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European Resuscitation Council Guidelines for Resuscitation 2010 Section 4. Adult advanced life support

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Summary of changes since 2005 Guidelines

The most important changes in the 2010 European Resuscitation Council Advanced Life Support (ALS) Guidelines include:

- Increased emphasis on the importance of minimally interrupted high-quality chest compressions throughout any ALS intervention: chest compressions are paused briefly only to enable specific interventions.
- Increased emphasis on the use of 'track and trigger systems' to detect the deteriorating patient and enable treatment to prevent in-hospital cardiac arrest.
- Increased awareness of the warning signs associated with the potential risk of sudden cardiac death out of hospital.
- Removal of the recommendation for a pre-specified period of cardiopulmonary resuscitation (CPR) before out-of-hospital defibrillation following cardiac arrest unwitnessed by the emergency medical services (EMS).
- Continuation of chest compressions while a defibrillator is charged—this will minimise the preshock pause.
- The role of the precordial thump is de-emphasised.
- The use of up to three quick successive (stacked) shocks for ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) occurring in the cardiac catheterisation laboratory or in the immediate post-operative period following cardiac surgery.
- Delivery of drugs via a tracheal tube is no longer recommended—if intravenous access cannot be achieved, drugs should be given by the intraosseous route.
- When treating VF/VT cardiac arrest, adrenaline 1 mg is given after the third shock once chest compressions have restarted and then every 3–5 min (during alternate cycles of CPR). Amiodarone 300 mg is also given after the third shock.
- Atropine is no longer recommended for routine use in asystole or pulseless electrical activity.
- Reduced emphasis on early tracheal intubation unless achieved by highly skilled individuals with minimal interruption to chest compressions.
- Increased emphasis on the use of capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of return of spontaneous circulation (ROSC).
- The potential role of ultrasound imaging during ALS is recognised.
- Recognition of the potential harm caused by hyperoxaemia after ROSC is achieved: once ROSC has been established and the oxygen saturation of arterial blood (SaO_2) can be monitored reliably (by pulse oximetry and/or arterial blood gas analysis), inspired oxygen is titrated to achieve a SaO_2 of 94–98%.
- Much greater detail and emphasis on the treatment of the post-cardiac arrest syndrome.
- Recognition that implementation of a comprehensive, structured post-resuscitation treatment protocol may improve survival in cardiac arrest victims after ROSC.
- Increased emphasis on the use of primary percutaneous coronary intervention in appropriate, but comatose, patients with sustained ROSC after cardiac arrest.
- Revision of the recommendation for glucose control: in adults with sustained ROSC after cardiac arrest, blood glucose values >10 mmol l^{-1} (>180 mg dl^{-1}) should be treated but hypoglycaemia must be avoided.
- Use of therapeutic hypothermia to include comatose survivors of cardiac arrest associated initially with non-shockable rhythms as well shockable rhythms. The lower level of evidence for use after cardiac arrest from non-shockable rhythms is acknowledged.
- Recognition that many of the accepted predictors of poor outcome in comatose survivors of cardiac arrest are unreliable,

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especially if the patient has been treated with therapeutic hypothermia.

4a Prevention of in-hospital cardiac arrest

Early recognition of the deteriorating patient and prevention of cardiac arrest is the first link in the chain of survival.[1] Once cardiac arrest occurs, fewer than 20% of patients suffering an in-hospital cardiac arrest will survive to go home.[2–4] Prevention of in-hospital cardiac arrest requires staff education, monitoring of patients, recognition of patient deterioration, a system to call for help and an effective response.[5]

The problem

Cardiac arrest in patients in unmonitored ward areas is not usually a sudden unpredictable event, nor is it usually caused by primary cardiac disease.[6] These patients often have slow and progressive physiological deterioration, involving hypoxaemia and hypotension that is unnoticed by staff, or is recognised but treated poorly.[7–9] Many of these patients have unmonitored arrests, and the underlying cardiac arrest rhythm is usually non-shockable.[3,10] Survival to hospital discharge is poor.[2,4,10]

The records of patients who have a cardiac arrest or unanticipated intensive care unit (ICU) admission often contain evidence of unrecognised, or untreated, respiratory and circulation problems.[6,8,11–16] The ACADEMIA study showed antecedents in 79% of cardiac arrests, 55% of deaths and 54% of unanticipated ICU admissions.[8] Early and effective treatment of seriously ill patients might prevent some cardiac arrests, deaths and unanticipated ICU admissions. Several studies show that up to a third of patients who have a false cardiac arrest call subsequently die.[17–19]

Nature of the deficiencies in the recognition and response to patient deterioration

These often include: infrequent, late or incomplete vital signs assessments; lack of knowledge of normal vital signs values; poor design of vital signs charts; poor sensitivity and specificity of 'track and trigger' systems; failure of staff to increase monitoring or escalate care, and staff workload.[20–28] There is also often a failure to treat abnormalities of the patient's airway, breathing and circulation, incorrect use of oxygen therapy, poor communication, lack of teamwork and insufficient use of treatment limitation plans.[7,14,29]

Education in acute care

Several studies show that medical and nursing staff lack knowledge and skills in acute care,[30] e.g., oxygen therapy,[31] fluid and electrolyte balance,[32] analgesia,[33] issues of consent,[34] pulse oximetry[35,36] and drug doses.[37] Medical school training provides poor preparation for doctors' early careers, and fails to teach them the essential aspects of applied physiology and acute care.[38] There is a need for an increased emphasis on acute care training of undergraduate and newly qualified doctors.[39,40] There is also little to suggest that the acute care training and knowledge of senior medical staff is better.[41,42] Staff often lack confidence when dealing with acute care problems, and rarely use a systematic approach to the assessment of critically ill patients.[43]

Staff education is an essential part of implementing a system to prevent cardiac arrest.[44] However, there are no randomised controlled studies addressing the impact of specific educational interventions on improvements in patient outcomes such as the earlier recognition or rescue of the deteriorating patient at risk of cardiac or respiratory arrest.

In an Australian study, virtually all the improvement in the hospital cardiac arrest rate occurred during the educational phase of implementation of a medical emergency team (MET) system.[45,46] In studies from Australian and American hospitals with established rapid response teams, education about the specific criteria for activating their teams led to proactive ICU admission of patients and a reduction in the number of ward cardiac arrests.[47–49] A UK study found that the number of cardiac arrest calls decreased while pre-arrest calls increased after implementing a standardised educational program in two hospitals; the intervention was associated with a decrease in true arrests, and increase in initial survival after cardiac arrest and survival to discharge.[50,51]

Monitoring and recognition of 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,[52] yet the measurement and recording of important physiological observations of sick patients occurs less frequently than is desirable.[6,8,13,16,24,53,54]

To assist in the early detection of critical illness, each patient should have a documented plan for vital signs monitoring that identifies which variables need to be measured and the frequency of measurement.[26] Many hospitals now use early warning scores (EWS) or calling criteria to identify the need to escalate monitoring, treatment, or to call for expert help.[13,24,55–57] The use of these systems has been shown to increase the frequency of patient vital signs measurements.[54,58,59]

These calling criteria or 'track-and-trigger' systems include single-parameter systems, multiple-parameter systems, aggregate weighted scoring systems or combination systems.[60] The aggregate weighted track-and-trigger systems offer a graded escalation of care, whereas single parameter track and trigger systems provide an all-or-nothing response.

Most of these systems lack robust data to suggest they have acceptable accuracy for use in the roles for which they are proposed. Low sensitivity of systems means that a significant number of patients at risk of deterioration leading to cardiac arrest are likely to be missed.[61,62] Hospitals should use a system validated for their specific patient population to identify individuals at increased risk of serious clinical deterioration, cardiac arrest, or death, both on admission and during hospital stay.

Alterations in physiological variables, singly or in combination are associated with, or can be used to predict the occurrence of cardiac arrest,[9,13,15,63,64] hospital death[22,23,65–82] and unplanned ICU admission,[15,80,83] with varying sensitivity and specificity. Differing criteria for ICU admission between hospitals make the use of unplanned ICU admission a less useful endpoint to study.

As one would expect, an increased number of derangements increases the likelihood of death.[11,15,20,63,77,84–91] The best combination and cut off values to allow early prediction is not known. For aggregate-weighted scoring systems, inclusion of heart rate (HR), respiratory rate (RR), systolic blood pressure (SBP), AVPU (alert, vocalizing, pain, unresponsive), temperature, age, and oxygen saturation achieve the best predictive value.[22,61] For single parameter track-and-trigger systems, cut-off points of HR <35 and >140 min^{-1}; RR <6 and >32 min^{-1}; and SBP < 80 mm Hg achieved the best positive predictive value.[23] Taking account of the patient's age improves the predictive value of both aggregate and single parameter scoring systems.[77] Aggregate-weighted scoring systems appear to have a rank order of performance that is relatively constant.[92] A newly devised, aggregate-weighted scoring system discriminates better than all others tested using mortality within 24 h of an early warning score as the outcome.[92]

The design of vital signs charts or the use of technology may have an important role in the detection of deterioration and requires further study.[21,93,94]

Calling for help

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.[95] However, the role of the cardiac arrest team has been questioned. In one small study, only patients who had return of spontaneous circulation before the cardiac arrest team arrived were discharged from hospital alive.[96] 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.

Nursing staff and junior doctors often find it difficult to ask for help or escalate treatment as they feel their clinical judgement may be criticised. Hospitals should ensure all staff are empowered to call for help and also trained to use structured communication tools such as RSVP (Reason-Story-Vital Signs-Plan)[97] or SBAR (Situation-Background-Assessment-Recommendation)[98] tools to ensure effective inter-professional communication.

The response to critical illness

The response to patients who are critically ill or who are at risk of becoming critically ill is usually provided by medical emergency teams (MET), rapid response teams (RRT), or critical care outreach teams (CCOT).[99–101] These teams replace or coexist with traditional cardiac arrest teams, which typically respond to patients already in cardiac arrest. MET/RRT usually comprise medical and nursing staff from intensive care and general medicine and respond to specific calling criteria. CCOT are common in the UK, based predominantly on individual or teams of nurses.[60] Outreach services exist in many forms, ranging from a single nurse to a 24-h, 7 days per week multiprofessional team. Any member of the healthcare team can initiate a MET/RRT/CCOT call. In some hospitals, the patient's family and friends are also encouraged to activate the team, if necessary.[102–104] Team interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids.[105–109] However, *post hoc* analysis of the MERIT study data suggests that all nearly all MET calls required 'critical care-type' interventions.[110] A circadian pattern of team 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.[111,112]

Studying the effect of the MET/RRT/CCOT systems on patient outcomes is difficult because of the complex nature of the intervention. During the period of most studies of rapid response teams, there has been a major international focus on improving other aspects of patient safety, e.g., hospital acquired infections, earlier treatment of sepsis and better medication management, all of which have the potential to influence patient deterioration and may have a beneficial impact on reducing cardiac arrests and hospital deaths. Additionally, a greater focus on improving 'end of life' care and the making of 'do not attempt resuscitation' (DNAR) decisions also impact cardiac arrest call rates. The available studies do not correct for these confounding factors.

Nevertheless, numerous single centre studies have reported reduced numbers of cardiac arrests after the implementation of RRT/MET systems.[45,47,107,111,113–125] However, a well-designed, cluster-randomised controlled trial of the MET system (MERIT study) involving 23 hospitals[24] did not show a reduction in cardiac arrest rate after introduction of a MET when analyzed on an intention-to-treat basis. This study was unable to demonstrate a difference between control and intervention hospitals in reduction in a composite outcome of (a) cardiac arrests without a pre-existing not-for-resuscitation (NFR) order, (b) unplanned ICU admissions, and (c) unexpected deaths (deaths without a pre-existing NFR order) taking place in general wards during the 6-month study MET period. Both the control and MET groups demonstrated improved outcome compared to baseline. *Post hoc* analysis of the MERIT study showed there was a decrease in cardiac arrest and unexpected mortality rate with increased activation of the MET system.[126] Several other studies have also been unable to show a reduction in cardiac arrest rates associated with the introduction of RRT/MET systems.[105,106,108,109,127–130] A single-centre study of the implementation of an early warning scoring system showed an increase in cardiac arrests among patients who had higher early warning scores, compared with similar scored patients before the intervention.[56]

A recent meta-analysis showed RRT/MET systems were associated with a reduction in rates of cardiopulmonary arrest outside the intensive care unit but are not associated with lower hospital mortality rates.[131]

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.[132] International organisations have offered definitions of levels of care and produced admission and discharge criteria for high dependency units (HDUs) and ICUs.[133,134]

Staffing levels

Hospital staffing tends to be at its lowest during the night and at weekends. This may influence patient monitoring, treatment and outcomes. Data from the US National Registry of CPR Investigators shows that survival rates from in-hospital cardiac arrest are lower during nights and weekends.[135] Admission to a general medical ward after 17.00 h[136] or to hospital at weekends[137] 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.[138,139] Several studies show that higher nurse staffing is associated with lower rates of failure-to-rescue, and reductions in rates of cardiac arrest rates, pneumonia, shock and death.[25,140,141]

Resuscitation decisions

The decision to start, continue and terminate resuscitation efforts is based on the balance between the risks, benefits and burdens these interventions place on patients, family members and healthcare providers. There are circumstances where resuscitation is inappropriate and should not be provided. Consider a 'do not attempt resuscitation' (DNAR) decision 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.[142] Even when there is clear evidence that cardiac arrest or death is likely, ward staff rarely make decisions about the patient's resuscitation status.[8] Many European countries have no formal policy for recording DNAR decisions and the practice of consulting patients about the decision is variable.[143,144] Improved knowledge, training and DNAR decision-making should improve

patient care and prevent futile CPR attempts (see Section 10).[145] Medical emergency teams may have an important role in improving end-of-life and DNAR decision-making.[142,146–148]

Guidelines for prevention of in-hospital cardiac arrest

Hospitals should provide a system of care that includes: (a) staff education about the signs of patient deterioration, and the rationale for rapid response to illness, (b) appropriate and regular vital signs monitoring of patients, (c) clear guidance (e.g., via calling criteria or early warning scores) to assist staff in the early detection of patient deterioration, (d) a clear, uniform system of calling for assistance, and (e) an appropriate and timely clinical response to calls for assistance.[5] 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: each patient should have a documented plan for vital signs monitoring that identifies which variables need to be measured and the frequency of measurement according to the severity of illness or the likelihood of clinical deterioration and cardiopulmonary arrest. Recent guidance suggests monitoring of simple physiological variables including pulse, blood pressure, respiratory rate, conscious level, temperature and arterial blood oxygen saturation by pulse oximetry (SpO_2).[26,149]
3. Use a track-and-trigger system (either 'calling criteria' or early warning 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 vital signs and, where used, early warning scores.
5. Have a clear and specific policy that requires a clinical response to abnormal physiology, based on the track and trigger system used. 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, RRT system) capable of responding in a timely fashion to acute clinical crises identified by the track-and-trigger system or other indicators. This service must be available 24 h per day. The team must include staff with the appropriate acute or critical care skills.
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. Ensure that staff know their role(s) in the rapid response system.
8. Hospitals must empower staff of all disciplines to call for help when they identify a patient at risk of deterioration or cardiac arrest. Staff should be trained in the use of structured communication tools to ensure effective handover of information between doctors, nurses and other healthcare professions.
9. 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.
10. 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.

Prevention of sudden cardiac death (SCD) out-of-hospital

Coronary artery disease is the commonest cause of SCD. Non-ischaemic cardiomyopathy and valvular disease account for most other SCD events. A small percentage of SCDs are caused by inherited abnormalities (e.g., Brugada syndrome, hypertrophic cardiomyopathy) or congenital heart disease.

Most SCD victims have a history of cardiac disease and warning signs, most commonly chest pain, in the hour before cardiac arrest.[150] In patients with a known diagnosis of cardiac disease, syncope (with or without prodrome—particularly recent or recurrent) is as an independent risk factor for increased risk of death.[151–161] Chest pain on exertion only, and palpitations associated with syncope only, are associated with hypertrophic cardiomyopathy, coronary abnormalities, Wolff–Parkinson–White, and arrhythmogenic right ventricular cardiomyopathy.

Apparently healthy children and young adults who suffer SCD can also have signs and symptoms (e.g., syncope/pre-syncope, chest pain and palpitations) that should alert healthcare professionals to seek expert help to prevent cardiac arrest.[162–170]

Children and young adults presenting with characteristic symptoms of arrhythmic syncope should have a specialist cardiology assessment, which should include an ECG and in most cases an echocardiogram and exercise test. Characteristics of arrhythmic syncope include: syncope in the supine position, occurring during or after exercise, with no or only brief prodromal symptoms, repetitive episodes, or in individuals with a family history of sudden death. In addition, non-pleuritic chest pain, palpitations associated with syncope, seizures (when resistant to treatment, occurring at night or precipitated by exercise, syncope, or loud noise), and drowning in a competent swimmer should raise suspicion of increased risk. Systematic evaluation in a clinic specializing in the care of those at risk for SCD is recommended in family members of young victims of SCD or those with a known cardiac disorder resulting in an increased risk of SCD.[151,171–175] A family history of syncope or SCD, palpitations as a symptom, supine syncope and syncope associated with exercise and emotional stress are more common in patients with long QT syndrome (LQTS).[176] In older adults[177,178] the absence of nausea and vomiting before syncope and ECG abnormalities is an independent predictor of arrhythmic syncope.

Inexplicable drowning and drowning in a strong swimmer may be due to LQTS or catecholaminergic polymorphic ventricular tachycardia (CPVT).[179] There is an association between LQTS and presentation with seizure phenotype.[180,181] Guidance has been published for the screening of competitive athletes to identify those at risk of sudden death.[182]

4b Prehospital resuscitation

EMS personnel

There is considerable variation across Europe in the structure and process of EMS systems. Some countries have adopted almost exclusively paramedic/emergency medical technician (EMT)-based systems while other incorporate prehospital physicians to a greater or lesser extent. In adult cardiac arrest, physician presence during resuscitation, compared with paramedics alone, has been reported to increase compliance with guidelines[183,184] and physicians in some systems can perform advanced resuscitation procedures more successfully.[183,185–188] When compared within individual systems, there are contradictory findings with some studies suggesting improved survival to hospital discharge when physicians are part of the resuscitation team[189–192] and other studies suggest-

ing no difference in short- or long-term survival.[183,189,191,193–199] in one study, survival of the event was lower when physicians were part of the resuscitation team.[199] Studies indirectly comparing resuscitation outcomes between physician-staffed and other systems are difficult to interpret because of the extremely high variability between systems, independent of physician-staffing.[200] Although some studies have documented higher survival rates after cardiac arrest in EMS systems that include experienced physicians,[186,188,201–203] compared with those that rely on non-physician providers,[201,202,204,205] other comparisons have found no difference in survival between systems using paramedics or physicians as part of the response.[206,207] Well-organised non-physician systems with highly trained paramedics have also reported high survival rates.[200] Given the inconsistent evidence, the inclusion or exclusion of physicians among prehospital personnel responding to cardiac arrests will depend largely on existing local policy.

Termination of resuscitation rules

One high-quality, prospective study has demonstrated that application of a 'basic life support termination of resuscitation rule' is predictive of death when applied by defibrillation-only emergency medical technicians.[208] The rule recommends termination when there is no return of spontaneous circulation, no shocks are administered, and the arrest is not witnessed by EMS personnel. Of 776 patients with cardiac arrest for whom the rule recommended termination, four survived [0.5% (95% CI 0.2–0.9)]. Implementation of the rule would reduce the transportation rate by almost two thirds. Four studies have shown external generalisability of this rule.[209–212]

Additional studies have shown associations with futility of certain variables such as no ROSC at scene; non-shockable rhythm; unwitnessed arrest; no bystander CPR, call response time and patient demographics.[213–218]

Two in-hospital studies and one emergency department study showed that the reliability of termination of resuscitation rules is limited in these settings.[219–221]

Prospectively validated termination of resuscitation rules such as the 'basic life support termination of resuscitation rule' can be used to guide termination of prehospital CPR in adults; however, these must be validated in an emergency medical services system similar to the one in which implementation is proposed. Other rules for various provider levels, including in-hospital providers, may be helpful to reduce variability in decision-making; however, rules should be prospectively validated prior to implementation.

CPR versus defibrillation first

There is evidence that performing chest compressions while retrieving and charging a defibrillator improves the probability of survival.[222] EMS personnel should provide good-quality CPR while a defibrillator is retrieved, applied and charged, but routine delivery of a pre-specified period of CPR (e.g., 2 or 3 min) before rhythm analysis and a shock is delivered is not recommended. Some emergency medical services have already fully implemented a pre-specified period of chest compressions before defibrillation; given the lack of convincing data either supporting or refuting this strategy, it is reasonable for them to continue this practice (see Section 3).[223]

4c 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 as rapidly as possible and certainly 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, RRT).

Location

Patients who have monitored arrests are usually diagnosed rapidly. Ward patients may have had a period of deterioration and an unwitnessed arrest.[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 only 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.[224,225]

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 or RRT) before cardiac arrest occurs. 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 Fig. 4.1.

- 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, RRT). 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.[226–235] 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.[236–239] Agonal breathing can also occur during chest compressions as cerebral perfusion improves, but is not indicative of a return of spontaneous 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 a finger sweep, 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. Efforts to protect the cervical spine must not jeopardise oxygenation and ventilation.

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 (consciousness, purposeful movement, normal breathing, or coughing), start CPR until more experience help arrives or the patient shows signs of life.

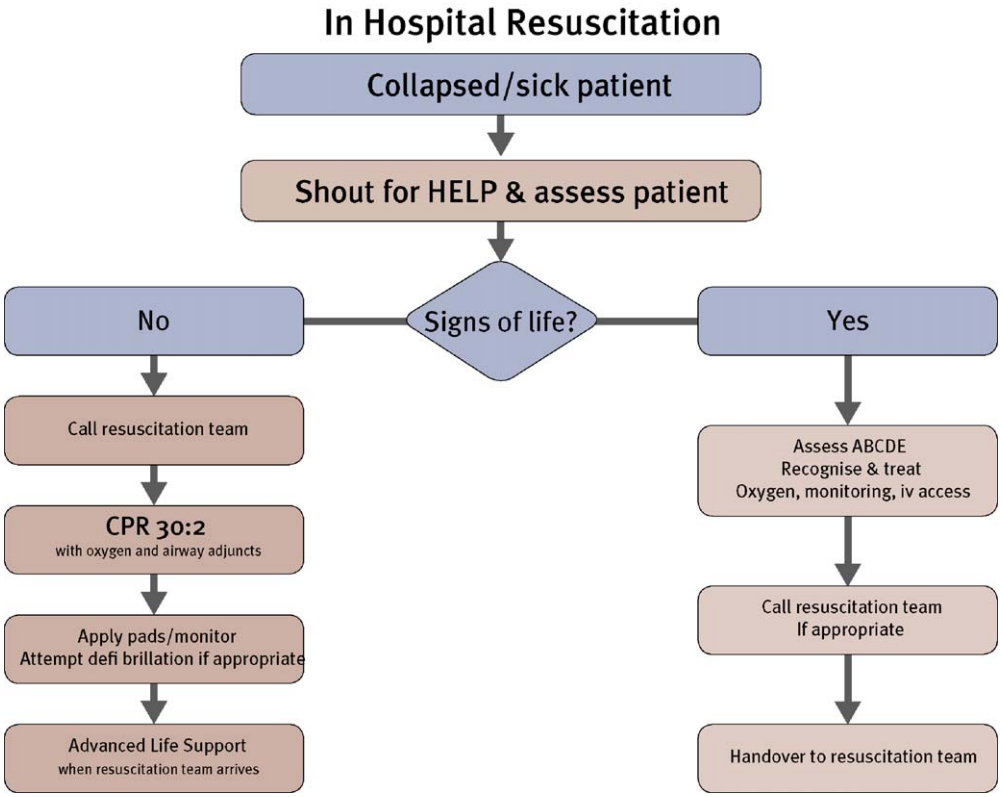

Fig. 4.1. Algorithm for the treatment of in-hospital cardiac arrest. © 2010 ERC.

- 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. Delivering chest compressions to a patient with a beating heart is unlikely to cause harm.[240] However, delays in diagnosis of cardiac arrest and starting CPR will adversely effect survival and 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. When a reliable measurement of oxygen saturation of arterial blood (e.g., pulse oximetry (SpO_2)) can be achieved, titrate the inspired oxygen concentration to achieve a SpO_2 of 94–98%.

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.
- Minimise interruptions and ensure high-quality compressions.
- Undertaking good-quality chest compressions for a prolonged time is tiring; with minimal interruption, 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 supraglottic airway device (SAD) 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. Waveform capnography should be routinely available for confirming tracheal tube placement (in the presence of a cardiac output) and subsequent monitoring of an intubated patient.
- 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 or a SAD has been inserted, continue chest compressions uninterrupted (except for defibrillation or pulse checks when indicated), at a rate of at least 100 min^{-1}, and ventilate the lungs at approximately 10 breaths min^{-1}. Avoid hyperventilation (both excessive rate and tidal volume), which may worsen outcome. Mechanical ventilators may free up a rescuer and ensure appropriate ventilation rates and volumes.
- If there is no airway and ventilation equipment available, consider giving 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. The use of adhesive electrode pads or a 'quick-look' paddles technique will enable rapid assessment of heart rhythm compared with attaching ECG electrodes.[241] Pause briefly to assess the heart rhythm. With a manual defibrillator, if the rhythm is VF/VT charge the defibrillator while another rescuer continues chest compressions. Once the defibrillator is charged, pause the chest compressions, ensure that all rescuers are clear of the patient and then give one shock. If using an automated external defibrillation (AED) follow the AED's audio-visual prompts.
- Restart chest compressions immediately after the defibrillation attempt. Minimise interruptions to chest compressions. Using a manual defibrillator it is possible to reduce the pause between stopping and restarting of chest compressions to less than 5 s.
- 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 4d).
- 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. Use a structured communication tool for handover (e.g., SBAR, RSVP).[97,98] Locate the patient's records.
- The quality of chest compressions during in-hospital CPR is frequently sub-optimal.[242,243] The importance of uninterrupted chest compressions cannot be over emphasised. Even short interruptions to chest compressions are disastrous for outcome and every effort must be made to ensure that continuous, effective chest compression is maintained throughout the resuscitation attempt. Chest compressions should commence at the beginning of a resuscitation attempt and continue uninterrupted unless they are briefly paused for a specific intervention (e.g., pulse check). The team leader should monitor the quality of CPR and alternate CPR providers if the quality of CPR is poor. Continuous $ETCO_2$ monitoring can be used to indicate the quality of CPR: although an optimal target for $ETCO_2$ during CPR has not been established, a value of less than 10 mm Hg (1.4 kPa) is associated with failure to achieve ROSC and may indicate that the quality of chest compressions should be improved. If possible, the person providing chest compressions should be alternated every 2 min, but without causing long pauses in chest compressions.

4d 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 and pulseless electrical activity (PEA)). The principal difference in the treatment of these two groups of arrhythmias is the need for attempted defibrillation in those patients with VF/VT. Subsequent actions, including high-quality chest compressions with minimal interruptions, 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 (Fig. 4.2) is applicable to all cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances (see Section 8).

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander basic life support (BLS), uninterrupted, high-quality chest compressions and early defibrillation for VF/VT. The use of adrenaline has been shown to increase return of spontaneous circulation (ROSC), but no resuscitation drugs or advanced airway interventions have been shown to increase survival to hospital discharge after cardiac arrest.[244–247] Thus, although drugs and advanced airways are still included among ALS interventions, they are of secondary importance to early defibrillation and high-quality, uninterrupted chest compressions.

As with previous guidelines, the ALS algorithm distinguishes between shockable and non-shockable rhythms. Each cycle is

broadly similar, with a total of 2 min of CPR being given before assessing the rhythm and where indicated, feeling for a pulse. Adrenaline 1 mg is given every 3–5 min until ROSC is achieved—the timing of the initial dose of adrenaline is described below. In VF/VT, a single dose of amiodarone is indicated after three unsuccessful shocks.

Shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia)

The first monitored rhythm is VF/VT in approximately 25% of cardiac arrests, both in-[4] or out-of-hospital.[248–250] VF/VT will also occur at some stage during resuscitation in about 25% of cardiac

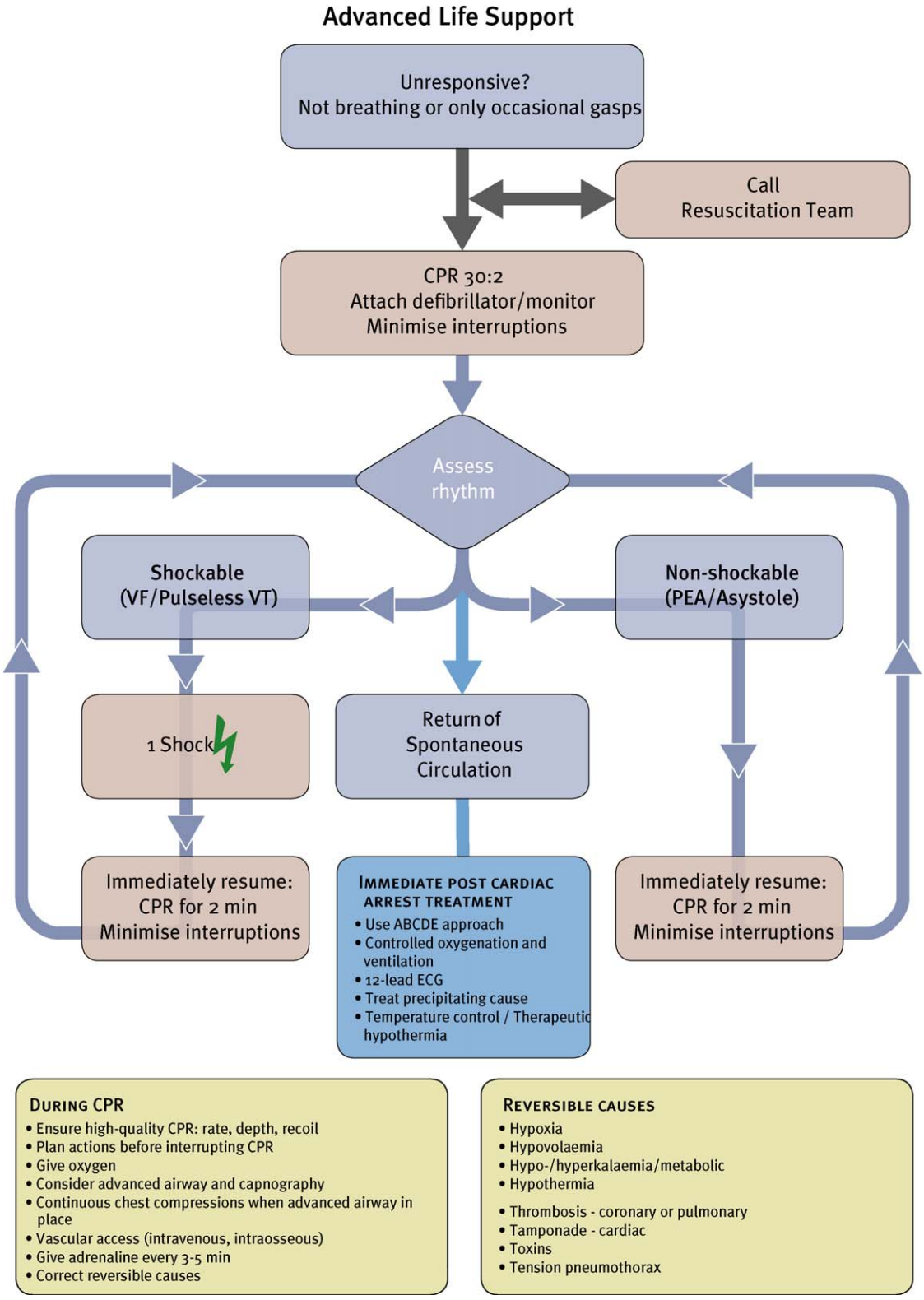

Fig. 4.2. Advanced life support cardiac arrest algorithm. © 2010 ERC.

arrests with an initial documented rhythm of asystole or PEA.[4] Having confirmed cardiac arrest, summon help (including the request for a defibrillator) and start CPR, beginning with chest compressions, with a compression:ventilation (CV) ratio of 30:2. When the defibrillator arrives, continue chest compressions while applying the paddles or self-adhesive pads. Identify the rhythm and treat according to the ALS algorithm.

- If VF/VT is confirmed, charge the defibrillator while another rescuer continues chest compressions. Once the defibrillator is charged, pause the chest compressions, quickly ensure that all rescuers are clear of the patient and then give one shock (360-J monophasic or 150–200 J biphasic).
- Minimise the delay between stopping chest compressions and delivery of the shock (the preshock pause); even 5–10 s delay will reduce the chances of the shock being successful.[251,252]
- 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 takes time until the post-shock circulation is established[253] and it is very rare for a pulse to be palpable immediately after defibrillation.[254] Furthermore, the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.[255]
- Continue CPR for 2 min, then pause briefly to assess the rhythm; if still VF/VT, give a second shock (360-J monophasic or 150–360-J biphasic). Without reassessing the rhythm or feeling for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions.
- Continue CPR for 2 min, then pause briefly to assess the rhythm; if still VF/VT, give a third shock (360-J monophasic or 150–360-J biphasic). Without reassessing the rhythm or feeling for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions. If IV/IO access has been obtained, give adrenaline 1 mg and amiodarone 300 mg once compressions have resumed. If ROSC has not been achieved with this 3rd shock the adrenaline will improve myocardial blood flow and may increase the chance of successful defibrillation with the next shock. In animal studies, peak plasma concentrations of adrenaline occur at about 90 s after a peripheral injection.[256] If ROSC has been achieved after the 3rd shock it is possible that the bolus dose of adrenaline will cause tachycardia and hypertension and precipitate recurrence of VF. However, naturally occurring adrenaline plasma concentrations are high immediately after ROSC,[257] and any additional harm caused by exogenous adrenaline has not been studied. Interrupting chest compressions to check for a perfusing rhythm midway in the cycle of compressions is also likely to be harmful. The use of waveform capnography may enable ROSC to be detected without pausing chect compressions and may be a way of avoiding a bolus injection of adenaline after ROSC has been achieved. Two prospective human studies have shown that a significant increase in end-tidal CO_2 occurs when return of spontaneous circulation occurs.[258,259]
- After each 2-min cycle of CPR, if the rhythm changes to asystole or PEA, see 'non-shockable rhythms' below. If a non-shockable rhythm is present and the rhythm is organised (complexes appear regular or narrow), try to palpate a pulse. Rhythm checks should be brief, and pulse checks should be undertaken only if an organised rhythm is observed. If there is any doubt about the presence of a pulse in the presence of an organised rhythm, resume CPR. If ROSC has been achieved, begin post-resuscitation care

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.[260] 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.[260,261] Reduction in the interval from compression to shock delivery by even a few seconds can increase the probability of shock success.[251,252]

Regardless of the arrest rhythm, give adrenaline 1 mg every 3–5 min until ROSC is achieved; in practice, this will be once every two cycles of the algorithm. If signs of life return during CPR (purposeful 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, while minimising the interruption in compressions.

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

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

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

This three-shock strategy may also be considered for an initial, witnessed VF/VT cardiac arrest if the patient is already connected to a manual defibrillator. Although there are no data supporting a three-shock strategy in any of these circumstances, it is unlikely that chest compressions will improve the already very high chance of return of spontaneous circulation when defibrillation occurs early in the electrical phase, immediately after onset of VF (see Section 3).[223]

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm[262–264] and is only likely to succeed if given within the first few seconds of the onset of a shockable rhythm.[265] There is more success with pulseless VT than with VF. Delivery of a precordial thump must not delay calling for help or accessing a defibrillator. It is therefore appropriate therapy only when several clinicians are present at a witnessed, monitored arrest, and when a defibrillator is not immediately to hand (see Section 3).[223,266] In practice, this is only likely to be in a critical care environment such as the emergency department or ICU.[264]

A precordial thump should be undertaken immediately after confirmation of cardiac arrest and only by healthcare professionals trained in the technique. Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, then retract the fist immediately to create an impulse-like stimulus. There are very rare reports of a precordial thump converting a perfusing to a non-perfusing rhythm.[267]

Airway and ventilation

During the treatment of persistent VF, ensure good-quality chest compressions between defibrillation attempts. Consider reversible causes (4 Hs and 4 Ts) 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 regular, ongoing experience with the technique. Personnel skilled in advanced airway management should attempt laryngoscopy and intubation without stopping chest compressions; a brief pause in chest compressions may be required as the tube is passed between the vocal cords, but this pause should not exceed 10 s. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until return of spontaneous circulation. No studies have shown that tracheal intubation increases survival after cardiac arrest. After intubation, confirm correct tube position and secure it adequately. Ventilate the lungs at 10 breaths min^{-1}; do not hyperventilate the patient. Once the patient's trachea has been intubated, continue chest compressions, at a rate of 100 min^{-1} without pausing during ventilation. A pause in the chest compressions enables 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 (or any reason) result in a substantially higher mean coronary perfusion pressure.

In the absence of personnel skilled in tracheal intubation, a supraglottic airway device (e.g., laryngeal mask airway) is an acceptable alternative (Section 4e). Once a supraglottic airway device 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).

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,[268] 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 IO route. Although normally considered as an alternative route for vascular access in children, it is now established as an effective route in adults.[269] Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a central venous catheter.[270] The recent availability of mechanical IO devices has increased the ease of performing this technique.[271]

Tracheal route

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.[272,273] 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.[274–277] Given the completely unreliable plasma concentrations achieved and increased availability of suitable IO devices, the tracheal route for drug delivery is no longer recommended. Drug delivery via a supraglottic airway device is even less reliable and should not be attempted.[278]

Adrenaline

Despite the widespread use of adrenaline during resuscitation, and several studies involving vasopressin, there is no placebo-controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases neurologically intact survival to hospital discharge. Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs. Despite the lack of human data, the use of adrenaline is still recommended, based largely on animal data and increased short-term survival in humans.[245,246] The alpha-adrenergic actions of adrenaline cause vasoconstriction, which increases myocardial and cerebral perfusion pressure. The higher coronary blood flow increases the frequency and amplitude of the VF waveform and should improve the chance of restoring a circulation when defibrillation is attempted.[260,279,280] Although adrenaline improves short-term survival, animal data indicate that it impairs the microcirculation[281,282] and post-cardiac arrest myocardial dysfunction,[283,284] which both might impact on long-term outcome. The optimal dose of adrenaline is not known, and there are no data supporting the use of repeated doses. There are few data on the pharmacokinetics of adrenaline during CPR. The optimal duration of CPR and number of shocks that should be given before giving drugs is unknown. On the basis of expert consensus, for VF/VT give adrenaline after the third shock once chest compressions have resumed, and then repeat every 3–5 min during cardiac arrest (alternate cycles). Do not interrupt CPR to give drugs.

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[285] and lidocaine,[286] 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 h. Lidocaine, 1 mg kg^{-1}, may be used as an alternative if amiodarone is not available, but do not give lidocaine if amiodarone has been given already.

Magnesium

The routine use of magnesium in cardiac arrest does not increase survival.[287–291] and is not recommended in cardiac arrest unless torsades de pointes is suspected (see peri-arrest arrhythmias).

Bicarbonate

Routine administration of sodium bicarbonate during cardiac arrest and CPR 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 the result of serial blood gas analysis. During cardiac arrest, arterial blood gas values do not reflect the acid–base state of the tissues[292]; the tissue pH will be lower than that in arterial blood. 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/pulseless ventricular tachycardia

In VF/VT persists, consider changing the position of the pads/paddles (see Section 3).[223] Review all potentially reversible causes (see below) and treat any that are identified. Persistent VF/VT may be an indication for percutaneous coronary intervention or thrombolysis—in these cases, a mechanical device CPR may help to maintain high-quality CPR for a prolonged period.[293]

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 arrest in the presence of electrical activity that would normally be associated with a palpable pulse. These patients often have some mechanical myocardial contractions, but these are too weak to produce a detectable pulse or blood pressure—this sometimes described as 'pseudo-PEA' (see below). PEA is often caused by reversible conditions, and can be treated if those conditions are identified and corrected. 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 venous access is achieved. If asystole is displayed, check without stopping CPR, that the leads are attached correctly. Once an advanced airway has been sited, continue chest compressions without pausing during ventilation. After 2 min of CPR, recheck the rhythm. If asystole is present, 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. Give adrenaline 1 mg (IV/IO) every alternate CPR cycle (i.e., about every 3–5 min) once vascular access is obtained. 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, following a 2-min cycle of CPR, if the rhythm has changed to VF, follow the algorithm for shockable rhythms. Otherwise, continue CPR and give adrenaline every 3–5 min following the failure to detect a palpable pulse with the pulse check. If VF is identified on the monitor midway through a 2-min cycle of CPR, complete the cycle of CPR before formal rhythm and shock delivery if appropriate—this strategy will minimise interruptions in chest compressions.

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 8.[294]

Use of ultrasound imaging during advanced life support

Several studies have examined the use of ultrasound during cardiac arrest to detect potentially reversible causes. Although no studies have shown that use of this imaging modality improves outcome, there is no doubt that echocardiography has the potential to detect reversible causes of cardiac arrest (e.g., cardiac tamponade, pulmonary embolism, ischaemia (regional wall motion abnormality), aortic dissection, hypovolaemia, pneumothorax).[295–302] When available for use by trained clinicians, ultrasound may be of use in assisting with diagnosis and treatment of potentially reversible causes of cardiac arrest. The integration of ultrasound into advanced life support requires considerable training if interruptions to chest compressions are to be minimised. A sub-xiphoid probe position has been recommended.[295,301,303] Placement of the probe just before chest compressions are paused for a planned rhythm assessment enables a well-trained operator to obtain views within 10 s.

Absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest is highly predictive of death[304–306] although sensitivity and specificity has not been reported.

The four 'Hs'

Minimise the risk of hypoxia by ensuring that the patient's lungs are ventilated adequately with 100% oxygen during CPR. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described in Section 4e, 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 8h),[294] gastrointestinal bleeding or rupture of an aortic aneurysm. Intravascular volume should be restored rapidly with warmed 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 8a).[294] 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 8c and d)[294]; 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. In the context of cardiac arrest from major trauma, bilateral thoracostomies may provide a more reliable way of decompressing a suspected tension pneumothorax.

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 8h).[294] The increasing use of ultrasound is making the diagnosis of cardiac tamponade much more reliable.

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 8b).[294] Where available, the

appropriate antidotes should be used, but most often treatment is supportive and standard ALS protocols should be followed.

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolus. If pulmonary embolism is a possible cause of the cardiac arrest, consider giving a fibrinolytic drug immediately (Section 4f).[307]

4e 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 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. In the unconscious patient, the commonest site of airway obstruction is at the soft palate and epiglottis.[308,309] Obstruction may also be caused 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 common, 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 laypeople. 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:

- Gurgling is caused by liquid or semisolid foreign material in the large 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 lifted gently to stretch the anterior neck structures (Fig. 4.3).[310–315]

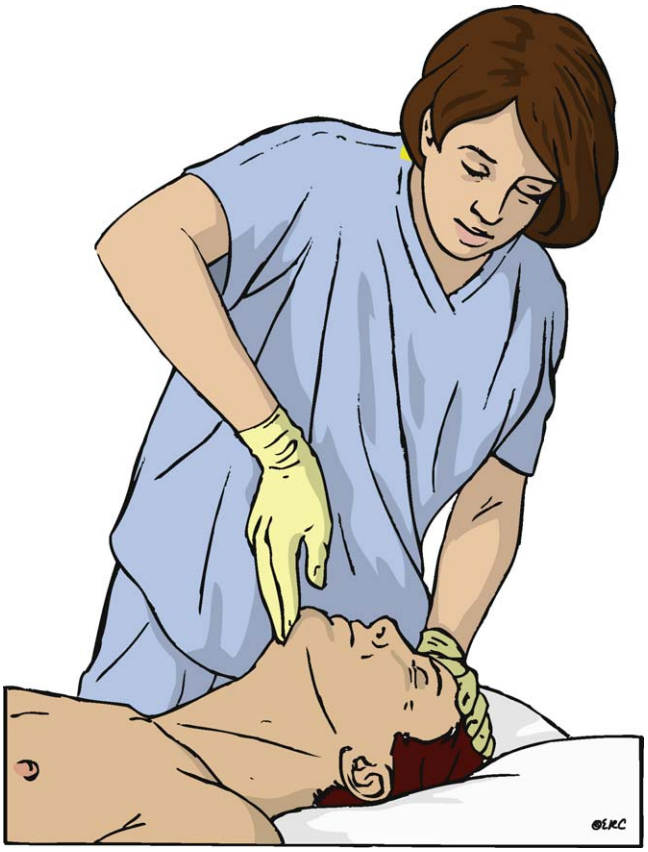

Fig. 4.3. Head tilt and chin lift.

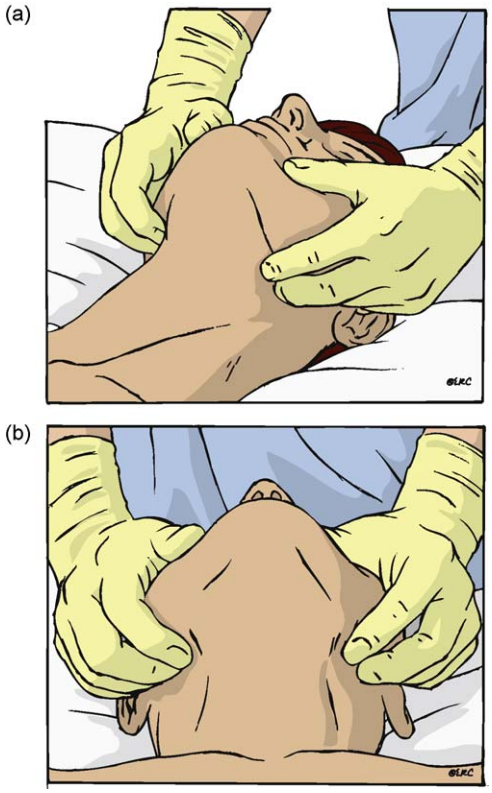

Fig. 4.4. Jaw thrust.

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 (Fig. 4.4).

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, forceps or suction 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;[316–320] 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.[321,322] If life-threatening airway obstruction persists despite effective application of jaw thrust or chin lift, add head tilt in small increments until the airway is open; establishing a patent airway takes priority over concerns about a potential cervical spine injury.

Adjuncts to basic airway techniques

Despite a total lack of published data on the use of nasopharyngeal and oropharyngeal airways during CPR, they 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 the patient's incisors and the angle of the jaw. The most common sizes are 2, 3 and 4 for small, medium and large adults, respectively.

If the glossopharyngeal and laryngeal reflexes are present, insertion of an oropharyngeal may cause airway vomiting or laryngospasm; thus, insertion should be attempted only in comatose patients (Fig. 4.5). The oropharyngeal airway can become obstructed at three possible sites:[323] 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.[324,325] 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).

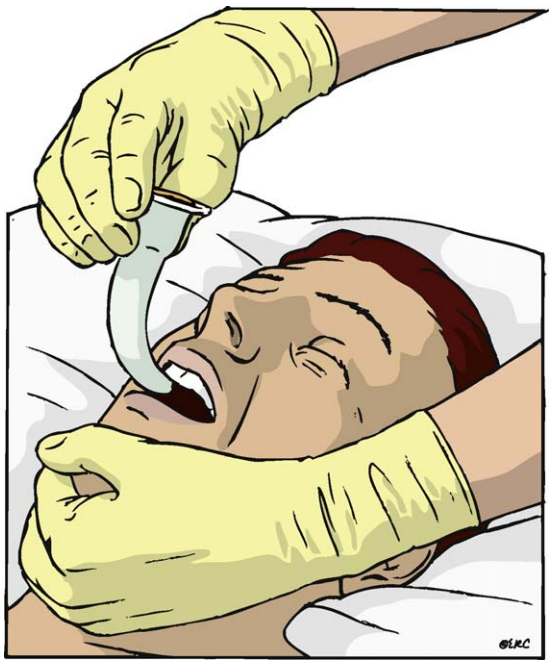

Fig. 4.5. Insertion of oropharyngeal airway.

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.[326] Sizes of 6–7 mm are suitable for adults. Insertion can cause damage to the mucosal lining of the nasal airway, resulting in bleeding in up to 30% of cases.[327] If the tube is too long it may stimulate the laryngeal or glossopharyngeal reflexes to produce laryngospasm or vomiting.

Oxygen

During CPR, give oxygen whenever it is available. There are no data to indicate the optimal arterial blood oxygen saturation (SaO_2) during CPR. There are animal data[328] and some observational clinical data indicating an association between high SaO_2 after ROSC and worse outcome.[329] 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. As soon as the arterial blood oxygen saturation can be measured reliably, by pulse oximeter (SpO_2) or arterial blood gas analysis, titrate the inspired oxygen concentration to achieve an arterial blood oxygen saturation in the range of 94–98%.

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; pharyngeal stimulation 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. 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 (Fig. 4.6).

High airway pressures can be generated if the tidal volume or inspiratory flow is 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 airway 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.

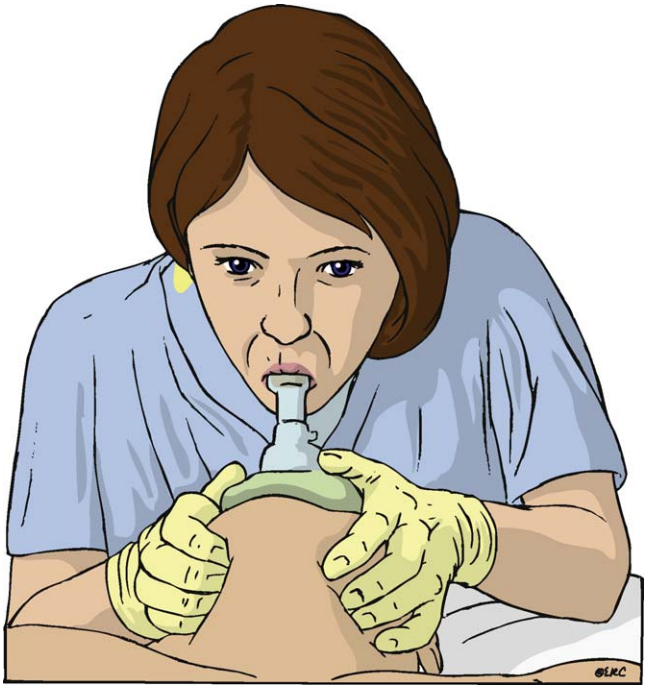

Fig. 4.6. Mouth-to-mask ventilation.

Self-inflating bag

The self-inflating bag can be connected to a facemask, tracheal tube or supraglottic airway device (SAD). Without supplemental oxygen, the self-inflating bag ventilates the patient's lungs with ambient air (21% oxygen). The delivered oxygen concentration can be increased to about 85% by using a reservoir system and attaching oxygen at a flow 10 l min^{-1}.

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.[330] Any significant leak will cause hypoventilation and, if the airway is not patent, gas may be forced into the stomach.[331,332] This will reduce ventilation further and greatly increase the risk of regurgitation and aspiration.[333] Cricoid pressure can reduce this risk[334,335] but requires the presence of a trained assistant. Poorly applied cricoid pressure may make it more difficult to ventilate the patient's lungs.[334,336–339]

The two-person technique for bag-mask ventilation is preferable (Fig. 4.7). One person holds the facemask in place using a jaw thrust with both 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 or a 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 laryngeal seal achieved with a supraglottic airway device 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

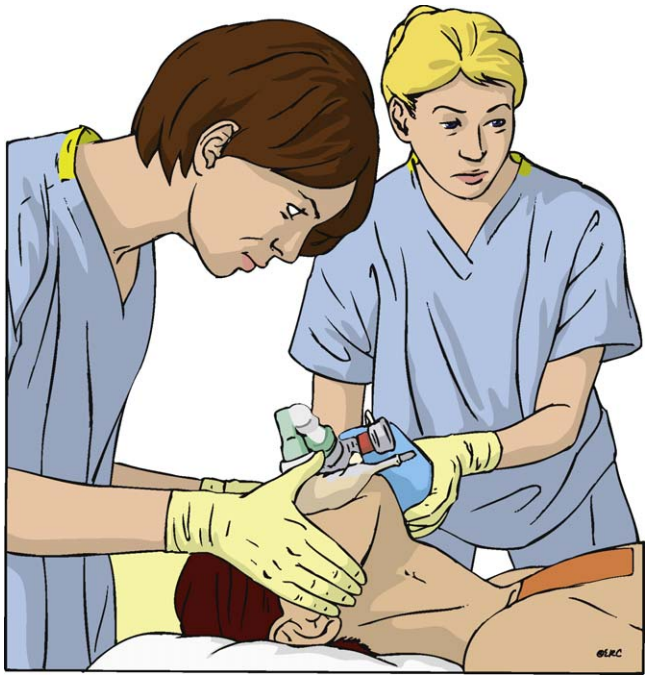

Fig. 4.7. The two-person technique for bag-mask ventilation.

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 is some data indicating that the ventilation rates delivered by healthcare personnel during cardiac arrest are excessive,[242,340,341] although other studies have shown more normal ventilation rates.[245,342,343] 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, supraglottic airway device).

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 parameters. 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.[344]
- Once set, they provide a constant tidal volume, respiratory rate and minute ventilation; thus, they may help to avoid excessive ventilation.
- Are associated with lower peak airway pressures than manual ventilation, which reduces intrathoracic pressure and facilitates improved venous return and subsequent cardiac output.

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.[345,346] 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.

Passive oxygen delivery

In the presence of a patent airway, chest compressions alone may result in some ventilation of the lungs.[347] Oxygen can be delivered passively, either via an adapted tracheal tube (Boussignac tube),[348,349] or with the combination of an oropharyngeal airway and standard oxygen mask with non-rebreather reservoir.[350] Although one study has shown higher neurologically intact survival with passive oxygen delivery (oral airway and oxygen mask) compared with bag-mask ventilation after out-of-hospital VF cardiac arrest, this was a retrospective analysis and is subject to numerous confounders.[350] There is insufficient evidence to support or refute the use of passive oxygen delivery during CPR to improve outcome when compared with oxygen delivery by positive pressure ventilation. Until further data are available, passive oxygen delivery without ventilation is not recommended for routine use during CPR.

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 (6–17% in several studies involving paramedics)[351–354] and dislodgement, is unacceptably high.[355] Prolonged attempts at tracheal intubation are harmful; the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Several alternative airway devices have been considered for airway management during CPR. There are published studies on the use during CPR of the Combitube, the classic laryngeal mask airway (cLMA), the laryngeal tube (LT) and the I-gel, but none of these studies have been powered adequately to enable survival to be studied as a primary endpoint; instead, most researchers have studied insertion and ventilation success rates. The supraglottic airway devices (SADs) are easier to insert than a tracheal tube and, unlike tracheal intubation, can generally be inserted without interrupting chest compressions.[356]

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 (Fig. 4.8) is quicker and easier to insert than a tracheal tube.[357–364] The original LMA (cLMA), which is reusable, has been studied during CPR, but none of these studies has compared it directly with the tracheal tube. A wide variety of single-use LMAs are used for CPR, but they have different characteristics to the cLMA and there are no published data on their performance in this setting.[365] Reported rates of successful ventilation during CPR with the LMA are very high for in-hospital studies (86–100%)[366–369] but generally less impressive (71–90%)[370–372] for out-of-hospital cardiac arrest (OHCA). The reason for the relatively disappointing results from the LMA in OHCA is not clear.

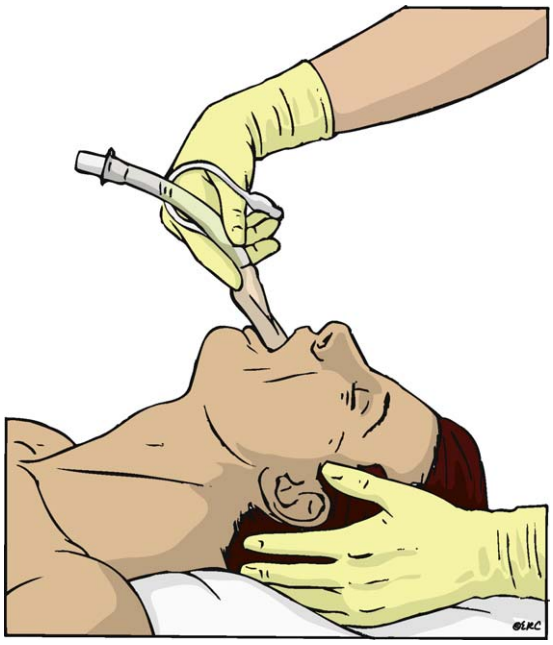

Fig. 4.8. Insertion of a laryngeal mask airway.

When used by inexperienced personnel, ventilation of the lungs of anaesthetised patients is more efficient and easier with an LMA than with a bag-mask.[330] When an LMA can be inserted without delay it is preferable to avoid bag-mask ventilation altogether. In comparison with bag-mask ventilation, use of a self-inflating bag and LMA during cardiac arrest reduces the incidence of regurgitation.[333] One study showed similar arterial blood gas values in patients successfully resuscitated after out-of-hospital cardiac arrest when either an LMA or bag mask was used.[373]

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.

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. There are many studies of the Combitube in CPR and successful ventilation was achieved in 79–98% of patients.[371,374–381] Two RCTs of the Combitube versus tracheal intubation for out-of-hospital cardiac arrest showed no difference in survival.[380,381] Use of the Combitube is waning and in many parts of the world it is being replaced by other devices such as the LT.

Laryngeal tube

The LT was introduced in 2001 (Fig. 4.9); it is known as the King LT airway in the United States. In anaesthetised patients, the performance of the LT is favourable in comparison with the classic LMA and ProSeal LMA.[382,383] After just 2 h of training, nurses successfully inserted a laryngeal tube and achieved ventilation in 24 of 30 (80%) of OHCAs.[384] A disposable version of the laryngeal tube (LT-D) is available and was inserted successfully by paramedics in 92 OHCAs (85 on the first attempt and 7 on the second attempt).[385]

Fig. 4.9. Laryngeal tube. © 2010 ERC.

In a manikin CPR study, use of the LT-D reduced the no-flow time significantly in comparison with use of a tracheal tube.[386]

I-gel

The cuff of the I-gel is made of thermoplastic elastomer gel (styrene ethylene butadene styrene) and does not require inflation; the stem of the I-gel incorporates a bite block and a narrow oesophageal drain tube (Fig. 4.10). It is very easy to insert, requiring only minimal training and a laryngeal seal pressure of 20–24 cm H_2O can be achieved.[387,388] In two manikin studies, insertion of the I-gel was significantly faster than several other airway devices.[356,389] The ease of insertion of the I-gel and its favourable leak pressure make it theoretically very attractive as a resuscitation airway device for those inexperienced in tracheal intubation. Use of the I-gel during cardiac arrest has been reported but more data on its use in this setting are awaited.[390,391]

Other airway devices

ProSeal LMA

The ProSeal LMA (PLMA) 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 cLMA for use during CPR: improved seal with the larynx enabling ventilation at higher airway

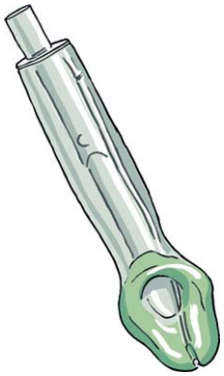

Fig. 4.10. I-gel. © 2010 ERC.

pressures,[392] 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 PLMA is slightly more difficult to insert than a cLMA and is relatively expensive. The Supreme LMA (SLMA) is a disposable version of the PLMA. Studies in anaesthetised patients indicate that it is relatively easy to insert and laryngeal seal pressures of 24–28 cm H_2O can be achieved.[393–395] Data on the use of the SLMA during cardiac arrest are awaited.

Intubating LMA

The intubating LMA (ILMA) is relatively easy to insert[396,397] but subsequent blind insertion of a tracheal tube generally requires more training.[398] One study has documented use of the ILMA after failed intubation by direct laryngoscopy in 24 cardiac arrests by prehospital physicians in France.[399]

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. A recent systematic review of randomised controlled trials (RCTs) of tracheal intubation versus alternative airway management in acutely ill and injured patients identified just three trials[400]: two were RCTs of the Combitube versus tracheal intubation for out-of-hospital cardiac arrest,[380,381] which showed no difference in survival. The third study was a RCT of prehospital tracheal intubation versus management of the airway with a bag-mask in children requiring airway management for cardiac arrest, primary respiratory disorders and severe injuries.[401] There was no overall benefit for tracheal intubation; on the contrary, of the children requiring airway management for a respiratory problem, those randomised to intubation had a lower survival rate that those in the bag-mask group. The Ontario Prehospital Advanced Life Support (OPALS) study documented no increase in survival to hospital discharge when the skills of tracheal intubation and injection of cardiac drugs were added to an optimised basic life support-automated external defibrillator (BLS-AED) system.[244]

The perceived advantages of tracheal intubation over bag-mask ventilation include: enabling ventilation without interrupting chest compressions[402]; enabling effective ventilation, particularly when lung and/or chest compliance is poor; minimising gastric inflation and therefore the risk of regurgitation; protection against pulmonary aspiration of gastric contents; and the potential to free the rescuer's hands for other tasks. Use of the bag-mask is more likely to cause gastric distension that, 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-valve-mask ventilation include:

- The risk of an unrecognised misplaced tracheal tube—in patients with out-of-hospital cardiac arrest the reliably documented incidence ranges from 0.5% to 17%: emergency physicians—0.5%[403]; paramedics—2.4%,[404] 6%,[351,352] 9%,[353] 17%.[354]
- A prolonged period without chest compressions while intubation is attempted—in a study of prehospital intubation by paramedics during 100 cardiac arrests the total duration of the interruptions in CPR associated with tracheal intubation attempts was 110 s (IQR 54–198 s; range 13–446 s) and in 25% the interruptions were more than 3 min.[405] Tracheal intubation attempts accounted for almost 25% of all CPR interruptions.
- A comparatively high failure rate. Intubation success rates correlate with the intubation experience attained by individual paramedics.[406] 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.[407,408]

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. Rescuers must weigh the risks and benefits of intubation against the need to provide effective chest compressions. The intubation attempt may require some 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.[350,409] No intubation attempt should interrupt chest compressions for more than 10 s; if intubation is achievable within these constraints, 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.

Clinical assessment

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. The reported sensitivity (proportion of tracheal intubations correctly identified) and specificity (proportion of oesophageal intubations correctly identified) of clinical assessment varies: sensitivity 74–100%; specificity 66–100%.[403,410–413]

Secondary confirmation of tracheal tube placement by an exhaled carbon dioxide or oesophageal detection device should reduce the risk of unrecognised oesophageal intubation but the performance of the available devices varies considerably. Furthermore, none of the secondary confirmation techniques will differentiate between a tube placed in a main bronchus and one placed correctly in the trachea.

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.[414] There are no data quantifying their ability to monitor tube position after initial placement.

Oesophageal detector device

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 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.[352,410,415–417] The performance of the syringe oesophageal detector device for identifying tracheal tube position has been reported in five cardiac arrest studies[352,418–421]: the sensitivity was 73–100% and the specificity 50–100%. The performance of the bulb oesophageal detector device for identifying tracheal tube position has been reported in three cardiac arrest studies[410,415,421]: the sensitivity was 71–75% and specificity 89–100%.

Carbon dioxide detectors

Carbon dioxide (CO_2) detector devices measure the concentration of exhaled carbon dioxide from the lungs. The persistence of exhaled CO_2 after six ventilations indicates placement of the tracheal tube in the trachea or a main bronchus.[403] Confirmation of correct placement above the carina will require auscultation of the chest bilaterally in the mid-axillary lines. Broadly, there three types of carbon dioxide detector device:

1. Disposable colorimetric end-tidal carbon dioxide ($ETCO_2$) detectors use a litmus paper to detect CO_2, and these devices generally give readings of purple ($ETCO_2$ < 0.5%), tan ($ETCO_2$ 0.5–2%) and yellow ($ETCO_2$ > 2%). In most studies, tracheal placement of the tube is considered verified if the tan colour persists after a few ventilations. In cardiac arrest patients, eight studies reveal 62–100% sensitivity in detecting tracheal placement of the tracheal tube and an 86–100% specificity in identifying non-tracheal position.[258,414,420,422–426] Although colorimetric CO_2 detectors identify placement in patients with good perfusion quite well, these devices are less accurate than clinical assessment in cardiac arrest patients because pulmonary blood flow may be so low that there is insufficient exhaled carbon dioxide. Furthermore, if the tracheal tube is in the oesophagus, six ventilations may lead to gastric distension, vomiting and aspiration.
2. Non-waveform electronic digital $ETCO_2$ devices generally measure $ETCO_2$ using an infrared spectrometer and display the results with a number; they do not provide a waveform graphical display of the respiratory cycle on a capnograph. Five studies of these devices for identification of tracheal tube position in cardiac arrest document 70–100% sensitivity and 100% specificity.[403,412,414,418,422,427]
3. End-tidal CO_2 detectors that include a waveform graphical display (capnographs) are the most reliable for verification of tracheal tube position during cardiac arrest. Two studies of waveform capnography to verify tracheal tube position in victims of cardiac arrest demonstrate 100% sensitivity and 100% specificity in identifying correct tracheal tube placement.[403,428] Three studies with a cumulative total of 194 tracheal and 22 oesophageal tube placements documented an overall 64% sensitivity and 100% specificity in identifying correct tracheal tube placement when using a capnograph in prehospital cardiac arrest victims.[410,415,421] However, in these studies intubation was undertaken only after arrival at hospital (time to intubation averaged more than 30 min) and many of the cardiac arrest victims studied had prolonged resuscitation times and very prolonged transport time.

Based on the available data, the accuracy of colorimetric CO_2 detectors, oesophageal detector devices and non-waveform capnometers does not exceed the accuracy of auscultation and direct visualization for confirming the tracheal position of a tube in victims of cardiac arrest. Waveform capnography is the most sensitive and specific way to confirm and continuously monitor the position of a tracheal tube in victims of cardiac arrest and should supplement clinical assessment (auscultation and visualization of tube through cords). Waveform capnography will not discriminate between tracheal and bronchial placement of the tube—careful auscultation is essential. Existing portable monitors make capnographic initial confirmation and continuous monitoring of tracheal tube position feasible in almost all settings, including out-of-hospital, emergency department, and in-hospital locations where intubation is performed. In the absence of a waveform capnograph it may be preferable to use a supraglottic airway device when advanced airway management is indicated.

Thoracic impedance

There are smaller changes in thoracic impedance with oesophageal ventilations than with ventilation of the lungs.[429–431] Changes in thoracic impedance may be used to detect ventilation[432] and oesphageal intubation[402,433] during cardiac arrest. It is possible that this technology can be used to measure tidal volume during CPR. The role of thoracic impedance as a tool to detect tracheal tube position and adequate ventilation during CPR is undergoing further research but is not yet ready for routine clinical use.

Cricoid pressure

In non-arrest patients cricoid pressure may offer some measure of protection to the airway from aspiration but it may also impede ventilation or interfere with intubation. The role of cricoid during cardiac arrest has not been studied. Application of cricoid pressure during bag-mask ventilation reduces gastric inflation.[334,335,434,435]

Studies in anaesthetised patients show that cricoid pressure impairs ventilation in many patients, increases peak inspiratory pressures and causes complete obstruction in up to 50% of patients depending on the amount of cricoid pressure (in the range of recommended effective pressure) that is applied.[334–339,436,437]

The routine use of cricoid pressure in cardiac arrest is not recommended. If cricoid pressure is used during cardiac arrest, the pressure should be adjusted, relaxed or released if it impedes ventilation or intubation.

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 caused by 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.

4f 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. The evidence for the optimal timing and order of drug delivery, and the optimal dose, is limited.

There are three groups of drugs relevant to the management of cardiac arrest that were reviewed during the 2010 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

Despite the continued widespread use of adrenaline and increased use of vasopressin during resuscitation in some countries, there is no placebo-controlled study that shows that the routine use of any vasopressor during human cardiac arrest increases survival to hospital discharge, although improved short-term survival has been documented.[245,246] 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.[438] Its alpha-adrenergic, vasoconstrictive effects cause 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), transient hypoxaemia due to pulmonary arteriovenous shunting, impaired microcirculation,[281] and worse post-cardiac arrest myocardial dysfunction[283,284] 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. Three randomised controlled trials[439–441] and a meta-analysis[442] demonstrated no difference in outcomes (ROSC, survival to discharge, or neurological outcome) with vasopressin versus adrenaline as a first line vasopressor in cardiac arrest. Two more recent studies comparing adrenaline alone or in combination with vasopressin also demonstrated no difference in ROSC, survival to discharge or neurological outcome.[443,444] There are no alternative vasopressors that provide survival benefit during cardiac arrest resuscitation when compared with adrenaline.

Participants at the 2010 Consensus Conference debated in depth the treatment recommendations that should follow from this evidence. Despite the absence of data demonstrating an increase in long-term survival, 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 any other vasopressor as an alternative to, or in combination with, adrenaline in any cardiac arrest rhythm to improve survival or neurological outcome. Current practice still supports adrenaline as the primary vasopressor for the treatment of cardiac arrest of all rhythms. Although the evidence of benefit from the use of adrenaline is limited, it was felt that the improved short-term survival documented in some studies[245,246] warranted its continued use, although in the absence of clinical evidence, the dose and timing have not been changed in the 2010 guidelines.

Adrenaline

Indications.

- Adrenaline is the first drug used in cardiac arrest of any cause: it is included in the ALS algorithm for use every 3–5 min of CPR (alternate cycles).
- Adrenaline is preferred in the treatment of anaphylaxis (Section 8g).[294]
- Adrenaline is a second-line treatment for cardiogenic shock.

Dose. During cardiac arrest, the initial IV/IO dose of adrenaline is 1 mg. There are no studies showing survival benefit for 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, even small doses of adrenaline (50–100 μg) 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 μg 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 Europe.

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.[285,286] 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 and benzyl alcohol), which causes histamine release, rather than the drug itself.[445] The use of an aqueous amiodarone preparation that is relatively free from these side effects has recently been approved for use in the United States.[446,447]

Following three initial shocks, amiodarone in shock-refractory VF improves the short-term outcome of survival to hospital admission compared with placebo[285] or lidocaine.[286] Amiodarone also appears to improve the response to defibrillation when given to humans or animals with VF or haemodynamically unstable ventricular tachycardia.[446–450] There is no evidence to indicate the optimal 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 4g).

Dose. Consider an initial intravenous dose of 300 mg amiodarone, diluted in 5% dextrose (or other suitable solvent) to a volume of 20 ml (or from a pre-filled syringe), if VF/VT persists after the third shock. Give a further dose of 150 mg if VF/VT persists. Amiodarone can cause thrombophlebitis when injected into a peripheral vein; use a central vein if a central venous catheter is *in situ* but, if not, use a large peripheral vein or the IO route followed by a generous flush. Details about the use of amiodarone for the treatment of other arrhythmias are given in Section 4g.

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 anti-arrhythmic drug of choice. Comparative studies with amiodarone[286] 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 at all out-of-hospital arrests attended by emergency medical services.

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 VF.

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

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.[451] 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[287–291,452] have failed to demonstrate any increase in the rate of ROSC when magnesium is given routinely during CPR.

Indications. Magnesium sulphate is indicated in

- ventricular or supraventricular tachycardia associated with hypomagnesaemia;
- torsades de pointes;
- digoxin toxicity.

Dose. 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

There is no evidence that routinely giving other drugs (e.g., atropine, procainamide, bretylium, calcium and hormones) during human cardiac arrest increases survival to hospital discharge. Recommendations for the use of these drugs are based on limited clinical studies, our understanding of the drug's 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.

Asystole during cardiac arrest is usually due to primary myocardial pathology rather than excessive vagal tone and there is no evidence that routine use of atropine is beneficial in the treatment of asystole or PEA. Several recent studies have failed to demonstrate any benefit from atropine in out-of-hospital or in-hospital cardiac arrests[244,453–458]; and its routine use for asystole or PEA is no longer recommended.

Atropine is indicated in:

- sinus, atrial, or nodal bradycardia when the haemodynamic condition of the patient is unstable (see Section 4g).

Calcium

Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. There is no data supporting any beneficial action for calcium after most cases of cardiac arrest[453,459–463]; conversely, other studies have suggested a possible adverse effect when given routinely during cardiac arrest (all rhythms).[464,465] 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 because pulmonary gas exchange ceases and cellular metabolism becomes anaerobic. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid–base state[292]; analysis of central venous blood may provide a better estimation of tissue pH (see Section 4d). Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. It has the following effects.

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

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.

Several animal and clinical studies have examined the use of buffers during cardiac arrest. Clinical studies using Tribonate®[466] or sodium bicarbonate as buffers have failed to demonstrate any advantage.[466–472] Only two studies have 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.[473,474] 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 8b).[294,475] Giving sodium bicarbonate routinely during cardiac arrest and CPR or after return of spontaneous circulation is not recommended. Consider sodium bicarbonate for

- life-threatening hyperkalaemia;
- cardiac arrest associated with hyperkalaemia;
- 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, central venous or marrow aspirate from IO needle) 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.

Fibrinolysis during CPR

Thrombus formation is a common cause of cardiac arrest, most commonly due to acute myocardial ischaemia following coronary artery occlusion by thrombus, but occasionally due to a dislodged venous thrombus causing a pulmonary embolism. The use of fibrinolytic drugs to break down coronary artery and pulmonary artery thrombus has been the subject of several studies. Fibrinolytics have also been demonstrated in animal studies to have beneficial effects on cerebral blood flow during cardiopulmonary resuscitation,[476,477] and a clinical study has reported less anoxic encephalopathy after fibrinolytic therapy during CPR.[478]

Several studies have examined the use of fibrinolytic therapy given during non-traumatic cardiac arrest unresponsive to

standard therapy.[307,479–484] and some have shown non-significant improvements in survival to hospital discharge,[307,481] and greater ICU survival.[478] A small series of case reports also reported survival to discharge in three cases refractory to standard therapy with VF or PEA treated with fibrinolytics.[485] Conversely, two large clinical trials[486,487] failed to show any significant benefit for fibrinolytics in out-of-hospital cardiac arrest unresponsive to initial interventions.

Results from the use of fibrinolytics in patients suffering cardiac arrest from suspected pulmonary embolus have been variable. A meta-analysis, which included patients with pulmonary embolus as a cause of the arrest, concluded that fibrinolytics increased the rate of ROSC, survival to discharge and long-term neurological function.[488] Several other studies have demonstrated an improvement in ROSC and admission to hospital or the intensive care unit, but not in survival to neurologically intact hospital discharge.[307,479–481,483,484,489–492]

Although several relatively small clinical studies[307,479,481,490] and case series[478,485,493–495] have not demonstrated any increase in bleeding complications with thrombolysis during CPR in non-traumatic cardiac arrest, a recent large study[487] and meta-analysis[488] have shown an increased risk of intracranial bleeding associated with the routine use of fibrinolytics during non-traumatic cardiac arrest. Successful fibrinolysis during cardiopulmonary resuscitation is usually associated with good neurological outcome.[488,490,491]

Fibrinolytic therapy should not be used routinely in cardiac arrest. Consider fibrinolytic therapy when cardiac arrest is caused by proven or suspected acute pulmonary embolus. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported in cases requiring in excess of 60 min of CPR. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts.[496,497] Mortality from surgical embolectomy is high if it follows cardiac arrest and should be avoided in patients requiring CPR. In patients who are not candidates for fibrinolytic therapy, percutaneous mechanical thromboembolectomy should be considered. Ongoing CPR is not a contraindication to fibrinolysis.

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 0.9% sodium chloride 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.[498–505]

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. Two animal studies[506,507] show that the increase in right atrial pressure produced by infusion of normothermic fluid during CPR decreases coronary perfusion pressure, and another animal study[508] shows that the coronary perfusion pressure rise with adrenaline during CPR is not improved with the addition of a fluid infusion.

Small clinical studies have not shown any benefit with hypertonic fluid[509] or chilled fluid.[510,511] One animal study shows that hypertonic saline improves cerebral blood flow during CPR.[512] Ensure normovolaemia, but in the absence of hypovolaemia, infusion of an excessive volume of fluid is likely to be harmful.[513] 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 within the first 2 min of resuscitation, consider gaining IO access. Intraosseous access has traditionally been used for children because of the difficulties in gaining intravenous access, but this route has now become established as a safe and effective route for gaining vascular access in adults too.[271,514–517] Tibial and humeral sites are readily accessible and provide equal flow rates for fluids.[514] Intraosseous delivery of resuscitation drugs will achieve adequate plasma concentrations. Several studies indicate that IO access is safe and effective for fluid resuscitation and drug delivery.[269,518–524]

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 very variable although generally considerably lower than those achieved by the IV or IO routes, particularly with adrenaline. Additionally, relatively large volumes of intratracheal fluid impair gas exchange. With the ease of gaining IO access and the lack of efficacy of tracheal drug administration, tracheal administration of drugs is no longer recommended

CPR techniques and devices

At best, standard manual CPR produces coronary and cerebral perfusion that is just 30% of normal.[525] Several CPR techniques and devices may improve haemodynamics or short-term survival when used by well-trained providers in selected cases. However, the success of any technique or device depends on the education and training of the rescuers and on resources (including personnel). In the hands of some groups, novel techniques and adjuncts may be better than standard CPR. However, a device or technique which provides good quality CPR when used by a highly trained team or in a test setting may show poor quality and frequent interruptions when used in an uncontrolled clinical setting.[526] While no circulatory adjunct is currently recommended for routine use instead of manual CPR, some circulatory adjuncts are being routinely used in both out-of-hospital and in-hospital resuscitation. It is prudent that rescuers are well-trained and that if a circulatory adjunct is used, a program of continuous surveillance be in place to ensure that use of the adjunct does not adversely affect survival. Although manual chest compressions are often performed very poorly,[527–529] no adjunct has consistently been shown to be superior to conventional manual CPR.

Open-chest CPR

Open-chest CPR produces better coronary perfusion coronary pressure than standard CPR[530] and may be indicated for patients with cardiac arrest caused by trauma, in the early postoperative phase after cardiothoracic surgery[531,532] (see Section 8i)[294] 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.[533,534] This enhances venous return during CPR[535,536] and improves ROSC and short-term survival.[537,538] Two studies showed improved survival to hospital discharge with IAC-CPR compared with standard CPR

for in-hospital cardiac arrest,[537,538] but another showed no survival advantage.[539]

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.[540–543] Results of ACD-CPR have been mixed. In some clinical studies ACD-CPR improved haemodynamics compared with standard CPR,[541,543–545] but in another study it did not.[546] In three randomised studies,[545,547,548] 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.[549–553] The efficacy of ACD-CPR may be highly dependent on the quality and duration of training.[554]

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.[205] Two post-mortem studies have shown more rib and sternal fractures after ACD-CPR compared with conventional CPR,[555,556] but another found no difference.[557]

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),[558–560] 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.[561] If rescuers can maintain a tight face-mask seal, the ITD may create the same negative intrathoracic pressure as when used with a tracheal tube.[561]

Most,[562–569] but not all,[570–573] animal studies have shown improved haemodynamics or outcomes during CPR when using the device. Several randomised trials have shown differing results. Two trials suggest that the use of an ITD in combination with ACD-CPR improves 24 h survival and survival to ICU admission in adult OHCA patients,[560,574] but these contrast with others which failed to show any improvement in ROSC or 24 h survival.[558,561] A recent meta-analysis demonstrated improved ROSC and short-term survival but no significant improvement in either survival to discharge or neurologically intact survival to discharge associated with the use of an ITD in the management of adult OHCA patients.[575] In the absence of data showing that the ITD increases survival to hospital discharge, its routine use in cardiac arrest is not recommended.

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,[576] 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.[577–579] One study has documented that the use of a piston CPR device compared with manual CPR increases interruption in CPR due to setting up and removal of the device from patients during transportation in out-of-hospital adult cardiac arrest.[580]

Lund University cardiac arrest system (LUCAS) CPR

The Lund University cardiac arrest system (LUCAS) is a gas-driven sternal compression device that incorporates a suction cup for active decompression. Although animal studies showed that LUCAS-CPR improves haemodynamic and short-term survival compared with standard CPR.[581,582] There are no published randomised human studies comparing LUCAS-CPR with standard CPR. A study using LUCAS for witnessed OHCA was unable to show any benefit (ROSC, survival to hospital or survival to hospital discharge) over standard CPR.[583] Case series totalling 200 patients have reported variable success in use of the LUCAS device, when implemented after an unsuccessful period of manual CPR.[347,581,584–586] One case series used LUCAS to perform CPR while PCI was being performed.[293] Eleven of 43 patients survived to hospital discharge neurologically intact. There are several other reports documenting use of LUCAS during PCI.[585,587,588] One post-mortem study showed similar injuries with LUCAS compared with standard CPR.[589] The early versions of the LUCAS device which were driven by high flow oxygen (LUCAS^{TM}1) should not be used in confined spaces where defibrillation in high ambient oxygen concentrations may risk a fire.[590]

Load-distributing band CPR (AutoPulse)

The load-distributing band (LDB) is a circumferential chest compression device comprising a pneumatically actuated constricting band and backboard. Although the use of LDB CPR improves haemodynamics,[591–593] results of clinical trials have been conflicting. Evidence from one multicenter randomised control trial in over 1000 adults documented no improvement in 4-h survival and worse neurological outcome when LDB-CPR was used by EMS providers for patients with primary out-of-hospital cardiac arrest.[594] However, a post hoc analysis of this study revealed significant heterogeneity between study sites.[598] A further study demonstrated lower odds of 30-day survival (OR 0.4) but subgroup analysis showed an increased rate of ROSC in LDB-CPR treated patients.[595] Other non-randomised human studies have reported increased rates of sustained ROSC,[596,597] increased survival to discharge following OHCA[597] and improved hemodynamics following failed resuscitation from in-hospital cardiac arrest.[591] Evidence from both clinical[594,598] and simulation[599] studies suggest that site-specific factors may influence resuscitation quality and efficacy of this device.

The current status of LUCAS and AutoPulse

Two large prospective randomised multicentre studies are currently underway to evaluate the load-distributing band (AutoPulse) and the Lund University cardiac arrest system (LUCAS). The results of these studies are awaited with interest. In hospital, mechanical devices have been used effectively to support patients undergoing primary coronary intervention (PCI)[293,585] and CT scans[600] and also for prolonged resuscitation attempts (e.g., hypothermia,[601,602] poisoning, thrombolysis for pulmonary embolism, prolonged transport, etc.) where rescuer fatigue may impair the effectiveness of manual chest compression. In the pre-hospital environment where extrication of patients, resuscitation in confined spaces and movement of patients on a trolley often preclude effective manual chest compressions, mechanical devices may also have an important role. During transport to hospital, manual CPR is often performed poorly; mechanical CPR can maintain good quality CPR during an ambulance transfer.[343,603] Mechanical devices also have the advantage of allowing defibrillation without interruption in external chest compression. The role of mechanical devices in all situations requires further evaluation.

4g Peri-arrest arrhythmias

The correct identification and treatment of arrhythmias in the critically ill patient may prevent cardiac arrest from occurring or from reoccurring after successful initial resuscitation. 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 senior doctors with the appropriate expertise.

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

Principles of treatment

The initial assessment and treatment of a patient with an arrhythmia should follow the ABCDE approach. Key elements in this process include assessing for adverse signs; administration of high flow oxygen; obtaining intravenous access, and establishing monitoring (ECG, blood pressure, SpO_2). Whenever possible, record a 12-lead ECG; this will help determine the precise rhythm, either before treatment or retrospectively. Correct any electrolyte abnormalities (e.g., K^+, Mg^{2+}, Ca^{2+}). Consider the cause and context of arrhythmias when planning treatment.

The assessment and treatment of all arrhythmias addresses two factors: the condition of the patient (stable versus unstable), and the nature of the arrhythmia. Anti-arrhythmic drugs are slower in onset 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.

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. Shock—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 mm Hg).
2. Syncope—loss of consciousness, which occurs as a consequence of reduced cerebral blood flow.
3. Heart failure—arrhythmias compromise myocardial performance by reducing coronary artery blood flow. In acute situations this is manifested by pulmonary oedema (failure of the left ventricle) and/or raised jugular venous pressure, and hepatic engorgement (failure of the right ventricle).
4. Myocardial ischaemia—this occurs when myocardial oxygen consumption exceeds delivery. Myocardial ischaemia may present with chest pain (angina) or may occur without pain as an isolated finding on the 12 lead ECG (silent ischaemia). The presence of myocardial ischaemia is especially important if there is underlying coronary artery disease or structural heart disease because it may cause further life-threatening complications including cardiac arrest.

Treatment options

Having determined the rhythm and the presence or absence of adverse signs, the options for immediate treatment are categorised as:

1. Electrical (cardioversion, pacing).
2. Pharmacological (anti-arrhythmic (and other) drugs).

Tachycardias

If the patient is unstable

If the patient is unstable and deteriorating, with any of the adverse signs and symptoms described above being caused by the tachycardia, attempt synchronised cardioversion immediately (Fig. 4.11). 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.

Repeated attempts at electrical cardioversion 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.[604] 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).[223] Atrial flutter and paroxysmal supraventricular tachycardia (SVT) will often convert with lower energies: start with 100-J monophasic or 70–120-J biphasic.

If the patient is stable

If the patient with tachycardia is stable (no adverse signs or symptoms) and is not deteriorating, pharmacological treatment may be possible. Evaluate the rhythm using a 12-lead ECG and assess the QRS duration. If the QRS duration is greater than 0.12 s (3 small squares on standard ECG paper) it is classified as a broad complex tachycardia. If the QRS duration is less than 0.12 s it is a narrow complex tachycardia.

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. 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. Expert help should be sought before using repeated doses or combinations of anti-arrhythmic drugs.

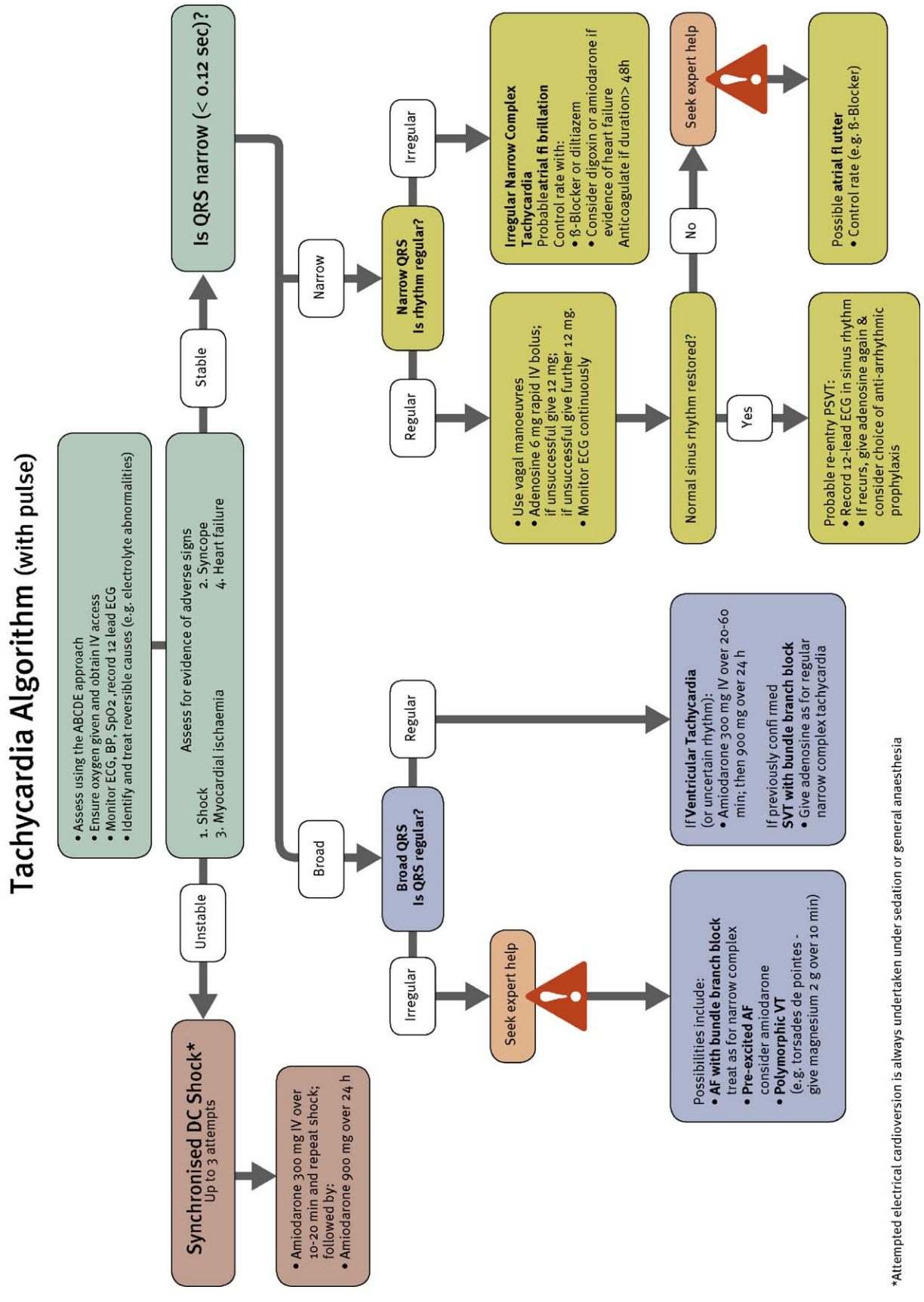

Fig. 4.11. Tachycardia algorithm. © 2010 ERC.

Broad-complex tachycardia

Broad-complex tachycardias are usually ventricular in origin.[605] 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. If there is uncertainty about the source of the arrhythmia, give intravenous adenosine (using the strategy described below) as it may convert the rhythm to sinus and help diagnose the underlying rhythm.[606]

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. Specialist advice should be sought before considering alternatives treatments such as procainamide, nifekalant or sotalol.

Irregular broad complex tachycardia

Irregular broad complex tachycardia is most likely to be AF with bundle branch block. Another possible cause is AF with ventricular pre-excitation (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., torsades de pointes), although this rhythm 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—this can provoke severe tachycardias. Electrical cardioversion is usually the safest treatment option.

Treat torsades de pointes VT immediately by stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate, 2 g, intravenously over 10 min.[607,608] 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

The first step in the assessment of a narrow complex tachycardia is to determine if it is regular or irregular.

The commonest regular narrow-complex tachycardias include:

- sinus tachycardia;
- AV nodal re-entry tachycardia (AVNRT, the commonest type of SVT);
- AV re-entry tachycardia (AVRT), which is associated with Wolff–Parkinson–White (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 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.[609] It causes a regular narrow-complex tachycardia, often with no clearly visible atrial activity on the ECG. Heart rates are usually well above the typical range of sinus rates at rest (60–120 beats min^{-1}). It is usually benign, unless there is additional co-incidental structural heart disease or coronary disease.

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 lead to slowing of the ventricular response and 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 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[609]: carotid sinus massage or the Valsalva manoeuvre will terminate up to a quarter of episodes of paroxysmal SVT. Carotid sinus massage stimulates baroreceptors, which increase vagal tone and reduces sympathetic drive, which slows conduction via the AV node. Carotid sinus massage is given by applying pressure over the carotid artery at the level of the cricoid cartilage. Massage the area with firm circular movements for about 5 s. If this does not terminate the arrhythmia, repeat on the opposite side. Avoid carotid massage if a carotid bruit is present: rupture of an atheromatous plaque could cause cerebral embolism and stroke. 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. 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. This strategy will terminate 90–95% of supraventricular arrhythmias.[610]
- 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 verapamil).
- 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 or diltiazem).

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 as described above. The European Society of Cardiology provides detailed guidelines on the management of AF.[611]

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 clinical scenario dictates that cardioversion is required and the duration of AF is greater than 48 h (or the duration is unknown) give an initial intravenous bolus injection of heparin followed by a continuous infusion to maintain the activated partial thromboplastin time at 1.5–2 times the reference control value. Anticoagulation should be continued for at least 4 weeks thereafter.[611]

If the aim is to control heart rate, the drugs of choice are beta-blockers[612,613] and diltiazem.[614,615] Digoxin and amiodarone may be used in patients with heart failure. Magnesium has also been used although the data supporting this is more limited.[616,617]

If the duration of AF is less than 48 h and rhythm control is considered appropriate, chemical cardioversion may be attempted. Seek expert help and consider ibutilide, flecainide or dofetilide. Amiodarone (300 mg intravenously over 20–60 min followed by 900 mg over 24 h) may also be used but is less effective. 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 in patients with pre-excited AF or atrial flutter, as these drugs block the AV node and cause a relative increase in pre-excitation.

Bradycardia

A bradycardia is defined as a heart rate of <60 beats min^{-1}. Bradycardia can have cardiac causes (e.g., myocardial infarction; myocardial ischaemia; sick sinus syndrome), non-cardiac causes (e.g., vasovagal response, hypothermia; hypoglycaemia; hypothyroidism, raised intracranial pressure) or be caused by drug toxicity (e.g., digoxin; beta-blockers; calcium channel blockers).

Bradycardias are caused by reduced sinoatrial node firing or failure of the atrial-ventricular conduction system. Reduced sinoatrial node firing is seen in sinus bradycardia (caused by excess vagal tone), sinus arrest, and sick sinus syndrome. 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 Mobitz types I and II. In Mobitz type I, the block is at the AV node, is often transient and may be asymptomatic. In Mobitz 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.

Initial assessment

Assess the patient with bradycardia using the ABCDE approach. Consider the potential cause of the bradycardia and look for the adverse signs. Treat any reversible causes of bradycardia identified in the initial assessment. If adverse signs are present start to treat the bradycardia. Initial treatments are pharmacological, with pacing being reserved for patients unresponsive to pharmacological treatments or with risks factors for asystole (Fig. 4.12).

Pharmacological treatment

If adverse signs are present, give atropine, 500 μg, intravenously and, if necessary, repeat every 3–5 min to a total of 3 mg. Doses of atropine of less than 500 μg, paradoxically, may cause further slowing of the heart rate.[618] In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart rate.[619] 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 treatment with atropine is ineffective, consider second line drugs. These include isoprenaline (5 μg min^{-1} starting dose), adrenaline (2–10 μg min^{-1}) and dopamine (2–10 μg kg^{-1} min^{-1}). Theophylline (100–200 mg slow intravenous injection) should be considered if the bradycardia is caused by inferior myocardial infarction, cardiac transplant or spinal cord injury. 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—it can cause a high-degree AV block or even sinus arrest.[620]

Pacing

Initiate transcutaneous pacing immediately if there is no response to atropine, or if atropine is unlikely to be effective.

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.

If atropine is ineffective and transcutaneous pacing is not immediately available, fist pacing can be attempted while waiting for

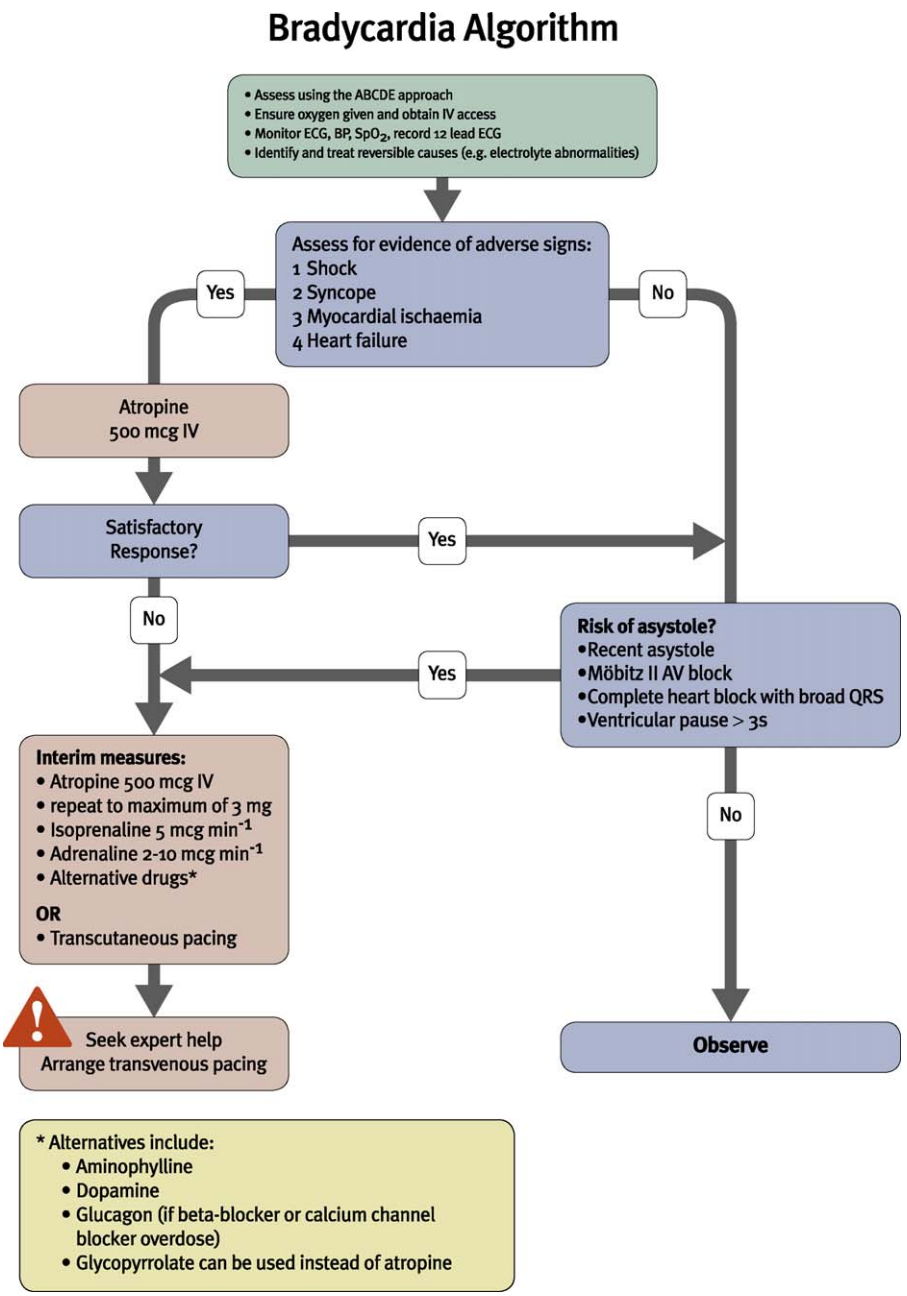

Fig. 4.12. Bradycardia algorithm. © 2010 ERC.

pacing equipment[621–623]: 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}.

Seek expert help to assess the need for temporary transvenous pacing. Temporary transvenous pacing should be considered if there are is a history of recent asystole; Möbitz type II AV block; complete (third-degree) heart block (especially with broad QRS or initial heart rate <40 beats min^{-1}) or evidence of ventricular standstill of more than 3 s.

Anti-arrhythmic 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.[624] 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 monomorphic 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;
- unsuccessful electrical cardioversion.

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 different countries). In patients with 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.[446] 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 can 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. The administration of calcium channel blockers to a patient with ventricular tachycardia may cause cardiovascular collapse.

Diltiazem at a dose of 250 $\mu g\,kg^{-1}$, followed by a second dose of 350 $\mu g\,kg^{-1}$, 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 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 $\mu g\,kg^{-1}$, 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 $\mu g\,kg^{-1}$ over 1 min, followed by an infusion of 50–200 $\mu g\,kg^{-1}\,min^{-1}$.

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

Magnesium

Magnesium is the first line treatment for polymorphic ventricular tachycardia. It may also reduce ventricular rate in atrial fibrillation.[617,625–627] Give magnesium sulphate 2 g (8 mmol) over 10 min. This can be repeated once if necessary.

4h Post-resuscitation care

Introduction

Successful ROSC is the just the first step toward the goal of complete recovery from cardiac arrest. The complex pathophysiological processes that occur following whole body ischaemia during cardiac arrest and the subsequent reperfusion response following successful resuscitation have been termed the post-cardiac arrest syndrome.[628] Many of these patients will require multiple organ support and the treatment they receive this post-resuscitation period influences significantly the ultimate neurological outcome.[184,629–633] 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. Of those patients admitted to intensive care units after cardiac arrest, approximately 25–56% will survive to be discharged from hospital depending on the system and quality of care.[498,629,632,634–638] Of the patients that survive to hospital

discharge, the vast majority have a good neurological outcome although many with some cognitive impairment.[639]

Post-cardiac arrest syndrome

The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and the persistent precipitating pathology.[628] The severity of this syndrome will vary with the duration and cause of cardiac arrest. It may not occur at all if the cardiac arrest is brief. Post-cardiac arrest brain injury manifests as coma, seizures, myoclonus, varying degrees of neurocognitive dysfunction and brain death. Among patients surviving to ICU admission but subsequently dying in-hospital, brain injury is the cause of death in 68% after out-of hospital cardiac arrest and in 23% after in-hospital cardiac arrest.[245,640] Post-cardiac arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypercarbia, hyperoxia, pyrexia, hyperglycaemia and seizures. Significant myocardial dysfunction is common after cardiac arrest but typically recovers by 2–3 days.[641,642] The whole body ischaemia/reperfusion of cardiac arrest activates immunological and coagulation pathways contributing to multiple organ failure and increasing the risk of infection.[643,644] Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion and vasodilation.[645,646]

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. Hypoxaemia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Several animal studies indicate that hyperoxaemia causes oxidative stress and harms post-ischaemic neurones.[647–650] One animal study has demonstrated that adjusting the fractional inspired concentration (FiO_2) to produce an arterial oxygen saturation of 94–96% in the first hour after ROSC ('controlled reoxygenation') achieved better neurological outcomes than achieved with the delivery of 100% oxygen.[328] A recent clinical registry study that included more than 6000 patients supports the animal data and shows post-resuscitation hyperoxaemia is associated with worse outcome, compared with both normoxaemia and hypoxaemia.[329] In clinical practice, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), it may be more practicable to titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%.

Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. Ensure the tracheal tube is positioned correctly well above the carina. Hypocarbia causes cerebral vasoconstriction and a decreased cerebral blood flow.[651] After cardiac arrest, hypocapnoea induced by hyperventilation causes cerebral ischaemia.[652–655] 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.

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. Give adequate doses of sedative, which will reduce oxygen consumption. Bolus doses of a neuromuscular blocking drug may be required, particularly if using therapeutic hypothermia (see below), but try to avoid infusions of neuromuscular blocking drugs because these may mask seizures. Obtain a chest radiograph to check the position of the tracheal tube and central venous lines, assess for pulmonary oedema, and detect complications from CPR such as a pneumothorax associated with rib fractures.

Circulation

The majority of out-of-hospital cardiac arrest patients have coronary artery disease.[656,657] Acute changes in coronary plaque morphology occur in 40–86% of cardiac arrest survivors and in 15–64% of autopsy studies.[658] It is well recognised that post-cardiac arrest patients with ST elevation myocardial infarction (STEMI) should undergo early coronary angiography and percutaneous coronary intervention (PCI) but, because chest pain and/or ST elevation are poor predictors of acute coronary occlusion in these patients,[659] this intervention should be considered in all post-cardiac arrest patients who are suspected of having coronary artery disease.[629,633,659–665] Several studies indicate that the combination of therapeutic hypothermia and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction.[629,633,638,665,666]

Post-cardiac arrest myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias.[641] Early echocardiography will enable the degree of myocardial dysfunction to be quantified.[642] In the ICU an arterial line for continuous blood pressure monitoring is essential. Treatment with fluid, inotropes and vasopressors may be guided by blood pressure, heart rate, urine output, and rate of plasma lactate clearance and central venous oxygen saturations. Non-invasive cardiac output monitors may help to guide treatment but there is no evidence that their use affects outcome. If treatment with fluid resuscitation and vasoactive drugs is insufficient to support the circulation, consider insertion of an intra-aortic balloon pump.[629,638] Infusion of relatively large volumes of fluids are tolerated remarkably well by patients with post-cardiac arrest syndrome.[513,629,630,641] Although early goal directed therapy is well-established in the treatment of sepsis,[667] and has been proposed as a treatment strategy after cardiac arrest,[630] there are no randomised, controlled data to support its routine use.

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 (MAP) of >100 mm Hg versus ≤100 mm Hg 5 min after ROSC; however, good functional recovery was associated with a higher blood pressure during the first 2 h after ROSC.[668] In a registry study of more than 6000 post-cardiac arrest patients, hypotension (systolic blood pressure <90 mm Hg) on admission to ICU was associated with worse outcome.[668a] Good outcomes have been achieved in studies of patients admitted after out-of-hospital cardiac arrest where the MAP target was low as 65–75 mm Hg[629] to as high as 90–100 mm Hg.[632,669] In the absence of definitive data, target the mean arterial blood pressure to achieve an adequate urine output ($1\ ml\ kg^{-1}\ h^{-1}$) and normal or decreasing plasma lactate values, taking into consideration the patient's normal blood pressure, the cause of the arrest and the severity of any myocardial dysfunction.[628] Importantly, hypothermia may increase urine output and impair lactate clearance.

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.[670] After asphyxial cardiac arrest, brain oedema may occur transiently after ROSC but it is rarely associated with clinically relevant increases in intracranial pressure.[671,672] Autoregulation of cerebral blood flow is impaired for some time after cardiac arrest, which means that cerebral perfusion varies with cerebral perfusion pressure instead of being linked to neuronal activity.[673,674] As discussed previously, following ROSC, maintain mean arterial pressure near the patient's normal level.

Sedation

Although it has been common practice to sedate and ventilate patients for at least 24 h after ROSC, there are no high-level data to support a defined period of ventilation, sedation and neuromuscular blockade after cardiac arrest. Patients need to be well-sedated during treatment with therapeutic hypothermia, and the duration of sedation and ventilation is therefore influenced by this treatment. There are no data to indicate whether or not the choice of sedation influences outcome, but a combination of opioids and hypnotics is usually used. Short-acting drugs (e.g., propofol, alfentanil, remifentanil) will enable earlier neurological assessment. Adequate sedation will reduce oxygen consumption. During hypothermia, optimal sedation can reduce or prevent shivering, which enable the target temperature to be achieved more rapidly. Use of published sedation scales for monitoring these patients (e.g., the Richmond or Ramsay Scales) may be helpful.[675,676]

Control of seizures

Seizures or myoclonus or both occur in 5–15% of adult patients who achieve ROSC and 10–40% of those who remain comatose.[498,677–680] Seizures increase cerebral metabolism by up to 3-fold[681] and may cause cerebral injury: treat promptly and effectively with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate. Myoclonus can be particularly difficult to treat; phenytoin is often ineffective. Clonazepam is the most effective antimyoclonic drug, but sodium valproate, levetiracetam and propofol may also be effective.[682] Maintenance therapy should be started after the first event once potential precipitating causes (e.g., intracranial haemorrhage, electrolyte imbalance) are excluded. No studies directly address the use of prophylactic anticonvulsant drugs after cardiac arrest in adults.

Glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome.[498–501,504,634,683,684] Although one randomised controlled trial in a cardiac surgical intensive care unit showed that tight control of blood glucose (4.4–6.1 mmol l^{-1} or 80–110 mg dl^{-1}) using insulin reduced hospital mortality in critically ill adults,[685] a second study by the same group in medical ICU patients showed no mortality benefit from tight glucose control.[686] In one randomised trial of patients resuscitated from OHCA with ventricular fibrillation, strict glucose control (72–108 mg dl^{-1}, 4–6 mmol l^{-1}]) gave no survival benefit compared with moderate glucose control (108–144 mg dl^{-1}, 6–8 mmol l^{-1}) and there were more episodes of hypoglycaemia in the strict glucose control group.[687] A large randomised trial of intensive glucose control (4.5–6.0 mmol l^{-1}) versus conventional glucose control (10 mmol l^{-1} or less) in general ICU patients reported increased 90-day mortality in patients treated with intensive glucose control.[688] Another recent study and two meta-analyses of studies of tight glucose control versus conventional glucose control in critically ill patients showed no significant difference in mortality but found tight glucose control was associated with a significantly increased risk of hypoglycaemia.[689–691] Severe hypoglycaemia is associated with increased mortality in critically ill patients,[692] and comatose patients are at particular risk from unrecognised hypoglycaemia. There is some evidence that, irrespective of the target range, variability in glucose values is associated with mortality.[693]

Based on the available data, following ROSC blood glucose should be maintained at $\leq$10 mmol l^{-1} (180 mg dl^{-1}).[694] Hypoglycaemia should be avoided. Strict glucose control should not be implemented in adult patients with ROSC after cardiac arrest because of the increased risk of hypoglycaemia.

Temperature control

Treatment of hyperpyrexia

A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest.[695–697] Several studies document an association between post-cardiac arrest pyrexia and poor outcomes.[498,695,697–700] There are no randomised controlled trials evaluating the effect of treatment of pyrexia (defined as $\geq$37.6 °C) compared to no temperature control in patients after cardiac arrest. Although the effect of elevated temperature on outcome is not proved, it seems prudent to treat any hyperthermia occurring after cardiac arrest with antipyretics or active cooling.

Therapeutic hypothermia

Animal and human data indicate that mild hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia.[701,702] Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen ($CMRO_2$) by about 6% for each 1 °C reduction in temperature[703] and this may reduce the release of excitatory amino acids and free radicals.[701] Hypothermia blocks the intracellular consequences of excitotoxin exposure (high calcium and glutamate concentrations) and reduces the inflammatory response associated with the post-cardiac arrest syndrome.

Which post-cardiac arrest patients should be cooled?. All studies of post-cardiac arrest therapeutic hypothermia have included only patients in coma. There is good evidence supporting the use of induced hypothermia in comatose survivors of out-of-hospital cardiac arrest caused by VF. One randomised trial[704] and a pseudo-randomised trial[669] demonstrated improved neurological outcome at hospital discharge or at 6 months in comatose patients after out-of-hospital VF cardiac arrest. Cooling was initiated within minutes to hours after ROSC and a temperature range of 32–34 °C was maintained for 12–24 h. Two studies with historical control groups showed improvement in neurological outcome after therapeutic hypothermia for comatose survivors of VF cardiac arrest.[705–707] Extrapolation of these data to other cardiac arrests (e.g., other initial rhythms, in-hospital arrests, paediatric patients) seems reasonable but is supported by only lower level data.

One small, randomised trial showed reduced plasma lactate values and oxygen extraction ratios in a group of comatose survivors after cardiac arrest with asystole or PEA who were cooled with a cooling cap.[708] Six studies with historical control groups have shown benefit using therapeutic hypothermia in comatose survivors of OHCA after all rhythm arrests.[629,632,709–712] Two non-randomised studies with concurrent controls indicate possible benefit of hypothermia following cardiac arrest from other initial rhythms in- and out-of-hospital.[713,714]

How to cool?. The practical application of therapeutic hypothermia is divided into three phases: induction, maintenance, and rewarming.[715] External and/or internal cooling techniques can be used to initiate cooling. An infusion of 30 ml kg^{-1} of 4 °C saline or Hartmann's solution decreases core temperature by approximately 1.5 °C[629,633,638,706,707,711,716–727] and this technique can be used initiate cooling prehospital.[511,728–731]

Other methods of inducing and/or maintaining hypothermia include:

- Simple ice packs and/or wet towels are inexpensive; however, these methods may be more time consuming for nursing staff, may result in greater temperature fluctuations, and do not enable controlled rewarming.[633,638,669,705,709,710,725,726,732–734] Ice cold fluids alone cannot be used to maintain hypothermia,[719] but even the addition of simple ice packs may control the temperature adequately.[725]
- Cooling blankets or pads.[727,735–740]
- Transnasal evaporative cooling.[740a]
- Water or air circulating blankets.[629,630,632,706,707,712,713,727,741–744]
- Water circulating gel-coated pads.[629,711,720,721,727,738,743,745]
- Intravascular heat exchanger, placed usually in the femoral or subclavian veins.[629,630,713,714,718,724,727,732,733,742,746–748]
- Cardiopulmonary bypass.[749]

In most cases, it is easy to cool patients initially after ROSC because the temperature normally decreases within this first hour.[498,698] Initial cooling is facilitated by neuromuscular blockade and sedation, which will prevent shivering.[750] Magnesium sulphate, a naturally occurring NMDA receptor antagonist, that reduces the shivering threshold slightly, can also be given to reduce the shivering threshold.[715,751]

In the maintenance phase, a cooling method with effective temperature monitoring that avoids temperature fluctuations is preferred. This is best achieved with external or internal cooling devices that include continuous temperature feedback to achieve a set target temperature. The temperature is typically monitored from a thermistor placed in the bladder and/or oesophagus.[715] As yet, there are no data indicating that any specific cooling technique increases survival when compared with any other cooling technique; however, internal devices enable more precise temperature control compared with external techniques.[727]

Plasma electrolyte concentrations, effective intravascular volume and metabolic rate can change rapidly during rewarming, as they do during cooling. Thus, rewarming must be achieved slowly: the optimal rate is not known, but the consensus is currently about 0.25–0.5 °C of warming per hour.[713]

When to cool?. Animal data indicate that earlier cooling after ROSC produces better outcomes.[752] Ultimately, starting cooling during cardiac arrest may be most beneficial—animal data indicate that this may facilitate ROSC.[753,754] Several clinical studies have shown that hypothermia can be initiated prehospital,[510,728,729,731,740,740a] but, as yet, there are no human data proving that time target temperature produces better outcomes. One registry-based case series of 986 comatose post-cardiac arrest patients suggested that time to initiation of cooling was not associated with improved neurological outcome post-discharge.[665] A case series of 49 consecutive comatose post-cardiac arrest patients intravascularly cooled after out-of-hospital cardiac arrest also documented that time to target temperature was not an independent predictor of neurologic outcome.[748]

Physiological effects and complications of hypothermia. The well-recognised physiological effects of hypothermia need to be managed carefully[715]:

- Shivering will increase metabolic and heat production, thus reducing cooling rates—strategies to reduce shivering are discussed above.
- Mild hypothermia increases systemic vascular resistance, causes arrhythmias (usually bradycardia).[714]
- It causes a diuresis and electrolyte abnormalities such as hypophosphatemia, hypokalemia, hypomagesemia and hypocalcemia.[715,755]
- Hypothermia decreases insulin sensitivity and insulin secretion, hyperglycemia,[669] which will need treatment with insulin (see glucose control).
- Mild hypothermia impairs coagulation and increases bleeding although this has not be confirmed in many clinical studies.[629,704] In one registry study an increased rate of minor bleeding occurred with the combination of coronary angiography and therapeutic hypothermia, but this combination of interventions was the also the best predictor of good outcome.[665]
- Hypothermia can impair the immune system and increase infection rates.[715,734,736]
- The serum amylase concentration is commonly increased during hypothermia but the significance of this unclear.
- The clearance of sedative drugs and neuromuscular blockers is reduced by up to 30% at a core temperature of 34 °C.[756]

Contraindications to hypothermia. Generally recognised contraindications to therapeutic hypothermia, but which are not applied universally, include: severe systemic infection, established multiple organ failure, and pre-existing medical coagulopathy (fibrinolytic therapy is not a contraindication to therapeutic hypothermia).

Other therapies

Neuroprotective drugs (coenzyme Q10,[737] thiopental,[757] glucocorticoids,[758,759] nimodipine,[760,761] lidoflazine,[762] or diazepam[452]) used alone, or as an adjunct to therapeutic hypothermia, have not been demonstrated to increase neurologically intact survival when included in the post-arrest treatment of cardiac arrest. There is also insufficient evidence to support the routine use of high-volume haemofiltration[763] to improve neurological outcome in patients with ROSC after cardiac arrest.

Prognostication

Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury; this has been shown both with[245] and without[640] therapeutic hypothermia. A quarter of those dying after admission to ICU following in-hospital cardiac arrest die from neurological injury. A means of predicting neurological outcome that can be applied to individual patients immediately after ROSC is required. Many studies have focused on prediction of poor long term outcome (vegetative state or death), based on clinical or test findings that indicate irreversible brain injury, to enable clinicians to limit care or withdraw organ support. The implications of these prognostic tests are such that they should have 100% specificity or zero false positive rate (FPR), i.e., proportion of individuals who eventually have a 'good' long-term outcome despite the prediction of a poor outcome. This topic of prognostication after cardiac arrest is controversial because: (1) many studies are confounded by self-fulfilling prophecy (treatment is rarely continued for long enough in sufficient patients to enable a true estimate of the false positive rate for any given prognosticator); (2) many studies include so few patients that even if the FPR is 0%, the upper limit of the 95% confidence interval may be high; and (3) most prognostication studies have been undertaken before implementation of therapeutic hypothermia

and there is evidence that this therapy makes these tests less reliable.

Clinical examination

There are no clinical neurological signs that reliably predict poor outcome (cerebral performance category [CPC] 3 or 4, or death) less than 24 h after cardiac arrest. In adult patients who are comatose after cardiac arrest, and who have not been treated with hypothermia and who do not have confounding factors (such as hypotension, sedatives or muscle relaxants), the absence of both pupillary light and corneal reflex at ≥72 h reliably predicts poor outcome (FPR 0%; 95% CI 0–9%).[680] Absence of vestibulo-ocular reflexes at ≥24 h (FPR 0%; 95% CI 0–14%)[764,765] and a GCS motor score of 2 or less at ≥72 h (FPR 5%; 95% CI 2–9%)[680] are less reliable. Other clinical signs, including myoclonus, are not recommended for predicting poor outcome. The presence of myoclonus status in adults is strongly associated with poor outcome,[679,680,766–768] but rare cases of good neurological recovery have been described and accurate diagnosis is problematic.[769–773]

Biochemical markers

Serum neuronal specific enolase elevations are associated with poor outcome for comatose patients after cardiac arrest.[680,748,774–792] Although specific cut-off values with a false positive rate of 0% have been reported, clinical application is limited due to variability in the 0% FPR cut-off values reported among various studies.

Serum S100 elevations are associated with poor outcome for comatose patients after cardiac arrest.[680,774–776,782,784,785,787,788,791,793–798]

Many other serum markers measured after sustained return of spontaneous circulation have been associated with poor outcome after cardiac arrest, including BNP,[799] vWF,[800] ICAM-1,[800] procalcitonin,[794] IL-1ra, RANTES, sTNFRII, IL-6, IL-8 and IL-10.[645] However, other studies found no relationship between outcome and serum IL-8,[793] and procalcitonin and sTREM-1.[801]

Worse outcomes for comatose survivors of cardiac arrest are also associated with increased levels of cerebrospinal fluid (CSF)-CK[802,803] and cerebrospinal fluid-CKBB.[774,775,777,789,803–807] However, one study found no relationship between cerebrospinal fluid-CKBB and prognosis.[808]

Outcomes are also associated with increased cerebrospinal fluid levels of other markers including NSE,[775,784,789]), S100,[784] LDH, GOT,[777,803] neurofilament,[809] acid phosphatase and lactate.[803] Cerebrospinal fluid levels of beta-D-N-acetylglucosaminidase, and pyruvate were not associated with the prognosis of cardiac arrest.[803]

In summary, evidence does not support the use of serum or CSF biomarkers alone as predictors of poor outcomes in comatose patients after cardiac arrest with or without treatment with therapeutic hypothermia (TH). Limitations included small numbers of patients and/or inconsistency in cut-off values for predicting poor outcome.

Electrophysiological studies

No electrophysiological study reliably predicts outcome of a comatose patient within the first 24 h after cardiac arrest. If somatosensory evoked potentials (SSEP) are measured after 24 h in comatose cardiac arrest survivors not treated with therapeutic hypothermia, bilateral absence of the N20 cortical response to median nerve stimulation predicts poor outcome (death or CPC 3 or 4) with a FPR of 0.7% (95% CI 0.1–3.7).[774] In the absence of confounding circumstances such as sedatives, hypotension, hypothermia or hypoxemia, it is reasonable to use unprocessed EEG interpretation (specifically identifying generalized suppression to less than 20 μV, burst suppression pattern with generalized epileptic activity, or diffuse periodic complexes on a flat background) observed between 24 and 72 h after ROSC to assist the prediction of a poor outcome (FPR 3%, 95% CI 0.9–11%) in comatose survivors of cardiac arrest not treated with hypothermia.[774] There is inadequate evidence to support the routine use of other electrophysiological studies (e.g., abnormal brainstem auditory evoked potentials) for prognostication of poor outcome in comatose cardiac arrest survivors.[606]

Imaging studies

Many imaging modalities (magnetic resonance imaging [MRI], computed tomography [CT], single photon emission computed tomography [SPECT], cerebral angiography, transcranial Doppler, nuclear medicine, near infra-red spectroscopy [NIRS]) have been studied to determine their utility for prediction of outcome in adult cardiac arrest survivors.[606] There are no level one or level two studies that support the use of any imaging modality to predict outcome of comatose cardiac arrest survivors. Overall, those imaging studies that have been undertaken were limited by small sample sizes, variable time of imaging (many very late in the course), lack of comparison with a standardised method of prognostication, and early withdrawal of care. Despite tremendous potential, neuroimaging has yet to be proven as an independently accurate modality for prediction of outcome in individual comatose cardiac arrest survivors and, at this time, its routine use for this purpose is not recommended.

Impact of therapeutic hypothermia on prognostication

There is inadequate evidence to recommend a specific approach to prognosticating poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia. There are no clinical neurological signs, electrophysiological studies, biomarkers, or imaging modalities that can reliably predict neurological outcome in the first 24 h after cardiac arrest. Based on limited available evidence, potentially reliable prognosticators of poor outcome in patients treated with therapeutic hypothermia after cardiac arrest include bilateral absence of N20 peak on SSEP ≥ 24 h after cardiac arrest (FPR 0%, 95% CI 0–69%) and the absence of both corneal and pupillary reflexes 3 or more days after cardiac arrest (FPR 0%, 95% CI 0–48%).[766,810] Limited available evidence also suggests that a Glasgow Motor Score of 2 or less at 3 days post-ROSC (FPR 14% [95% CI 3–44%])[766] and the presence of status epilepticus (FPR of 7% [95% CI 1–25%] to 11.5% [95% CI 3–31%])[811,812] are potentially unreliable prognosticators of poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia. One study of 111 post-cardiac arrest patients treated with therapeutic hypothermia attempted to validate prognostic criteria proposed by the American Academy of Neurology.[774,813] This study demonstrated that clinical exam findings at 36–72 h were unreliable predictors of poor neurological outcome while bilaterally absent N20 peak on somatosensory evoked potentials (false positive rate 0%, 95% CI 0–13%) and unreactive electroencephalogram background (false positive rate 0%, 95% CI 0–13%) were the most reliable. A decision rule derived using this dataset demonstrated that the presence of two independent predictors of poor neurological outcome (incomplete recovery brainstem reflexes, early myoclonus, unreactive electroencephalogram and bilaterally absent cortical SSEPs) predicted poor neurological outcome with a false positive rate of 0% (95% CI 0–14%). Serum biomarkers such as NSE are potentially valuable as adjunctive studies in prognostication of poor outcome in patients treated with hypothermia, but their reliability is limited because few patients have been studied and the assay is not well

standardisation.[814,815] Given the limited available evidence, decisions to limit care should not be made based on the results of a single prognostication tool.

Organ donation

Solid organs have been successfully transplanted after cardiac death.[816] This group of patients offers an untapped opportunity to increase the organ donor pool. Organ retrieval from non-heart beating donors is classified as controlled or uncontrolled.[817] Controlled donation occurs after planned withdrawal of treatment following non-survivable injuries/illnesses. Uncontrolled donation describes donation after a patient is brought in dead or with on-going CPR that fails to restore a spontaneous circulation.

Graft function after transplantation is influenced by the duration of warm ischaemia time from cessation of cardiac output until organ preservation is undertaken. Where delays in the initiation of organ preservation are anticipated mechanical chest compression devices may be useful for maintaining effective circulation and organ perfusion whilst the necessary regulatory steps to allow organ donation to occur are undertaken.[818–820]

Cardiac arrest centres

There is wide variability in survival among hospitals caring for patients after resuscitation from cardiac arrest.[498,631,635,636,821–823] There is some low-level evidence that ICUs admitting more than 50 post-cardiac arrest patients per year produce better survival rates than those admitting less than 20 cases per year.[636] Another observational study showed that unadjusted survival to discharge was greater in hospitals that received ≥40 cardiac arrest patients/year compared with those that received <40 per year, but this difference disappeared after adjustment for patient factors.[824]

Several studies with historic control groups have shown improved survival after implementation of a comprehensive package of post-resuscitation care that includes therapeutic hypothermia and percutaneous coronary intervention.[629,632,633] There is also evidence of improved survival after out-of-hospital cardiac arrest in large hospitals with cardiac catheter facilities compared with smaller hospitals with no cardiac catheter facilities.[631] Several studies of out-of-hospital adult cardiac arrest failed to demonstrate any effect of transport interval from the scene to the receiving hospital on survival to hospital discharge if return of spontaneous circulation was achieved at the scene and transport intervals were short (3–11 min).[825–827] This implies that it may be safe to bypass local hospitals and transport the post-cardiac arrest patient to a regional cardiac arrest centre.

There is indirect evidence that regional cardiac resuscitation systems of care improve outcome after ST elevation myocardial infarction (STEMI).[828–850]

The implication from all these data is that specialist cardiac arrest centres and systems of care may be effective but, as yet, there is no direct evidence to support this hypothesis.[851–853]

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