amount of lower level data, including case reports that have reported the use of a number of additional therapeutic interventions especially in situations of severe cardiac toxicity.

One antidote recommended, is for cyanide toxicity. The mining industry has used cyanide to process ore for more than 120 years. Strict regulations are used to prevent cyanide toxicity and there have been no documented accidental human deaths due to cyanide poisoning in Australia for 100 years. Patients with severe cardiotoxicity (cardiac arrest, cardiovascular instability, metabolic acidosis, or altered mental status) caused by known or suspected cyanide poisoning should receive cyanide antidote therapy in addition to standard resuscitation guidelines. It is recommended for adult patients with suspected severe cyanide poisoning (including those in cardiac arrest) should receive immediate parenteral hydroxocobalamin, 5mg with repeat dosing up to 15mg.

In opiate induced respiratory arrest the dose of naloxone varies between small aliquots of 100 microgram (a more conservative approach to achieve return of spontaneous respiration and airway control), and a large 2mg dose aimed at achieving immediate and complete reversal. The conservative approach has the advantage of maintaining a safe airway without precipitating an acute withdrawal response with associated risk of harm to both patient and clinician.

In tricyclic toxicity sodium bicarbonate bolus is the mainstay of therapy in the setting of tricyclic-induced cardiac conduction abnormalities. This treatment strategy should be considered during the arrest and in the post arrest period of care for patients surviving cardiac arrest caused by tricyclic antidepressant toxicity associated with wide QRS complexes.

Recreational drug use inducing cardiac arrest is complex and standard resuscitation protocols should be followed. Deaths involving methamphetamines in Australia have been steadily increasing since 2010, and its use among injecting drug users has increased by 50 per cent over the past 10 years, according to research from the National Drug and Alcohol Research Centre at UNSW in 2015. This is further complicated by increased polypharmacy among users and unknown additives or bulking agents mixed with the drugs. If recreational drugs are responsible for a cardiac arrest, the exact toxic agent may not be known at the time. Complicating this is that the drugs/ toxins may induce other reversible causes.

Cardiotoxicity of amphetamine and its synthetic derivates can manifest itself as, acute myocardial infarction or necrosis, arrhythmias, cardiomyopathy and acute heart failure. Amphetamines can precipitate vascular spasm and therefore cause ischemic infarction. Myocardial ischemia causes a massive efflux of potassium. This efflux of potassium can in turn lead to cardiac arrhythmias. Also amphetamines themselves can induce myocardial necrosis. Patients suffering cardiac arrest from amphetamines have been demonstrated to have significant coronary artery stenosis in their 20's and 30's.

Once ROSC is achieved routine post resuscitation should be commenced. This will include consideration for ongoing reversal of the toxins with antidotes, specific toxin protocols, fluids and possible dialysis.

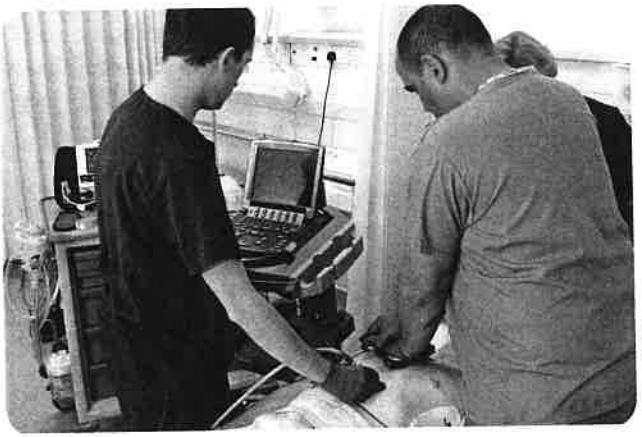


Figure 6.10 Use of focused cardiac ultrasound during advanced life support

Ultrasound during advanced life support

In skilled hands, focused echocardiography/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 6.10). Placement of the probe just before chest compressions are paused for a planned rhythm assessment

common place and have been used for enables a well-trained operator to obtain views within 10 s.

The use of automated mechanical chest compression devices

Automated mechanical chest compression devices should not be used routinely to replace manual chest compressions. However, they may be a reasonable alternative to high-quality manual chest compressions in situations where sustained high-quality manual chest compressions are impractical or compromise provider safety – CPR in a moving ambulance where safety is at risk, prolonged CPR (e.g. hypothermic arrest), and CPR during certain procedures (e.g. coronary angiography or preparation for extracorporeal CPR (ECPR)). Avoid interruptions to compressions during device deployment. Healthcare personnel who use mechanical CPR devices should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills.

Extracorporeal CPR

Extracorporeal resuscitation techniques require vascular access and a circuit with a pump and oxygenator and can provide a circulation of oxygenated blood to restore tissue perfusion. This has the potential to buy time for restoration of an adequate spontaneous circulation, and treatment of reversible underlying conditions. This is commonly called extracorporeal life support (ECLS), and more specifically ECPR when used during cardiac arrest. These techniques are becoming more both in-hospital and out-of-hospital despite limited observational data in selected patient groups. Observational studies suggest ECPR is associated with improved survival when there is a reversible cause for cardiac arrest (e.g. myocardial infarction, pulmonary embolism, severe hypothermia, poisoning), there is little co-morbidity, the cardiac arrest is witnessed, the individual receives immediate high quality CPR. ECPR is best implemented early and certainly within 1 h of collapse. The implementation of ECPR requires considerable resource and training, and availability is therefore limited.

The duration of a resuscitation attempt

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

The duration of any resuscitation attempt should be based on the individual circumstances of the case and is a matter of clinical judgement, taking into consideration the circumstances and 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 VF/pVT, or there is a potentially reversible cause that can be treated. The use of mechanical compression devices and ECPR techniques make prolonged attempts at resuscitation feasible in selected patients. It is generally accepted that asystole for more than 20 min in the absence of a reversible cause and with ongoing ALS constitutes reasonable grounds for stopping further resuscitation attempts, although a shorter or longer time could be appropriate based on the circumstances of cardiac arrest.

Diagnosing death after unsuccessful resuscitation

If CPR is unsuccessful at achieving ROSC and a decision is made to discontinue CPR efforts, 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 cardiorespiratory arrest. After 5 min of continued cardiorespiratory arrest, the absence of pupillary responses to light, corneal reflexes, and motor response to supra-orbital pressure should be confirmed. The time of death is recorded as the time at which these criteria are fulfilled.

Post-event tasks

At the end of the resuscitation further tasks include:

1. Ongoing care of the patient, and allocation of further team roles and responsibilities including handover to other teams, using ISBAR or RSVP.
2. Documentation of the resuscitation attempt. This may be assisted by information from the 'scribe', defibrillators and monitors to help document events and times.
3. Communication with relatives, family and significant others (Chapter 17).
4. An immediate post-event debriefing ('Hot' debriefing). This is normally led by the resuscitation team leader, focuses on immediate issues and concerns, and is usually of short duration. This can be difficult if the patient has a ROSC, as focus then inevitably shifts to post-resuscitation care. A delayed facilitated debriefing ('Cold' debriefing) is also useful (Chapter 2).
5. Ensuring audit forms (and any other documentation) are completed according to local practices.
6. Ensuring equipment and drug trolleys are replenished.

Summary learning

- The ALS algorithm provides a framework for the standardised resuscitation of all adult patients in cardiac arrest.
- The delivery of high quality chest compressions with minimal interruptions is an important determinant of outcome.
- Treatment depends on the underlying rhythm.
- Look for reversible causes and treat early.
- Secure the airway early to enable continuous chest compressions.
- Use waveform capnography to help assess and guide resuscitation interventions.

My key take-home messages from this chapter

Further reading

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