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European Resuscitation Council Guidelines for Resuscitation 2005 Section 4. Adult advanced life support

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4a. Prevention of in-hospital cardiac arrest

The problem

This new section of the guidelines stresses the importance of preventing in-hospital cardiac arrest. Fewer than 20% of patients suffering an in-hospital cardiac arrest will survive to go home.[1,2] Most survivors have a witnessed and monitored VF arrest, primary myocardial ischaemia as the cause, and receive immediate defibrillation.

Cardiac arrest in patients in unmonitored ward areas is not usually a sudden unpredictable event, nor is it usually caused by primary cardiac disease. These patients often have slow and progressive physiological deterioration, involving hypoxia and hypotension, that is unnoticed by staff, or is recognised but poorly treated.[3,4] The underlying cardiac arrest rhythm in this group is usually non-shockable and survival to hospital discharge is very poor.[1,5]

The records of patients who have a cardiac arrest or unanticipated intensive care unit (ICU) admission often contain evidence of unrecognised, or untreated, breathing and circulation problems.[3,4,6—8] The ACADEMIA study showed antecedents in 79% of cardiac arrests, 55% of deaths and 54% of unanticipated ICU admissions.[4] Early and effective treatment of seriously ill patients might prevent some cardiac arrests, deaths and unanticipated ICU admissions. A third of patients who have a false cardiac arrest call die subsequently.[9]

Nature of the deficiencies in acute care

These often involve simple aspects of care including: the failure to treat abnormalities of the patient's airway, breathing and circulation, incorrect use of oxygen therapy, failure to monitor patients, failure to involve experienced senior staff, poor communication, lack of teamwork and insufficient use of treatment limitation plans.[3,7]

Several studies show that medical and nursing staff lack knowledge and skills in acute care. For example, trainee doctors may lack knowledge about oxygen therapy,[10] fluid and electrolyte balance,[11] analgesia,[12] issues of consent,[13] pulse oximetry[14] and drug doses.[15] Medical students may be unable to recognise abnormal breathing patterns.[16] Medical school training provides poor preparation for doctors' early careers, and fails to teach them the essential aspects of applied physiology and acute care.[17] There is also little to suggest that the acute care training and knowledge of

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senior medical staff is better.[18,19] Staff often lack confidence when dealing with acute care problems, and rarely use a systematic approach to the assessment of critically ill patients.[20]

Recognising the critically ill patient

In general, the clinical signs of acute illness are similar whatever the underlying process, as they reflect failing respiratory, cardiovascular and neurological systems. Abnormal physiology is common on general wards,[21] yet the measurement and recording of important physiological observations of sick patients occurs less frequently than is desirable.[3,4,8] This is surprising, as respiratory rate abnormalities may predict cardiorespiratory arrest.[22] To assist in the early detection of critical illness, many hospitals now use early warning scores (EWS) or calling criteria.[23–25] Early warning scoring systems allocate points to routine vital signs measurements on the basis of their derangement from an arbitrarily agreed 'normal' range.[23–25] The weighted score of one or more vital sign observations, or the total EWS, may be used to suggest increasing the frequency of vital signs monitoring to nurses, or to call ward doctors or critical care outreach teams to the patient. Alternatively, systems incorporating 'calling criteria' are based on routine observations, which activate a response when one or more variables reach an extremely abnormal value.[23,26] There are no data to establish the superiority of one system over another, but it may be preferable to use an EWS system, which can track changes in physiology and warn of impending physiological collapse, rather than the ''calling criteria'' approach, which is triggered only when an extreme value of physiology has been reached.

There is a clinical rationale to the use of EWS or calling criteria systems to identify sick patients early. However, their sensitivity, specificity and accuracy in predicting clinical outcomes has yet to be validated convincingly.[27,28] Several studies have identified abnormalities of heart rate, blood pressure, respiratory rate and conscious level as markers of impending critical events.[22,23,29] The suggestion that their incidence has predictive value must be questioned, as not all important vital signs are, or can be, recorded continuously in general ward areas. Several studies show that charting of vital signs is poor, with gaps in data recording.[3,4,8,30] Although the use of physiological systems can increase the frequency of vital signs monitoring,[31] they will be useful for outcome prediction only if widespread monitoring of hospitalised patients becomes available. Even when medical staff are alerted to a patient's abnormal physiology, there is often delay in attending the patient or referring to higher levels of care.[3,4,7] Whereas the use of a warning score based on physiological abnormalities is attractive, it is possible that a more subjective approach, based on staff experience and expertise, may also be effective.[32]

Response to critical illness

The traditional response to cardiac arrest is a reactive one in which hospital staff ('the cardiac arrest team') attend the patient after the cardiac arrest has occurred. Cardiac arrest teams appear to improve survival after cardiac arrest in circumstances where no team has previously existed.[33] However, the role of the cardiac arrest team has been questioned. In one study, only patients who had return of spontaneous circulation before the cardiac arrest team arrived were discharged from hospital alive.[34] When combined with the poor survival rate after in-hospital cardiac arrest, this emphasises the importance of early recognition and treatment of critically ill patients to prevent cardiac arrest. The name 'cardiac arrest team' implies that the team will be called only after cardiac arrest has occurred.

In some hospitals the cardiac arrest team has been replaced by a medical emergency team (MET) that responds, not only to patients in cardiac arrest, but also to those with acute physiological deterioration.[26] The MET usually comprises medical and nursing staff from intensive care and general medicine. and responds to specific calling criteria. Any member of the healthcare team can initiate a MET call. Early involvement of the MET may reduce cardiac arrests, deaths and unanticipated ICU admissions.[35,36] The MET may also be useful in detecting medical error, improving treatment limitation decisions and reducing postoperative ward deaths.[37,38] MET interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids.[39] A circadian pattern of MET activation has been reported, which may suggest that systems for identifying and responding to medical emergencies may not be uniform throughout the 24-h period.[40] Studying the effect of the MET on patient outcomes is difficult. Many of the study findings to date can be criticised because of poor study design. A recent, well-designed, cluster-randomised controlled trial of the MET system demonstrated that the introduction of a MET increased the calling incidence for the team. However, it failed to show a reduction in the incidence of cardiac arrest, unexpected death or unplanned ICU admission.[41]

In the UK, a system of pre-emptive ward care, based predominantly on individual or teams of nurses known as critical care outreach, has developed.[42] Outreach services exist in many forms, ranging from a single nurse to a 24-h, 7 days per week multiprofessional team. An outreach team or system may reduce ward deaths, postoperative adverse events, ICU admissions and readmissions, and increase survival.[43–45]

Other attempts to improve the general ward care of patients and prevent physiological deterioration and cardiac arrest include new admission processes, early physiological monitoring and clinical intervention in the emergency department (ED), and the appointment of new grades of emergency physicians. Many of these models attempt to support the primary admitting team with the skills of 'resuscitation' specialists.[46] Medical and surgical assessment units act as a single location for all acute admissions until their required level of care is evaluated. Patients are monitored and observed for periods of up to 72 h, and there is usually rapid access to senior medical staff, diagnostics and urgent treatment.[47] The single location provides a central focus for on-call medical, nursing and physiotherapy staff, in contrast to the traditional system in which staff and patients are dispersed throughout the hospital.

Many acutely ill patients present to hospital via the ED and are obviously in need of immediate ICU-type interventions. Early goal-directed therapy in the ED reverses physiological derangement and appears to improve patient survival.[48]

Appropriate placement of patients

Ideally, the sickest patients should be admitted to an area that can provide the greatest supervision and the highest level of organ support and nursing care. This often occurs, but some patients are placed incorrectly.[49] International organisations have offered definitions of levels of care and produced admission and discharge criteria for high dependency units (HDUs) and ICUs.[50,51]

Staffing levels

Hospital staffing tends to be at its lowest during the night and at weekends. This may influence patient monitoring, treatment and outcomes. Admission to a general medical ward after 17:00 h[52] or to hospital at weekends[53] is associated with increased mortality. Patients who are discharged from ICUs to general wards at night have an increased risk of in-hospital death compared with those discharged during the day and those discharged to HDUs.[54] One study shows that higher nurse staffing is associated with reduction in cardiac arrest rates, as well as rates of pneumonia, shock and death.[55]

Resuscitation decisions

Consider 'do not attempt resuscitation' (DNAR) when the patient:

- does not wish to have CPR
- will not survive cardiac arrest even if CPR is attempted

Hospital staff often fail to consider whether resuscitation attempts are appropriate and resuscitation attempts in futile cases are common.[37] Even when there is clear evidence that cardiac arrest or death is likely, ward staff rarely make decisions about the patient's resuscitation status.[4] Many European countries have no formal policy for recording DNAR decisions and the practice of consulting patients about the decision is variable.[56] Improved knowledge, training and DNAR decision-making should improve patient care and prevent futile CPR attempts (see Section 8).

Guidelines for prevention of in-hospital cardiac arrest

The following strategies may prevent avoidable in-hospital cardiac arrests.

1. Provide care for patients who are critically ill or at risk of clinical deterioration in appropriate areas, with the level of care provided matched to the level of patient sickness.
2. Critically ill patients need regular observations: match the frequency and type of observations to the severity of illness or the likelihood of clinical deterioration and cardiopulmonary arrest. Often only simple vital sign observations (pulse, blood pressure, respiratory rate) are needed.
3. Use an EWS system to identify patients who are critically ill and or at risk of clinical deterioration and cardiopulmonary arrest.
4. Use a patient charting system that enables the regular measurement and recording of EWS.
5. Have a clear and specific policy that requires a clinical response to EWS systems. This should include advice on the further clinical management of the patient and the specific responsibilities of medical and nursing staff.
6. The hospital should have a clearly identified response to critical illness. This may include a designated outreach service or resuscitation team (e.g. MET) capable of responding to acute clinical crises identified by clinical triggers or

other indicators. This service must be available 24 h per day.
7. Train all clinical staff in the recognition, monitoring and management of the critically ill patient. Include advice on clinical management while awaiting the arrival of more experienced staff.
8. Identify patients for whom cardiopulmonary arrest is an anticipated terminal event and in whom CPR is inappropriate, and patients who do not wish to be treated with CPR. Hospitals should have a DNAR policy, based on national guidance, which is understood by all clinical staff.
9. Ensure accurate audit of cardiac arrest, 'false arrest', unexpected deaths and unanticipated ICU admissions using common datasets. Audit also the antecedents and clinical response to these events.

4b. In-hospital resuscitation

After in-hospital cardiac arrest, the division between basic life support and advanced life support is arbitrary; in practice, the resuscitation process is a continuum and is based on common sense. The public expect that clinical staff can undertake cardiopulmonary resuscitation (CPR). For all in-hospital cardiac arrests, ensure that:

- cardiorespiratory arrest is recognised immediately
- help is summoned using a standard telephone number
- CPR is started immediately using airway adjuncts, e.g. a pocket mask and, if indicated, defibrillation attempted within 3 min

The exact sequence of actions after in-hospital cardiac arrest will depend on many factors, including:

- location (clinical/non-clinical area; monitored/unmonitored area)
- training of the first responders
- number of responders
- equipment available
- hospital response system to cardiac arrest and medical emergencies, (e.g. MET) cardiac arrest team

Location

Patients who have monitored arrests are usually diagnosed rapidly. Ward patients may have had a period of deterioration and an unwitnessed arrest.[3,4,6–8] Ideally, all patients who are at high risk of cardiac arrest should be cared for in a monitored area where facilities for immediate resuscitation are available.

Training of first responders

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

Number of responders

The single responder must ensure that help is coming. If other staff are nearby, several actions can be undertaken simultaneously.

Equipment available

All clinical areas should have immediate access to resuscitation equipment and drugs to facilitate rapid resuscitation of the patient in cardiopulmonary arrest. Ideally, the equipment used for CPR (including defibrillators) and the layout of equipment and drugs should be standardised throughout the hospital.[57]

Resuscitation team

The resuscitation team may take the form of a traditional cardiac arrest team, which is called only when cardiac arrest is recognised. Alternatively, hospitals may have strategies to recognise patients at risk of cardiac arrest and summon a team (e.g., MET) before cardiac arrest occurs.[35,36,39,41,58] The term 'resuscitation team' reflects the range of response teams. In hospital cardiac arrests are rarely sudden or unexpected. A strategy of recognising patients at risk of cardiac arrest may enable some of these arrests to be prevented, or may prevent futile resuscitation attempts in those who are unlikely to benefit from CPR.

Immediate actions for a collapsed patient in a hospital

An algorithm for the initial management of in-hospital cardiac arrest is shown in Figure 4.1.

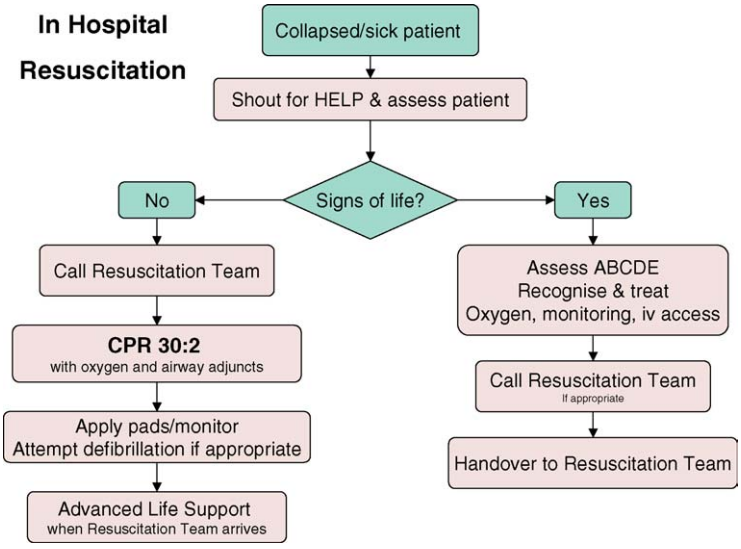

Figure 4.1 Algorithm for the treatment of in-hospital cardiac arrest.

- Ensure personal safety.
- Check the victim for a response.
- When healthcare professionals see a patient collapse or find a patient apparently unconscious in a clinical area, they should first shout for help, then assess if the patient is responsive. Gently shake the shoulders and ask loudly: ''Are you all right?''
- If other members of staff are nearby, it will be possible to undertake actions simultaneously.

The responsive patient

Urgent medical assessment is required. Depending on the local protocols, this may take the form of a resuscitation team (e.g., MET). While awaiting this team, give the patient oxygen, attach monitoring and insert an intravenous cannula.

The unresponsive patient

The exact sequence will depend on the training of staff and experience in assessment of breathing and circulation. Trained healthcare staff cannot assess the breathing and pulse sufficiently reliably to confirm cardiac arrest.[16,59,60] Agonal breathing (occasional gasps, slow, laboured or noisy breathing) is common in the early stages of cardiac arrest and is a sign of cardiac arrest and should not be confused as a sign of life/circulation.

- Shout for help (if not already)

Turn the victim on to his back and then open the airway:

- Open Airway and check breathing:
  - Open the airway using a head tilt chin lift
  - Look in the mouth. If a foreign body or debris is visible attempt to remove with forceps or suction as appropriate
  - If you suspect that there may have been an injury to the neck, try to open the airway using a jaw thrust. Remember that maintaining an airway and adequate ventilation is the overriding priority in managing a patient with a suspected spinal injury. If this is unsuccessful, use just enough head tilt to clear the airway. Use manual in-line stabilisation to minimise head movement if sufficient rescuers are available.

Keeping the airway open, look, listen, and feel for normal breathing (an occasional gasp, slow, laboured or noisy breathing is not normal):

- Look for chest movement
- Listen at the victim's mouth for breath sounds
- Feel for air on your cheek

Look, listen, and feel for no more than 10 s to determine if the victim is breathing normally

- Check for signs of a circulation:
  - It may be difficult to be certain that there is no pulse. If the patient has no signs of life (lack of movement, normal breathing, or coughing), start CPR until more experience help arrives or the patient shows signs of life.
  - Those experienced in clinical assessment should assess the carotid pulse whilst simultaneously looking for signs of life for not more than 10 s.
  - If the patient appears to have no signs of life, or if there is doubt, start CPR immediately. Delays

in diagnosis of cardiac arrest and starting CPR will adversely effect survival must be avoided.

If there is a pulse or signs of life, urgent medical assessment is required. Depending on the local protocols, this may take the form of a resuscitation team. While awaiting this team, give the patient oxygen, attach monitoring, and insert an intravenous cannula.

If there is no breathing, but there is a pulse (respiratory arrest), ventilate the patient's lungs and check for a circulation every 10 breaths.

Starting in-hospital CPR

- One person starts CPR as others call the resuscitation team and collect the resuscitation equipment and a defibrillator. If only one member of staff is present, this will mean leaving the patient.
- Give 30 chest compressions followed by 2 ventilations.
- Undertaking chest compressions properly is tiring; try to change the person doing chest compressions every 2 min.
- Maintain the airway and ventilate the lungs with the most appropriate equipment immediately to hand. A pocket mask, which may be supplemented with an oral airway, is usually readily available. Alternatively, use a laryngeal mask airway (LMA) and self-inflating bag, or bag-mask, according to local policy. Tracheal intubation should be attempted only by those who are trained, competent and experienced in this skill.
- Use an inspiratory time of 1 s and give enough volume to produce a normal chest rise. Add supplemental oxygen as soon as possible.
- Once the patient's trachea has been intubated, continue chest compressions uninterrupted (except for defibrillation or pulse checks when indicated), at a rate of 100 min^{-1}, and ventilate the lungs at approximately 10 breaths min^{-1}. Avoid hyperventilation.
- If there is no airway and ventilation equipment available, give mouth-to-mouth ventilation. If there are clinical reasons to avoid mouth-to-mouth contact, or you are unwilling or unable to do this, do chest compressions until help or airway equipment arrives.
- When the defibrillator arrives, apply the paddles to the patient and analyse the rhythm. If self-adhesive defibrillation pads are available, apply these without interrupting chest compressions. Pause briefly to assess the heart rhythm. If indicated, attempt either manual or automated external defibrillation (AED).
- Recommence chest compressions immediately after the defibrillation attempt. Minimise interruptions to chest compressions.
- Continue resuscitation until the resuscitation team arrives or the patient shows signs of life. Follow the voice prompts if using an AED. If using a manual defibrillator, follow the universal algorithm for advanced life support (Section 4c).
- Once resuscitation is underway, and if there are sufficient staff present, prepare intravenous cannulae and drugs likely to be used by the resuscitation team (e.g. adrenaline).
- Identify one person to be responsible for handover to the resuscitation team leader. Locate the patient's records.
- The quality of chest compressions during in-hospital CPR is frequently sub-optimal.[61,62] The team leader should monitor the quality of CPR and change CPR providers if the quality of CPR is poor. The person providing chest compressions should be changed every 2 min.

The monitored and witnessed cardiac arrest

If a patient has a monitored and witnessed cardiac arrest, act as follows.

- Confirm cardiac arrest and shout for help.
- Consider a precordial thump if the rhythm is VF/VT and a defibrillator is not immediately available.
- If the initial rhythm is VF/VT and a defibrillator is immediately available, give a shock first. The use of adhesive electrode pads or a 'quick-look' paddles technique will enable rapid assessment of heart rhythm compared with attaching ECG electrodes.[63]

Training for healthcare professionals

The Immediate Life Support course trains healthcare professionals in the skills required to start resuscitation, including defibrillation, and to be members of a cardiac arrest team (see Section 9).[64] The Advanced Life Support (ALS) course teaches the skills required for leading a resuscitation team.[65,66]

4c. ALS treatment algorithm

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

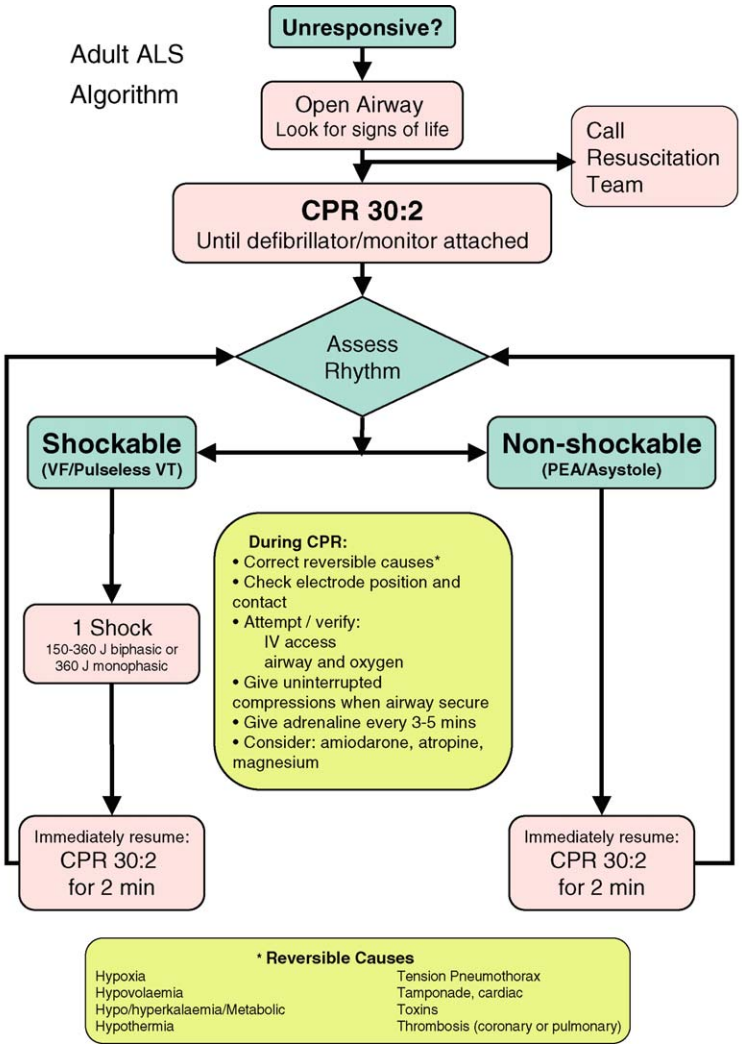

Figure 4.2 Advanced life support cardiac arrest algorithm.

and pulseless electrical activity (PEA)). The principal difference in the management of these two groups of arrhythmias is the need for attempted defibrillation in those patients with VF/VT. Subsequent actions, including chest compressions, airway management and ventilation, venous access, administration of adrenaline and the identification and correction of reversible factors, are common to both groups.

Although the ALS cardiac arrest algorithm (Figure 4.2) is applicable to all cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances (Section 7).

The interventions that unquestionably contribute to improved survival after cardiac arrest are early defibrillation for VF/VT and prompt and effective bystander basic life support (BLS). Advanced airway intervention and the delivery of drugs have not been shown to increase survival to hospital discharge after cardiac arrest, although they are still included among ALS interventions. Thus, during advanced life support, attention must be focused on early defibrillation and high-quality, uninterrupted BLS.

Shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia)

In adults, the commonest rhythm at the time of cardiac arrest is VF, which may be preceded by a period of VT or even supraventricular tachycardia (SVT).[67] Having confirmed cardiac arrest, summon help (including the request for a defibrillator) and start CPR, beginning with external chest compression, with a compression:ventilation (CV) ratio of 30:2. As soon as the defibrillator arrives, diagnose the rhythm by applying paddles or self-adhesive pads to the chest.

If VF/VT is confirmed, charge the defibrillator and give one shock (150—200-J biphasic or 360-J monophasic). Without reassessing the rhythm or feeling for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions. 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,[68] and the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.[69] If a perfusing rhythm has been restored, giving chest compressions does not increase the chance of VF recurring.[70] In the presence of post-shock asystole, chest compressions may usefully induce VF.[70] Continue CPR for 2 min, then pause briefly to check the monitor: if there is still VF/VT, give a second shock (150—360-J biphasic or 360-J monophasic). Resume CPR immediately after the second shock.

Pause briefly after 2 min of CPR to check the monitor: if there is still VF/VT, give adrenaline followed immediately by a third shock (150—360-J biphasic or 360-J monophasic) and resumption of CPR (drug-shock-CPR-rhythm check sequence). Minimise the delay between stopping chest compressions and delivery of the shock. The adenaline that is given immediately before the shock will be circulated by the CPR that immediately follows the shock. After drug delivery and 2 min of CPR, analyse the rhythm and be prepared to deliver another shock immediately if indicated. If VF/VT persists after the third shock, give an intravenous bolus of amiodarone 300 mg. Inject the amiodarone during the brief rhythm analysis before delivery of the fourth shock.

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

During treatment of VF/VT, healthcare providers must practice efficient coordination between CPR and shock delivery. When VF is present for more than a few minutes, the myocardium is depleted of oxygen and metabolic substrates. A brief period of chest compressions will deliver oxygen and energy substrates and increase the probability of restoring a perfusing rhythm after shock delivery.[71] Analyses of VF waveform characteristics predictive of shock success indicate that the shorter the time between chest compression and shock delivery, the more likely the shock will be successful.[71,72] Reduction in the interval from compression to shock delivery by even a few seconds can increase the probability of shock success.[73]

Regardless of the arrest rhythm, give adrenaline 1 mg every 3—5 min until ROSC is achieved; this will be once every two loops of the algorithm. If signs of life return during CPR (movement, normal breathing, or coughing), 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 arrhythmia. If no pulse is present, continue CPR. Providing CPR with a CV ratio of 30:2 is tiring; change the individual undertaking compressions every 2 min.

Precordial thump

Consider giving a single precordial thump when cardiac arrest is confirmed rapidly after a witnessed, sudden collapse and a defibrillator is not immediately to hand (Section 3).[74] These circumstances are most likely to occur when the patient is monitored. 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. A precordial thump is most likely to be successful in converting VT to sinus rhythm. Successful treatment of VF by precordial thump is much less likely: in all the reported successful cases, the precordial thump was given within the first 10 s of VF.[75] There are very rare reports of a precordial thump converting a perfusing to a non-perfusing rhythm.[76]

Airway and ventilation

During the treatment of persistent VF, ensure good-quality chest compressions between defibrillation attempts. Consider reversible causes (4 H's and 4 T's) and, if identified, correct them. Check the electrode/defibrillating paddle positions and contacts, and the adequacy of the coupling medium, e.g. gel pads. Tracheal intubation provides the most reliable airway, but should be attempted only if the healthcare provider is properly trained

and has adequate ongoing experience with the technique. Personnel skilled in advanced airway management should attempt laryngoscopy without stopping chest compressions; a brief pause in chest compressions may be required as the tube is passed through the vocal cords. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until return of spontaneous circulation. No intubation attempt should take longer than 30 s: if intubation has not been achieved after this time, recommence bag-mask ventilation. After intubation, confirm correct tube position and secure it adequately. Once the patient's trachea has been intubated, continue chest compressions, at a rate of 100 min^{-1}, without pausing during ventilation. Ventilate the lungs at 10 breaths min^{-1}; do not hyperventilate the patient. A pause in the chest compressions allows the coronary perfusion pressure to fall substantially. On resuming compressions there is some delay before the original coronary perfusion pressure is restored, thus chest compressions that are not interrupted for ventilation result in a substantially higher mean coronary perfusion pressure.

In the absence of personnel skilled in tracheal intubation, acceptable alternatives are the Combitube, laryngeal mask airway (LMA), ProSeal LMA, or Laryngeal Tube (Section 4d). Once one of these airways has been inserted, attempt to deliver continuous chest compressions, uninterrupted during ventilation. If excessive gas leakage causes inadequate ventilation of the patient's lungs, chest compressions will have to be interrupted to enable ventilation (using a CV ratio of 30:2).

During continuous chest compressions, ventilate the lungs at 10 breaths min^{-1}.

Intravenous access and drugs

Peripheral versus central venous drug delivery. Establish intravenous access if this has not already been achieved. 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,[77] insertion of a central venous catheter requires interruption of CPR and is associated with several complications. Peripheral venous cannulation is quicker, easier to perform and safer. Drugs injected peripherally must be followed by a flush of at least 20 ml of fluid and elevation of the extremity for 10—20 s to facilitate drug delivery to the central circulation.

Intraosseous route. If intravenous access is difficult or impossible, consider the intraosseous route. Although normally considered as an alternative route for vascular access in children, it can also be effective in adults.[78] Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a central venous catheter. The intraosseous route also enables withdrawal of marrow for venous blood gas analysis and measurement of electrolytes and haemoglobin concentration.

Tracheal route. If neither intravenous nor intraosseous access can be established, some drugs can be given by the tracheal route. However, unpredictable plasma concentrations are achieved when drugs are given via a tracheal tube, and the optimal tracheal dose of most drugs is unknown. During CPR, the equipotent dose of adrenaline given via the trachea is three to ten times higher than the intravenous dose.[79,80] Some animal studies suggest that the lower adrenaline concentrations achieved when the drug is given via the trachea may produce transient beta-adrenergic effects, which will cause hypotension and lower coronary artery perfusion pressure.[81—84] If given via the trachea, the dose of adrenaline is 3 mg diluted to at least 10 ml with sterile water. Dilution with water instead of 0.9% saline may achieve better drug absorption.[85] The solutions in prefilled syringes are acceptable for this purpose.

Adrenaline. Despite the widespread use of adrenaline during resuscitation, and several studies involving vasopressin, there is no placebo-controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases 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. The alpha-adrenergic actions of adrenaline cause vasoconstriction, which increases myocardial and cerebral perfusion pressure. The higher coronary blood flow increases the frequency of the VF waveform and should improve the chance of restoring a circulation when defibrillation is attempted.[86—88] The optimal duration of CPR and number of shocks that should be given before giving drugs is unknown. On the basis of expert consensus, if VF/VT persists after two shocks, give adrenaline and repeat every 3—5 min during cardiac arrest. Do not interrupt CPR to give drugs.

Anti-arrhythmic drugs. There is no evidence that giving any anti-arrhythmic drug routinely during human cardiac arrest increases survival to hospital discharge. In comparison with placebo[89] and lidocaine,[90] the use of amiodarone in shock-

refractory VF improves the short-term outcome of survival to hospital admission. In these studies, the anti-arrhythmic therapy was given if VF/VT persisted after at least three shocks; however, these were delivered using the conventional three-stacked shocks strategy. There are no data on the use of amiodarone for shock-refractory VF/VT when single shocks are used. On the basis of expert consensus, if VF/VT persists after three shocks, give 300 mg amiodarone by bolus injection. A further dose of 150 mg may be given for recurrent or refractory VF/VT, followed by an infusion of 900 mg over 24. Lidocaine 1 mg kg^{-1} may be used as an alternative if amiodarone is not available, but do not give lidocaine if amiodarone has been given already.

Magnesium. Although the routine use of magnesium in cardiac arrest does not increase survival,[91–95] give magnesium (8 mmol = 4 ml 50% magnesium sulphate or 2 g) for refractory VF if there is any suspicion of hypomagnesaemia (e.g., patients on potassium-losing diuretics).

Bicarbonate. Administering sodium bicarbonate routinely during cardiac arrest and CPR (especially in out-of-hospital cardiac arrests) or after return of spontaneous circulation is not recommended. Give sodium bicarbonate (50 mmol) if cardiac arrest is associated with hyperkalaemia or tricyclic antidepressant overdose; repeat the dose according to the clinical condition and result of repeated blood gas analysis. Some experts give bicarbonate if the arterial pH is less than 7.1, but this is controversial. During cardiac arrest, arterial blood gas values do not reflect the acid—base state of the tissues[96]; the tissue pH will be lower than that in arterial blood. Mixed venous blood values give a more accurate estimate of the pH in the tissues,[96] but it is rare for a pulmonary artery catheter to be in situ at the time of cardiac arrest. If a central venous catheter is in situ, central venous blood gas analysis will provide a closer estimate of tissue acid/base state than that provided by arterial blood.

Persistent ventricular fibrillation

In VF persists, consider changing the position of the paddles (Section 3). Review all potentially reversible causes (see below) and treat any that are identified.

The duration of any individual resuscitation attempt 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/VT.

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as cardiac electrical activity in the absence of any palpable pulses. These patients often have some mechanical myocardial contractions, but these are too weak to produce a detectable pulse or blood pressure. PEA is often caused by reversible conditions, and can be treated if those conditions are identified and corrected (see below). Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively.

If the initial monitored rhythm is PEA or asystole, start CPR 30:2 and give adrenaline 1 mg as soon as intravascular access is achieved. If asystole is displayed, check without stopping CPR that the leads are attached correctly. Asystole is a condition that could be exacerbated or precipitated by excessive vagal tone and, theoretically, this could be reversed by a vagolytic drug; therefore, despite the lack of evidence that routine atropine for asystolic cardiac arrest increases survival, give atropine 3 mg (the dose that will provide maximum vagal blockade) if there is asystole or the rhythm is slow PEA (rate <60 min^{-1}). Secure the airway as soon as possible, to enable chest compressions to be delivered without pausing during ventilation. After 2 min of CPR, recheck the rhythm. If no rhythm is present (asystole), or if there is no change in the ECG appearance, resume CPR immediately. If an organised rhythm is present, attempt to palpate a pulse. If no pulse is present (or if there is any doubt about the presence of a pulse), continue CPR. If a pulse is present, begin post-resuscitation care. If signs of life return during CPR, check the rhythm and attempt to palpate a pulse.

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves, because this may respond to cardiac pacing. There is no benefit in attempting to pace true asystole.

If there is doubt about whether the rhythm is asystole or fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Fine VF that is difficult to distinguish from asystole will not be shocked successfully into a perfusing rhythm. Continuing good-quality CPR may improve the amplitude and frequency of the VF and improve the chance of successful defibrillation to a perfusing rhythm. Delivering repeated shocks in an attempt to defibrillate what is thought to be fine VF will increase myocardial injury, both directly from the electricity and indirectly from the interruptions in coronary blood flow.

During the treatment of asystole or PEA, if the rhythm changes to VF, follow the left side of

the algorithm. Otherwise, continue CPR and give adrenaline every 3—5 min (every other loop of the algorithm).

Potentially 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. More details on many of these conditions are covered in Section 7.

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 Section 4d, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by hypovolaemia is due usually to severe haemorrhage. This may be precipitated by trauma (Section 7i), gastrointestinal bleeding or rupture of an aortic aneurysm. Intravascular volume should be restored rapidly with fluid, coupled with urgent surgery to stop the haemorrhage.

Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history, e.g. renal failure (Section 7a). A 12-lead ECG may be diagnostic. Intravenous calcium chloride is indicated in the presence of hyperkalaemia, hypocalcaemia and calcium channel-blocker overdose.

Suspect hypothermia in any drowning incident (Sections 7c and d); use a low-reading thermometer.

The four Ts

A tension pneumothorax may be the primary cause of PEA and may follow attempts at central venous catheter insertion. The diagnosis is made clinically. Decompress rapidly by needle thoracocentesis, and then insert a chest drain.

Cardiac tamponade is difficult to diagnose because the typical signs of distended neck veins and hypotension are usually obscured by the arrest itself. Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for needle pericardiocentesis or resuscitative thoracotomy (see Section 7i).

In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or toxic substances may be revealed only by laboratory investigations (Section 7b). Where available, the appropriate antidotes should be used, but most often treatment is supportive.

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolus. If cardiac arrest is thought to be caused by pulmonary embolism, consider giving a thrombolytic drug immediately (Section 4e).[97]

4d. Airway management and ventilation

Introduction

Patients requiring resuscitation often have an obstructed airway, usually secondary to loss of consciousness, but occasionally it may be the primary cause of cardiorespiratory arrest. Prompt assessment, with control of the airway and ventilation of the lungs, is essential. This will help to prevent secondary hypoxic damage to the brain and other vital organs. Without adequate oxygenation it may be impossible to restore a spontaneous cardiac output. These principles may not apply to the witnessed primary cardiac arrest in the vicinity of a defibrillator; in this case, the priority is immediate attempted defibrillation.

Airway obstruction

Causes of airway obstruction

Obstruction of the airway may be partial or complete. It may occur at any level, from the nose and mouth down to the trachea (Figure 4.3). In the unconscious patient, the commonest site of airway obstruction is at the level of the pharynx. Until recently this obstruction had been attributed to posterior displacement of the tongue caused by decreased muscle tone; with the tongue ultimately touching the posterior pharyngeal wall. The precise cause of airway obstruction in the unconscious state has been identified by studying patients under general anaesthesia.[98,99] These studies of anaesthetised patients have shown that the site of airway obstruction is at the soft palate and epiglottis and not the tongue. Obstruction may be caused also by vomit or blood (regurgitation of gastric contents or trauma), or by foreign bodies. Laryngeal obstruction may be caused by oedema from burns, inflammation or anaphylaxis. Upper airway stimulation may cause laryngeal spasm. Obstruction of the airway below the larynx is less com-

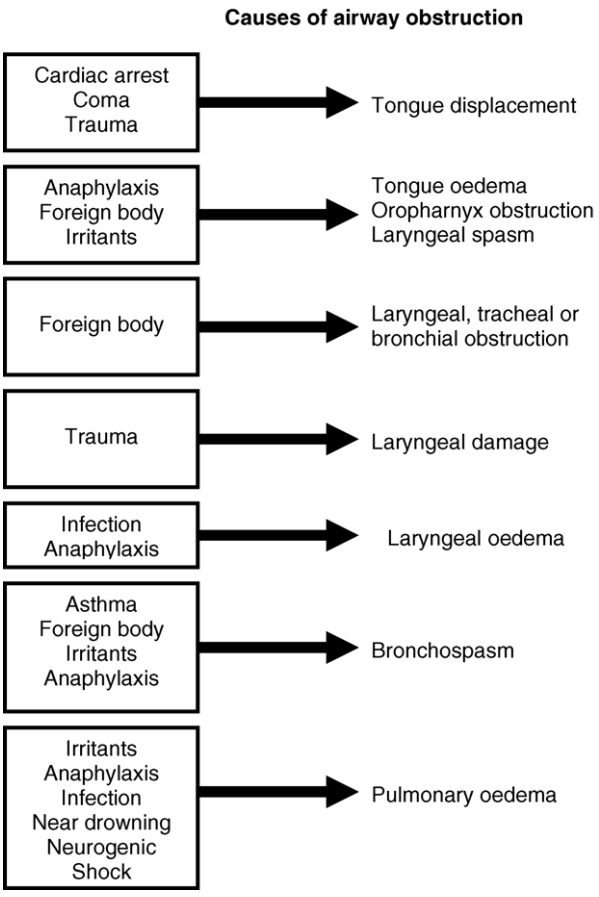

Figure 4.3 Causes of airway obstruction.

mon, but may arise from excessive bronchial secretions, mucosal oedema, bronchospasm, pulmonary oedema or aspiration of gastric contents.

Recognition of airway obstruction

Airway obstruction can be subtle and is often missed by healthcare professionals, let alone by lay people. The 'look, listen and feel' approach is a simple, systematic method of detecting airway obstruction.

- Look for chest and abdominal movements.
- Listen and feel for airflow at the mouth and nose.

In partial airway obstruction, air entry is diminished and usually noisy. Inspiratory stridor is caused by obstruction at the laryngeal level or above. Expiratory wheeze implies obstruction of the lower airways, which tend to collapse and obstruct during expiration. Other characteristic sounds include the following:

- Gurgling is caused by liquid or semisolid foreign material in the main airways.
- Snoring arises when the pharynx is partially occluded by the soft palate or epiglottis.
- Crowing is the sound of laryngeal spasm.

In a patient who is making respiratory efforts, complete airway obstruction causes paradoxical chest and abdominal movement, often described as 'see-saw breathing'. As the patient attempts to breathe in, the chest is drawn in and the abdomen expands; the opposite occurs during expiration. This is in contrast to the normal breathing pattern of synchronous movement upwards and outwards of the abdomen (pushed down by the diaphragm) with the lifting of the chest wall. During airway obstruction, other accessory muscles of respiration are used, with the neck and the shoulder muscles contracting to assist movement of the thoracic cage. Full examination of the neck, chest and abdomen is required to differentiate the paradoxical movements that may mimic normal respiration. The examination must include listening for the absence of breath sounds in order to diagnose complete airway obstruction reliably; any noisy breathing indicates partial airway obstruction. During apnoea, when spontaneous breathing movements are absent, complete airway obstruction is recognised by failure to inflate the lungs during attempted positive pressure ventilation. Unless airway patency can be re-established to enable adequate lung ventilation within a period of a very few minutes, neurological and other vital organ injury may occur, leading to cardiac arrest.

Basic airway management

Once any degree of obstruction is recognised, immediate measures must be taken to create and maintain a clear airway. There are three manoeuvres that may improve the patency of an airway obstructed by the tongue or other upper airway structures: head tilt, chin lift, and jaw thrust.

Head tilt and chin lift

The rescuer's hand is placed on the patient's forehead and the head gently tilted back; the fingertips of the other hand are placed under the point of the patient's chin, which is gently lifted to stretch the anterior neck structures (Figure 4.4).[100–105]

Jaw thrust

Jaw thrust is an alternative manoeuvre for bringing the mandible forward and relieving obstruction by the soft palate and epiglottis. The rescuer's index and other fingers are placed behind the angle of the mandible, and pressure is applied upwards and forwards. Using the thumbs, the mouth is opened slightly by downward displacement of the chin (Figure 4.5).

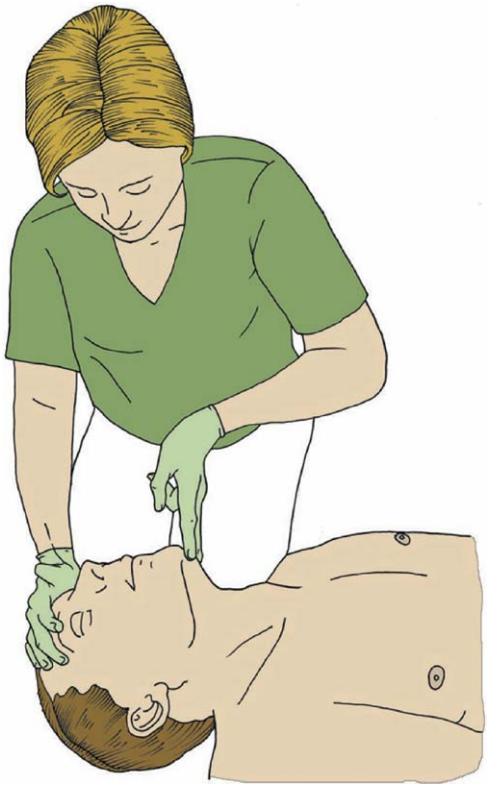

Figure 4.4 Head tilt and chin lift. © 2005 European Resuscitation Council.

These simple positional methods are successful in most cases where airway obstruction results from relaxation of the soft tissues. If a clear airway cannot be achieved, look for other causes of airway obstruction. Use a finger sweep to remove any solid foreign body seen in the mouth. Remove broken or displaced dentures, but leave well-fitting dentures as they help to maintain the contours of the mouth, facilitating a good seal for ventilation.

Airway management in patients with suspected cervical spine injury

If spinal injury is suspected (e.g., if the victim has fallen, been struck on the head or neck, or has been rescued after diving into shallow water), maintain the head, neck, chest and lumbar region in the neutral position during resuscitation. Excessive head tilt could aggravate the injury and damage the cervical spinal cord[106–110]; however, this complication has not been documented and the relative risk is unknown. When there is a risk of cervical spine injury, establish a clear upper airway by using jaw thrust or chin lift in combination with manual in-line stabilisation (MILS) of the head and neck by an assistant.[111,112] If life-threatening airway obstruction persists despite effective application of jaw thrust or chin lift, add head tilt a small amount at a time until the airway is open; establishing a patent airway takes priority over concerns about a potential cervical spine injury.

Adjuncts to basic airway techniques

Simple airway adjuncts are often helpful, and sometimes essential, to maintain an open airway, particularly when resuscitation is prolonged. The position of the head and neck must be maintained to keep the airway aligned. Oropharyngeal and nasopharyngeal airways overcome backward displacement of the soft palate and tongue in an unconscious patient, but head tilt and jaw thrust may also be required.

Oropharyngeal airways. Oropharyngeal airways are available in sizes suitable for the newborn to large adults. An estimate of the size required is obtained by selecting an airway with a length corresponding to the vertical distance between

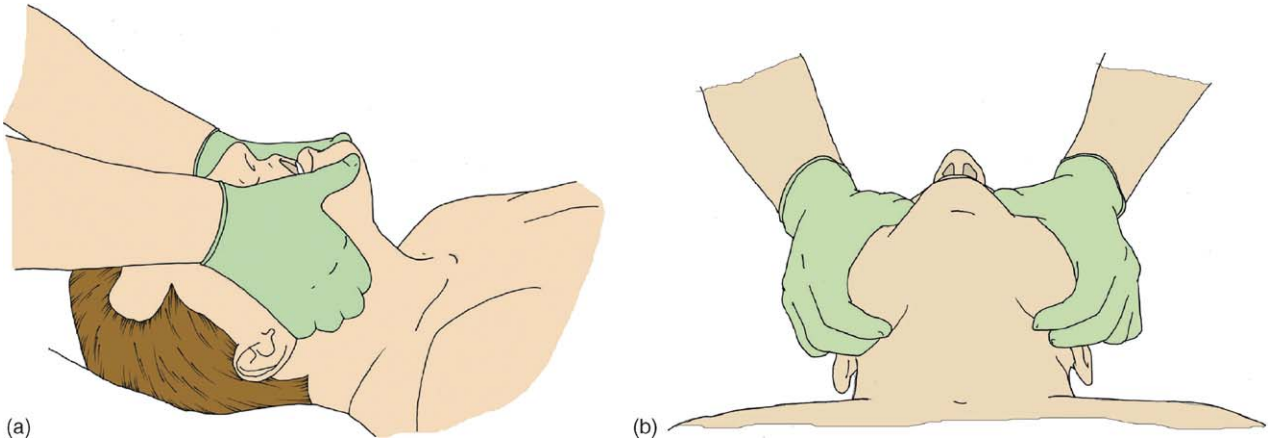

Figure 4.5 Jaw thrust. © 2005 European Resuscitation Council.

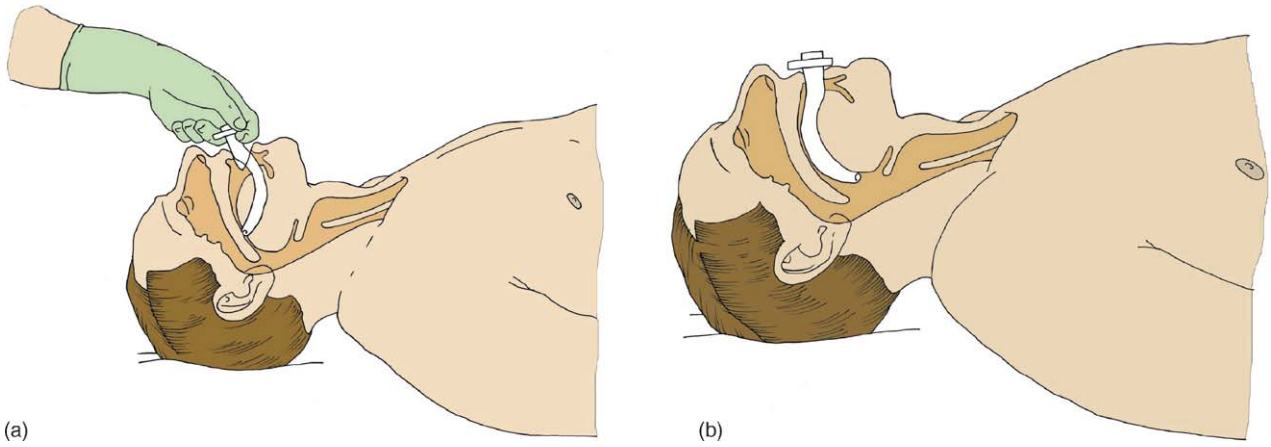

Figure 4.6 Insertion of oropharyngeal airway. © 2005 European Resuscitation Council.

the patient's incisors and the angle of the jaw (Figure 4.6). The most common sizes are 2, 3 and 4 for small, medium and large adults, respectively.

If the glossopharyngeal and laryngeal reflexes are present, vomiting or laryngospasm may be caused by inserting an oropharyngeal airway; thus, insertion should be attempted only in comatose patients. The oropharyngeal airway can become obstructed at three possible sites:[113] part of the tongue can occlude the end of the airway; the airway can lodge in the vallecula; and the airway can be obstructed by the epiglottis.

Nasopharyngeal airways. In patients who are not deeply unconscious, a nasopharyngeal airway is tolerated better than an oropharyngeal airway. The nasopharyngeal airway may be life saving in patients with clenched jaws, trismus or maxillofacial injuries, when insertion of an oral airway is impossible. Inadvertent insertion of a nasopharyngeal airway through a fracture of the skull base and into the cranial vault is possible, but extremely rare.[114,115] In the presence of a known or suspected basal skull fracture an oral airway is preferred but, if this is not possible and the airway is obstructed, gentle insertion of a nasopharyngeal airway may be life saving (i.e., the benefits may far outweigh the risks).

The tubes are sized in millimetres according to their internal diameter, and the length increases with diameter. The traditional methods of sizing a nasopharyngeal airway (measurement against the patient's little finger or anterior nares) do not correlate with the airway anatomy and are unreliable.[116] Sizes of 6—7 mm are suitable for adults. Insertion can cause damage to the mucosal lining of the nasal airway, with bleeding in up to 30% of cases.[117] If the tube is too long it may stimulate the laryngeal or glossopharyngeal reflexes to produce laryngospasm or vomiting.

Oxygen

Give oxygen whenever it is available. A standard oxygen mask will deliver up to 50% oxygen concentration, providing the flow of oxygen is high enough. A mask with a reservoir bag (non-rebreathing mask), can deliver an inspired oxygen concentration of 85% at flows of 10—15 l min^{-1}. Initially, give the highest possible oxygen concentration, which can then be titrated to the oxygen saturation by pulse oximeter (SpO_2) or arterial blood gases.

Suction

Use a wide-bore rigid sucker (Yankauer) to remove liquid (blood, saliva and gastric contents) from the upper airway. Use the sucker cautiously if the patient has an intact gag reflex; the sucker can provoke vomiting.

Ventilation

Provide artificial ventilation as soon as possible for any patient in whom spontaneous ventilation is inadequate or absent. Expired air ventilation (rescue breathing) is effective, but the rescuer's expired oxygen concentration is only 16—17%, so it must be replaced as soon as possible by ventilation with oxygen-enriched air. Although mouth-to-mouth ventilation has the benefit of not requiring any equipment, the technique is aesthetically unpleasant, particularly when vomit or blood is present, and rescuers may be reluctant to place themselves in intimate contact with a victim who may be unknown to them.[118—121] There are only isolated reports of individuals acquiring infections after providing CPR, e.g. tuberculosis[122] and severe acute respiratory distress syndrome (SARS).[123] Transmission of human immunodefi-

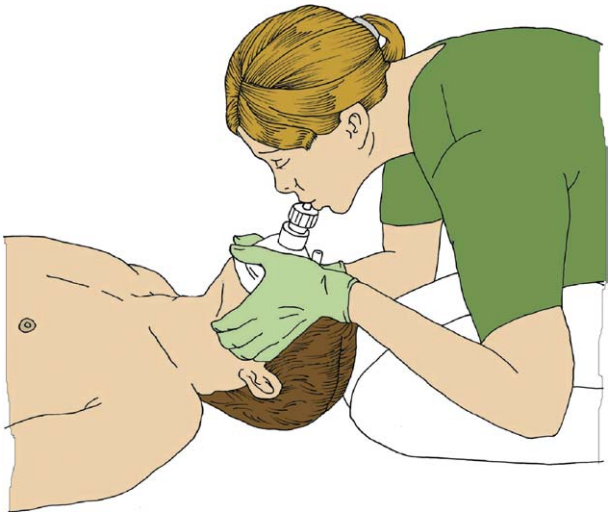

Figure 4.7 Mouth-to-mask ventilation. © 2005 European Resuscitation Council.

ciency virus (HIV) during provision of CPR has never been reported. Simple adjuncts are available to enable direct person-to-person contact to be avoided; some of these devices may reduce the risk of cross-infection between patient and rescuer, although they are unlikely to offer significant protection from SARS.[123] The pocket resuscitation mask is used widely. It is similar to an anaesthetic facemask, and enables mouth-to-mask ventilation. It has a unidirectional valve, which directs the patient's expired air away from the rescuer. The mask is transparent so that vomit or blood from the patient can be seen. Some masks have a connector for the addition of oxygen. When using masks without a connector, supplemental oxygen can be given by placing the tubing underneath one side and ensuring an adequate seal. Use a two-hand technique to maximise the seal with the patient's face (Figure 4.7).

High airway pressures can be generated if the tidal volumes or inspiratory flows are excessive, predisposing to gastric inflation and subsequent risk of regurgitation and pulmonary aspiration. The possibility of gastric inflation is increased by

- malalignment of the head and neck, and an obstructed airway
- an incompetent oesophageal sphincter (present in all patients with cardiac arrest)
- a high inflation pressure

Conversely, if inspiratory flow is too low, inspiratory time will be prolonged and the time available to give chest compressions is reduced. Deliver each breath over approximately 1 s and transfer a volume that corresponds to normal chest movement; this represents a compromise between giving an adequate volume, minimising the risk of gastric inflation, and allowing adequate time for chest compressions. During CPR with an unprotected airway, give two ventilations after each sequence of 30 chest compressions.

Self-inflating bag

The self-inflating bag can be connected to a facemask, tracheal tube or alternative airway device such as the LMA or Combitube. Without supplemental oxygen, the self-inflating bag ventilates the patient's lungs with ambient air (21% oxygen). This can be increased to about 45% by attaching oxygen directly to the bag. If a reservoir system is attached and the oxygen flow is increased to approximately 10 l min^{-1}, an inspired oxygen concentration of approximately 85% can be achieved.

Although the bag-mask device enables ventilation with high concentrations of oxygen, its use by a single person requires considerable skill. When used with a face mask, it is often difficult to achieve a gas-tight seal between the mask and the patient's face, and to maintain a patent airway with one hand while squeezing the bag with the other.[124] Any significant leak will cause hypoventilation and, if the airway is not patent, gas may be forced into the stomach.[125,126] This will reduce ventilation further and greatly increase the risk of regurgitation and aspiration.[127] Cricoid pressure can reduce this risk but requires the presence of a trained assistant. Poorly applied cricoid pressure may make it more difficult to ventilate the patient's lungs.[128]

The two-person technique for bag-mask ventilation is preferable (Figure 4.8). One person holds the facemask in place using a jaw thrust with both

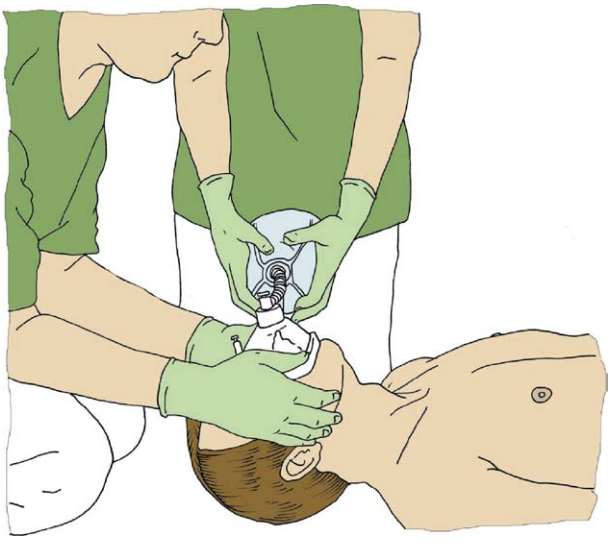

Figure 4.8 The two-person technique for bag-mask ventilation. © 2005 European Resuscitation Council.

hands, and an assistant squeezes the bag. In this way, a better seal can be achieved and the patient's lungs can be ventilated more effectively and safely.

Once a tracheal tube, Combitube or supraglottic airway device has been inserted, ventilate the lungs at a rate of 10 breaths min^{-1} and continue chest compressions without pausing during ventilations. The seal of the LMA around the larynx is unlikely to be good enough to prevent at least some gas leaking when inspiration coincides with chest compressions. Moderate gas leakage is acceptable, particularly as most of this gas will pass up through the patient's mouth; if excessive gas leakage results in 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.

Automatic ventilators

Very few studies address specific aspects of ventilation during advanced life support. There are some data indicating that the ventilation rates delivered by healthcare personnel during cardiac arrest are excessive.[61,129] Automatic ventilators or resuscitators provide a constant flow of gas to the patient during inspiration; the volume delivered is dependent on the inspiratory time (a longer time provides a greater tidal volume). Because pressure in the airway rises during inspiration, these devices are often pressure limited to protect the lungs against barotrauma. An automatic ventilator can be used with either a facemask or other airway device (e.g., tracheal tube, LMA).

An automatic resuscitator should be set initially to deliver a tidal volume of 6—7 ml kg^{-1} at 10 breaths min^{-1}. Some ventilators have coordinated markings on the controls to facilitate easy and rapid adjustment for patients of different sizes, and others are capable of sophisticated variation in respiratory pattern. In the presence of a spontaneous circulation, the correct setting will be determined by analysis of the patient's arterial blood gases.

Automatic resuscitators provide many advantages over alternative methods of ventilation.

- In unintubated patients, the rescuer has both hands free for mask and airway alignment.
- Cricoid pressure can be applied with one hand while the other seals the mask on the face.
- In intubated patients they free the rescuer for other tasks.
- Once set, they provide a constant tidal volume, respiratory rate and minute ventilation; thus, they may help to avoid excessive ventilation.

A manikin study of simulated cardiac arrest and a study involving fire-fighters ventilating the lungs of anaesthetised patients both showed a significant decrease in gastric inflation with manually-triggered flow-limited oxygen-powered resuscitators and mask compared with a bag-mask.[130,131] However, the effect of automatic resuscitators on gastric inflation in humans in cardiac arrest has not been studied, and there are no data demonstrating clear benefit over bag-valve-mask devices.

Alternative airway devices

The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest. There is evidence that, without adequate training and experience, the incidence of complications, such as unrecognised oesophageal intubation (6—14% in some studies)[132—135] and dislodgement, is unacceptably high.[136] Prolonged attempts at tracheal intubation are harmful; the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Several alternative airway devices have been considered for airway management during CPR. The Combitube, the LMA, and the Laryngeal Tube (LT) are the only alternative devices to be studied during CPR, but none of these studies have been powered adequately to enable survival to be studied as a primary endpoint; instead, most researchers have studied insertion and ventilation success rates. There are no data supporting the routine use of any specific approach to airway management during cardiac arrest. The best technique is dependent on the precise circumstances of the cardiac arrest and the competence of the rescuer.

Laryngeal mask airway (LMA)

The laryngeal mask airway comprises a wide-bore tube with an elliptical inflated cuff designed to seal around the laryngeal opening (Figure 4.9). It is easier to insert than a tracheal tube.[137—143] The LMA has been studied during CPR, but none of these studies has compared it directly with the tracheal tube. During CPR, successful ventilation is achieved with the LMA in 72—98% of cases.[144—150]

Ventilation using the LMA is more efficient and easier than with a bag-mask.[124] When an LMA can be inserted without delay it is preferable to avoid bag-mask ventilation altogether. When used for intermittent positive pressure ventilation, provided high inflation pressures (>20 cm H_2O) are avoided, gastric inflation can be minimised. In comparison with bag-mask ventilation, use of a self-inflating bag and

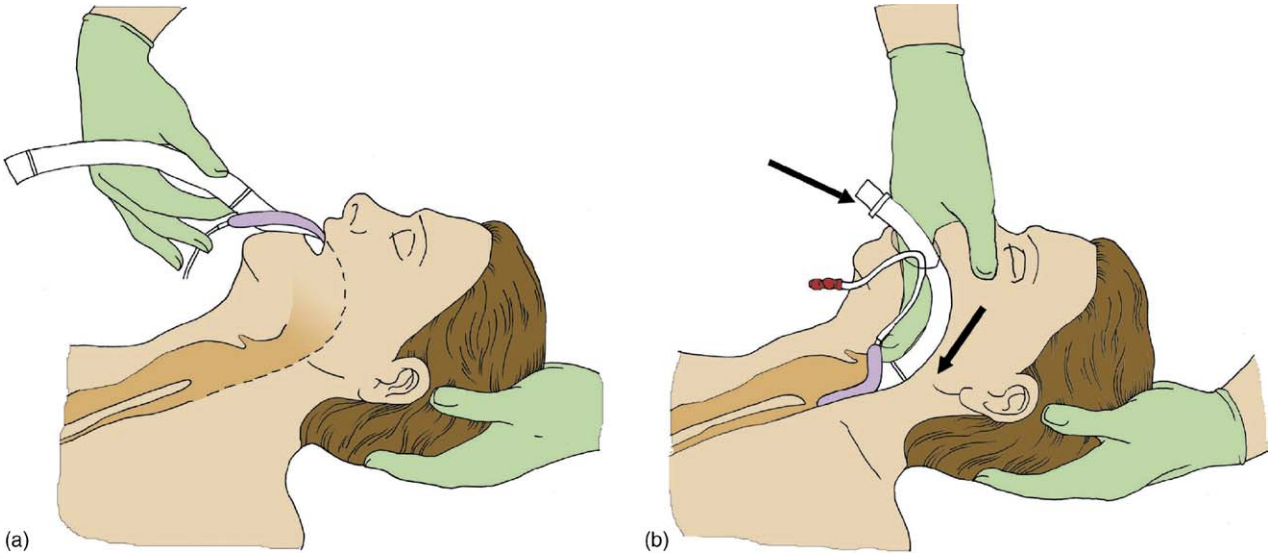

Figure 4.9 Insertion of a laryngeal mask airway. © 2005 European Resuscitation Council.

LMA during cardiac arrest reduces the incidence of regurgitation.[127]

In comparison with tracheal intubation, the perceived disadvantages of the LMA are the increased risk of aspiration and inability to provide adequate ventilation in patients with low lung and/or chest-wall compliance. There are no data demonstrating whether or not it is possible to provide adequate ventilation via an LMA without interruption of chest compressions. The ability to ventilate the lungs adequately while continuing to compress the chest may be one of the main benefits of a tracheal tube. There are remarkably few cases of pulmonary aspiration reported in the studies of the LMA during CPR.

The Combitube

The Combitube is a double-lumen tube introduced blindly over the tongue, and provides a route for ventilation whether the tube has passed into the oesophagus (Figure 4.10a) or the tra-

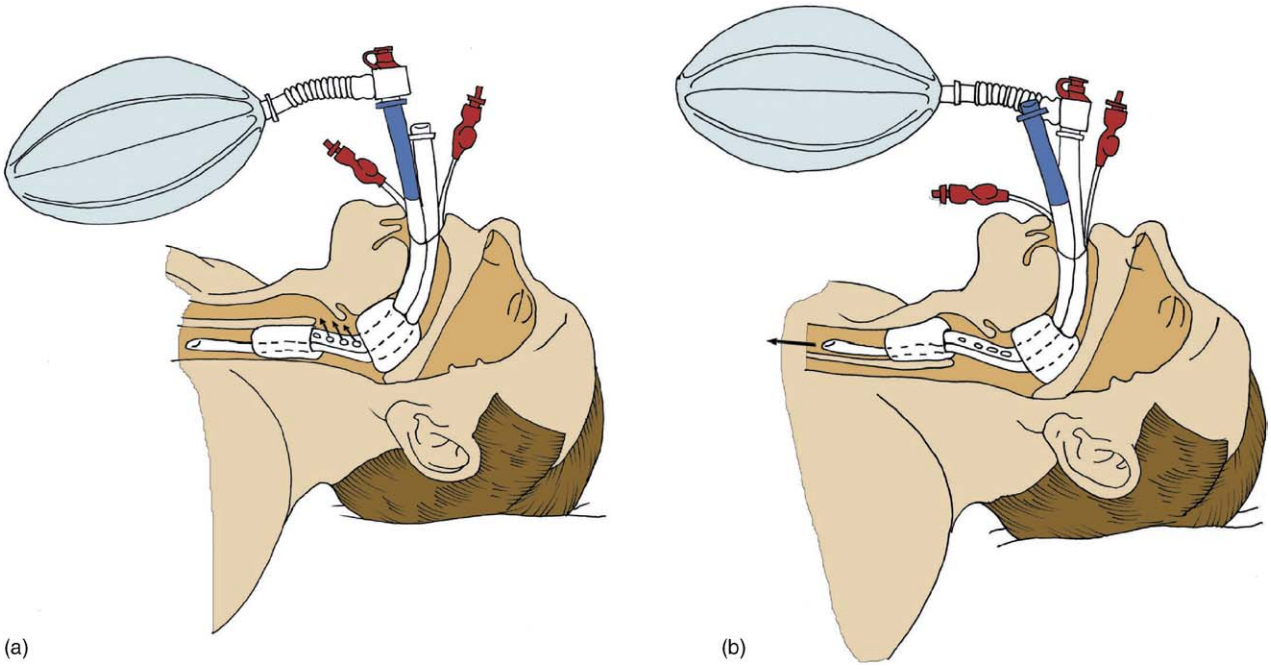

Figure 4.10 (a) Combitube in the oesophageal position. (b) Combitube in the tracheal position. © 2005 European Resuscitation Council.

chea (Figure 4.10b). There are many studies of the Combitube in CPR and successful ventilation was achieved in 79–98% of patients.[146,151–157] All except one[151] of these studies involved out-of-hospital cardiac arrest, which reflects the infrequency with which the Combitube is used in hospitals. On the basis of these studies, the Combitube appears as safe and effective as tracheal intubation for airway management during cardiac arrest; however, there are inadequate survival data to be able to comment with certainty on the impact on outcome. It is possible to attempt to ventilate the lungs through the wrong port of the Combitube (2.2% in one study)[152]: This is equivalent to unrecognised oesophageal intubation with a standard tracheal tube.

Other airway devices

Laryngeal Tube. The LT is a relatively new airway device; its function in anaesthetised patients has been reported in several studies. The performance of the LT is favourable in comparison with the classic LMA and LMA,[158,159] and successful insertion rates have been reported even in studies of paramedics.[160] There are sporadic case reports relating to use of the laryngeal tube during CPR.[161,162] In a recent study, the LT was placed in 30 patients in cardiac arrest out of hospital by minimally trained nurses.[163] LT insertion was successful within two attempts in 90% of patients, and ventilation was adequate in 80% of cases. No regurgitation occurred in any patient.

ProSeal LMA. The ProSeal LMA has been studied extensively in anaesthetised patients, but there are no studies of its function and performance during CPR. It has several attributes that, in theory, make it more suitable than the classic LMA for use during CPR: improved seal with the larynx enabling ventilation at higher airway pressures,[164,165] the inclusion of a gastric drain tube enabling venting of liquid regurgitated gastric contents from the upper oesophagus and passage of a gastric tube to drain liquid gastric contents, and the inclusion of a bite block. The Proseal LMA has potential weaknesses as an airway device for CPR: it is slightly more difficult to insert than a classic LMA, it is not available in disposable form and is relatively expensive, and solid regurgitated gastric contents will block the gastric drainage tube. Data are awaited on its performance during CPR.

Airway management device. In anaesthetised patients, the airway management device (AMD) performed poorly in one study,[166] but a modified version appeared to function slightly better.[167] The pharyngeal airway express (PAX) also performed poorly in one study of anaesthetised patients.[168] There are no data on the use of either of these devices during CPR.

Intubating LMA. The intubating LMA (ILMA) is valuable for managing the difficult airway during anaesthesia, but it has not been studied during CPR. Although it is relatively easy to insert the ILMA,[169,170] reliable, blind insertion of a tracheal tube requires considerable training[171] and, for this reason, it is not an ideal technique for the inexperienced provider.

Tracheal intubation

There is insufficient evidence to support or refute the use of any specific technique to maintain an airway and provide ventilation in adults with cardiopulmonary arrest. Despite this, tracheal intubation is perceived as the optimal method of providing and maintaining a clear and secure airway. It should be used only when trained personnel are available to carry out the procedure with a high level of skill and confidence. The only randomised controlled trial comparing tracheal intubation with bag-mask ventilation was undertaken in children requiring airway management out-of-hospital.[172] In this investigation there was no difference in survival to discharge, but it is unclear how applicable this paediatric study is to adult resuscitation. Two reports compared outcomes from out-of-hospital cardiac arrest in adults when treated by either emergency medical technicians or paramedics.[173,174] The skills provided by the paramedics, including intubation and intravenous cannulation and drug administration,[174] made no difference to survival to hospital discharge.

The perceived advantages of tracheal intubation over bag-mask ventilation include: maintenance of a patent airway, which is protected from aspiration of gastric contents or blood from the oropharynx; ability to provide an adequate tidal volume reliably even when chest compressions are uninterrupted; the potential to free the rescuer's hands for other tasks; the ability to suction airway secretions; and the provision of a route for giving drugs. Use of the bag-mask is more likely to cause gastric distension which, theoretically, is more likely to cause regurgitation with risk of aspiration. However, there are no reliable data to indicate that the incidence of aspiration is any more in cardiac arrest patients ventilated with bag-mask versus those that are ventilated via tracheal tube.

The perceived disadvantages of tracheal intubation over bag-mask ventilation include: the risk of an unrecognised misplaced tracheal tube, which in patients with out-of-hospital cardiac arrest in some studies ranges from 6%[132–134] to 14%[135]; a prolonged period without chest compressions while intubation is attempted; and a comparatively high failure rate. Intubation success rates correlate with the intubation experience attained by individual paramedics.[175] Rates for failure to intubate are as high as 50% in prehospital systems with a low patient volume and providers who do not perform intubation frequently.[134] The cost of training prehospital staff to undertake intubation should also be considered. Healthcare personnel who undertake prehospital intubation should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills.

In some cases, laryngoscopy and attempted intubation may prove impossible or cause life-threatening deterioration in the patient's condition. Such circumstances include acute epiglottal conditions, pharyngeal pathology, head injury (where straining may occur further rise in intracranial pressure) or cervical spine injury. In these circumstances, specialist skills such as the use of anaesthetic drugs or fibreoptic laryngoscopy may be required. These techniques require a high level of skill and training.

Rescuers must weigh the risks and benefits of intubation against the need to provide effective chest compressions. The intubation attempt will require interruption of chest compressions but, once an advanced airway is in place, ventilation will not require interruption of chest compressions. Personnel skilled in advanced airway management should be able to undertake laryngoscopy without stopping chest compressions; a brief pause in chest compressions will be required only as the tube is passed through the vocal cords. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until return of spontaneous circulation. No intubation attempt should take longer than 30 s; if intubation has not been achieved after this time, recommence bag-mask ventilation. After intubation, tube placement must be confirmed and the tube secured adequately.

Confirmation of correct placement of the tracheal tube

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine use of primary and secondary techniques to confirm correct placement of the tracheal tube should reduce this risk. Primary assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and adequate) and over the epigastrium (breath sounds should not be heard). Clinical signs of correct tube placement (condensation in the tube, chest rise, breath sounds on auscultation of lungs, and inability to hear gas entering the stomach) are not completely reliable. Secondary confirmation of tracheal tube placement by an exhaled carbon dioxide or oesophageal detection device should reduce the risk of unrecognised oesophageal intubation. If there is doubt about correct tube placement, use the laryngoscope and look directly to see if the tube passes through the vocal cords.

None of the secondary confirmation techniques will differentiate between a tube placed in a main bronchus and one placed correctly in the trachea. There are inadequate data to identify the optimal method of confirming tube placement during cardiac arrest, and all devices should be considered as adjuncts to other confirmatory techniques.[176] There are no data quantifying their ability to monitor tube position after initial placement.

The oesophageal detector device creates a suction force at the tracheal end of the tracheal tube, either by pulling back the plunger on a large syringe or releasing a compressed flexible bulb. Air is aspirated easily from the lower airways through a tracheal tube placed in the cartilage-supported rigid trachea. When the tube is in the oesophagus, air cannot be aspirated because the oesophagus collapses when aspiration is attempted. The oesophageal detector device is generally reliable in patients with both a perfusing and a non-perfusing rhythm, but it may be misleading in patients with morbid obesity, late pregnancy or severe asthma or when there are copious tracheal secretions; in these conditions the trachea may collapse when aspiration is attempted.[133,177–180]

Carbon dioxide detector devices measure the concentration of exhaled carbon dioxide from the lungs. The persistence of exhaled carbon dioxide after six ventilations indicates placement of the tracheal tube in the trachea or a main bronchus.[181] Confirmation of correct placement above the carina will require auscultation of the chest bilaterally in the mid-axillary lines. In patients with a spontaneous circulation, a lack of exhaled carbon dioxide indicates that the tube is in the oesophagus. During cardiac arrest, pulmonary blood flow may be so low that there is insufficient exhaled carbon dioxide, so the detector does not identify a correctly placed tracheal tube. When exhaled carbon dioxide

is detected in cardiac arrest, it indicates reliably that the tube is in the trachea or main bronchus but, when it is absent, tracheal tube placement is best confirmed with an oesophageal detector device. A variety of electronic as well as simple, inexpensive, colorimetric carbon dioxide detectors are available for both in-hospital and out-of-hospital use.

Cricoid pressure

During bag-mask ventilation and attempted intubation, cricoid pressure applied by a trained assistant should prevent passive regurgitation of gastric contents and the consequent risk of pulmonary aspiration. If the technique is applied imprecisely or with excessive force, ventilation and intubation can be made more difficult.[128] If ventilation of the patient's lungs is not possible, reduce the pressure applied to the cricoid cartilage or remove it completely. If the patient vomits, release the cricoid immediately.

Securing the tracheal tube

Accidental dislodgement of a tracheal tube can occur at any time, but may be more likely during resuscitation and during transport. The most effective method for securing the tracheal tube has yet to be determined; use either conventional tapes or ties, or purpose-made tracheal tube holders.

Cricothyroidotomy

Occasionally, it will be impossible to ventilate an apnoeic patient with a bag-mask, or to pass a tracheal tube or alternative airway device. This may occur in patients with extensive facial trauma or laryngeal obstruction due to oedema or foreign material. In these circumstances, delivery of oxygen through a needle or surgical cricothyroidotomy may be life-saving. A tracheostomy is contraindicated in an emergency, as it is time consuming, hazardous and requires considerable surgical skill and equipment.

Surgical cricothyroidotomy provides a definitive airway that can be used to ventilate the patient's lungs until semi-elective intubation or tracheostomy is performed. Needle cricothyroidotomy is a much more temporary procedure providing only short-term oxygenation. It requires a wide-bore, non-kinking cannula, a high-pressure oxygen source, runs the risk of barotrauma and can be particularly ineffective in patients with chest trauma. It is also prone to failure because of kinking of the cannula, and is unsuitable for patient transfer.

4e. Assisting the circulation

Drugs and fluids for cardiac arrest

This topic is divided into: drugs used during the management of a cardiac arrest; anti-arrhythmic drugs used in the peri-arrest period; other drugs used in the peri-arrest period; fluids; and routes for drug delivery. Every effort has been made to provide accurate information on the drugs in these guidelines, but literature from the relevant pharmaceutical companies will provide the most up-to-date data.

Drugs used during the treatment of cardiac arrest

Only a few drugs are indicated during the immediate management of a cardiac arrest, and there is limited scientific evidence supporting their use. Drugs should be considered only after initial shocks have been delivered (if indicated) and chest compressions and ventilation have been started.

There are three groups of drugs relevant to the management of cardiac arrest that were reviewed during the 2005 Consensus Conference: vasopressors, anti-arrhythmics and other drugs. Routes of drug delivery other than the optimal intravenous route were also reviewed and are discussed.

Vasopressors

There are currently no placebo-controlled studies showing that the routine use of any vasopressor at any stage during human cardiac arrest increases survival to hospital discharge. The primary goal of cardiopulmonary resuscitation is to re-establish blood flow to vital organs until the restoration of spontaneous circulation. Despite the lack of data from cardiac arrest in humans, vasopressors continue to be recommended as a means of increasing cerebral and coronary perfusion during CPR.

Adrenaline (epinephrine) versus vasopressin. Adrenaline has been the primary sympathomimetic agent for the management of cardiac arrest for 40 years.[182] Its primary efficacy is due to its alpha-adrenergic, vasoconstrictive effects causing systemic vasoconstriction, which increases coronary and cerebral perfusion pressures. The beta-adrenergic actions of adrenaline (inotropic, chronotropic) may increase coronary and cerebral blood flow, but concomitant increases in myocardial oxygen consumption, ectopic ventricular arrhythmias (particularly when the myocardium is acidotic) and transient hypoxaemia due to

pulmonary arteriovenous shunting may offset these benefits.

The potentially deleterious beta-effects of adrenaline have led to exploration of alternative vasopressors. Vasopressin is a naturally occurring antidiuretic hormone. In very high doses it is a powerful vasoconstrictor that acts by stimulation of smooth muscle V1 receptors. The importance of vasopressin in cardiac arrest was first recognised in studies of out-of-hospital cardiac arrest patients, where vasopressin levels were found to be higher in successfully resuscitated patients.[183,184] Although clinical[185,186] and animal[187–189] studies demonstrated improved haemodynamic variables when using vasopressin as an alternative to adrenaline during resuscitation from cardiac arrest, some,[186] but not all, demonstrated improved survival.[190,191]

The first clinical use of vasopressin during cardiac arrest was reported in 1996 and appeared promising. In a study of cardiac arrest patients refractory to standard therapy with adrenaline, vasopressin restored a spontaneous circulation in all eight patients, three of whom were discharged neurologically intact.[186] The following year, the same group published a small randomised trial of out-of-hospital ventricular fibrillation, in which the rates of successful resuscitation and survival for 24 h were significantly higher in patients treated with vasopressin than in those treated with adrenaline.[192] Following these two studies, the American Heart Association (AHA) recommended that vasopressin could be used as an alternative to adrenaline for the treatment of adult shock-refractory VF.[182] The success of these small studies led to two large randomised studies comparing vasopressin with adrenaline for in-hospital[193] and out-of-hospital[194] cardiac arrest. Both studies randomised patients to receive vasopressin or adrenaline initially, and used adrenaline as a rescue treatment in patients refractory to the initial drug. Both studies were unable to demonstrate an overall increase in the rates of ROSC or survival for vasopressin 40 U,[193] with the dose repeated in one study,[194] when compared with adrenaline (1 mg, repeated), as the initial vasopressor. In the large out-of-hospital cardiac arrest study,[194] post-hoc analysis suggested that the subset of patients with asystole had significant improvement in survival to discharge, but survival neurologically intact was no different.

A recent meta-analysis of five randomised trials[195] showed no statistically significant difference between vasopressin and adrenaline for ROSC, death within 24 h or death before hospital discharge. The subgroup analysis based on initial cardiac rhythm did not show any statistically significant difference in the rate of death before hospital discharge.[195]

Participants at the 2005 Consensus Conference debated in depth the treatment recommendations that should follow from this evidence. Despite the absence of placebo-controlled trials, adrenaline has been the standard vasopressor in cardiac arrest. It was agreed that there is currently insufficient evidence to support or refute the use of vasopressin as an alternative to, or in combination with, adrenaline in any cardiac arrest rhythm. Current practice still supports adrenaline as the primary vasopressor for the treatment of cardiac arrest of all rhythms.

Adrenaline

Indications

- Adrenaline is the first drug used in cardiac arrest of any aetiology: it is included in the ALS algorithm for use every 3–5 min of CPR.
- Adrenaline is preferred in the treatment of anaphylaxis (Section 7g).
- Adrenaline is second-line treatment for cardiogenic shock.

Dose. During cardiac arrest, the initial intravenous dose of adrenaline is 1 mg. When intravascular (intravenous or intra-osseous) access is delayed or cannot be achieved, give 2–3 mg, diluted to 10 ml with sterile water, via the tracheal tube. Absorption via the tracheal route is highly variable.

There is no evidence supporting the use of higher doses of adrenaline for patients in refractory cardiac arrest. In some cases, an adrenaline infusion is required in the post-resuscitation period.

Following return of spontaneous circulation, excessive ($\geq$1 mg) doses of adrenaline may induce tachycardia, myocardial ischaemia, VT and VF. Once a perfusing rhythm is established, if further adrenaline is deemed necessary, titrate the dose carefully to achieve an appropriate blood pressure. Intravenous doses of 50–100 mcg are usually sufficient for most hypotensive patients. Use adrenaline cautiously in patients with cardiac arrest associated with cocaine or other sympathomimetic drugs.

Use. Adrenaline is available most commonly in two dilutions:

- 1 in 10,000 (10 ml of this solution contains 1 mg of adrenaline)
- 1 in 1000 (1 ml of this solution contains 1 mg of adrenaline)

Both these dilutions are used routinely in European countries.

Various other pressor drugs (e.g., noradrenaline)[196] have been used experimentally as an alternative to adrenaline for the treatment of cardiac arrest.

Anti-arrhythmics

As with vasopressors, the evidence that anti-arrhythmic drugs are of benefit in cardiac arrest is limited. No anti-arrhythmic drug given during human cardiac arrest has been shown to increase survival to hospital discharge, although amiodarone has been shown to increase survival to hospital admission.[89,90] Despite the lack of human long-term outcome data, the balance of evidence is in favour of the use anti-arrhythmic drugs for the management of arrhythmias in cardiac arrest.

Amiodarone. Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Atrioventricular conduction is slowed, and a similar effect is seen with accessory pathways. Amiodarone has a mild negative inotropic action and causes peripheral vasodilation through non-competitive alpha-blocking effects. The hypotension that occurs with intravenous amiodarone is related to the rate of delivery and is due more to the solvent (Polysorbate 80), which causes histamine release, rather than the drug itself.[197] The use of an aqueous amiodarone preparation that is relatively free from these side effects is encouraged but is not yet widely available [198,199].

Following three initial shocks, amiodarone in shock-refractory VF improves the short-term outcome of survival to hospital admission compared with placebo[89] or lignocaine.[90] Amiodarone also appears to improve the response to defibrillation when given to humans or animals with VF or haemodynamically unstable ventricular tachycardia.[198–202] There is no evidence to indicate the time at which amiodarone should be given when using a single shock strategy. In the clinical studies to date, the amiodarone was given if VF/VT persisted after at least three shocks. For this reason, and in the absence of any other data, amiodarone 300 mg is recommended if VF/VT persists after three shocks.

Indications. Amiodarone is indicated in

- refractory VF/VT
- haemodynamically stable ventricular tachycardia (VT) and other resistant tachyarrhythmias (Section 4f)

Dose. Consider an initial intravenous dose of 300 mg amiodarone, diluted in 5% dextrose to a volume of 20 ml (or from a pre-filled syringe), if VF/VT persists after the third shock. Amiodarone can cause thrombophlebitis when injected into a peripheral vein; use a central venous catheter if one is in situ but,if not, use a large peripheral vein and a generous flush. Details about the use of amiodarone for the treatment of other arrhythmias are given in Section 4f.

Clinical aspects of use. Amiodarone may paradoxically be arrhythmogenic, especially if given concurrently with drugs that prolong the QT interval. However, it has a lower incidence of pro-arrhythmic effects than other anti-arrhythmic drugs under similar circumstances. The major acute adverse effects from amiodarone are hypotension and bradycardia, which can be prevented by slowing the rate of drug infusion, or can be treated with fluids and/or inotropic drugs. The side effects associated with prolonged oral use (abnormalities of thyroid function, corneal microdeposits, peripheral neuropathy, and pulmonary/hepatic infiltrates) are not relevant in the acute setting.

Lidocaine. Until the publication of the 2000 ILCOR guidelines, lidocaine was the antiarrhythmic drug of choice. Comparative studies with amiodarone[90] have displaced it from this position, and lidocaine is now recommended only when amiodarone is unavailable. Amiodarone should be available at all hospital arrests and to all out-of-hospital arrests attended by ambulance crew.

Lidocaine is a membrane-stabilising anti-arrhythmic drug that acts by increasing the myocyte refractory period. It decreases ventricular automaticity, and its local anaesthetic action suppresses ventricular ectopic activity. Lidocaine suppresses activity of depolarised, arrhythmogenic tissues while interfering minimally with the electrical activity of normal tissues. Therefore, it is effective in suppressing arrhythmias associated with depolarisation (e.g. ischaemia, digitalis toxicity) but is relatively ineffective against arrhythmias occurring in normally polarised cells (e.g., atrial fibrillation/flutter). Lidocaine raises the threshold for ventricular fibrillation.

Lidocaine toxicity causes paraesthesia, drowsiness, confusion and muscular twitching progressing to convulsions. It is considered generally that a safe dose of lidocaine must not exceed 3 mg kg^{-1} over the first hour. If there are signs of toxicity, stop the infusion immediately; treat seizures if they occur. Lidocaine depresses myocardial function, but to a much lesser extent than amiodarone. The myocardial depression is usually transient and can be treated with intravenous fluids or vasopressors.

Indications. Lidocaine is indicated in refractory VF/VT (when amiodarone is unavailable).

Dose. When amiodarone is unavailable, consider an initial dose of 100 mg (1—1.5 mg kg^{-1}) of lidocaine for VF/pulseless VT refractory to three shocks. Give an additional bolus of 50 mg if necessary. The total dose should not exceed 3 mg kg^{-1} during the first hour.

Clinical aspects of use. Lidocaine is metabolised by the liver, and its half-life is prolonged if the hepatic blood flow is reduced, e.g. in the presence of reduced cardiac output, liver disease or in the elderly. During cardiac arrest normal clearance mechanisms do not function, thus high plasma concentrations may be achieved after a single dose. After 24 h of continuous infusion, the plasma half-life increases significantly. Reduce the dose in these circumstances, and regularly review the indication for continued therapy. Lidocaine is less effective in the presence of hypokalaemia and hypomagnesaemia, which should be corrected immediately.

Magnesium sulphate. Magnesium is an important constituent of many enzyme systems, especially those involved with ATP generation in muscle. It plays a major role in neurochemical transmission, where it decreases acetylcholine release and reduces the sensitivity of the motor endplate. Magnesium also improves the contractile response of the stunned myocardium, and limits infarct size by a mechanism that has yet to be fully elucidated.[203] The normal plasma range of magnesium is 0.8—1.0 mmol l^{-1}.

Hypomagnesaemia is often associated with hypokalaemia, and may contribute to arrhythmias and cardiac arrest. Hypomagnesaemia increases myocardial digoxin uptake and decreases cellular Na^+/K^+-ATP-ase activity. Patients with hypomagnesaemia, hypokalaemia, or both may become cardiotoxic even with therapeutic digitalis levels. Magnesium deficiency is not uncommon in hospitalised patients and frequently coexists with other electrolyte disturbances, particularly hypokalaemia, hypophosphataemia, hyponatraemia and hypocalcaemia.

Although the benefits of giving magnesium in known hypomagnesaemic states are recognised, the benefit of giving magnesium routinely during cardiac arrest is unproven. Studies in adults in and out of hospital[91—95,204] have failed to demonstrate any increase in the rate of ROSC when magnesium is given routinely during CPR. There is some evidence that magnesium may be beneficial in refractory VF.[205]

Indications. Magnesium sulphate is indicated in

- shock-refractory VF in the presence of possible hypomagnesaemia
- ventricular tachyarrhythmias in the presence of possible hypomagnesaemia
- torsades de pointes
- digoxin toxicity

Dose. In shock-refractory VF, give an initial intravenous dose of 2 g (4 ml (8 mmol)) of 50% magnesium sulphate) peripherally over 1—2 min; it may be repeated after 10—15 min. Preparations of magnesium sulphate solutions differ among European countries.

Clinical aspects of use. Hypokalaemic patients are often hypomagnesaemic. If ventricular tachyarrhythmias arise, intravenous magnesium is a safe, effective treatment. The role of magnesium in acute myocardial infarction is still in doubt. Magnesium is excreted by the kidneys, but side effects associated with hypermagnesaemia are rare, even in renal failure. Magnesium inhibits smooth muscle contraction, causing vasodilation and a dose-related hypotension, which is usually transient and responds to intravenous fluids and vasopressors.

Other drugs

The evidence for the benefits of other drugs, including atropine, aminophylline and calcium, given routinely during human cardiac arrest, is limited. Recommendations for the use of these drugs are based on our understanding of their pharmacodynamic properties and the pathophysiology of cardiac arrest.

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

Side effects of atropine are dose-related (blurred vision, dry mouth and urinary retention); they are not relevant during a cardiac arrest. Acute confusional states may occur after intravenous injection, particularly in elderly patients. After cardiac arrest, dilated pupils should not be attributed solely to atropine.

Atropine is indicated in:

- asystole
- pulseless electrical activity (PEA) with a rate <60 min^{-1}
- sinus, atrial, or nodal bradycardia when the haemodynamic condition of the patient is unstable

The recommended adult dose of atropine for asystole or PEA with a rate <60 min^{-1} is 3 mg intravenously in a single bolus. Its use in the treatment of bradycardia is covered in Section 4f. Several recent studies have failed to demonstrate any benefit from atropine in out-of-hospital or in-hospital cardiac arrests[174,206–210]; however, asystole carries a grave prognosis and there are anecdotal accounts of success after giving atropine. It is unlikely to be harmful in this situation.

Theophylline (aminophylline). Theophylline is a phosphodiesterase inhibitor that increases tissue concentrations of cAMP and releases adrenaline from the adrenal medulla. It has chronotropic and inotropic actions. The limited studies of aminophylline in bradyasystolic cardiac arrest have failed to demonstrate an increase in ROSC or survival to hospital discharge[211–214]; the same studies have not shown that harm is caused by aminophylline.

Aminophylline is indicated in:

- asystolic cardiac arrest
- peri-arrest bradycardia refractory to atropine

Theophylline is given as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone. The recommended adult dose is 250–500 mg (5 mg kg^{-1}) given by slow intravenous injection.

Theophylline has a narrow therapeutic window with an optimal plasma concentration of 10–20 mg l^{-1} (55–110 mmol l^{-1}). Above this concentration, side effects such as arrhythmias and convulsions may occur, especially when given rapidly by intravenous injection.

Calcium. Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. There are very few data supporting any beneficial action for calcium after most cases of cardiac arrest. High plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may impair cerebral recovery. Give calcium during resuscitation only when indicated specifically, i.e. in pulseless electrical activity caused by

- hyperkalaemia
- hypocalcaemia
- overdose of calcium channel-blocking drugs

The initial dose of 10 ml 10% calcium chloride (6.8 mmol Ca^{2+}) may be repeated if necessary. Calcium can slow the heart rate and precipitate arrhythmias. In cardiac arrest, calcium may be given by rapid intravenous injection. In the presence of a spontaneous circulation give it slowly. Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.

Buffers. Cardiac arrest results in combined respiratory and metabolic acidosis caused by cessation of pulmonary gas exchange and the development of anaerobic cellular metabolism, respectively. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. If the arterial blood pH is less than 7.1 (or base excess more negative than −10 mmol l^{-1}) during or following resuscitation from cardiac arrest, consider giving small doses of sodium bicarbonate (50 ml of an 8.4% solution). During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid–base state[96]; analysis of central venous blood may provide a better estimation of tissue pH (see Section 4c). Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. This has the following effects.

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

Mild acidaemia causes vasodilation and can increase cerebral blood flow. Therefore, full correction of the arterial blood pH may theoretically reduce cerebral blood flow at a particularly critical time. As the bicarbonate ion is excreted as carbon dioxide via the lungs, ventilation needs to be increased. For all these reasons, metabolic acidosis must be severe to justify giving sodium bicarbonate.

Several animal and clinical studies have examined the use of buffers during cardiac arrest. Clinical studies using Tribonate®[215] or sodium bicarbonate as buffers have failed to demonstrate any advantage.[216–220] Only one study has found clinical benefit, suggesting that EMS systems using sodium bicarbonate earlier and more frequently had significantly higher ROSC and hospital discharge rates and better long-term neurological outcome.[221] Animal studies have generally been inconclusive, but some have shown benefit in giving sodium bicarbonate to treat cardiovascular toxicity (hypotension, cardiac arrhythmias) caused by tricyclic antidepressants and other fast sodium channel blockers (Section 7b).[222] Giving sodium bicarbonate routinely during cardiac arrest and CPR (especially in out-

of-hospital cardiac arrests) or after return of spontaneous circulation is not recommended. Consider sodium bicarbonate for life-threatening hyperkalaemia or cardiac arrest associated with hyperkalaemia, severe metabolic acidosis, or tricyclic overdose. Give 50 mmol (50 ml of an 8.4% solution) of sodium bicarbonate intravenously. Repeat the dose as necessary, but use acid/base analysis (either arterial or central venous) to guide therapy. Severe tissue damage may be caused by subcutaneous extravasation of concentrated sodium bicarbonate. The solution is incompatible with calcium salts as it causes the precipitation of calcium carbonate.

Thrombolysis during CPR. Adult cardiac arrest is usually caused by acute myocardial ischaemia following coronary artery occlusion by thrombus. There are several reports on the successful use of thrombolytics during cardiac arrest, particularly when the arrest was caused by pulmonary embolism. The use of thrombolytic drugs to break down coronary artery and pulmonary artery thrombus has been the subject of several studies. Thrombolytics have also been demonstrated in animal studies to have beneficial effects on cerebral blood flow during cardiopulmonary resuscitation,[223,224] and a clinical study has reported less anoxic encephalopathy after thrombolytic therapy during CPR.[225]

Several studies have examined the use of thrombolytic therapy given during non-traumatic cardiac arrest refractory to standard therapy. Two studies have shown an increase in ROSC with non-significant improvements in survival to hospital discharge,[97,226] and a further study demonstrated greater ICU survival.[225] A small series of case reports has also reported survival to discharge in three cases refractory to standard therapy with VF or PEA treated with thrombolytics[227]; conversely, one large clinical trial[228] failed to show any significant benefit for thrombolytics in cases of undifferentiated PEA out-of-hospital cardiac arrest unresponsive to initial interventions.

When given to cardiac arrest patients with suspected or proven pulmonary embolus, two studies have demonstrated possible benefits[229,230]; one found an improvement in 24-h survival.[229] Several clinical studies[97,226,229,231] and case series[227,230,232–234] have not demonstrated any increase in bleeding complications with thrombolysis during CPR in non-traumatic cardiac arrest.

There are insufficient clinical data to recommend the routine use of thrombolysis during non-traumatic cardiac arrest. Consider thrombolytic therapy when cardiac arrest is thought to be due to proven or suspected acute pulmonary embolus. Thrombolysis may be considered in adult cardiac arrest on a case by case basis following initial failure of standard resuscitation in patients in whom an acute thrombotic aetiology for the arrest is suspected. Ongoing CPR is not a contraindication to thrombolysis.

Following thrombolysis 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 thrombolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts.[235,236]

Intravenous fluids

Hypovolaemia is a potentially reversible cause of cardiac arrest. Infuse fluids rapidly if hypovolaemia is suspected. In the initial stages of resuscitation there are no clear advantages to using colloid, so use saline or Hartmann's solution. Avoid dextrose, which is redistributed away from the intravascular space rapidly and causes hyperglycaemia, which may worsen neurological outcome after cardiac arrest.[237–244]

Whether fluids should be infused routinely during cardiac arrest is controversial. There are no published human studies of routine fluid use compared to no fluids during normovolaemic cardiac arrest. Four animal studies[245–248] of experimental ventricular fibrillation neither support nor refute the use of intravenous fluids routinely. In the absence of hypovolaemia, infusion of an excessive volume of fluid is likely to be harmful. Use intravenous fluid to flush peripherally injected drugs into the central circulation.

Alternative routes for drug delivery

Intraosseous route

If intravenous access cannot be established, intraosseous delivery of resuscitation drugs will achieve adequate plasma concentrations. Several studies indicate that intraosseous access is safe and effective for fluid resuscitation, drug delivery and laboratory evaluation.[78,249–255] Traditionally, the intraosseous route is used mainly for children, but it is also effective in adults.

Drugs given via the tracheal tube

Resuscitation drugs can also be given via the tracheal tube, but the plasma concentrations achieved using this route are variable and substantially

lower than those achieved by the intravenous or intraosseous routes.

Doses of adrenaline 3—10 times higher than when given intravenously are required to achieve similar plasma concentrations.[79,80] During CPR, lung perfusion is only 10—30% of the normal value, resulting in a pulmonary adrenaline depot. When cardiac output is restored after a high dose of endobronchial adrenaline, prolonged reabsorption of adrenaline from the lungs into the pulmonary circulation may occur, causing arterial hypertension, malignant arrhythmias and recurrence of VF.[80] Lidocaine and atropine can also be given via a tracheal tube, but the plasma concentrations achieved are also variable.[256—258] If intravenous access is delayed or cannot be achieved, consider obtaining intraosseous access. Give drugs via the tracheal tube if intravascular (intravenous or intraosseous) access is delayed or cannot be achieved. There are no benefits from endobronchial injection compared with injection of the drug directly into the tracheal tube.[256] Dilution with water instead of 0.9% saline may achieve better drug absorption and cause less reduction in PaO_2.[85,259]

CPR techniques and devices

At best, standard manual CPR produces coronary and cerebral perfusion that is just 30% of normal.[260] Several CPR techniques and devices may improve haemodynamics or short-term survival when used by well-trained providers in selected cases. To date, no adjunct has consistently been shown to be superior to conventional manual CPR. CPR techniques include the following.

High-frequency chest compressions (HFCC)

High-frequency (>100 compressions min^{-1}) manual or mechanical chest compressions improve haemodynamics but have not been shown to improve long-term outcome.[261—265]

Open-chest CPR

Open-chest CPR produces better coronary perfusion coronary pressure than standard CPR[266] and may be indicated for patients with cardiac arrest due to trauma (see Section 7i), in the early postoperative phase after cardiothoracic surgery[267,268] (see Section 7h) or when the chest or abdomen is already open (transdiaphragmatic approach), for example, in trauma surgery.

Interposed abdominal compression (IAC-CPR)

The IAC-CPR technique involves compression of the abdomen during the relaxation phase of chest compression.[269,270] This enhances venous return during CPR[271,272] and improves ROSC and short-term survival.[273,274] One study showed improved survival to hospital discharge with IAC-CPR compared with standard CPR for out-of-hospital cardiac arrest,[274] but another showed no survival advantage.[275] CPR devices include the following.

Active compression-decompression CPR (ACD-CPR)

ACD-CPR is achieved with a hand-held device equipped with a suction cup to lift the anterior chest actively during decompression. Decreasing intrathoracic pressure during the decompression phase increases venous return to the heart and increases cardiac output and subsequent coronary and cerebral perfusion pressures during the compression phase.[276—279]Results of ACD-CPR have been mixed. In some clinical studies ACD-CPR improved haemodynamics compared with standard CPR,[173,277,279,280] but in another study it did not.[281] In three randomised studies,[280,282,283] ACD-CPR improved long-term survival after out-of-hospital cardiac arrest; however, in five other randomised studies, ACD-CPR made no difference to outcome.[284—288] The efficacy of ACD-CPR may be highly dependent on the quality and duration of training.[289]

A meta-analysis of 10 trials of out-of-hospital cardiac arrest and two of in-hospital cardiac arrest showed no early or late survival benefit to ACD-CPR over conventional CPR.[290] Two post-mortem studies have shown more rib and sternal fractures after ACD-CPR compared with conventional CPR,[291,292] but another found no difference.[293]

Impedance threshold device (ITD)

The impedance threshold device (ITD) is a valve that limits air entry into the lungs during chest recoil between chest compressions; this decreases intrathoracic pressure and increases venous return to the heart. When used with a cuffed tracheal tube and active compression-decompression (ACD),[294—296] the ITD is thought to act synergistically to enhance venous return during active decompression. The ITD has also been used during conventional CPR with a tracheal tube or facemask.[297] If rescuers can maintain a tight facemask seal, the ITD may create the same negative

intrathoracic pressure as when used with a tracheal tube.[297]

In two randomised studies of out-of-hospital cardiac arrest, ACD-CPR plus the ITD improved ROSC and 24-h survival compared with standard CPR alone.[296,298] When used during standard CPR, the ITD increased 24-h survival after PEA out-of-hospital cardiac arrest.[297]

Mechanical piston CPR

Mechanical piston devices depress the sternum by means of a compressed gas-powered plunger mounted on a backboard. In several studies in animals,[299,300] mechanical piston CPR improved end-tidal carbon dioxide, cardiac output, cerebral blood flow, MAP and short-term neurological outcome. Studies in humans also document improvement in end-tidal carbon dioxide and mean arterial pressure when using mechanical piston CPR compared with conventional CPR.[301–303]

Lund University cardiac arrest system (LUCAS) CPR

The Lund University cardiac arrest system (LUCAS) is a gas-driven sternal compression device that incorporates a suction cup for active decompression. There are no published randomised human studies comparing LUCAS-CPR with standard CPR. A study of pigs with VF showed that LUCAS-CPR improves haemodynamic and short-term survival compared with standard CPR.[304] The LUCAS was also used in 20 patients, but incomplete outcome data were reported.[304] In another pig study, in comparison with standard CPR, LUCAS-CPR increased cerebral blood flow and cardiac output.[305] The LUCAS enables delivery of continuous compressions during transport and defibrillation.

Mechanical piston CPR or LUCAS CPR may be particularly useful when prolonged CPR is required; this might include during transport to hospital or after cardiac arrest following hypothermia[306] or poisoning.

Load-distributing band CPR or vest CPR

The load distributing band (LDB) is a circumferential chest compression device comprising a pneumatically actuated constricting band and backboard. The use of LDB CPR improves haemodynamics.[307–309] A case–control study documented improvement in survival to the emergency department when LDB-CPR was delivered after out-of-hospital cardiac arrest.[310]

Phased thoracic–abdominal compression–decompression CPR (PTACD-CPR)

Phased thoracic–abdominal compression–decompression CPR combines the concepts of IAC-CPR and ACD-CPR. It comprises a hand-held device that alternates chest compression and abdominal decompression with chest decompression and abdominal compression. One randomised study of adults in cardiac arrest documented no improvement in survival from use of PTACD-CPR.[311]

Minimally invasive direct cardiac massage

Minimally invasive direct cardiac massage (MIDCM) is accomplished by insertion of a small plunger-like device through a 2–4-cm incision in the chest wall. In one clinical study the MIDCM generated improved blood pressure over standard CPR, but the device caused cardiac rupture in one postoperative cardiovascular surgical patient.[312] The plunger device is no longer manufactured.

4f. Peri-arrest arrhythmias

Introduction

A successful strategy to reduce the mortality and morbidity of cardiac arrest includes measures to prevent other potentially serious arrhythmias, and optimal treatment should they occur. Cardiac arrhythmias are well recognised complications of myocardial infarction. They may precede ventricular fibrillation or follow successful defibrillation. The treatment algorithms described in this section have been designed to enable the non-specialist ALS provider to treat the patient effectively and safely in an emergency; for this reason, they have been kept as simple as possible. If patients are not acutely ill there may be several other treatment options, including the use of drugs (oral or parenteral), that will be less familiar to the non-expert. In this situation there will be time to seek advice from cardiologists or other doctors with the appropriate expertise.

More comprehensive information on the management of arrhythmias can be found at www.escardio.org.

Principles of treatment

In all cases, give oxygen and insert an intravenous cannula while the arrhythmia is assessed. Whenever possible, record a 12-lead ECG; this will help determine the precise rhythm, either before treatment

or retrospectively, if necessary with the help of an expert. Correct any electrolyte abnormalities (e.g., K^+, Mg^{2+}, Ca^{2+}) (Section 7a).

The assessment and treatment of all arrhythmias addresses two factors: the condition of the patient (stable versus unstable), and the nature of the arrhythmia.

Adverse signs

The presence or absence of adverse signs or symptoms will dictate the appropriate treatment for most arrhythmias. The following adverse factors indicate a patient who is unstable because of the arrhythmia.

1. Clinical evidence of low cardiac output. This is seen as pallor, sweating, cold and clammy extremities (increased sympathetic activity), impaired consciousness (reduced cerebral blood flow), and hypotension (e.g., systolic blood pressure <90 mmHg).
2. Excessive tachycardia. Coronary blood flow occurs predominantly during diastole. Very high heart rates (e.g., >150 min^{-1}) reduce diastole critically, decreasing coronary blood flow and causing myocardial ischaemia. Broad, complex tachycardias are tolerated less well by the heart than narrow, complex tachycardias.
3. Excessive bradycardia. This is defined as a heart rate of <40 beats min^{-1}, but rates of <60 beats min^{-1} may not be tolerated by patients with poor cardiac reserve. Even a higher heart rate may be inappropriately slow for a patient with a low stroke-volume.
4. Heart failure. By reducing coronary artery blood flow, arrhythmias compromise myocardial performance. In acute situations this is manifested by pulmonary oedema (failure of the left ventricle) or raised jugular venous pressure, and hepatic engorgement (failure of the right ventricle).
5. Chest pain. The presence of chest pain implies that the arrhythmia (particularly a tachyarrhythmia) is causing myocardial ischaemia. This is especially important if there is underlying coronary artery disease or structural heart disease in which myocardial ischaemia is likely to lead to further life-threatening complications including cardiac arrest.

Treatment options

Having determined the rhythm and the presence or absence of adverse signs, there are broadly three options for immediate treatment:

1. anti-arrhythmic (and other) drugs
2. attempted electrical cardioversion
3. cardiac pacing

All anti-arrhythmic treatments—physical manoeuvres, drugs, or electrical treatment—can also be pro-arrhythmic, so that clinical deterioration may be caused by the treatment rather than lack of effect. Furthermore, the use of multiple anti-arrhythmic drugs or high doses of a single drug can cause myocardial depression and hypotension. This may cause a deterioration of the cardiac rhythm. Anti-arrhythmic drugs are slower in effect and less reliable than electrical cardioversion in converting a tachycardia to sinus rhythm; thus, drugs tend to be reserved for stable patients without adverse signs, and electrical cardioversion is usually the preferred treatment for the unstable patient displaying adverse signs.

Once the arrhythmia has been treated successfully, repeat the 12-lead ECG to enable detection of any underlying abnormalities that may require long-term therapy.

Bradycardia

A bradycardia is defined strictly as a heart rate of <60 beats min^{-1}. However, it is more helpful to classify a bradycardia as absolute (<40 beats min^{-1}) or relative, when the heart rate is inappropriately slow for the haemodynamic state of the patient.

The first step in the assessment of bradycardia is to determine if the patient is unstable (Figure 4.11). The following adverse signs may indicate instability:

- systolic blood pressure <90 mmHg
- heart rate <40 beats min^{-1}
- ventricular arrhythmias requiring suppression
- heart failure

If adverse signs are present, give atropine, 500 mcg, intravenously and, if necessary, repeat every 3—5 min to a total of 3 mg. Doses of atropine of less than 500 mcg paradoxically may cause further slowing of the heart rate.[313] In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart rate.[314] Use atropine cautiously in the presence of acute coronary ischaemia or myocardial infarction; increased heart rate may worsen ischaemia or increase the zone of infarction. If a satisfactory response is achieved with atropine, or the patient is stable, next determine the risk of asystole, which is indicated by:

- recent asystole

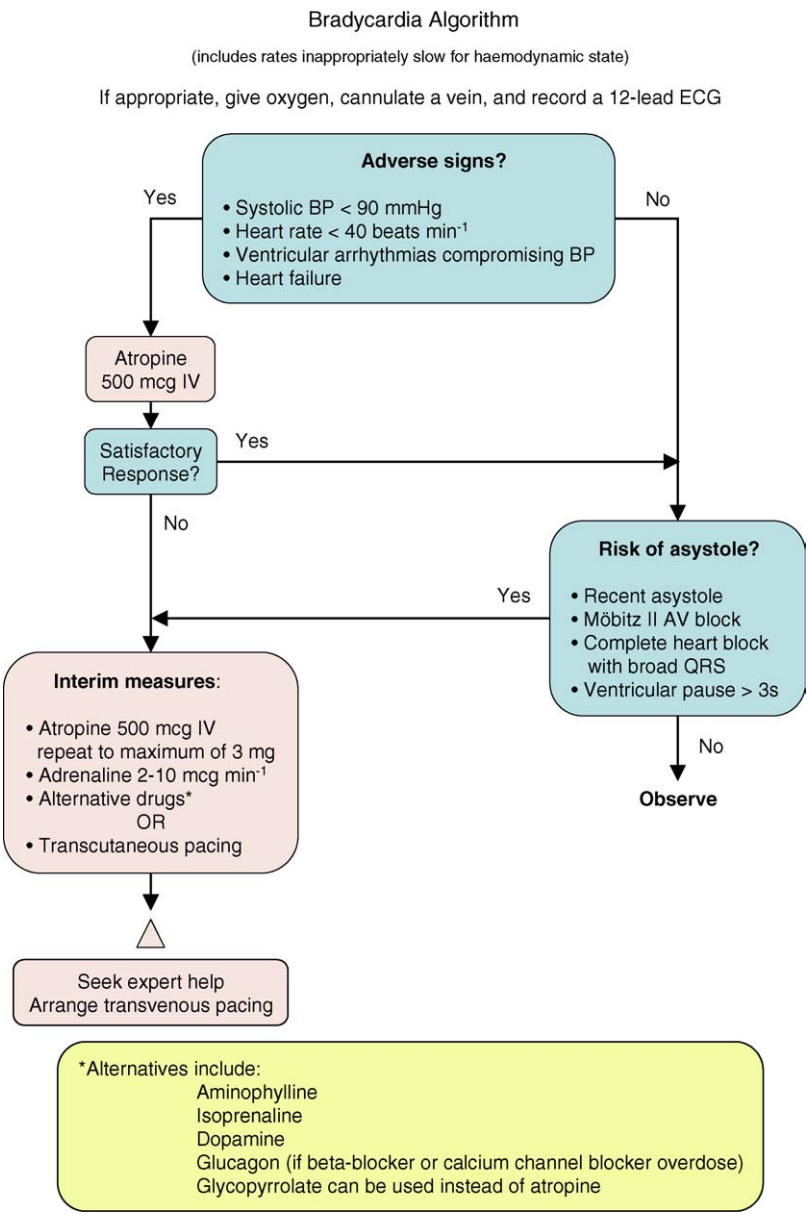

Figure 4.11 Bradycardia algorithm.

- Möbitz type II AV block
- complete (third-degree) heart block (especially with broad QRS or initial heart rate <40 beats min^{-1})
- ventricular standstill of more than 3 s

Atrioventricular (AV) blocks are divided into first, second, and third degrees and may be associated with multiple medications or electrolyte disturbances, as well as structural problems caused by acute myocardial infarction and myocarditis. A first-degree AV block is defined by a prolonged P—R interval (>0.20 s), and is usually benign. Second-degree AV block is divided into Möbitz types I and II. In Möbitz type I, the block is at the AV node, is often transient and may be asymptomatic. In Möbitz type II, the block is most often below the AV node at the bundle of His or at the bundle branches, and is often symptomatic, with the potential to progress to complete AV block. Third-degree heart block is defined by AV dissociation which may be permanent or transient, depending on the underlying cause.

Pacing is likely to be required if there is a risk of asystole, or if the patient is unstable and has failed to respond satisfactorily to atropine. Under these circumstances, the definitive treatment is transvenous pacing. One or more of the following interventions can be used to improve the patient's condition while waiting for the appropriate personnel and facilities:

- transcutaneous pacing
- adrenaline infusion in the range of 2—10 mcg min^{-1} titrated to response

Other drugs that can be given for symptomatic bradycardia include dopamine, isoprenaline and theophylline. Consider giving intravenous glucagon if beta-blockers or calcium channel blockers are a potential cause of the bradycardia. Do not give atropine to patients with cardiac transplants—paradoxically, it can cause a high-degree AV block or even sinus arrest.[315]

Complete heart block with a narrow QRS is not an absolute indication for pacing, because AV junctional ectopic pacemakers (with a narrow QRS) may provide a reasonable and stable heart rate.

Pacing

Transcutaneous pacing. Initiate transcutaneous pacing immediately if there is no response to atropine, if atropine is unlikely to be effective or if the patient is severely symptomatic, particularly if there is high-degree block (Möbitz Type II second- or third-degree block). Transcutaneous pacing can be painful and may fail to produce effective mechanical capture. Verify mechanical capture and reassess the patient's condition. Use analgesia and sedation to control pain, and attempt to identify the cause of the bradyarrhythmia.

Fist pacing. If atropine is ineffective and transcutaneous pacing is not immediately available, fist pacing can be attempted while waiting for pacing equipment[316—318]: give serial rhythmic blows with the closed fist over the left lower edge of the sternum to pace the heart at a physiological rate of 50—70 beats min^{-1}.

Tachycardias

Previous ERC guidelines have included three separate tachycardia algorithms: broad-complex tachycardia, narrow-complex tachycardia and atrial fibrillation. In the peri-arrest setting, many treatment principles are common to all the tachycardias; for this reason, they have been combined into a single tachycardia algorithm (Figure 4.12).

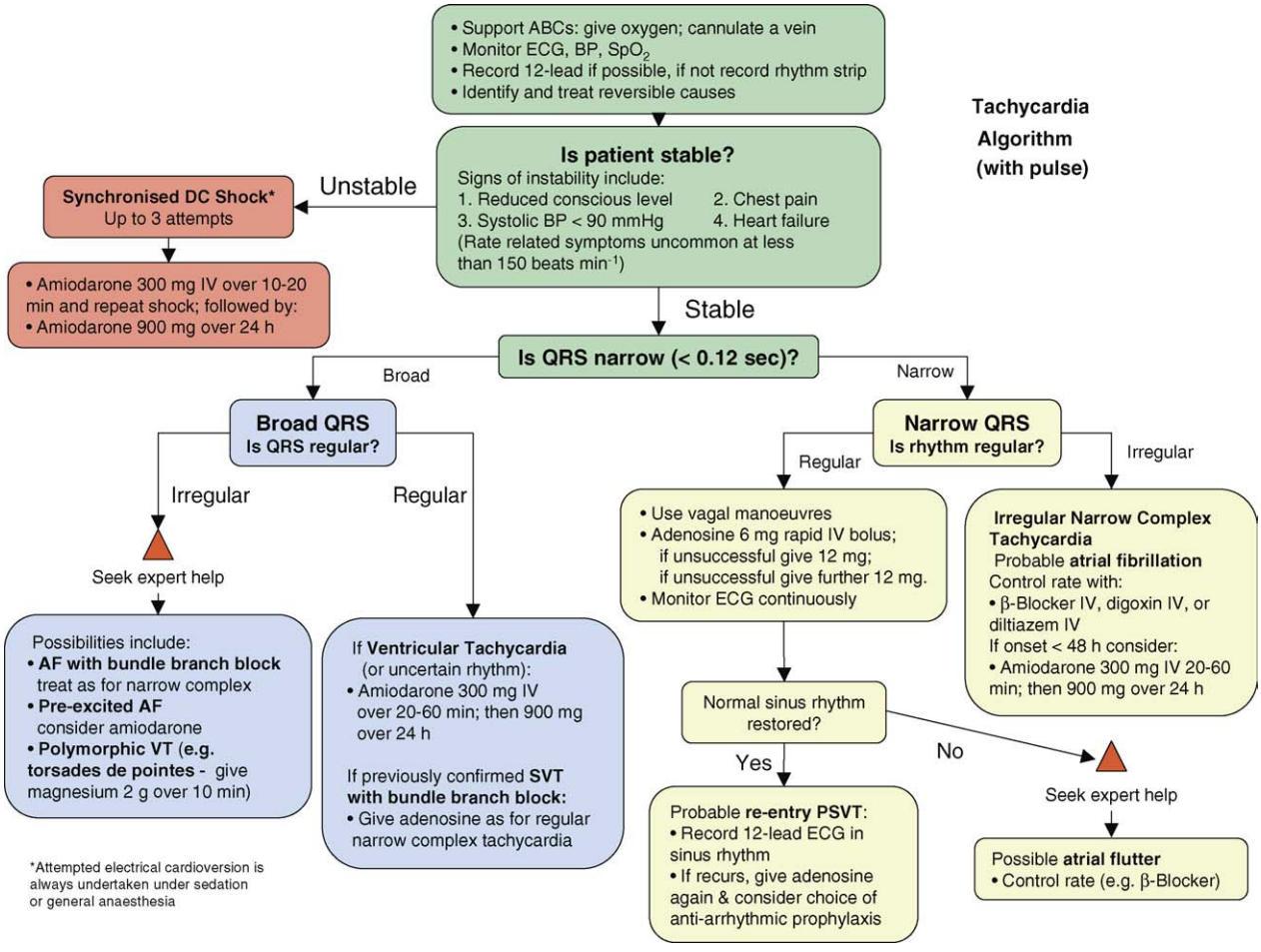

Figure 4.12 Tachycardia algorithm.

If the patient is unstable and deteriorating, with signs and symptoms caused by the tachycardia (e.g., impaired conscious level, chest pain, heart failure, hypotension or other signs of shock), attempt synchronised cardioversion immediately. In patients with otherwise normal hearts, serious signs and symptoms are uncommon if the ventricular rate is <150 beats min^{-1}. Patients with impaired cardiac function or significant comorbidity may be symptomatic and unstable at lower heart rates. If cardioversion fails to restore sinus rhythm and the patient remains unstable, give amiodarone 300 mg intravenously over 10—20 min and re-attempt electrical cardioversion. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 h. Serial DC shocks are not appropriate for recurrent (within hours or days) paroxysms (self-terminating episodes) of atrial fibrillation. This is relatively common in critically ill patients who may have ongoing precipitating factors causing the arrhythmia (e.g., metabolic disturbance, sepsis). Cardioversion does not prevent subsequent arrhythmias. If there are recurrent episodes, treat them with drugs.

Synchronised electrical cardioversion

If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronised with the R wave of the ECG rather than with the T wave. By avoiding the relative refractory period in this way, the risk of inducing ventricular fibrillation is minimised. Conscious patients must be anaesthetised or sedated before synchronised cardioversion is attempted. For a broad-complex tachycardia and AF, start with 200-J monophasic or 120—150 J biphasic and increase in increments if this fails (see Section 3). Atrial flutter and paroxysmal SVT will often convert with lower energies: start with 100-J monophasic or 70—120-J biphasic.

If the patient with tachycardia is stable (no serious signs or symptoms caused by the tachycardia) and is not deteriorating, there is time to evaluate the rhythm using the 12-lead ECG and determine treatment options. The ALS provider may not have the expertise to diagnose the tachycardia precisely, but should be capable of differentiating between sinus tachycardia, narrow-complex SVT and broad-complex tachycardia. If the patient is stable there is normally time to consult an expert. If the patient becomes unstable, proceed immediately to synchronised electrical cardioversion. Management of patients with significant comorbid conditions and symptomatic tachycardia requires treatment of the comorbid conditions.

Broad-complex tachycardia

In broad-complex tachycardias the QRS complexes are >0.12 s and are usually ventricular in origin. Although broad-complex tachycardias may be caused by supraventricular rhythms with aberrant conduction, in the unstable patient in the peri-arrest context assume they are ventricular in origin. In the stable patient with broad-complex tachycardia, the next step is to determine if the rhythm is regular or irregular.

Regular broad complex tachycardia. A regular broad-complex tachycardia is likely to be ventricular tachycardia or SVT with bundle branch block. Stable ventricular tachycardia can be treated with amiodarone 300 mg intravenously over 20—60 min followed by an infusion of 900 mg over 24 h. If the broad-complex regular tachycardia is thought to be SVT with bundle branch block, give adenosine, using the strategy indicated for narrow-complex tachycardia (below).

Irregular broad complex tachycardia. Irregular broad complex tachycardia is most likely to be AF with bundle branch block, but careful examination of a 12-lead ECG (if necessary by an expert) may enable confident identification of the rhythm. Another possible cause is AF with ventricular pre-excitation (in patients with Wolff—Parkinson—White (WPW) syndrome). There is more variation in the appearance and width of the QRS complexes than in AF with bundle branch block. A third possible cause is polymorphic VT (e.g., torsade de pointes), but polymorphic VT is relatively unlikely to be present without adverse features.

Seek expert help with the assessment and treatment of irregular broad-complex tachyarrhythmia. If treating AF with bundle branch block, treat as for AF (see below). If pre-excited AF (or atrial flutter) is suspected, avoid adenosine, digoxin, verapamil and diltiazem. These drugs block the AV node and cause a relative increase in pre-excitation. Electrical cardioversion is usually the safest treatment option.

Treat torsades de pointes VT immediately by stopping all drugs known to prolong QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate, 2 g, intravenously over 10 min.[319,320] Obtain expert help, as other treatment (e.g., overdrive pacing) may be indicated to prevent relapse once the arrhythmia has been corrected. If adverse features develop (which is usual), arrange immediate synchronised cardioversion. If the patient becomes pulseless, attempt defibrillation immediately (cardiac arrest algorithm).

Narrow-complex tachycardia

Regular narrow-complex tachycardias include:

- sinus tachycardia
- AV nodal re-entry tachycardia (AVNRT, the commonest type of SVT)
- AV re-entry tachycardia (AVRT (due to WPW syndrome))
- atrial flutter with regular AV conduction (usually 2:1)

Irregular narrow-complex tachycardia is most commonly AF or sometimes atrial flutter with variable AV conduction ('variable block').

Regular narrow-complex tachycardia

Sinus tachycardia. Sinus tachycardia is a common physiological response to a stimulus such as exercise or anxiety. In a sick patient it may be seen in response to many stimuli, such as pain, fever, anaemia, blood loss and heart failure. Treatment is almost always directed at the underlying cause; trying to slow sinus tachycardia that has occurred in response to most of these situations will make the situation worse.

AVNRT and AVRT (*paroxysmal SVT*). AVNRT is the commonest type of paroxysmal SVT, often seen in people without any other form of heart disease and is relatively uncommon in a peri-arrest setting. It causes a regular narrow-complex tachycardia, often with no clearly visible atrial activity on the ECG, with heart rates usually well above the typical range of sinus rates at rest (60—120 beats min^{-1}). It is usually benign, unless there is additional coincidental structural heart disease or coronary disease, but may cause symptoms that the patient finds frightening.

AV re-entry tachycardia (AVRT) is seen in patients with the WPW syndrome and is also usually benign unless there happens to be additional structural heart disease. The common type of AVRT is a regular narrow-complex tachycardia, also often having no visible atrial activity on the ECG.

Atrial flutter with regular AV conduction (*often 2:1 block*). Atrial flutter with regular AV conduction (often 2:1 block) produces a regular narrow-complex tachycardia in which it may be difficult to see atrial activity and identify flutter waves with confidence, so it may be indistinguishable initially from AVNRT and AVRT. When atrial flutter with 2:1 block or even 1:1 conduction is accompanied by bundle branch block, it produces a regular broad-complex tachycardia that will usually be very difficult to distinguish from VT; treatment of this rhythm as if it were VT will usually be effective, or will slow the ventricular response enabling identification of the rhythm. Most typical atrial flutter has an atrial rate of about 300 beats min^{-1}, so atrial flutter with 2:1 block tends to produce a tachycardia of about 150 beats min^{-1}. Much faster rates (170 beats min^{-1} or more) are unlikely to be due to atrial flutter with 2:1 block.

Treatment of regular narrow complex tachycardia. If the patient is unstable with adverse features caused by the arrhythmia, attempt synchronised electrical cardioversion. It is reasonable to give adenosine to an unstable patient with a regular narrow-complex tachycardia while preparations are made for synchronised cardioversion; however, do not delay electrical cardioversion if the adenosine fails to restore sinus rhythm. In the absence of adverse features, proceed as follows.

- Start with vagal manoeuvres. Carotid sinus massage or the Valsalva manoeuvre will terminate up to a quarter of episodes of paroxysmal SVT. A Valsalva manoeuvre (forced expiration against a closed glottis) in the supine position may be the most effective technique. A practical way of achieving this without protracted explanation is to ask the patient to blow into a 20-ml syringe with enough force to push back the plunger. Avoid carotid massage if a carotid bruit is present; rupture of an atheromatous plaque could cause cerebral embolism and stroke. In the context of acute ischaemia or digitalis toxicity, sudden bradycardia may trigger VF. Record an ECG (preferably multi-lead) during each manoeuvre. If the rhythm is atrial flutter, slowing of the ventricular response will often occur and demonstrate flutter waves.
- If the arrhythmia persists and is not atrial flutter, use adenosine. Give 6 mg as a rapid intravenous bolus. Record an ECG (preferably multi-lead) during each injection. If the ventricular rate slows transiently but the arrhythmia then persists, look for atrial activity such as atrial flutter or other atrial tachycardia and treat accordingly. If there is no response to adenosine 6 mg, give a 12-mg bolus; if there is no response, give one further 12 mg-bolus.
- Successful termination of a tachyarrhythmia by vagal manoeuvres or adenosine indicates that it was almost certainly AVNRT or AVRT. Monitor the patients for further rhythm abnormalities. Treat recurrence either with further adenosine or with a longer-acting drug with AV nodal-blocking action (e.g., diltiazem or beta-blocker).
- Vagal manoeuvres or adenosine will terminate almost all AVNRT or AVRT within seconds. Failure to terminate a regular narrow-complex tachycar-

dia with adenosine suggests an atrial tachycardia such as atrial flutter.
- If adenosine is contraindicated or fails to terminate a regular narrow-complex tachycardia without demonstrating that it is atrial flutter, give a calcium channel blocker (e.g., verapamil 2.5—5 mg intravenously over 2 min).

Irregular narrow-complex tachycardia

An irregular narrow-complex tachycardia is most likely to be AF with an uncontrolled ventricular response or, less commonly, atrial flutter with variable AV block. Record a 12-lead ECG to identify the rhythm. If the patient is unstable with adverse features caused by the arrhythmia, attempt synchronised electrical cardioversion.

If there are no adverse features, treatment options include:

- rate control by drug therapy
- rhythm control using drugs to encourage chemical cardioversion
- rhythm control by electrical cardioversion
- treatment to prevent complications (e.g., anticoagulation)

Obtain expert help to determine the most appropriate treatment for the individual patient. The longer a patient remains in AF, the greater is the likelihood of atrial clot developing. In general, patients who have been in AF for more than 48 h should not be treated by cardioversion (electrical or chemical) until they have received full anticoagulation or absence of atrial clot has been shown by transoesophageal echocardiography. If the aim is to control heart rate, options include a beta-blocker,[321,322] digoxin, diltiazem,[323,324] magnesium[325,326] or combinations of these.

If the duration of AF is less than 48 h and rhythm control is considered appropriate, this may be attempted using amiodarone (300 mg intravenously over 20—60 min followed by 900 mg over 24 h). Ibutilide or flecainide can also be given for rhythm control, but expert advice should be obtained before using these drugs for this purpose. Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

Seek expert help if any patient with AF is known or found to have ventricular pre-excitation (WPW syndrome). Avoid using adenosine, diltiazem, verapamil or digoxin to patients with pre-excited AF or atrial flutter, as these drugs block the AV node and cause a relative increase in pre-excitation.

Antiarrhythmic drugs

Adenosine

Adenosine is a naturally occurring purine nucleotide. It slows transmission across the AV node but has little effect on other myocardial cells or conduction pathways. It is highly effective for terminating paroxysmal SVT with re-entrant circuits that include the AV node (AVNRT). In other narrow-complex tachycardias, adenosine will reveal the underlying atrial rhythms by slowing the ventricular response. It has an extremely short half-life of 10—15 s and, therefore, is given as a rapid bolus into a fast running intravenous infusion or followed by a saline flush. The smallest dose likely to be effective is 6 mg (which is outside some current licences for an initial dose) and, if unsuccessful this can be followed with up to two doses each of 12 mg every 1—2 min. Patients should be warned of transient unpleasant side effects, in particular nausea, flushing, and chest discomfort.[327] Adenosine is not available in some European countries, but adenosine triphosphate (ATP) is an alternative. In a few European countries neither preparation may be available; verapamil is probably the next best choice. Theophylline and related compounds block the effect of adenosine. Patients receiving dipyridamole or carbamazepine, or with denervated (transplanted) hearts, display a markedly exaggerated effect that may be hazardous. In these patients, or if injected into a central vein, reduce the initial dose of adenosine to 3 mg. In the presence of WPW syndrome, blockage of conduction across the AV node by adenosine may promote conduction across an accessory pathway. In the presence of supraventricular arrhythmias this may cause a dangerously rapid ventricular response. In the presence of WPW syndrome, rarely, adenosine may precipitate atrial fibrillation associated with a dangerously rapid ventricular response.

Amiodarone

Intravenous amiodarone has effects on sodium, potassium and calcium channels as well as alpha- and beta-adrenergic blocking properties. Indications for intravenous amiodarone include:

- control of haemodynamically stable VT, polymorphic VT and wide-complex tachycardia of uncertain origin
- paroxysmal SVT uncontrolled by adenosine, vagal manoeuvres or AV nodal blockade
- to control rapid ventricular rate due to accessory pathway conduction in pre-excited atrial arrhythmias

Give amiodarone, 300 mg intravenously, over 10—60 min depending on the circumstances and haemodynamic stability of the patient. This loading dose is followed by an infusion of 900 mg over 24 h. Additional infusions of 150 mg can be repeated as necessary for recurrent or resistant arrhythmias to a maximum manufacturer-recommended total daily dose of 2 g (this maximum licensed dose varies between countries). In patients known to have severely impaired heart function, intravenous amiodarone is preferable to other anti-arrhythmic drugs for atrial and ventricular arrhythmias. Major adverse effects from amiodarone are hypotension and bradycardia, which can be prevented by slowing the rate of drug infusion. The hypotension associated with amiodarone is caused by vasoactive solvents (Polysorbate 80 and benzyl alcohol). A new aqueous formulation of amiodarone does not contain these solvents and causes no more hypotension than lidocaine.[198] Whenever possible, intravenous amiodarone should be given via a central venous catheter; it causes thrombophlebitis when infused into a peripheral vein. In an emergency it should be injected into a large peripheral vein.

Calcium channel blockers: verapamil and diltiazem

Verapamil and diltiazem are calcium channel blocking drugs that slow conduction and increase refractoriness in the AV node. Intravenous diltiazem is not available in some countries. These actions may terminate re-entrant arrhythmias and control ventricular response rate in patients with a variety of atrial tachycardias. Indications include:

- stable regular narrow-complex tachycardias uncontrolled or unconverted by adenosine or vagal manoeuvres
- to control ventricular rate in patients with AF or atrial flutter and preserved ventricular function when the duration of the arrhythmia is less than 48 h

The initial dose of verapamil is 2.5—5 mg intravenously given over 2 min. In the absence of a therapeutic response or drug-induced adverse event, give repeated doses of 5—10 mg every 15—30 min to a maximum of 20 mg. Verapamil should be given only to patients with narrow-complex paroxysmal SVT or arrhythmias known with certainty to be of supraventricular origin.

Diltiazem at a dose of 250 mcg kg^{-1}, followed by a second dose of 350 mcg kg^{-1}, is as effective as verapamil. Verapamil and, to a lesser extent, diltiazem may decrease myocardial contractility and critically reduce cardiac output in patients with severe LV dysfunction. For the reasons stated under adenosine (above), calcium channel blockers are considered harmful when given to patients with AF or atrial flutter associated with known pre-excitation (WPW) syndrome.

Beta-adrenergic blockers

Beta-blocking drugs (atenolol, metoprolol, labetalol (alpha- and beta-blocking effects), propranolol, esmolol) reduce the effects of circulating catecholamines and decrease heart rate and blood pressure. They also have cardioprotective effects for patients with acute coronary syndromes. Beta-blockers are indicated for the following tachycardias:

- narrow-complex regular tachycardias uncontrolled by vagal manoeuvres and adenosine in the patient with preserved ventricular function
- to control rate in AF and atrial flutter when ventricular function is preserved

The intravenous dose of atenolol ($beta_1$) is 5 mg given over 5 min, repeated if necessary after 10 min. Metoprolol ($beta_1$) is given in doses of 2—5 mg at 5-min intervals to a total of 15 mg. Propranolol ($beta_1$ and $beta_2$ effects), 100 mcg kg^{-1}, is given slowly in three equal doses at 2—3-min intervals.

Intravenous esmolol is a short-acting (half-life of 2—9 min) $beta_1$-selective beta-blocker. It is given as an intravenous loading dose of 500 mcg kg^{-1} over 1 min, followed by an infusion of 50—200 mcg kg^{-1} min^{-1}.

Side effects of beta-blockade include bradycardias, AV conduction delays and hypotension. Contraindications to the use of beta-adrenergic blocking agents include second- or third-degree heart block, hypotension, severe congestive heart failure and lung disease associated with bronchospasm.

Magnesium

Magnesium can be given for control of ventricular rate in atrial fibrillation.[326,328—330] Give magnesium sulphate 2 g (8 mmol) over 10 min. This can be repeated once if necessary.

4g. Post-resuscitation care

Introduction

ROSC is the just the first step toward the goal of complete recovery from cardiac arrest. Interventions in the post-resuscitation period are likely

to influence the final outcome significantly,[237,331] yet there are relatively few data relating to this phase. Of 22,105 patients admitted to intensive care units in the UK after cardiac arrest, 9974 (45%) survived to leave intensive care and 6353 (30%) survived to hospital discharge (data from Intensive Care National Audit and Research Centre (ICNARC), London, December 1995 to October 2004). To return the patient to a state of normal cerebral function with no neurological deficit, a stable cardiac rhythm and normal haemodynamic function, further resuscitation tailored to each patient's individual needs is required. The post-resuscitation phase starts at the location where ROSC is achieved but, once stabilised, the patient is transferred to the most appropriate high-care area (e.g., intensive care unit, coronary care unit) for continued monitoring and treatment.

Airway and breathing

Patients who have had a brief period of cardiac arrest responding immediately to appropriate treatment may achieve an immediate return of normal cerebral function. These patients do not require tracheal intubation and ventilation but should be given oxygen via a facemask. Hypoxia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. Ensure the tracheal tube is positioned correctly well above the carina. Hypocarbia causes cerebral vasoconstriction and a decreased cerebral blood flow.[332] After cardiac arrest, hypocapnia induced by hyperventilation causes cerebral ischaemia.[333–336] There are no data to support the targeting of a specific arterial PCO_2 after resuscitation from cardiac arrest, but it is reasonable to adjust ventilation to achieve normocarbia and to monitor this using the end-tidal PCO_2 and arterial blood gas values. Adjust the inspired oxygen concentrations to achieve adequate arterial oxygen saturation.

Insert a gastric tube to decompress the stomach; gastric distension caused by mouth-to-mouth or bag-mask-valve ventilation will splint the diaphragm and impair ventilation. Avoid coughing; this will increase intracranial pressure and may cause transient hypoxaemia. Give adequate doses of sedative and, if absolutely necessary, give a neuromuscular blocking drug. Obtain a chest radiograph to check the position of the tracheal tube and central venous lines, etc., assess for pulmonary oedema and to detect complications from CPR such as a pneumothorax associated with rib fractures.

Circulation

If there is evidence of coronary occlusion, consider the need for immediate revascularisation by thrombolysis or percutaneous coronary intervention (see acute coronary syndromes).

Haemodynamic instability is common after cardiac arrest and manifests as hypotension, low cardiac index and arrhythmias.[337] This post-resuscitation myocardial dysfunction (or myocardial stunning) is usually transient and often reverses within 24–48 h.[338] The post-resuscitation period is associated with marked elevations in plasma cytokine concentrations, manifesting as a sepsis-like syndrome and multiple organ dysfunction.[339]

Infusion of fluids may be required to increase right heart filling pressures or, conversely, diuretics and vasodilators may be needed to treat left ventricular failure. In the ICU an arterial line for continuous blood pressure monitoring is essential, and the use of a non-invasive or invasive (pulmonary artery catheter) cardiac output monitor may be helpful. There are very few randomised trials evaluating the role of blood pressure on the outcome after cardiac arrest. One randomised study demonstrated no difference in the neurological outcome among patients randomised to a mean arterial blood pressure of >100 mmHg versus $\leq$100 mmHg 5 min after ROSC; however, good functional recovery was associated with a higher blood pressure during the first 2 h after ROSC.[340] In the absence of definitive data, target the mean arterial blood pressure to achieve an adequate urine output, taking into consideration the patient's normal blood pressure.

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

Disability (optimising neurological recovery)

Cerebral perfusion

Immediately after ROSC there is a period of cerebral hyperaemia.[341] After 15–30 min of reperfusion, however, global cerebral blood flow decreases and there is generalised hypoperfusion. Normal cerebral autoregulation is lost, leaving cerebral perfusion dependent on mean arterial pressure. Under these circumstances, hypotension will compromise cerebral blood flow severely and will compound any neurological injury. Thus, after ROSC,

maintain mean arterial pressure at the patient's normal level.

Sedation

Although it has been common practice to sedate and ventilate patients for up to 24 h after ROSC, there are no data to support a defined period of ventilation, sedation and neuromuscular blockade after cardiac arrest. The duration of sedation and ventilation may be influenced by the use of therapeutic hypothermia (see below). There are no data to indicate whether or not the choice of sedation influences outcome, but short-acting drugs (e.g., propofol, alfentanil, remifentanil) will enable earlier neurological assessment. There is an increased incidence of pneumonia when sedation is prolonged beyond 48 h after prehospital or in-hospital cardiac arrest.[342]

Control of seizures

Seizures and/or myoclonus occur in 5—15% of adult patients who achieve ROSC, and in approximately 40% of those who remain comatose.[343] Seizures increase cerebral metabolism by up to four-fold. Prolonged seizure activity may cause cerebral injury, and should be controlled with benzodiazepines, phenytoin, propofol or a barbiturate. Each of these drugs can cause hypotension, and this must be treated appropriately. Seizures and myoclonus per se are not related significantly to outcome, but status epilepticus and, in particular, status myoclonus are associated with a poor outcome.[343,344]

Temperature control

Treatment of hyperpyrexia. A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest.[345—347] The risk of a poor neurological outcome increases for each degree of body temperature >37 °C.[348] Antipyretics and/or physical cooling methods decrease infarct volumes in animal models of global ischaemia.[349,350] Treat any hyperthermia occurring in the first 72 h after cardiac arrest with antipyretics or active cooling.

Therapeutic hypothermia. Mild therapeutic hypothermia is thought to suppress many of the chemical reactions associated with reperfusion injury. These reactions include free-radical production, excitatory amino acid release, and calcium shifts, which can in turn lead to mitochondrial damage and apoptosis (programmed cell death).[351—353] Two randomised clinical trials showed improved outcome in adults remaining comatose after initial resuscitation from out-of-hospital VF cardiac arrest, who were cooled within minutes to hours after ROSC.[354,355] The subjects were cooled to 32—34 °C for 12—24 h. One study documented improved metabolic endpoints (lactate and O_2 extraction) when comatose adult patients were cooled after ROSC from out-of-hospital cardiac arrest in which the initial rhythm was PEA/asystole.[356] A small study showed benefit after therapeutic hypothermia in comatose survivors of non-VF arrest.[357]

External and/or internal cooling techniques can be used to initiate cooling.[354—356,358—361] An infusion of 30 mg kg^{-1} of 4 °C-saline decreases core temperature by 1.5 °C.[358,359,361,362] Intravascular cooling enables more precise control of core temperature than external methods, but it is unknown whether this improves outcome.[360,363—365]

Complications of mild therapeutic hypothermia include increased infection, cardiovascular instability, coagulopathy, hyperglycaemia and electrolyte abnormalities such as hypophosphataemia and hypomagnesaemia.[366,367]

Unconscious adult patients with spontaneous circulation after out-of-hospital VF cardiac arrest should be cooled to 32—34 °C. Cooling should be started as soon as possible and continued for at least 12—24 h.[368—374] Induced hypothermia might also benefit unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest from a non-shockable rhythm, or cardiac arrest in hospital. Treat shivering by ensuring adequate sedation and giving neuromuscular blocking drugs. Bolus doses of neuromuscular blockers are usually adequate, but infusions are necessary occasionally. Rewarm the patient slowly (0.25—0.5 °C h^{-1}) and avoid hyperthermia. The optimum target temperature, rate of cooling, duration of hypothermia and rate of rewarming have yet to be determined; further studies are essential.

Blood glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome.[237—244] Persistent hyperglycaemia after stroke is also associated with a worse neurological outcome.[375—378] Tight control of blood glucose (4.4—6.1 mmol l^{-1} or 80—110 mg dl^{-1}) using insulin reduces hospital mortality in critically ill adults,[379,380] but this has not been demonstrated in post-cardiac arrest patients specifically. The benefit is thought to result from the strict glycaemic control rather than the dose of insulin infused.[381] One rat study has shown

that glucose plus insulin improves cerebral outcome after asphyxial cardiac arrest.[382] There are no randomised controlled human trials of glucose control after cardiac arrest. The optimal blood glucose target in critically ill patients has not been determined. Comatose patients are at particular risk from unrecognised hypoglycaemia, and the risk of this complication occurring increases as the target blood glucose concentration is lowered.

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

Prognostication

Once a heart has been resuscitated to a stable rhythm and cardiac output, the organ that influences an individual's survival most significantly is the brain. Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury.[383] A quarter of those dying after admission to ICU following in-hospital cardiac arrest die from neurological injury. A means of predicting neurological outcome that can be applied to individual patients immediately after ROSC is required. Such a test of prognosis must have 100% specificity.

Clinical tests

There are no neurological signs that can predict outcome in the first hours after ROSC. By 3 days after the onset of coma relating to cardiac arrest, 50% of patients with no chance of ultimate recovery have died. In the remaining patients, the absence of pupil light reflexes on day 3 and an absent motor response to pain on day 3 are both independently predictive of a poor outcome (death or vegetative state) with very high specificity.[384–386]

Biochemical tests

Measurement of serum neuron-specific enolase (NSE) and protein S-100b may be useful in determining the outcome of a cardiac arrest.[237,243,244,387–399] However, the 95% confidence interval (CI) in the trials undertaken to date is wide, and in many of the studies return to consciousness (without comment on level of function) was considered a ''good'' outcome. The only meta-analysis to look at this topic estimated that to obtain 95% CI with 5% false-positive rate would require a study population of approximately 600 patients.[400] No study this large has been conducted, and these biochemical tests remain unreliable for predicting outcome in individual cases.

Electrophysiological tests

Median nerve somatosensory evoked potentials in normothermic patients, comatose for at least 72 h after cardiac arrest, predict poor outcome with 100% specificity.[384] Bilateral absence of the N20 component of the evoked potentials in comatose patients with coma of hypoxic-anoxic origin is uniformly fatal. When recorded at least 24–48 h after ROSC, the electroencephalogram (EEG), provides limited prognostic information.[401–413] A normal or grossly abnormal EEG predicts outcome reliably, but an EEG between these extremes is unreliable for prognostication.

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