

European Resuscitation Council Guidelines for Resuscitation 2010 Section 1. Executive summary

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Introduction

The publication of these European Resuscitation Council (ERC) Guidelines for cardiopulmonary resuscitation (CPR) updates those that were published in 2005 and maintains the established 5-yearly cycle of guideline changes.¹ Like the previous guidelines, these 2010 guidelines are based on the most recent International Consensus on CPR Science with Treatment Recommendations (CoSTR),² which incorporated the results of systematic reviews of a wide range of topics relating to CPR. Resuscitation science continues to advance, and clinical guidelines must be updated regularly to reflect these developments and advise healthcare providers on best practice. In between the 5-yearly guideline updates, interim scientific statements can inform the healthcare provider about new therapies that might influence outcome significantly.³

This executive summary provides the essential treatment algorithms for the resuscitation of children and adults and highlights the main guideline changes since 2005. Detailed guidance is provided in each of the remaining nine sections, which are published as individual papers within this issue of Resuscitation. The sections of the 2010 guidelines are:

1. Executive summary;
2. Adult basic life support and use of automated external defibrillators;⁴
3. Electrical therapies: automated external defibrillators, defibrillation, cardioversion and pacing;⁵
4. Adult advanced life support;⁶

5. Initial management of acute coronary syndromes;⁷
6. Paediatric life support;⁸
7. Resuscitation of babies at birth;⁹
8. Cardiac arrest in special circumstances: electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution;¹⁰
9. Principles of education in resuscitation;¹¹
10. The ethics of resuscitation and end-of-life decisions.¹²

The guidelines that follow do not define the only way that resuscitation can be delivered; they merely represent a widely accepted view of how resuscitation should be undertaken both safely and effectively. The publication of new and revised treatment recommendations does not imply that current clinical care is either unsafe or ineffective.

Summary of main changes since 2005 Guidelines

Basic life support

Changes in basic life support (BLS) since the 2005 guidelines include:^{4,13}

- Dispatchers should be trained to interrogate callers with strict protocols to elicit information. This information should focus on the recognition of unresponsiveness and the quality of breathing. In combination with unresponsiveness, absence of breathing or any abnormality of breathing should start a dispatch protocol for suspected cardiac arrest. The importance of gasping as sign of cardiac arrest is emphasised.
- All rescuers, trained or not, should provide chest compressions to victims of cardiac arrest. A strong emphasis on delivering

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high quality chest compressions remains essential. The aim should be to push to a depth of at least 5 cm at a rate of at least 100 compressions min^{-1} , to allow full chest recoil, and to minimise interruptions in chest compressions. Trained rescuers should also provide ventilations with a compression–ventilation (CV) ratio of 30:2. Telephone-guided chest compression-only CPR is encouraged for untrained rescuers.

- The use of prompt/feedback devices during CPR will enable immediate feedback to rescuers and is encouraged. The data stored in rescue equipment can be used to monitor and improve the quality of CPR performance and provide feedback to professional rescuers during debriefing sessions.

Electrical therapies: automated external defibrillators, defibrillation, cardioversion and pacing^{5,14}

The most important changes in the 2010 ERC Guidelines for electrical therapies include:

- The importance of early, uninterrupted chest compressions is emphasised throughout these guidelines.
- Much greater emphasis on minimising the duration of the pre-shock and post-shock pauses; the continuation of compressions during charging of the defibrillator is recommended.
- Emphasis on resumption of chest compressions following defibrillation; in combination with continuation of compressions during defibrillator charging, the delivery of defibrillation should be achievable with an interruption in chest compressions of no more than 5 s.
- The safety of the rescuer remains paramount, but there is recognition in these guidelines that the risk of harm to a rescuer from a defibrillator is very small, particularly if the rescuer is wearing gloves. The focus is now on a rapid safety check to minimise the pre-shock pause.
- When treating out-of-hospital cardiac arrest, emergency medical services (EMS) personnel should provide good-quality CPR while a defibrillator is retrieved, applied and charged, but routine delivery of a specified period of CPR (e.g., 2 or 3 min) before rhythm analysis and a shock is delivered is no longer recommended. For some emergency medical services that have already fully implemented a 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.
- The use of up to three-stacked shocks may be considered if VF/VT occurs during cardiac catheterisation or in the early post-operative period following cardiac surgery. This three-shock strategy may also be considered for an initial, witnessed VF/VT cardiac arrest when the patient is already connected to a manual defibrillator.
- Encouragement of the further development of AED programmes – there is a need for further deployment of AEDs in both public and residential areas.

Adult advanced life support

The most important changes in the 2010 ERC Advanced Life Support (ALS) Guidelines include^{6,15}:

- 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 specified period of cardiopulmonary resuscitation (CPR) before out-of-hospital defibrillation following cardiac arrest unwitnessed by EMS personnel.
- Continuation of chest compressions while a defibrillator is charged – this will minimise the pre-shock 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 (IO) 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 (PEA).
- 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 (including 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 $>10 \text{ mmol l}^{-1}$ ($>180 \text{ mg dl}^{-1}$) 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, especially if the patient has been treated with therapeutic hypothermia.

Initial management of acute coronary syndromes

Changes in the management of acute coronary syndrome since the 2005 guidelines include^{7,16}:

- The term non-ST elevation myocardial infarction-acute coronary syndrome (NSTEMI-ACS) has been introduced for both NSTEMI and unstable angina pectoris because the differential diagnosis is dependent on biomarkers that may be detectable only after

several hours, whereas decisions on treatment are dependent on the clinical signs at presentation.

- History, clinical examinations, biomarkers, ECG criteria and risk scores are unreliable for the identification of patients who may be safely discharged early.
- The role of chest pain observation units (CPUs) is to identify, by using repeated clinical examinations, ECG and biomarker testing, those patients who require admission for invasive procedures. This may include provocative testing and, in selected patients, imaging procedures such as cardiac computed tomography, magnetic resonance imaging, etc.
- Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided.
- Nitrates should not be used for diagnostic purposes.
- Supplementary oxygen is to be given only to those patients with hypoxaemia, breathlessness or pulmonary congestion. Hyperoxaemia may be harmful in uncomplicated infarction.
- Guidelines for treatment with acetyl salicylic acid (ASA) have been made more liberal: ASA may now be given by bystanders with or without EMS dispatcher assistance.
- Revised guidance for new anti-platelet and anti-thrombin treatment for patients with ST elevation myocardial infarction (STEMI) and non-STEMI-ACS based on therapeutic strategy.
- Gp IIb/IIIa inhibitors before angiography/percutaneous coronary intervention (PCI) are discouraged.
- The reperfusion strategy in STEMI has been updated:
 - Primary PCI (PPCI) is the preferred reperfusion strategy provided it is performed in a timely manner by an experienced team.
 - A nearby hospital may be bypassed by the EMS provided PPCI can be achieved without too much delay.
 - The acceptable delay between start of fibrinolysis and first balloon inflation varies widely between about 45 and 180 min depending on infarct localisation, age of the patient, and duration of symptoms.
 - 'Rescue PCI' should be undertaken if fibrinolysis fails.
 - The strategy of routine PCI immediately after fibrinolysis ('facilitated PCI') is discouraged.
 - Patients with successful fibrinolysis but not in a PCI-capable hospital should be transferred for angiography and eventual PCI, performed optimally 6–24 h after fibrinolysis (the 'pharmaco-invasive' approach).
 - Angiography and, if necessary, PCI may be reasonable in patients with ROSC after cardiac arrest and may be part of a standardised post-cardiac arrest protocol.
 - To achieve these goals, the creation of networks including EMS, non PCI capable hospitals and PCI hospitals is useful.
- Recommendations for the use of beta-blockers are more restricted: there is no evidence for routine intravenous beta-blockers except in specific circumstances such as for the treatment of tachyarrhythmias. Otherwise, beta-blockers should be started in low doses only after the patient is stabilised.
- Guidelines on the use of prophylactic anti-arrhythmics angiotensin, converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs) and statins are unchanged.

Paediatric life support

Major changes in these new guidelines for paediatric life support include^{8,17}:

- Recognition of cardiac arrest – Healthcare providers cannot reliably determine the presence or absence of a pulse in less than 10s in infants or children. Healthcare providers should look for signs of life and if they are confident in the technique,

they may add pulse palpation for diagnosing cardiac arrest and decide whether they should begin chest compressions or not. The decision to begin CPR must be taken in less than 10s. According to the child's age, carotid (children), brachial (infants) or femoral pulse (children and infants) checks may be used.

- The CV ratio used for children should be based on whether one, or more than one rescuer is present. Lay rescuers, who usually learn only single-rescuer techniques, should be taught to use a ratio of 30 compressions to 2 ventilations, which is the same as the adult guidelines and enables anyone trained in BLS to resuscitate children with minimal additional information. Rescuers with a duty to respond should learn and use a 15:2 CV ratio; however, they can use the 30:2 ratio if they are alone, particularly if they are not achieving an adequate number of compressions. Ventilation remains a very important component of CPR in asphyxial arrests. Rescuers who are unable or unwilling to provide mouth-to-mouth ventilation should be encouraged to perform at least compression-only CPR.
- The emphasis is on achieving quality compressions of an adequate depth with minimal interruptions to minimise no-flow time. Compress the chest to at least one third of the anterior-posterior chest diameter in all children (i.e., approximately 4 cm in infants and approximately 5 cm in children). Subsequent complete release is emphasised. For both infants and children, the compression rate should be at least 100 but not greater than 120 min⁻¹. The compression technique for infants includes two-finger compression for single rescuers and the two-thumb encircling technique for two or more rescuers. For older children, a one- or two-hand technique can be used, according to rescuer preference.
- Automated external defibrillators (AEDs) are safe and successful when used in children older than 1 year of age. Purpose-made paediatric pads or software attenuate the output of the machine to 50–75 J and these are recommended for children aged 1–8 years. If an attenuated shock or a manually adjustable machine is not available, an unmodified adult AED may be used in children older than 1 year. There are case reports of successful use of AEDs in children aged less than 1 year; in the rare case of a shockable rhythm occurring in a child less than 1 year, it is reasonable to use an AED (preferably with dose attenuator).
- To reduce the no flow time, when using a manual defibrillator, chest compressions are continued while applying and charging the paddles or self-adhesive pads (if the size of the child's chest allows this). Chest compressions are paused briefly once the defibrillator is charged to deliver the shock. For simplicity and consistency with adult BLS and ALS guidance, a single-shock strategy using a non-escalating dose of 4 J kg⁻¹ (preferably biphasic, but monophasic is acceptable) is recommended for defibrillation in children.
- Cuffed tracheal tubes can be used safely in infants and young children. The size should be selected by applying a validated formula.
- The safety and value of using cricoid pressure during tracheal intubation is not clear. Therefore, the application of cricoid pressure should be modified or discontinued if it impedes ventilation or the speed or ease of intubation.
- Monitoring exhaled carbon dioxide (CO₂), ideally by capnography, is helpful to confirm correct tracheal tube position and recommended during CPR to help assess and optimize its quality.
- Once spontaneous circulation is restored, inspired oxygen should be titrated to limit the risk of hyperoxaemia.
- Implementation of a rapid response system in a paediatric inpatient setting may reduce rates of cardiac and respiratory arrest and in-hospital mortality.

- New topics in the 2010 guidelines include channelopathies and several new special circumstances: trauma, single ventricle pre- and post-1st stage repair, post-Fontan circulation, and pulmonary hypertension.

Resuscitation of babies at birth

The following are the main changes that have been made to the guidelines for resuscitation at birth in 2010^{9,18}:

- For uncompromised babies, a delay in cord clamping of at least 1 min from the complete delivery of the infant, is now recommended. As yet there is insufficient evidence to recommend an appropriate time for clamping the cord in babies who are severely compromised at birth.
- For term infants, air should be used for resuscitation at birth. If, despite effective ventilation, oxygenation (ideally guided by oximetry) remains unacceptable, use of a higher concentration of oxygen should be considered.
- Preterm babies less than 32 weeks gestation may not reach the same transcutaneous oxygen saturations in air as those achieved by term babies. Therefore blended oxygen and air should be given judiciously and its use guided by pulse oximetry. If a blend of oxygen and air is not available use what is available.
- Preterm babies of less than 28 weeks gestation should be completely covered up to their necks in a food-grade plastic wrap or bag, without drying, immediately after birth. They should then be nursed under a radiant heater and stabilised. They should remain wrapped until their temperature has been checked after admission. For these infants delivery room temperatures should be at least 26 °C.
- The recommended CV ratio for CPR remains at 3:1 for newborn resuscitation.
- Attempts to aspirate meconium from the nose and mouth of the unborn baby, while the head is still on the perineum, are not recommended. If presented with a floppy, apnoeic baby born through meconium it is reasonable to rapidly inspect the oropharynx to remove potential obstructions. If appropriate expertise is available, tracheal intubation and suction may be useful. However, if attempted intubation is prolonged or unsuccessful, start mask ventilation, particularly if there is persistent bradycardia.
- If adrenaline is given then the intravenous route is recommended using a dose of 10–30 $\mu\text{g kg}^{-1}$. If the tracheal route is used, it is likely that a dose of at least 50–100 $\mu\text{g kg}^{-1}$ will be needed to achieve a similar effect to 10 $\mu\text{g kg}^{-1}$ intravenously.
- Detection of exhaled carbon dioxide in addition to clinical assessment is recommended as the most reliable method to confirm placement of a tracheal tube in neonates with spontaneous circulation.
- Newly born infants born at term or near-term with evolving moderate to severe hypoxic–ischaemic encephalopathy should, where possible, be treated with therapeutic hypothermia. This does not affect immediate resuscitation but is important for post-resuscitation care.

Principles of education in resuscitation

The key issues identified by the Education, Implementation and Teams (EIT) task force of the International Liaison Committee on Resuscitation (ILCOR) during the Guidelines 2010 evidence evaluation process are^{11,19}:

- Educational interventions should be evaluated to ensure that they reliably achieve the learning objectives. The aim is to ensure that learners acquire and retain the skills and knowledge that will

enable them to act correctly in actual cardiac arrests and improve patient outcomes.

- Short video/computer self-instruction courses, with minimal or no instructor coaching, combined with hands-on practice can be considered as an effective alternative to instructor-led basic life support (CPR and AED) courses.
- Ideally all citizens should be trained in standard CPR that includes compressions and ventilations. There are circumstances however where training in compression-only CPR is appropriate (e.g., opportunistic training with very limited time). Those trained in compression-only CPR should be encouraged to learn standard CPR.
- Basic and advanced life support knowledge and skills deteriorate in as little as 3–6 months. The use of frequent assessments will identify those individuals who require refresher training to help maintain their knowledge and skills.
- CPR prompt or feedback devices improve CPR skill acquisition and retention and should be considered during CPR training for laypeople and healthcare professionals.
- An increased emphasis on non-technical skills (NTS) such as leadership, teamwork, task management and structured communication will help improve the performance of CPR and patient care.
- Team briefings to plan for resuscitation attempts, and debriefings based on performance during simulated or actual resuscitation attempts should be used to help improve resuscitation team and individual performance.
- Research about the impact of resuscitation training on actual patient outcomes is limited. Although manikin studies are useful, researchers should be encouraged to study and report the impact of educational interventions on actual patient outcomes.

Epidemiology and outcome of cardiac arrest

Ischaemic heart disease is the leading cause of death in the world.²⁰ In Europe, cardiovascular disease accounts for around 40% of all deaths under the age of 75 years.²¹ Sudden cardiac arrest is responsible for more than 60% of adult deaths from coronary heart disease.²² Summary data from 37 communities in Europe indicate that the annual incidence of EMS-treated out-of-hospital cardiopulmonary arrests (OHCA) for all rhythms is 38 per 100,000 population.^{22a} Based on these data, the annual incidence of EMS-treated ventricular fibrillation (VF) arrest is 17 per 100,000 and survival to hospital discharge is 10.7% for all-rhythm and 21.2% for VF cardiac arrest. Recent data from 10 North American sites are remarkably consistent with these figures: median rate of survival to hospital discharge was 8.4% after EMS-treated cardiac arrest from any rhythm and 22.0% after VF.²³ There is some evidence that long-term survival rates after cardiac arrest are increasing.^{24,25} On initial heart rhythm analysis, about 25–30% of OHCA victims have VF, a percentage that has declined over the last 20 years.^{26–30} It is likely that many more victims have VF or rapid ventricular tachycardia (VT) at the time of collapse but, by the time the first electrocardiogram (ECG) is recorded by EMS personnel, the rhythm has deteriorated to asystole.^{31,32} When the rhythm is recorded soon after collapse, in particular by an on-site AED, the proportion of patients in VF can be as high as 59%³³ to 65%.³⁴

The reported incidence of in-hospital cardiac arrest is more variable, but is in the range of 1–5 per 1000 admissions.³⁵ Recent data from the American Heart Association's National Registry of CPR indicate that survival to hospital discharge after in-hospital cardiac arrest is 17.6% (all rhythms).³⁶ The initial rhythm is VF or pulseless VT in 25% of cases and, of these, 37% survive to leave hospital; after PEA or asystole, 11.5% survive to hospital discharge.

The International Consensus on Cardiopulmonary Science

The International Liaison Committee on Resuscitation (ILCOR) includes representatives from the American Heart Association (AHA), the European Resuscitation Council (ERC), the Heart and Stroke Foundation of Canada (HSFC), the Australian and New Zealand Committee on Resuscitation (ANZCOR), Resuscitation Council of Southern Africa (RCSA), the Inter-American Heart Foundation (IAHF), and the Resuscitation Council of Asia (RCA). Since 2000, researchers from the ILCOR member councils have evaluated resuscitation science in 5-yearly cycles. The conclusions and recommendations of the 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care With Treatment Recommendations were published at the end of 2005.^{37,38} The most recent International Consensus Conference was held in Dallas in February 2010 and the published conclusions and recommendations from this process form the basis of these 2010 ERC Guidelines.²

Each of the six ILCOR task forces [basic life support (BLS); advanced life support (ALS); acute coronary syndromes (ACS); paediatric life support (PLS); neonatal life support (NLS); and education, implementation and teams (EIT)] identified topics requiring evidence evaluation and invited international experts to review them. The literature reviews followed a standardised 'worksheet' template including a specifically designed grading system to define the level of evidence of each study.³⁹ When possible, two expert reviewers were invited to undertake independent evaluations for each topic. The 2010 International Consensus Conference involved 313 experts from 30 countries. During the 3 years leading up to this conference, 356 worksheet authors reviewed thousands of relevant, peer-reviewed publications to address 277 specific resuscitation questions, each in standard PICO (Population, Intervention, Comparison Outcome) format.² Each science statement summarised the experts' interpretation of all relevant data on a specific topic and consensus draft treatment recommendations were added by the relevant ILCOR task force. Final wording of science statements and treatment recommendations was completed after further review by ILCOR member organisations and the editorial board.²

The comprehensive conflict of interest (COI) policy that was created for the 2005 International Consensus Conference⁴⁰ was revised for 2010.⁴¹ Representatives of manufacturers and industry did not participate in either of the 2005 and the 2010 conferences.

From science to guidelines

As in 2005, the resuscitation organisations forming ILCOR will publish individual resuscitation guidelines that are consistent

with the science in the consensus document, but will also consider geographic, economic and system differences in practice, and the availability of medical devices and drugs. These 2010 ERC Resuscitation Guidelines are derived from the 2010 CoSTR document but represent consensus among members of the ERC Executive Committee. The ERC Executive Committee considers these new recommendations to be the most effective and easily learned interventions that can be supported by current knowledge, research and experience. Inevitably, even within Europe, differences in the availability of drugs, equipment, and personnel will necessitate local, regional and national adaptation of these guidelines. Many of the recommendations made in the ERC Guidelines 2005 remain unchanged in 2010, either because no new studies have been published or because new evidence since 2005 has merely strengthened the evidence that was already available.

Conflict of interest policy for the 2010 ERC Guidelines

All authors of these 2010 ERC Resuscitation Guidelines have signed COI declarations ([Appendix B](#)).

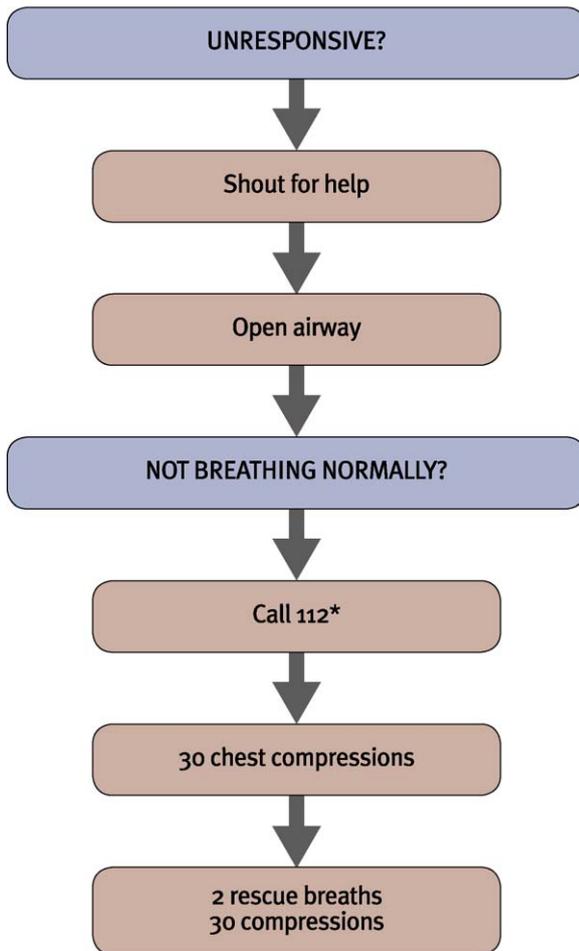
The Chain of Survival

The actions linking the victim of sudden cardiac arrest with survival are called the Chain of Survival ([Fig. 1.1](#)). The first link of this chain indicates the importance of recognising those at risk of cardiac arrest and calling for help in the hope that early treatment can prevent arrest. The central links depict the integration of CPR and defibrillation as the fundamental components of early resuscitation in an attempt to restore life. Immediate CPR can double or triple survival from VF OHCA.^{42–45} Performing chest-compression-only CPR is better than giving no CPR at all.^{46,47} Following VF OHCA, cardiopulmonary resuscitation plus defibrillation within 3–5 min of collapse can produce survival rates as high as 49–75%.^{48–55} Each minute of delay before defibrillation reduces the probability of survival to discharge by 10–12%.^{42,56} The final link in the Chain of Survival, effective post-resuscitation care, is targeted at preserving function, particularly of the brain and heart. In hospital, the importance of early recognition of the critically ill patient and activation of a medical emergency or rapid response team, with treatment aimed at preventing cardiac arrest, is now well accepted.⁶ Over the last few years, the importance of the post-cardiac arrest phase of treatment, depicted in the fourth ring of the Chain of Survival, has been increasingly recognised.³ Differences in post-cardiac arrest treatment may account for some of the inter-hospital variability in outcome after cardiac arrest.^{57–63}



Fig. 1.1. Chain of Survival.

Adult Basic Life Support



*or national emergency number

Fig. 1.2. Basic life support algorithm.

4. Keeping the airway open, look, listen and feel for breathing:

- look for chest movement;
- listen at the victim's mouth for breath sounds;
- feel for air on your cheek;
- decide if breathing is normal, not normal or absent.

In the first few minutes after cardiac arrest, a victim may be barely breathing, or taking infrequent, slow and noisy gasps. Do not confuse this with normal breathing. Look, listen and feel for *no more* than 10 s to determine whether the victim is breathing normally. If you have any doubt whether breathing is normal, act as if it is not normal.

5a. If he is breathing normally:

- turn him into the recovery position (see below);
- send or go for help – call 112 or local emergency number for an ambulance;
- continue to assess that breathing remains normal.

5b. If the breathing is *not* normal or absent:

- send someone for help and to find and bring an AED if available; or if you are on your own, use your mobile phone to alert the ambulance service – leave the victim only when there is no other option;
- start chest compression as follows:

- kneel by the side of the victim;
- place the heel of one hand in the centre of the victim's chest; (which is the lower half of the victim's breastbone (sternum));
- place the heel of your other hand on top of the first hand;
- interlock the fingers of your hands and ensure that pressure is not applied over the victim's ribs. Keep your arms straight. Do not apply any pressure over the upper abdomen or the bottom end of the sternum.
- position yourself vertically above the victim's chest and press down on the sternum at least 5 cm (but not exceeding 6 cm);
- after each compression, release all the pressure on the chest without losing contact between your hands and the sternum; repeat at a rate of at least 100 min⁻¹ (but not exceeding 120 min⁻¹);
- compression and release should take equal amounts of time.

6a. Combine chest compression with rescue breaths.

- After 30 compressions open the airway again using head tilt and chin lift.
- Pinch the soft part of the nose closed, using the index finger and thumb of your hand on the forehead.
- Allow the mouth to open, but maintain chin lift.
- Take a normal breath and place your lips around his mouth, making sure that you have a good seal.
- Blow steadily into the mouth while watching for the chest to rise, taking about 1 s as in normal breathing; this is an effective rescue breath.
- Maintaining head tilt and chin lift, take your mouth away from the victim and watch for the chest to fall as air comes out.
- Take another normal breath and blow into the victim's mouth once more to achieve a total of two effective rescue breaths. The two breaths should not take more than 5 s in all. Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions.
- Continue with chest compressions and rescue breaths in a ratio of 30:2.
- Stop to recheck the victim only if he starts to wake up: to move, opens eyes and to breathe normally. Otherwise, do not interrupt resuscitation.

If your initial rescue breath does not make the chest rise as in normal breathing, then before your next attempt:

- look into the victim's mouth and remove any obstruction;
- recheck that there is adequate head tilt and chin lift;
- do not attempt more than two breaths each time before returning to chest compressions.

Adult basic life support

Adult BLS sequence

Throughout this section, the male gender implies both males and females.

Basic life support comprises the following sequence of actions (Fig. 1.2).

1. Make sure you, the victim and any bystanders are safe.
2. Check the victim for a response:
 - gently shake his shoulders and ask loudly: "Are you all right?"
- 3a. If he responds:
 - leave him in the position in which you find him, provided there is no further danger;
 - try to find out what is wrong with him and get help if needed;
 - reassess him regularly.
- 3b. If he does *not* respond:
 - shout for help
 - turn the victim onto his back and then open the airway using head; tilt and chin lift;
 - place your hand on his forehead and gently tilt his head back;
 - with your fingertips under the point of the victim's chin, lift the chin to open the airway.

If there is more than one rescuer present, another rescuer should take over delivering CPR every 2 min to prevent fatigue. Ensure that interruption of chest compressions is minimal during the changeover of rescuers.

6b. Chest-compression-only CPR may be used as follows:

- if you are not trained, or are unwilling to give rescue breaths, give chest compressions only;
- if only chest compressions are given, these should be continuous, at a rate of at least 100 min⁻¹ (but not exceeding 120 min⁻¹).

7. Do not interrupt resuscitation until:

- professional help arrives and takes over; or
- the victim starts to wake up: to move, opens eyes and to breathe normally; or
- you become exhausted.

Recognition of cardiorespiratory arrest

Checking the carotid pulse (or any other pulse) is an inaccurate method of confirming the presence or absence of circulation, both for lay rescuers and for professionals.^{64–66} Healthcare professionals, as well as lay rescuers, have difficulty determining the presence or absence of adequate or normal breathing in unresponsive victims.^{67,68} This may be because the victim is making occasional (agonal) gasps, which occur in the first minutes after onset in up to 40% of cardiac arrests.⁶⁹ Laypeople should be taught to begin CPR if the victim is unconscious (unresponsive) and not breathing normally. It should be emphasised during training that the presence of agonal gasps is an indication for starting CPR immediately.

Initial rescue breaths

In adults needing CPR, the cardiac arrest is likely to have a primary cardiac cause – CPR should start with chest compression rather than initial ventilations. Time should not be spent checking the mouth for foreign bodies unless attempted rescue breathing fails to make the chest rise.

Ventilation

During CPR, the optimal tidal volume, respiratory rate and inspired oxygen concentration to achieve adequate oxygenation and CO₂ removal is unknown. During CPR, blood flow to the lungs is substantially reduced, so an adequate ventilation–perfusion ratio can be maintained with lower tidal volumes and respiratory rates than normal.⁷⁰ Hyperventilation is harmful because it increases intrathoracic pressure, which decreases venous return to the heart and reduces cardiac output. Interruptions in chest compression reduce survival.⁷¹

Rescuers should give each rescue breath over about 1 s, with enough volume to make the victim's chest rise, but to avoid rapid or forceful breaths. The time taken to give two breaths should not exceed 5 s. These recommendations apply to all forms of ventilation during CPR, including mouth-to-mouth and bag-mask ventilation with and without supplementary oxygen.

Chest compression

Chest compressions generate a small but critical amount of blood flow to the brain and myocardium and increase the likelihood that defibrillation will be successful. Optimal chest compression technique comprises: compressing the chest at a rate of at least 100 min⁻¹ and to a depth of at least 5 cm (for an adult), but not exceeding 6 cm; allowing the chest to recoil completely after each compression^{72,73}; taking approximately the same amount of time for compression as relaxation. Rescuers can be assisted

to achieve the recommended compression rate and depth by prompt/feedback devices that are either built into the AED or manual defibrillator, or are stand-alone devices.

Compression-only CPR

Some healthcare professionals as well as lay rescuers indicate that they would be reluctant to perform mouth-to-mouth ventilation, especially in unknown victims of cardiac arrest.^{74,75} Animal studies have shown that chest-compression-only CPR may be as effective as combined ventilation and compression in the first few minutes after non-asphyxial arrest.^{76,77} If the airway is open, occasional gasps and passive chest recoil may provide some air exchange, but this may result in ventilation of the dead space only.^{69,78–80} Animal and mathematical model studies of chest-compression-only CPR have shown that arterial oxygen stores deplete in 2–4 min.^{81,82} In adults, the outcome of chest compression without ventilation is significantly better than the outcome of giving no CPR at all in non-asphyxial arrest.^{46,47} Several studies of human cardiac arrest suggest equivalence of chest-compression-only CPR and chest compressions combined with rescue breaths, but none of these studies exclude the possibility that chest-compression-only is inferior to chest compressions combined with ventilations.^{47,83} Chest compression-only may be sufficient only in the first few minutes after collapse. Chest-compression-only CPR is not as effective as conventional CPR for cardiac arrests of non-cardiac origin (e.g., drowning or suffocation) in adults and children.^{84,85} Chest compression combined with rescue breaths is, therefore, the method of choice for CPR delivered by both trained lay rescuers and professionals. Laypeople should be encouraged to perform compression-only CPR if they are unable or unwilling to provide rescue breaths, or when instructed during an emergency call to an ambulance dispatcher centre.

Risks to the rescuer

Physical effects

The incidence of adverse effects (muscle strain, back symptoms, shortness of breath, hyperventilation) on the rescuer from CPR training and actual performance is very low.⁸⁶ Several manikin studies have found that, as a result of rescuer fatigue, chest compression depth can decrease as little as 2 min after starting chest compressions.⁸⁷ Rescuers should change about every 2 min to prevent a decrease in compression quality due to rescuer fatigue. Changing rescuers should not interrupt chest compressions.

Risks during defibrillation

A large randomised trial of public access defibrillation showed that AEDs can be used safely by laypeople and first responders.⁸⁸ A systematic review identified only eight papers that reported a total of 29 adverse events associated with defibrillation.⁸⁹ Only one of these adverse events was published after 1997.⁹⁰

Disease transmission

There are only very few cases reported where performing CPR has been linked to disease transmission. Three studies showed that barrier devices decreased transmission of bacteria in controlled laboratory settings.^{91,92} Because the risk of disease transmission is very low, initiating rescue breathing without a barrier device is reasonable. If the victim is known to have a serious infection appropriate precautions are recommended.

Recovery position

There are several variations of the recovery position, each with its own advantages. No single position is perfect for all victims.^{93,94} The position should be stable, near to a true lateral position with

Adult Foreign Body Airway Obstruction Treatment

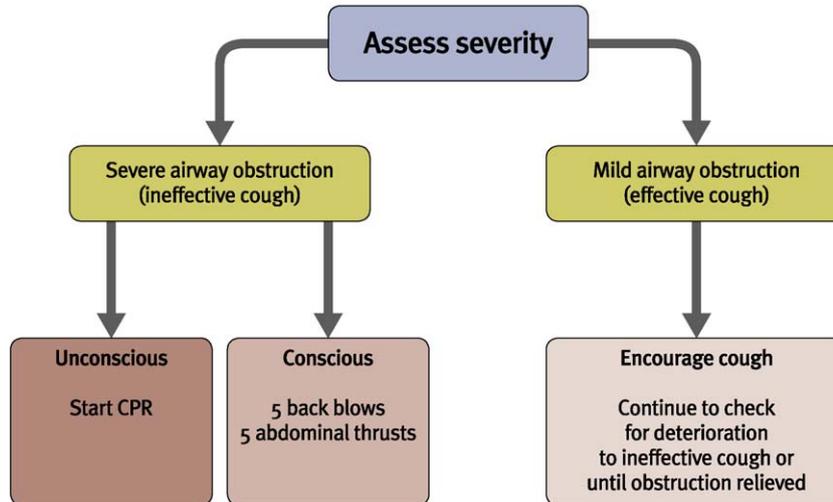


Fig. 1.3. Adult foreign-body airway obstruction (choking) sequence. © 2010 ERC.

Table 1.1

Differentiation between mild and severe foreign body airway obstruction (FBAO)^a

Sign	Mild obstruction	Severe obstruction
“Are you choking?”	“Yes”	Unable to speak, may nod
Other signs	Can speak, cough, breathe	Cannot breathe/wheezy breathing/silent attempts to cough/unconsciousness

^a General signs of FBAO: attack occurs while eating; victim may clutch his neck.

the head dependent, and with no pressure on the chest to impair breathing.⁹⁵

Foreign-body airway obstruction (choking)

Foreign-body airway obstruction (FBAO) is an uncommon but potentially treatable cause of accidental death.⁹⁶ The signs and symptoms enabling differentiation between mild and severe airway obstruction are summarised in Table 1.1. The adult foreign-body airway obstruction (choking) sequence is shown in Fig. 1.3.

Electrical therapies: automated external defibrillators, defibrillation, cardioversion and pacing

Automated external defibrillators

Automated external defibrillators (AEDs) are safe and effective when used by either laypeople or healthcare professionals (in- or out-of-hospital). Use of an AED by a layperson makes it possible to defibrillate many minutes before professional help arrives.

Sequence for use of an AED

The ERC AED algorithm is shown in Fig. 1.4.

1. Make sure you, the victim, and any bystanders are safe.
2. Follow the Adult BLS sequence:

- if the victim is unresponsive and not breathing normally, send someone for help and to find and bring an AED if available;
 - if you are on your own, use your mobile phone to alert the ambulance service – leave the victim only when there is no other option.
3. Start CPR according to the adult BLS sequence. If you are on your own and the AED is in your immediate vicinity, start with applying the AED.
 4. As soon as the AED arrives:
 - switch on the AED and attach the electrode pads on the victim’s bare chest;
 - if more than one rescuer is present, CPR should be continued while electrode pads are being attached to the chest;
 - follow the spoken/visual directions immediately;
 - ensure that nobody is touching the victim while the AED is analysing the rhythm.
 - 5a. If a shock is indicated:
 - ensure that nobody is touching the victim;
 - push shock button as directed;
 - immediately restart CPR 30:2;
 - continue as directed by the voice/visual prompts.
 - 5b. If no shock is indicated:
 - immediately resume CPR, using a ratio of 30 compressions to 2 rescue breaths;
 - continue as directed by the voice/visual prompts.
 6. Continue to follow the AED prompts until:
 - professional help arrives and takes over;
 - the victim starts to wake up: moves, opens eyes and breathes normally;
 - you become exhausted.

Public access defibrillation programmes

Automated external defibrillator programmes should be actively considered for implementation in public places such as airports,⁵² sport facilities, offices, in casinos⁵⁵ and on aircraft,⁵³ where cardiac arrests are usually witnessed and trained rescuers are quickly on scene. Lay rescuer AED programmes with very rapid response times, and uncontrolled studies using police officers as first responders,^{97,98} have achieved reported survival rates as high as 49–74%.

Automated External Defibrillation Algorithm

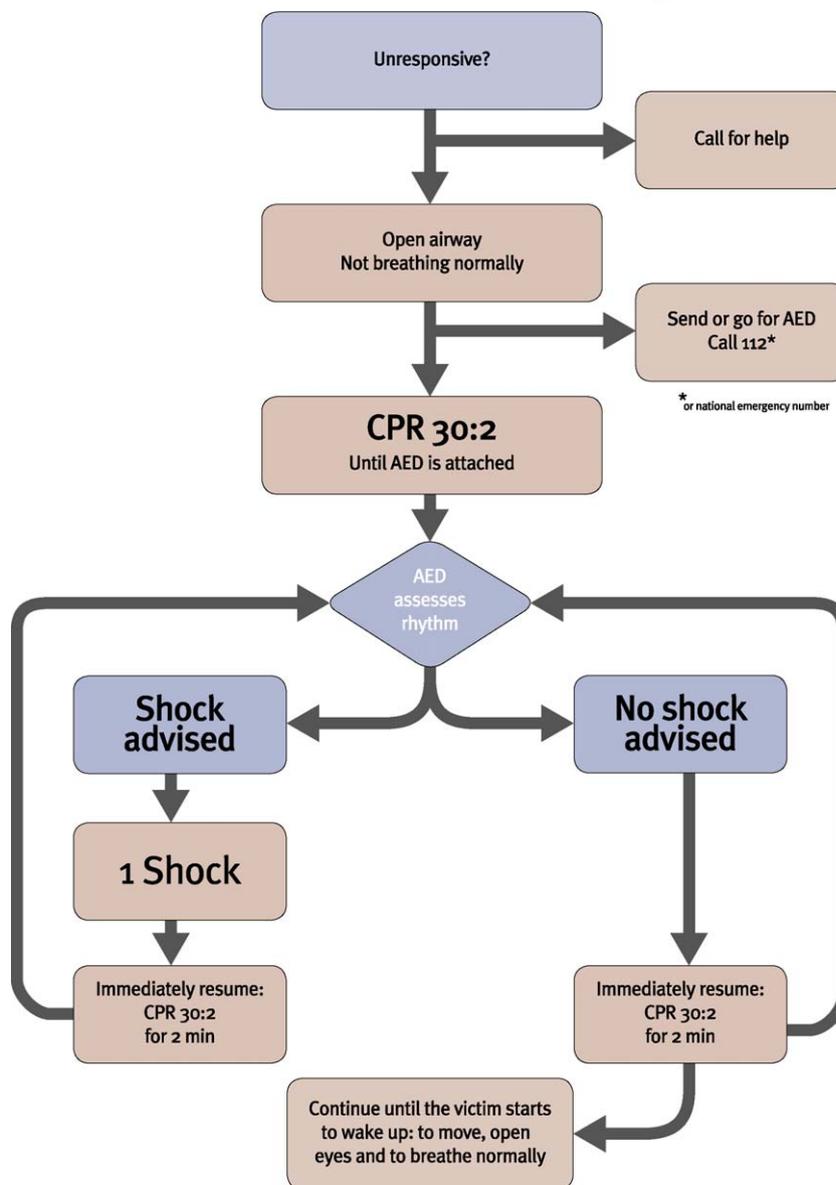


Fig. 1.4. AED algorithm. © 2010 ERC.

The full potential of AEDs has not yet been achieved, because they are used mostly in public settings, yet 60–80% of cardiac arrests occur at home. Public access defibrillation (PAD) and first responder AED programmes may increase the number of victims who receive bystander CPR and early defibrillation, thus improving survival from out-of-hospital SCA.⁹⁹ Recent data from nationwide studies in Japan and the USA^{33,100} showed that when an AED was available, victims were defibrillated much sooner and with a better chance of survival. Programmes that make AEDs publicly available in residential areas have not yet been evaluated. The acquisition of an AED for individual use at home, even for those considered at high risk of sudden cardiac arrest, has proved not to be effective.¹⁰¹

In-hospital use of AEDs

At the time of the 2010 Consensus on CPR Science Conference, there were no published randomised trials comparing in-hospital use of AEDs with manual defibrillators. Two lower-level studies of adults with in-hospital cardiac arrest from shockable rhythms

showed higher survival-to-hospital discharge rates when defibrillation was provided through an AED programme than with manual defibrillation alone.^{102,103} Despite limited evidence, AEDs should be considered for the hospital setting as a way to facilitate early defibrillation (a goal of <3 min from collapse), especially in areas where healthcare providers have no rhythm recognition skills or where they use defibrillators infrequently. An effective system for training and retraining should be in place.¹⁰⁴ Enough healthcare providers should be trained to enable the first shock to be given within 3 min of collapse anywhere in the hospital. Hospitals should monitor collapse-to-first shock intervals and monitor resuscitation outcomes.

Shock in manual versus semi-automatic mode

Many AEDs can be operated in both manual and semi-automatic mode but few studies have compared these two options. The semi-automatic mode has been shown to reduce time to first shock when used both in-hospital¹⁰⁵ and pre-hospital¹⁰⁶ settings, and results

in higher VF conversion rates,¹⁰⁶ and delivery of fewer inappropriate shocks.¹⁰⁷ Conversely, semi-automatic modes result in less time spent performing chest compressions,^{107,108} mainly because of a longer pre-shock pause associated with automated rhythm analysis. Despite these differences, no overall difference in ROSC, survival, or discharge rate from hospital has been demonstrated in any study.^{105,106,109} The defibrillation mode that affords the best outcome will depend on the system, skills, training and ECG recognition skills of rescuers. A shorter pre-shock pause and lower total hands-off-ratio increases vital organ perfusion and the probability of ROSC.^{71,110,111} With manual defibrillators and some AEDs it is possible to perform chest compressions during charging and thereby reduce the pre-shock pause to less than 5 s. Trained individuals may deliver defibrillation in manual mode but frequent team training and ECG recognition skills are essential.

Strategies before defibrillation

Minimising the pre-shock pause

The delay between stopping chest compressions and delivery of the shock (the pre-shock pause) must be kept to an absolute minimum; even 5–10 s delay will reduce the chances of the shock being successful.^{71,110,112} The pre-shock pause can easily be reduced to less than 5 s by continuing compressions during charging of the defibrillator and by having an efficient team coordinated by a leader who communicates effectively. The safety check to ensure that nobody is in contact with the patient at the moment of defibrillation should be undertaken rapidly but efficiently. The negligible risk of a rescuer receiving an accidental shock is minimised even further if all rescuers wear gloves.¹¹³ The post-shock pause is minimised by resuming chest compressions immediately after shock delivery (see below). The entire process of defibrillation should be achievable with no more than a 5 s interruption to chest compressions.

Pads versus paddles

Self-adhesive defibrillation pads have practical benefits over paddles for routine monitoring and defibrillation.^{114–118} They are safe and effective and are preferable to standard defibrillation paddles.¹¹⁹

Fibrillation waveform analysis

It is possible to predict, with varying reliability, the success of defibrillation from the fibrillation waveform.^{120–139} If optimal defibrillation waveforms and the optimal timing of shock delivery can be determined in prospective studies, it should be possible to prevent the delivery of unsuccessful high energy shocks and minimise myocardial injury. This technology is under active development and investigation but current sensitivity and specificity is insufficient to enable introduction of VF waveform analysis into clinical practice.

CPR before defibrillation

Several studies have examined whether a period of CPR prior to defibrillation is beneficial, particularly in patients with an unwitnessed arrest or prolonged collapse without resuscitation. A review of evidence for the 2005 guidelines resulted in the recommendation that it was reasonable for EMS personnel to give a period of about 2 min of CPR before defibrillation in patients with prolonged collapse (>5 min).¹⁴⁰ This recommendation was based on clinical studies, which showed that when response times

exceeded 4–5 min, a period of 1.5–3 min of CPR before shock delivery improved ROSC, survival to hospital discharge^{141,142} and 1 year survival¹⁴² for adults with out-of-hospital VF or VT compared with immediate defibrillation.

More recently, two randomised controlled trials documented that a period of 1.5–3 min of CPR by EMS personnel before defibrillation did not improve ROSC or survival to hospital discharge in patients with out-of-hospital VF or pulseless VT, regardless of EMS response interval.^{143,144} Four other studies have also failed to demonstrate significant improvements in overall ROSC or survival to hospital discharge with an initial period of CPR,^{141,142,145,146} although one did show a higher rate of favourable neurological outcome at 30 days and 1 year after cardiac arrest.¹⁴⁵ Performing chest compressions while retrieving and charging a defibrillator has been shown to improve the probability of survival.¹⁴⁷

In any cardiac arrest they have not witnessed, EMS personnel should provide good-quality CPR while a defibrillator is retrieved, applied and charged, but routine delivery of a 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 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.

Delivery of defibrillation

One shock versus three-stacked shock sequence

Interruptions in external chest compression reduces the chances of converting VF to another rhythm.⁷¹ Studies have shown a significantly lower hands-off-ratio with a one-shock instead of a three-stacked shock protocol¹⁴⁸ and some,^{149–151} but not all,^{148,152} have suggested a significant survival benefit from this single-shock strategy.

When defibrillation is warranted, give a single shock and resume chest compressions immediately following the shock. Do not delay CPR for rhythm analysis or a pulse check immediately after a shock. Continue CPR (30 compressions:2 ventilations) for 2 min until rhythm analysis is undertaken and another shock given (if indicated) (see *Advanced life support*).⁶

If VF/VT occurs during cardiac catheterisation or in the early post-operative period following cardiac surgery (when chest compressions could disrupt vascular sutures), consider delivering up to three-stacked shocks before starting chest compressions (see *Special circumstances*).¹⁰ 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.

Waveforms

Monophasic defibrillators are no longer manufactured, and although many will remain in use for several years, biphasic defibrillators have now superseded them.

Monophasic versus biphasic defibrillation

Although biphasic waveforms are more effective at terminating ventricular arrhythmias at lower energy levels, have demonstrated greater first shock efficacy than monophasic waveforms, and have greater first shock efficacy for long duration VF/VT.^{153–155} No randomised studies have demonstrated superiority in terms of

neurologically intact survival to hospital discharge. Biphasic waveforms have been shown to be superior to monophasic waveforms for elective cardioversion of atrial fibrillation, with greater overall success rates, using less cumulative energy and reducing the severity of cutaneous burns,^{156–159} and are the waveform of choice for this procedure.

Energy levels

Optimal energy levels for both monophasic and biphasic waveforms are unknown. The recommendations for energy levels are based on a consensus following careful review of the current literature.

First shock

There are no new published studies looking at the optimal energy levels for monophasic waveforms since publication of the 2005 guidelines. Relatively few studies on biphasic waveforms have been published in the past 5 years on which to refine the 2005 guidelines. There is no evidence that one biphasic waveform or device is more effective than another. First shock efficacy of the biphasic truncated exponential (BTE) waveform using 150–200 J has been reported as 86–98%.^{153,154,160–162} First shock efficacy of the rectilinear biphasic (RLB) waveform using 120 J is up to 85% (data not published in the paper but supplied by personnel communication).¹⁵⁵ Two studies have suggested equivalence with lower and higher starting energy biphasic defibrillation.^{163,164} Although human studies have not shown harm (raised biomarkers, ECG changes, ejection fraction) from any biphasic waveform up to 360 J,^{163,165} several animal studies have suggested the potential for harm with higher energy levels.^{166–169}

The initial biphasic shock should be no lower than 120 J for RLB waveforms and 150 J for BTE waveforms. Ideally, the initial biphasic shock energy should be at least 150 J for all waveforms.

Second and subsequent shocks

The 2005 guidelines recommended either a fixed or escalating energy strategy for defibrillation and there is no evidence to change this recommendation.

Cardioversion

If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave of the electrocardiogram rather than with the T wave: VF can be induced if a shock is delivered during the relative refractory portion of the cardiac cycle.¹⁷⁰ Biphasic waveforms are more effective than monophasic waveforms for cardioversion of AF.^{156–159} Commencing at high energy levels does not improve cardioversion rates compared with lower energy levels.^{156,171–176} An initial synchronised shock of 120–150 J, escalating if necessary is a reasonable strategy based on current data. Atrial flutter and paroxysmal SVT generally require less energy than atrial fibrillation for cardioversion.¹⁷⁵ Give an initial shock of 100 J monophasic or 70–120 J biphasic. Give subsequent shocks using stepwise increases in energy.¹⁷⁷ The energy required for cardioversion of VT depends on the morphological characteristics and rate of the arrhythmia.¹⁷⁸ Use biphasic energy levels of 120–150 J for the initial shock. Consider stepwise increases if the first shock fails to achieve sinus rhythm.¹⁷⁸

Pacing

Consider pacing in patients with symptomatic bradycardia refractory to anti-cholinergic drugs or other second line therapy

(see *Advanced life support*).⁶ Immediate pacing is indicated especially when the block is at or below the His-Purkinje level. If transthoracic pacing is ineffective, consider transvenous pacing.

Implantable cardioverter defibrillators

Implantable cardioverter defibrillators (ICDs) are implanted because a patient is considered to be at risk from, or has had, a life-threatening shockable arrhythmia. On sensing a shockable rhythm, an ICD will discharge approximately 40 J through an internal pacing wire embedded in the right ventricle. On detecting VF/VT, ICD devices will discharge no more than eight times, but may reset if they detect a new period of VF/VT. Discharge of an ICD may cause pectoral muscle contraction in the patient, and shocks to the rescuer have been documented.¹⁷⁹ In view of the low energy levels discharged by ICDs, it is unlikely that any harm will come to the rescuer, but the wearing of gloves and minimising contact with the patient while the device is discharging is prudent.

Adult advanced life support

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.¹⁸⁰ Once cardiac arrest occurs, fewer than 20% of patients having an in-hospital cardiac arrest will survive to go home.^{36,181,182} 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.¹⁸³

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.¹⁸⁴ These patients often have slow and progressive physiological deterioration, involving hypoxaemia and hypotension that is unnoticed by staff, or is recognised but treated poorly.^{185–187} Many of these patients have unmonitored arrests, and the underlying cardiac arrest rhythm is usually non-shockable^{182,188}; survival to hospital discharge is poor.^{36,181,188}

Education in acute care

Staff education is an essential part of implementing a system to prevent cardiac arrest.¹⁸⁹ 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.^{190,191}

Monitoring and recognition of the critically ill patient

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.¹⁹² 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 ('track and trigger').^{193–197}

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).^{198–200} 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 based predominantly on individual or teams of nurses.²⁰¹ 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.²⁰² Medical emergency teams have an important role in improving end-of-life and do-not-attempt resuscitation (DNAR) decision-making, which at least partly accounts for the reduction in cardiac arrest rates.^{203–206}

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.¹⁸³ 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 SpO₂.^{192,207}
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 (e.g., SBAR – Situation-Background-Assessment-Recommendation)²⁰⁸ 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.²⁰⁹ 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.^{210–218}

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. 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.²³ 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.²¹⁹ The rule recommends termination when there is no ROSC, no shocks are administered, and the arrest is not witnessed by EMS personnel. 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 before implementation.

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 CPR. For all in-hospital cardiac arrests, ensure that:

- cardiorespiratory arrest is recognised immediately;
- help is summoned using a standard telephone number;

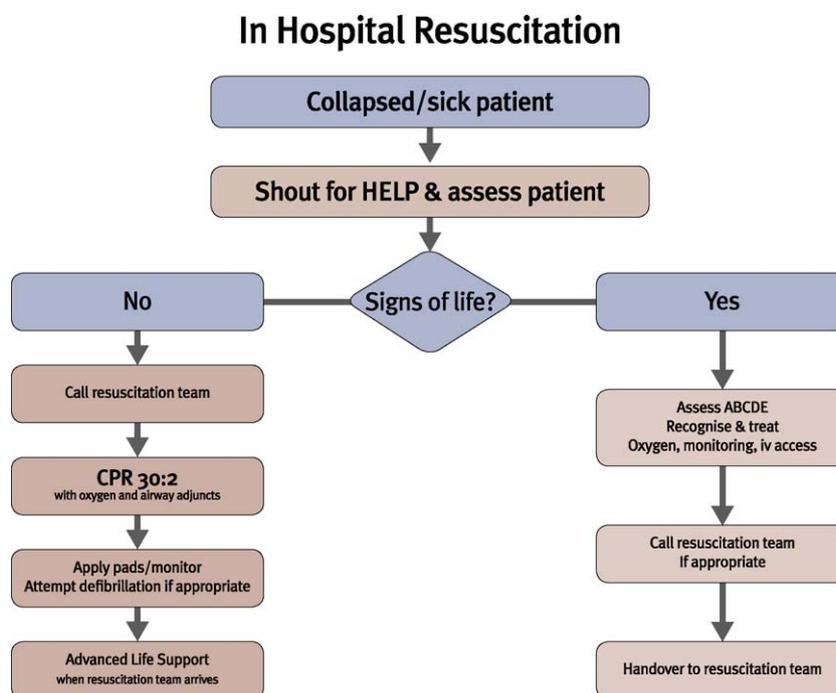


Fig. 1.5. Algorithm for the initial management of in-hospital cardiac arrest. © 2010 ERC.

- CPR is started immediately using airway adjuncts if indicated, defibrillation attempted as rapidly as possible and certainly within 3 min.

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.^{220,221}

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.

An algorithm for the initial management of in-hospital cardiac arrest is shown in Fig. 1.5.

- 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.
- 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.²²² 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 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.
- 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).^{208,223} Locate the patient's records.

Advanced Life Support

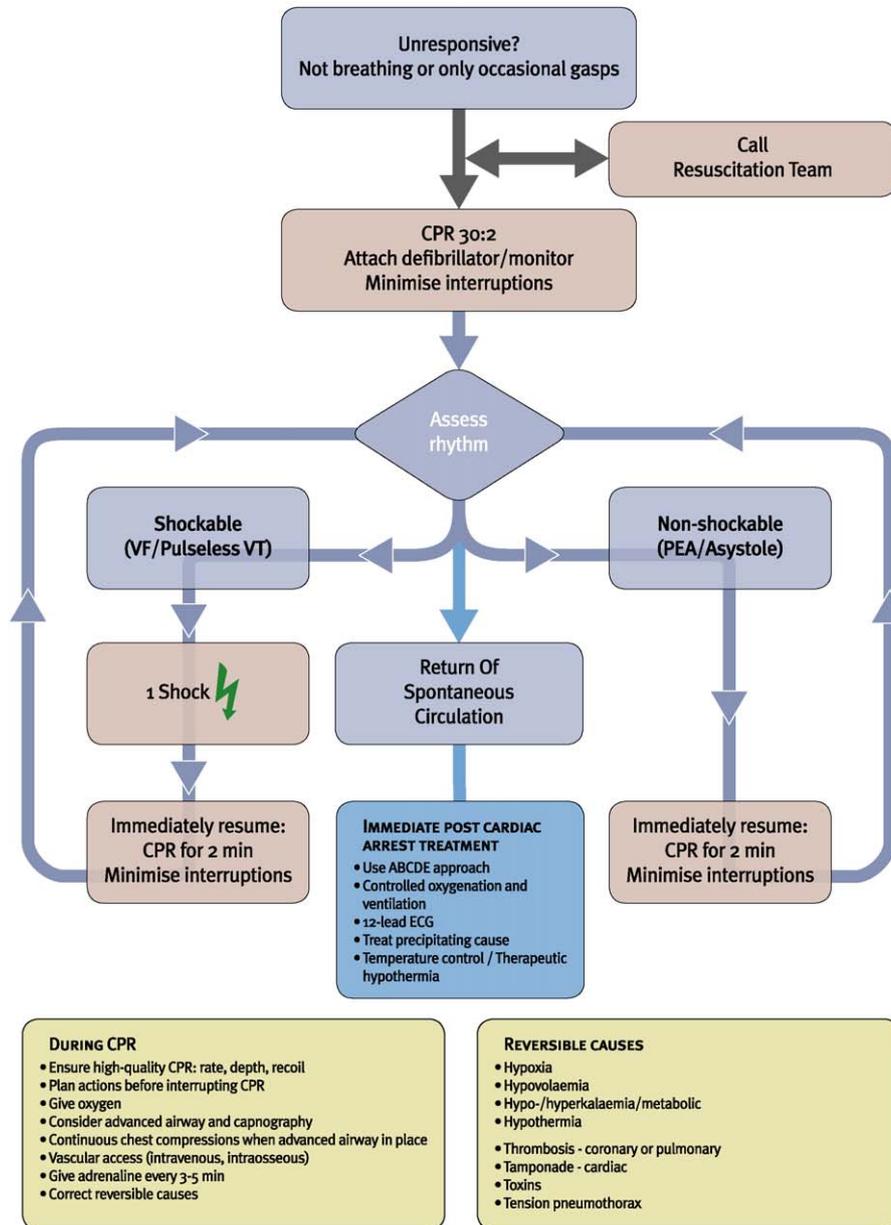


Fig. 1.6. ALS cardiac arrest algorithm. © 2010 ERC.

- The quality of chest compressions during in-hospital CPR is frequently sub-optimal.^{224,225} 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. The team leader should monitor the quality of CPR and alternate CPR providers if the quality of CPR is poor. Continuous ETCO₂ monitoring can be used to indicate the quality of CPR: although an optimal target for ETCO₂ 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 changed every 2 min, but without causing long pauses in chest compressions.

ALS treatment algorithm

Although the ALS cardiac arrest algorithm (Fig. 1.6) is applicable to all cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances (see Section 8).¹⁰

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander BLS, uninterrupted, high-quality chest compressions and early defibrillation for VF/VT. The use of adrenaline has been shown to increase ROSC, but no resuscitation drugs or advanced airway interventions have been shown to increase survival to hospital discharge after cardiac arrest.^{226–229} 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.

Shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia)

The first monitored rhythm is VF/VT in approximately 25% of cardiac arrests, both in-³⁶ or out-of-hospital.^{24,25,146} VF/VT will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA.³⁶ Having confirmed cardiac arrest, summon help (including the request for a defibrillator) and start CPR, beginning with chest compressions, with a 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 pre-shock pause); even 5–10 s delay will reduce the chances of the shock being successful.^{71,110}
- 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 established²³⁰ and it is very rare for a pulse to be palpable immediately after defibrillation.²³¹ Furthermore, the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.²³²
- 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.²³³ 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,²³⁴ 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 chest compressions and may be a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. Two prospective human studies have

shown that a significant increase in end-tidal CO₂ occurs when return of spontaneous circulation occurs.^{235,236}

- 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.

Regardless of the arrest rhythm, give further doses of 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.

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm^{237–239} and is likely to succeed only if given within the first few seconds of the onset of a shockable rhythm.²⁴⁰ 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.²⁴¹ In practice, this is only likely to be in a critical care environment such as the emergency department or ICU.²³⁹

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 through 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 hyper-ventilate the patient. Once the patient's trachea has been intubated, continue chest compressions, at a rate of 100 min⁻¹ without pausing during ventilation.

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).

Intravascular access

Establish intravenous access if this has not already been achieved. Peripheral venous cannulation is quicker, easier to perform and safer than central venous cannulation. Drugs injected peripherally must be followed by a flush of at least 20 ml of fluid. If intravenous access is difficult or impossible, consider the IO route. Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a central venous catheter.²⁴² The recent availability of mechanical IO devices has increased the ease of performing this technique.²⁴³

Unpredictable plasma concentrations are achieved when drugs are given via a tracheal tube, and the optimal tracheal dose of most drugs is unknown, thus, the tracheal route for drug delivery is no longer recommended.

Drugs

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. 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.^{227,228} 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. 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. 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 placebo²⁴⁴ and lidocaine,²⁴⁵ the use of amiodarone in shock-refractory VF improves the short-term outcome of survival to hospital admission. 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.^{246–250} 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 ROSC is not recommended. Give sodium bicarbonate (50 mmol) if cardiac arrest is associated with hyperkalaemia or tricyclic antidepressant overdose; repeat the dose according to the clinical condition and the result of serial blood gas analysis.

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. 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.

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.

Atropine

Asystole during cardiac arrest is usually caused by 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^{226,251–256}; and its routine use for asystole or PEA is no longer recommended.

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 8.¹⁰

Fibrinolysis during CPR

Fibrinolytic therapy should not be used routinely in cardiac arrest.²⁵⁷ 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.^{258,259} 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. Whether fluids should be infused routinely during primary cardiac arrest is controversial. Ensure normovolaemia, but in the absence of hypovolaemia, infusion of an excessive volume of fluid is likely to be harmful.²⁶⁰

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, aortic dissection, hypovolaemia, pneumothorax).^{261–268} 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.^{261,267,269} 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^{270–272} although sensitivity and specificity has not been reported.

Airway management and ventilation

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. 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.

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.

During CPR, give oxygen whenever it is available. There are no data to indicate the optimal arterial blood oxygen saturation (SaO₂) during CPR. There are animal data²⁷³ and some observational clinical data indicating an association between high SaO₂ after ROSC and worse outcome.²⁷⁴ Initially, give the highest possible oxygen concentration. As soon as the arterial blood oxygen saturation can be measured reliably, by pulse oximeter (SpO₂) or arterial blood gas analysis, titrate the inspired oxygen concentration to achieve an arterial blood oxygen saturation in the range of 94–98%.

Alternative airway devices versus 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. There is evidence that, without adequate training and experience, the incidence of complications, is unacceptably high.²⁷⁵ In patients with out-of-hospital cardiac arrest the reliably documented incidence of unrecognised oesophageal intubation ranges from 0.5% to 17%: emergency physicians – 0.5%²⁷⁶; paramedics – 2.4%,²⁷⁷ 6%,^{278,279} 9%,²⁸⁰ 17%.²⁸¹ Prolonged attempts at tracheal intubation are harmful; stopping chest compressions during this time will compromise coronary and cerebral perfusion. 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.²⁸² Tracheal intubation attempts accounted for almost 25% of all CPR interruptions. 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. 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. No intubation attempt should interrupt chest compressions for more than 10 s. After intubation, tube placement must be confirmed and the tube secured adequately.

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.²⁸³

Confirmation of correct placement of the tracheal tube

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine use of primary and secondary techniques to confirm correct placement of the tracheal tube should reduce this risk. Primary assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and adequate) and over the epigastrium (breath sounds should not be heard). Clinical signs of correct tube placement are not completely reliable. Secondary confirmation of tracheal tube placement by an exhaled carbon dioxide or oesophageal detection device should reduce the risk of unrecognised oesophageal intubation but the performance of the available devices varies considerably and all of them should be considered as adjuncts to other confirmatory techniques.²⁸⁴ None of the secondary confirmation techniques will differentiate between a tube placed in a main bronchus and one placed correctly in the trachea.

The accuracy of colorimetric CO₂ 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). 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.

CPR techniques and devices

At best, standard manual CPR produces coronary and cerebral perfusion that is just 30% of normal.²⁸⁵ 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.²⁸⁶ 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,^{287–289} no adjunct has consistently been shown to be superior to conventional manual CPR.

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. 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.²⁹⁰ In the absence of data showing that the ITD increases survival to hospital discharge, its routine use in cardiac arrest is not recommended.

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,^{291,292} there are no published randomised human studies comparing LUCAS-CPR with standard CPR.

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,^{293–295} results of clinical trials have been conflicting. Evidence from one multicentre 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.²⁹⁶ A non-randomised human study reported increased survival to discharge following OHCA.²⁹⁷

The current status of LUCAS and AutoPulse

Two large prospective randomised multicentre studies are currently underway to evaluate the LDB (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)^{298,299} and CT scans³⁰⁰ and also for prolonged resuscitation attempts (e.g., hypothermia,^{301,302} poisoning, thrombolysis for pulmonary embolism, prolonged transport etc) where rescuer fatigue may impair the effectiveness of manual chest compression. In the prehospital 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.^{303,304} 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.

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. These treatment algorithms should enable the non-specialist ALS provider to treat the patient effectively and safely in an emergency. 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.

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, SpO₂). 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⁺, Mg²⁺, Ca²⁺). 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.

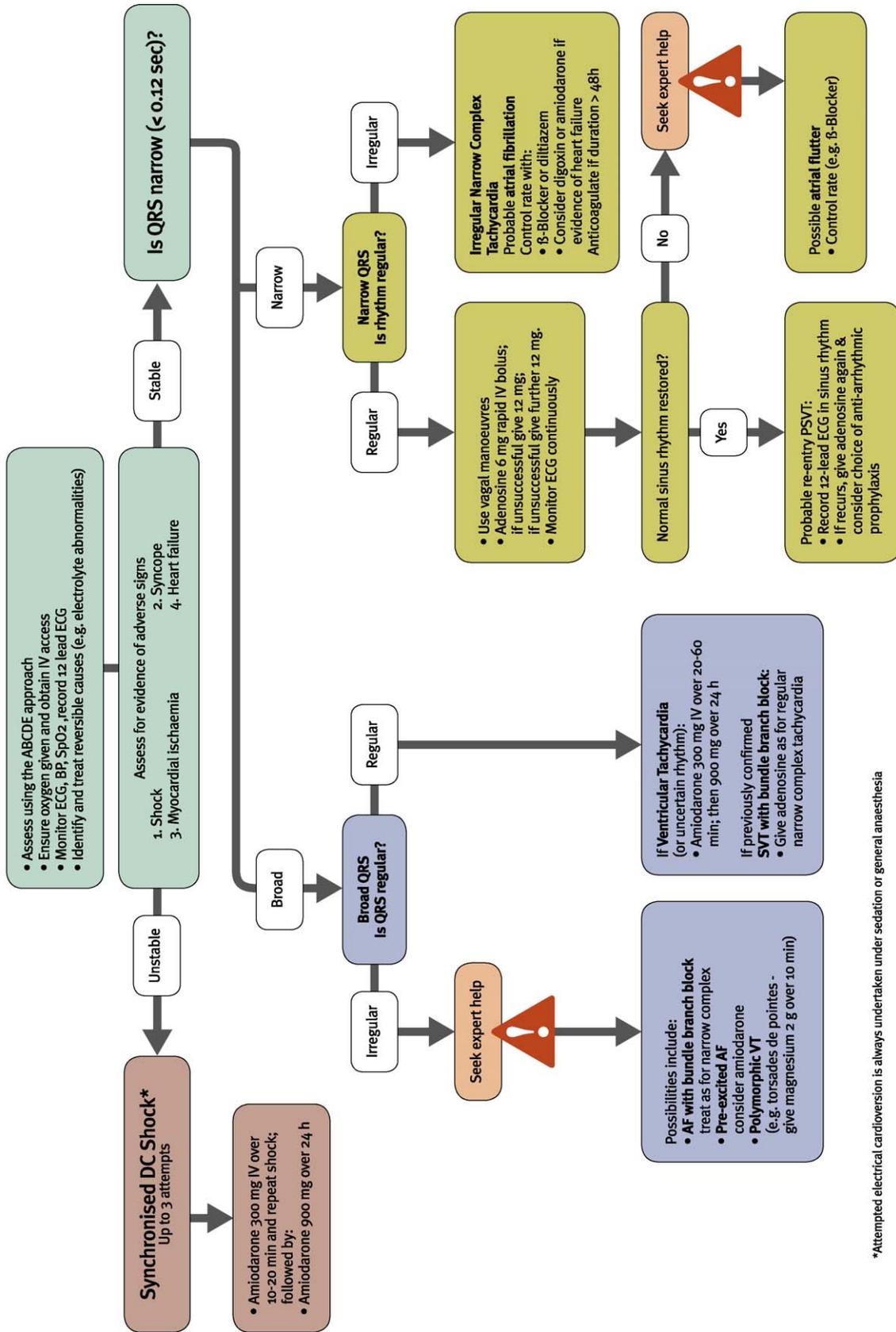
1. Shock – this is seen as pallor, sweating, cold and clammy extremities (increased sympathetic activity), impaired consciousness (reduced cerebral blood flow), and hypotension (e.g., systolic blood pressure <90 mm Hg).
2. Syncope – loss of consciousness, which occurs as a consequence of reduced cerebral 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).

Tachycardia Algorithm (with pulse)



*Attempted electrical cardioversion is always undertaken under sedation or general anaesthesia

Fig. 1.7. Tachycardia algorithm. © 2010 ERC.

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. 1.7). 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.

If the patient is stable

If the patient with tachycardia is stable (no adverse signs or symptoms) and is not deteriorating, drug treatment is likely to be appropriate (Fig. 1.7). Vagal manoeuvres may be appropriate initial treatment for a supraventricular tachycardia.

Bradycardia

A bradycardia is defined as a heart rate of <60 beats min⁻¹. 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

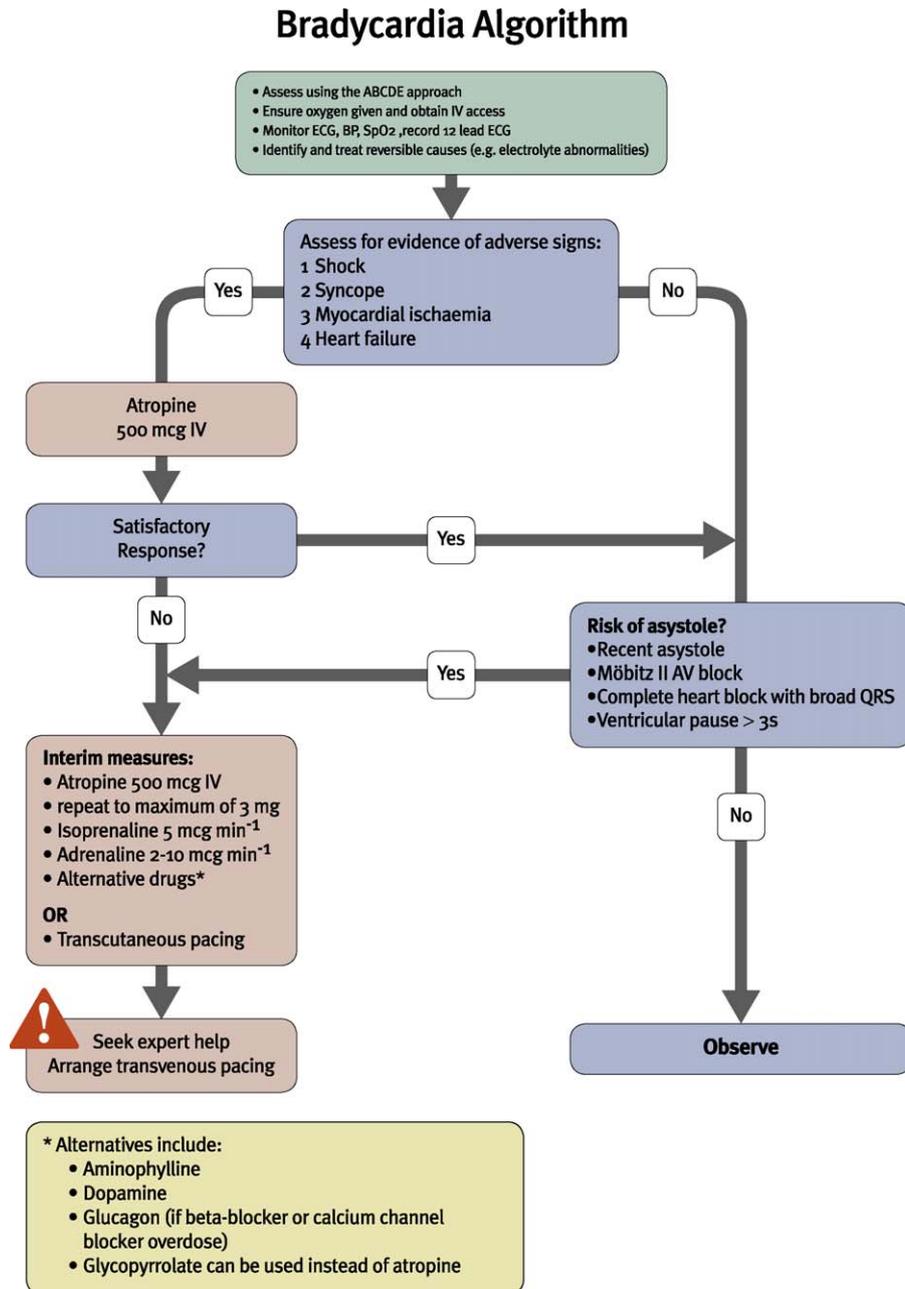


Fig. 1.8. Bradycardia algorithm. © 2010 ERC.

to pharmacological treatments or with risks factors for asystole (Fig. 1.8).

Post-resuscitation care

Successful ROSC is the just the first step toward the goal of complete recovery from cardiac arrest. The post-cardiac arrest syndrome, which comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and the persistent precipitating pathology, often complicates the post-resuscitation phase.³ 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.^{227,305} 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.^{306,307} The whole body ischaemia/reperfusion of cardiac arrest activates immunological and coagulation pathways contributing to multiple organ failure and increasing the risk of infection.^{308,309} Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion and vasodilation.^{310,311}

Airway and breathing

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.^{273,312–315} A clinical registry study documented that post-resuscitation hyperoxaemia was associated with worse outcome, compared with both normoxaemia and hypoxaemia.²⁷⁴ 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. There are no data to support the targeting of a specific arterial PCO₂ after resuscitation from cardiac arrest, but it is reasonable to adjust ventilation to achieve normocarbidaemia and to monitor this using the end-tidal PCO₂ and arterial blood gas values.

Circulation

It is well recognised that post-cardiac arrest patients with 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,³¹⁶ this intervention should be considered in all post-cardiac arrest patients who are suspected of having coronary artery disease.^{316–324} Several studies indicate that the combination of therapeutic hypothermia and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction.^{317,323–326}

Post-cardiac arrest myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias.³⁰⁶ If treatment with fluid resuscitation and vasoactive drugs is insufficient to support the circulation, consider insertion of an intra-aortic balloon pump.^{317,325} 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.³

Disability (optimizing neurological recovery)

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.^{58,327–330} Seizures increase cerebral metabolism by up to 3-fold³³¹ and may cause cerebral injury: treat promptly and effectively with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate. 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.^{58,332–338} 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.³³⁹ 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.^{340–342} Severe hypoglycaemia is associated with increased mortality in critically ill patients,³⁴³ 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.³⁴⁴ Based on the available data, following ROSC blood glucose should be maintained at ≤10 mmol l⁻¹ (180 mg dl⁻¹).³⁴⁵ 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.^{346–348} Several studies document an association between post-cardiac arrest pyrexia and poor outcomes.^{58,346,348–351} There are no randomised controlled trials evaluating the effect of treatment of pyrexia (defined as ≥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.^{352,353} Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen (CMRO₂) by about 6% for each 1 °C reduction in temperature³⁵⁴ and this may reduce the release of excitatory amino acids and free radicals.³⁵² 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.

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 trial³⁵⁵ and a pseudo-randomised trial³⁵⁶ 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. 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.^{317,357–363}

The practical application of therapeutic hypothermia is divided into three phases: induction, maintenance, and rewarming.³⁶⁴ Animal data indicate that earlier cooling after ROSC produces better outcomes.³⁶⁵ 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. Other methods of inducing and/or maintaining hypothermia include: simple ice packs and/or wet towels; cooling blankets or pads; water or air circulating blankets; water circulating gel-coated pads; intravascular heat exchanger; and cardiopulmonary bypass.

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. 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.³⁶²

The well-recognised physiological effects of hypothermia need to be managed carefully.³⁶⁴

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 with²²⁷ and without³⁰⁵ 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.

Clinical examination

There are no clinical neurological signs that predict poor outcome (Cerebral Performance Category [CPC] 3 or 4, or death) reliably 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%).³³⁰ Absence of vestibulo-ocular reflexes at ≥ 24 h (FPR 0%; 95% CI 0–14%)^{366,367} and a GCS motor score of 2 or less at ≥ 72 h (FPR 5%; 95% CI 2–9%)³³⁰ 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,^{329,330,368–370} but rare cases of good neurological recovery have been described and accurate diagnosis is problematic.^{371–375}

Biochemical markers

Evidence does not support the use of serum (e.g., neuronal specific enolase, S100 protein) 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 studied 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%).³⁷⁶

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.¹⁵ There are no high-level studies that support the use of any imaging modality to predict outcome of comatose cardiac arrest survivors.

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%).^{368,377} 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%])³⁶⁸ and the presence of status epilepticus (FPR of 7% [95% CI 1–25%] to 11.5% [95% CI 3–31%])^{378,379} are potentially unreliable prognosticators of poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia. 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.³⁸⁰ 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.³⁸¹ 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.

Cardiac arrest centres

There is wide variability in survival among hospitals caring for patients after resuscitation from cardiac arrest.^{57–63} 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.⁶¹ There is indirect evidence that regional cardiac resuscitation systems of care improve outcome ST elevation myocardial infarction (STEMI).^{382–404}

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.^{405–407}

Initial management of acute coronary syndromes

Introduction

The incidence of acute STEMI is decreasing in many European countries⁴⁰⁸; however, the incidence of non-STEMI acute coronary syndrome (non-STEMI-ACS) is increasing.^{409,410} Although in-hospital mortality from STEMI has been reduced significantly by modern reperfusion therapy and improved secondary prophylaxis, the overall 28-day mortality is virtually unchanged because about two-thirds of those who die do so before hospital arrival, mostly from lethal arrhythmias triggered by ischaemia.⁴¹¹ Thus, the best chance of improving survival from an ischaemic attack is reducing the delay from symptom onset to first medical contact and targeted treatment started in the early out-of-hospital phase.

The term acute coronary syndrome (ACS) encompasses three different entities of the acute manifestation of coronary heart disease: STEMI, NSTEMI and unstable angina pectoris (UAP). Non-ST elevation myocardial infarction and UAP are usually combined in the term non-STEMI-ACS. The common pathophysiology of ACS is a ruptured or eroded atherosclerotic plaque.⁴¹² Electrocardiographic (ECG) characteristics (absence or presence of ST elevation) differentiate STEMI from NSTEMI-ACS. The latter may present with ST segment depression, nonspecific ST segment wave abnormalities, or even a normal ECG. In the absence of ST elevation, an increase in the plasma concentration of cardiac biomarkers, particularly troponin T or I as the most specific markers of myocardial cell necrosis, indicates NSTEMI.

Acute coronary syndromes are the commonest cause of malignant arrhythmias leading to sudden cardiac death. The therapeutic goals are to treat acute life-threatening conditions, such as VF or extreme bradycardia, and to preserve left ventricular function and prevent heart failure by minimising the extent of myocardial damage. The current guidelines address the first hours after onset of symptoms. Out-of-hospital treatment and initial therapy in the emergency department (ED) may vary according to local capabilities, resources and regulations. The data supporting out-of-hospital treatment are often extrapolated from studies of initial treatment after hospital admission; there are few high-quality out-of-hospital studies. Comprehensive guidelines for the diagnosis and treatment of ACS with and without ST elevation have been published by the European Society of Cardiology and the American College of Cardiology/American Heart Association. The current recommendations are in line with these guidelines (Figs. 1.9 and 1.10).^{413,414}

Diagnosis and risk stratification in acute coronary syndromes

Patients at risk, and their families, should be able to recognize characteristic symptoms such as chest pain, which may radiate into other areas of the upper body, often accompanied by other symptoms including dyspnoea, sweating, nausea or vomiting and syncope. They should understand the importance of early activation of the EMS system and, ideally, should be trained in basic life support (BLS). Optimal strategies for increasing layperson awareness of the various ACS presentations and improvement of ACS recognition in vulnerable populations remain to be determined. Moreover, EMS dispatchers must be trained to recognize ACS symptoms and to ask targeted questions.

Signs and symptoms of ACS

Typically ACS appears with symptoms such as radiating chest pain, shortness of breath and sweating; however, atypical symptoms or unusual presentations may occur in the elderly, in females, and in diabetics.^{415,416} None of these signs and symptoms of ACS can be used alone for the diagnosis of ACS.

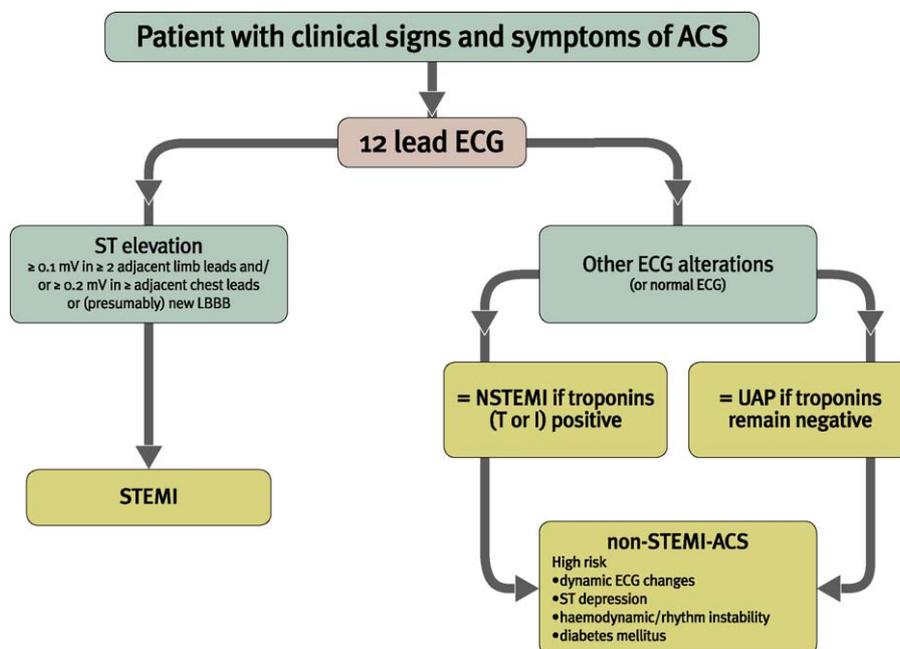


Fig. 1.9. Definitions of acute coronary syndromes (ACS); STEMI, ST elevation myocardial infarction; NSTEMI, non-ST elevation myocardial infarction; UAP, unstable angina pectoris.

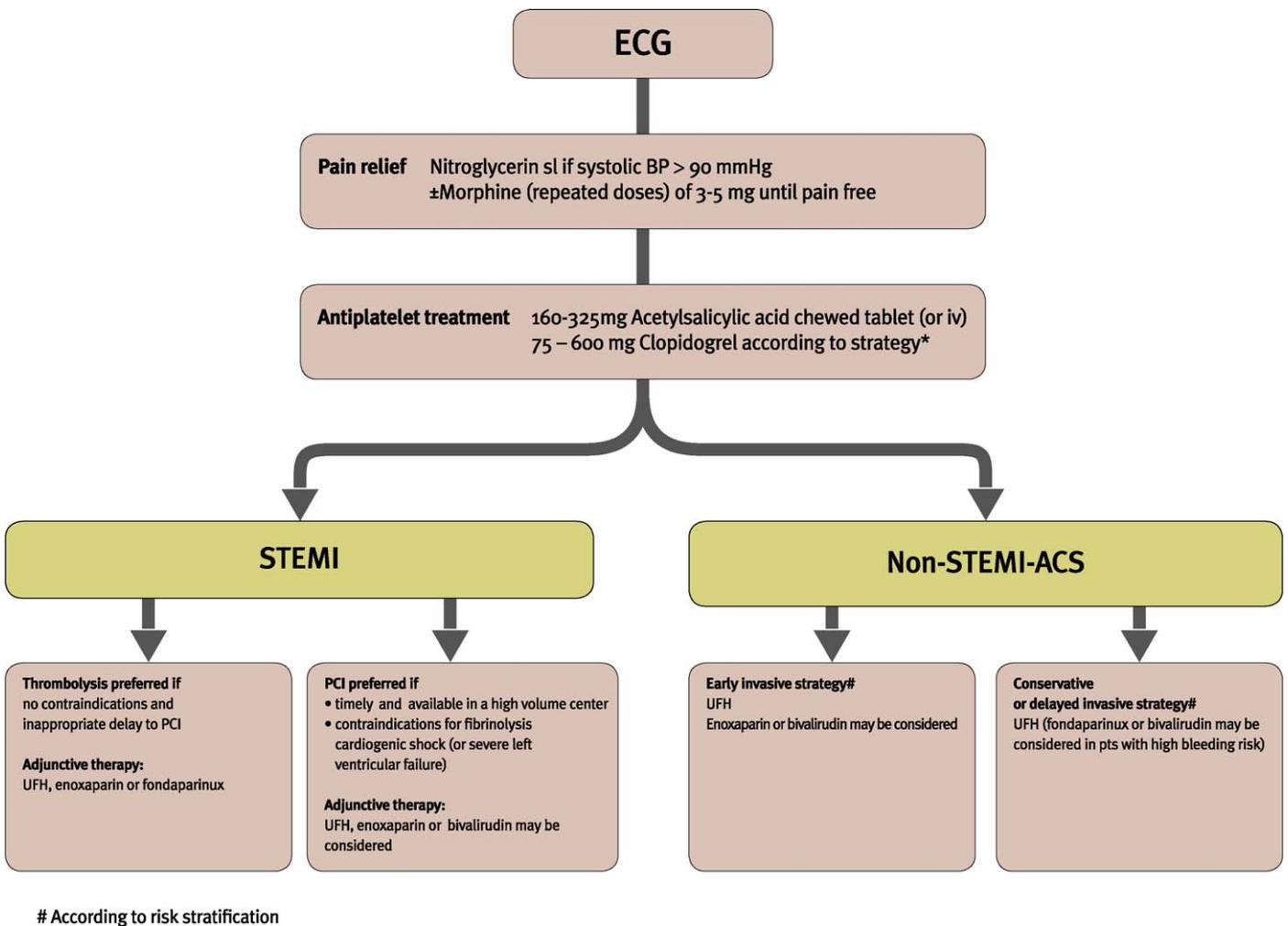


Fig. 1.10. Treatment algorithm for acute coronary syndromes; BP, blood pressure; PCI, percutaneous coronary intervention; UFH, unfractionated heparin. *Prasugrel, 60 mg loading dose, may be chosen as an alternative to clopidogrel in patients with STEMI and planned PPCI provided there is no history of stroke or transient ischaemic attack. At the time of writing, ticagrelor has not yet been approved as an alternative to clopidogrel.

12-lead ECG

A 12-lead ECG is the key investigation for assessment of an ACS. In case of STEMI, it indicates the need for immediate reperfusion therapy (i.e., primary percutaneous coronary intervention (PCI) or prehospital fibrinolysis). When an ACS is suspected, a 12-lead ECG should be acquired and interpreted as soon as possible after first patient contact, to facilitate earlier diagnosis and triage. Prehospital or ED ECG yields useful diagnostic information when interpreted by trained health care providers.⁴¹⁷

Recording of a 12-lead ECG out-of-hospital enables advanced notification to the receiving facility and expedites treatment decisions after hospital arrival. Paramedics and nurses can be trained to diagnose STEMI without direct medical consultation, as long as there is strict concurrent provision of medically directed quality assurance. If interpretation of the prehospital ECG is not available on-site, computer interpretation^{418,419} or field transmission of the ECG is reasonable.

Biomarkers

In the absence of ST elevation on the ECG, the presence of a suggestive history and elevated concentrations of biomarkers (troponin T and troponin I, CK, CK-MB, myoglobin) characterise non-STEMI and distinguish it from STEMI and unstable angina

respectively. Measurement of a cardiac-specific troponin is preferable. Elevated concentrations of troponin are particularly helpful in identifying patients at increased risk of adverse outcome.⁴²⁰

Decision rules for early discharge

Attempts have been made to combine evidence from history, physical examination serial ECGs and serial biomarker measurement in order to form clinical decision rules that would help triage of ED patients with suspected ACS.

None of these rules is adequate and appropriate to identify ED chest pain patients with suspected ACS who can be safely discharged from the ED.⁴²¹

Chest pain observation protocols

In patients presenting to the ED with a history suggestive of ACS, but normal initial workup, chest pain (observation) units may represent a safe and effective strategy for evaluating patients. They reduce length of stay, hospital admissions and health-care costs, improve diagnostic accuracy and improve quality of life.⁴²² There is no direct evidence demonstrating that chest pain units or observation protocols reduce adverse cardiovascular outcomes, particularly mortality, for patients presenting with possible ACS.

Treatment of acute coronary syndromes—symptoms

Glyceryl trinitrate is an effective treatment for ischaemic chest pain and has beneficial haemodynamic effects, such as dilation of the venous capacitance vessels, dilation of the coronary arteries and, to a minor extent, the peripheral arteries. Glyceryl trinitrate may be considered if the systolic blood pressure is above 90 mm Hg and the patient has ongoing ischaemic chest pain. Glyceryl trinitrate can also be useful in the treatment of acute pulmonary congestion. Nitrates should not be used in patients with hypotension (systolic blood pressure \leq 90 mm Hg), particularly if combined with bradycardia, and in patients with inferior infarction and suspected right ventricular involvement. Use of nitrates under these circumstances can decrease the blood pressure and cardiac output.

Morphine is the analgesic of choice for nitrate-refractory pain and also has calming effects on the patient making sedatives unnecessary in most cases. Since morphine is a dilator of venous capacitance vessels, it may have additional benefit in patients with pulmonary congestion. Give morphine in initial doses of 3–5 mg intravenously and repeat every few minutes until the patient is pain-free. Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided for analgesia because of their pro-thrombotic effects.⁴²³

Monitoring of the arterial oxygen saturation (SaO₂) with pulse oximetry will help to determine the need for supplemental oxygen. These patients do not need supplemental oxygen unless they are hypoxaemic. Limited data suggest that high-flow oxygen may be harmful in patients with uncomplicated myocardial infarction.^{424–426} Aim to achieve an oxygen saturation of 94–98%, or 88–92% if the patient is at risk of hypercapnic respiratory failure.⁴²⁷

Treatment of acute coronary syndromes—cause

Inhibitors of platelet aggregation

Inhibition of platelet aggregation is of primary importance for initial treatment of coronary syndromes as well as for secondary prevention, since platelet activation and aggregation is the key process initiating an ACS.

Acetylsalicylic acid (ASA)

Large randomised controlled trials indicate decreased mortality when ASA (75–325 mg) is given to hospitalised patients with ACS. A few studies have suggested reduced mortality if ASA is given earlier.^{428,429} Therefore, give ASA as soon as possible to all patients with suspected ACS unless the patient has a known true allergy to ASA. ASA may be given by the first healthcare provider, bystander or by dispatcher assistance according to local protocols. The initial dose of chewable ASA is 160–325 mg. Other forms of ASA (soluble, IV) may be as effective as chewed tablets.

ADP receptor inhibitors

Thienopyridines (clopidogrel, prasugrel) and the cyclo-pentyl-triazolo-pyrimidine, ticagrelor, inhibit the ADP receptor irreversibly, which further reduces platelet aggregation in addition to that produced by ASA.

If given in addition to heparin and ASA in high-risk non-STEMI-ACS patients, clopidogrel improves outcome.^{430,431} Clopidogrel should be given as early as possible in addition to ASA and an antithrombin to all patients presenting with non-STEMI-ACS. If a conservative approach is selected, give a loading dose of 300 mg; with a planned PCI strategy, an initial dose of 600 mg may be preferred. Prasugrel or ticagrelor can be given instead of clopidogrel.

Although there is no large study on the use of clopidogrel for pre-treatment of patients presenting with STEMI and planned PCI, it is likely that this strategy is beneficial. Since platelet inhibition is more profound with a higher dose, a 600 mg loading dose given

as soon as possible is recommended for patients presenting with STEMI and planned PCI. Prasugrel or ticagrelor can be used instead of clopidogrel before planned PCI. Patients with STEMI treated with fibrinolysis should be treated with clopidogrel (300 mg loading dose up to an age of 75 years and 75 mg without loading dose if >75 years of age) in addition to ASA and an antithrombin.

Glycoprotein (Gp) IIB/IIIa inhibitors

Gp IIB/IIIa receptor is the common final link of platelet aggregation. Eptifibatid and tirofiban lead to reversible inhibition, while abciximab leads to irreversible inhibition of the Gp IIB/IIIa receptor. There are insufficient data to support routine pre-treatment with Gp IIB/IIIa inhibitors in patients with STEMI or non-STEMI-ACS.

Antithrombins

Unfractionated heparin (UFH) is an indirect inhibitor of thrombin, which in combination with ASA is used as an adjunct with fibrinolytic therapy or primary PCI (PPCI) and is an important part of treatment of unstable angina and STEMI. There are now several alternative antithrombins for the treatment of patients with ACS. In comparison with UFH, these alternatives have a more specific factor Xa activity (low molecular weight heparins [LMWH], fondaparinux) or are direct thrombin inhibitors (bivalirudin). With these newer antithrombins, in general, there is no need to monitor the coagulation system and there is a reduced risk of thrombocytopenia.

In comparison with UFH, enoxaparin reduces the combined endpoint of mortality, myocardial infarction and the need for urgent revascularisation, if given within the first 24–36 h of onset of symptoms of non-STEMI-ACS.^{432,433} For patients with a planned initial conservative approach, fondaparinux and enoxaparin are reasonable alternatives to UFH. For patients with an increased bleeding risk consider giving fondaparinux or bivalirudin, which cause less bleeding than UFH.^{434–436} For patients with a planned invasive approach, enoxaparin or bivalirudin are reasonable alternatives to UFH.

Several randomised studies of patients with STEMI undergoing fibrinolysis have shown that additional treatment with enoxaparin instead of UFH produced better clinical outcomes (irrespective of the fibrinolytic used) but a slightly increased bleeding rate in elderly (\geq 75 years) and low weight patients (BW < 60 kg).^{437–439}

Enoxaparin is a safe and effective alternative to UFH for contemporary PPCI (i.e., broad use of thienopyridines and/or Gp IIB/IIIa receptor blockers).^{440,441} There are insufficient data to recommend any LMWH other than enoxaparin for PPCI in STEMI. Bivalirudin is also a safe alternative to UFH for STEMI and planned PCI.

Strategies and systems of care

Several systematic strategies to improve quality of out-of-hospital care for patients with ACS have been investigated. These strategies are principally intended to promptly identify patients with STEMI in order to shorten the delay to reperfusion treatment. Also triage criteria have been developed to select high-risk patients with non-STEMI-ACS for transport to tertiary care centres offering 24/7 PCI services. In this context, several specific decisions have to be made during initial care beyond the basic diagnostic steps necessary for clinical evaluation of the patient and interpretation of a 12-lead ECG. These decisions relate to:

- (1) Reperfusion strategy in patients with STEMI i.e., PPCI vs. pre-hospital fibrinolysis.

- (2) Bypassing a closer but non-PCI capable hospital and taking measures to shorten the delay to intervention if PPCI is the chosen strategy.
- (3) Procedures in special situations e.g., for patients successfully resuscitated from non-traumatic cardiac arrest, patients with shock or patients with non-STEMI-ACS who are unstable or have signs of very high risk.

Reperfusion strategy in patients presenting with STEMI

For patients presenting with STEMI within 12 h of symptom onset, reperfusion should be initiated as soon as possible independent of the method chosen.^{414,442–444} Reperfusion may be achieved with fibrinolysis, with PPCI, or a combination of both. Efficacy of reperfusion therapy is profoundly dependent on the duration of symptoms. Fibrinolysis is effective specifically in the first 2–3 h after symptom onset; PPCI is less time sensitive.⁴⁴⁵ Giving fibrinolytics out-of-hospital to patients with STEMI or signs and symptoms of an ACS with presumed new LBBB is beneficial. Fibrinolytic therapy can be given safely by trained paramedics, nurses or physicians using an established protocol.^{446–451} The efficacy is greatest within the first 3 h of the onset of symptoms.⁴⁵² Patients with symptoms of ACS and ECG evidence of STEMI (or presumably new LBBB or true posterior infarction) presenting directly to the ED should be given fibrinolytic therapy as soon as possible unless there is timely access to PPCI. Healthcare professionals who give fibrinolytic therapy must be aware of its contraindications and risks.

Primary percutaneous intervention

Coronary angioplasty with or without stent placement has become the first-line treatment for patients with STEMI, because it has been shown to be superior to fibrinolysis in the combined endpoints of death, stroke and reinfarction in several studies and meta-analyses.^{453,454}

Fibrinolysis versus primary PCI

Several reports and registries comparing fibrinolytic (including pre-hospital administration) therapy with PPCI showed a trend of improved survival if fibrinolytic therapy was initiated within 2 h of onset of symptoms and was combined with rescue or delayed PCI.^{455–457} If PPCI cannot be accomplished within an adequate timeframe, independent of the need for emergent transfer, then immediate fibrinolysis should be considered unless there is a contraindication. For those STEMI patients presenting in shock, primary PCI (or coronary artery bypass surgery) is the preferred reperfusion treatment. Fibrinolysis should be considered only if there is a substantial delay to PCI.

Triage and inter-facility transfer for primary PCI

The risk of death, reinfarction or stroke is reduced if patients with STEMI are transferred promptly from community hospitals to tertiary care facilities for PPCI.^{383,454,458} It is less clear whether immediate fibrinolytic therapy (in- or out-of-hospital) or transfer for PPCI is superior for younger patients presenting with anterior infarction and within a short duration of <2–3 h.⁴⁵⁹ Transfer of STEMI patients for PPCI is reasonable for those presenting more than 3 h but less than 12 h after the onset of symptoms, provided that the transfer can be achieved rapidly.

Combination of fibrinolysis and percutaneous coronary intervention

Fibrinolysis and PCI may be used in a variety of combinations to restore coronary blood flow and myocardial perfusion. There are several ways in which the two therapies can be

combined. Facilitated PCI is PCI performed immediately after fibrinolysis, a pharmaco-invasive strategy refers to PCI performed routinely 3–24 h after fibrinolysis, and rescue PCI is defined as PCI performed for a failed reperfusion (as evidenced by <50% resolution of ST segment elevation at 60–90 min after completion of fibrinolytic treatment). These strategies are distinct from a routine PCI approach where the angiography and intervention is performed several days after successful fibrinolysis. Several studies and meta-analyses demonstrate worse outcome with routine PCI performed immediately or as early as possible after fibrinolysis.^{458,460} Therefore routine facilitated PCI is not recommended even if there may be some specific subgroups of patients that may benefit from this procedure.⁴⁶¹ It is reasonable to perform angiography and PCI when necessary in patients with failed fibrinolysis according to clinical signs and/or insufficient ST-segment resolution.⁴⁶²

In the case of clinically successful fibrinolysis (evidenced by clinical signs and ST-segment resolution >50%), angiography delayed by several hours after fibrinolysis (the ‘pharmaco-invasive’ approach) has been shown to improve outcome. This strategy includes early transfer for angiography and PCI if necessary after fibrinolytic treatment.^{463,464}

Reperfusion after successful CPR

Coronary heart disease is the most frequent cause of out-of-hospital cardiac arrest. Many of these patients will have an acute coronary occlusion with signs of STEMI on the ECG, but cardiac arrest due to ischaemic heart disease can also occur in absence of these findings. In patients with STEMI or new LBBB on ECG following ROSC after out-of-hospital cardiac arrest, immediate angiography and PCI or fibrinolysis should be considered.^{316,321} It is reasonable to perform immediate angiography and PCI in selected patients despite the lack of ST elevation on the ECG or prior clinical findings such as chest pain. It is reasonable to include reperfusion treatment in a standardised post-cardiac arrest protocol as part of a strategy to improve outcome.³¹⁷ Reperfusion treatment should not preclude other therapeutic strategies including therapeutic hypothermia.

Primary and secondary prevention

Preventive interventions in patients presenting with an ACS should be initiated early after hospital admission and should be continued if already in place. Preventive measures improve prognosis by reducing the number of major adverse cardiac events. Prevention with drugs encompasses beta-blockers, angiotensin converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARB) and statins, as well as basic treatment with ASA and, if indicated, thienopyridines.

Paediatric life support

Paediatric basic life support

Sequence of actions

Rescuers who have been taught adult BLS and have no specific knowledge of paediatric resuscitation may use the adult sequence, as outcome is worse if they do nothing. Non-specialists who wish to learn paediatric resuscitation because they have responsibility for children (e.g., teachers, school nurses, lifeguards), should be taught that it is preferable to modify adult BLS and perform five initial breaths followed by approximately one minute of CPR before they go for help (see adult BLS guideline).

Paediatric basic life support

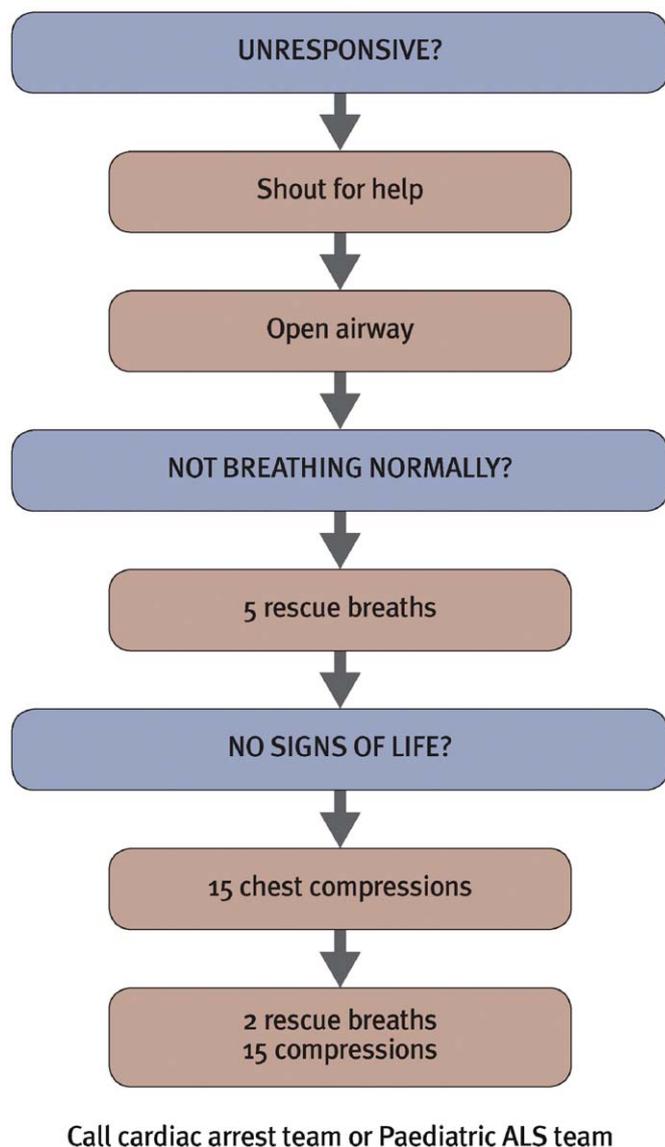


Fig. 1.11. Paediatric basic life support algorithm for those with a duty to respond.

The following sequence is to be followed by those with a duty to respond to paediatric emergencies (usually health professional teams) (Fig. 1.11).

1. Ensure the safety of rescuer and child.
2. Check the child's responsiveness.
 - Gently stimulate the child and ask loudly: Are you all right?
- 3A. If the child responds by answering or moving:
 - Leave the child in the position in which you find him (provided he is not in further danger).
 - Check his condition and get help if needed.
 - Reassess him regularly.
- 3B. If the child does not respond:
 - Shout for help.
 - Turn carefully the child on his back.
 - Open the child's airway by tilting the head and lifting the chin.
 - Place your hand on his forehead and gently tilt his head back.

- At the same time, with your fingertip(s) under the point of the child's chin, lift the chin. Do not push on the soft tissues under the chin as this may obstruct the airway.
- If you still have difficulty in opening the airway, try a jaw thrust: place the first two fingers of each hand behind each side of the child's mandible and push the jaw forward.

4. Keeping the airway open, look, listen and feel for normal breathing by putting your face close to the child's face and looking along the chest:

- Look for chest movements.
- Listen at the child's nose and mouth for breath sounds.
- Feel for air movement on your cheek.

In the first few minutes after a cardiac arrest a child may be taking slow infrequent gasps. Look, listen and feel for no more than 10 s before deciding—if you have any doubt whether breathing is normal, act as if it is not normal:

5A. If the child is breathing normally:

- Turn the child on his side into the recovery position (see below)
- Send or go for help—call the local emergency number for an ambulance.
- Check for continued breathing.

5B. If breathing is not normal or absent:

- Remove carefully any obvious airway obstruction.
- Give five initial rescue breaths.
- While performing the rescue breaths note any gag or cough response to your action. These responses or their absence will form part of your assessment of 'signs of life', which will be described later.

Rescue breaths for a child over 1 year of age:

- Ensure head tilt and chin lift.
- Pinch the soft part of the nose closed with the index finger and thumb of your hand on his forehead.
- Allow the mouth to open, but maintain chin lift.
- Take a breath and place your lips around the mouth, making sure that you have a good seal.
- Blow steadily into the mouth over about 1–1.5 s watching for chest rise.
- Maintain head tilt and chin lift, take your mouth away from the victim and watch for his chest to fall as air comes out.
- Take another breath and repeat this sequence five times. Identify effectiveness by seeing that the child's chest has risen and fallen in a similar fashion to the movement produced by a normal breath.

Rescue breaths for an infant:

- Ensure a neutral position of the head and a chin lift.
- Take a breath and cover the mouth and nose of the infant with your mouth, making sure you have a good seal. If the nose and mouth cannot be covered in the older infant, the rescuer may attempt to seal only the infant's nose or mouth with his mouth (if the nose is used, close the lips to prevent air escape).
- Blow steadily into the infant's mouth and nose over 1–1.5 s, sufficient to make the chest visibly rise.
- Maintain head position and chin lift, take your mouth away from the victim and watch for his chest to fall as air comes out.
- Take another breath and repeat this sequence five times.

For both infants and children, if you have difficulty achieving an effective breath, the airway may be obstructed:

- Open the child's mouth and remove any visible obstruction. Do not perform a blind finger sweep.

- Ensure that there is adequate head tilt and chin lift but also that the neck is not over extended.
- If head tilt and chin lift has not opened the airway, try the jaw thrust method.
- Make up to five attempts to achieve effective breaths, if still unsuccessful, move on to chest compressions.

6. Assess the child's circulation

Take no more than 10 s to:

- Look for signs of life—this includes any movement, coughing or normal breathing (not abnormal gasps or infrequent, irregular breaths).
If you check the pulse, ensure you take no more than 10 s.
In a child over 1 year—feel for the carotid pulse in the neck.
In an infant—feel for the brachial pulse on the inner aspect of the upper arm.

The femoral pulse in the groin, which is half way between the anterior superior iliac spine and the symphysis pubis, can also be used in infant and children.

7A. If you are confident that you can detect signs of life within 10 s:

- Continue rescue breathing, if necessary, until the child starts breathing effectively on his own.
- Turn the child on to his side (into the recovery position) if he remains unconscious.
- Re-assess the child frequently.

7B. If there are no signs of life, unless you are CERTAIN you can feel a definite pulse of greater than 60 beats min^{-1} within 10 s:

- Start chest compressions.
- Combine rescue breathing and chest compressions:

Chest compressions

For all children, compress the lower half of the sternum.

To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Compress the sternum one finger's breadth above this; the compression should be sufficient to depress the sternum by at least one-third of the depth of the chest. Don't be afraid to push too hard: "Push Hard and Fast". Release the pressure completely and repeat at a rate of at least 100 min^{-1} (but not exceeding 120 min^{-1}). After 15 compressions, tilt the head, lift the chin, and give two effective breaths. Continue compressions and breaths in a ratio of 15:2. The best method for compression varies slightly between infants and children.

Chest compression in infants

The lone rescuer compresses the sternum with the tips of two fingers. If there are two or more rescuers, use the encircling technique. Place both thumbs flat side by side on the lower half of the sternum (as above) with the tips pointing towards the infant's head. Spread the rest of both hands with the fingers together to encircle the lower part of the infant's rib cage with the tips of the fingers supporting the infant's back. For both methods, depress the lower sternum by at least one-third of the depth of the infant's chest (approximately 4 cm).

Chest compression in children over 1 year of age

Place the heel of one hand over the lower half of the sternum (as above). Lift the fingers to ensure that pressure is not applied over the child's ribs. Position yourself vertically above the victim's chest and, with your arm straight, compress the sternum to depress it by at least one-third of the depth of the chest (approximately 5 cm). In larger children or for small rescuers, this is achieved most easily by using both hands with the fingers interlocked.

8. Do not interrupt resuscitation until:

- The child shows signs of life (starts to wake up, to move, opens eyes and to breathe normally or a definite pulse of greater than 60 min^{-1} is palpated).

- Further qualified help arrives and takes over.
- You become exhausted.

When to call for assistance

It is vital for rescuers to get help as quickly as possible when a child collapses.

- When more than one rescuer is available, one starts resuscitation while another rescuer goes for assistance.
- If only one rescuer is present, undertake resuscitation for about 1 min before going for assistance. To minimise interruption in CPR, it may be possible to carry an infant or small child while summoning help.
- The only exception to performing 1 min of CPR before going for help is in the case of a child with a witnessed, sudden collapse when the rescuer is alone. In this case, cardiac arrest is likely to be caused by an arrhythmia and the child will need defibrillation. Seek help immediately if there is no one to go for you.

Recovery position

An unconscious child whose airway is clear, and who is breathing normally, should be turned on his side into the recovery position. The adult recovery position is suitable for use in children.

Foreign body airway obstruction (FBAO)

Back blows, chest thrusts and abdominal thrusts all increase intra-thoracic pressure and can expel foreign bodies from the airway. In half of the episodes more than one technique is needed to relieve the obstruction.⁴⁶⁵ There are no data to indicate which measure should be used first or in which order they should be applied. If one is unsuccessful, try the others in rotation until the object is cleared.

The FBAO algorithm for children was simplified and aligned with the adult version in 2005 guidelines; this continues to be the recommended sequence for managing FBAO (Fig. 1.12).

The most significant difference from the adult algorithm is that abdominal thrusts should not be used for infants. Although abdominal thrusts have caused injuries in all age groups, the risk is particularly high in infants and very young children. This is because of the horizontal position of the ribs, which leaves the upper abdominal viscera much more exposed to trauma. For this reason, the guidelines for the treatment of FBAO are different between infants and children. Signs for the recognition of FBAO in a child are listed in Table 1.2.

Table 1.2
Signs of foreign body airway obstruction.

General signs of FBAO	
Witnessed episode	
Coughing/choking	
Sudden onset	
Recent history of playing with/eating small objects	
Ineffective coughing	Effective cough
Unable to vocalise	Crying or verbal response to questions
Quiet or silent cough	Loud cough
Unable to breathe	Able to take a breath before coughing
Cyanosis	Fully responsive
Decreasing level of consciousness	

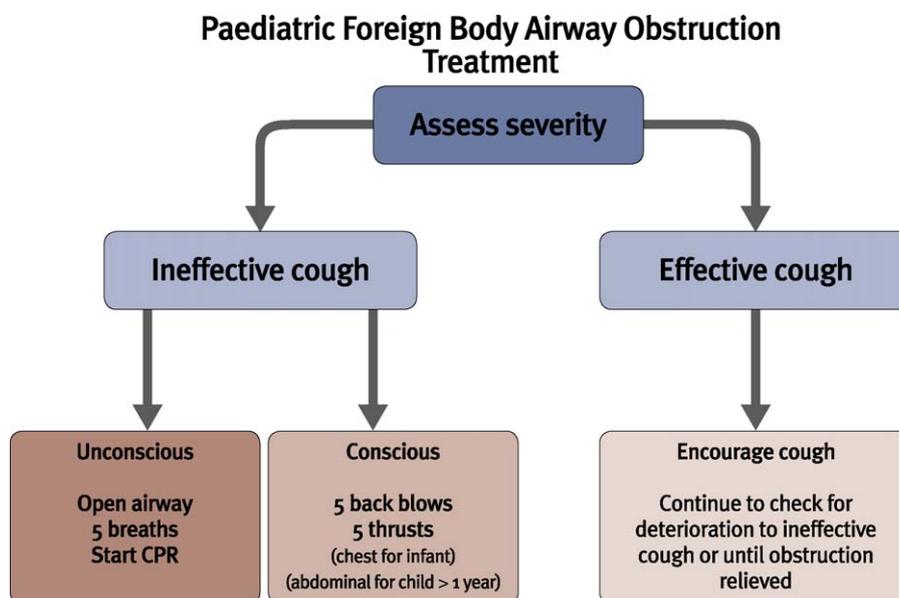


Fig. 1.12. Paediatric foreign body airway obstruction algorithm. © 2010 ERC.

Paediatric advanced life support

Prevention of cardiopulmonary arrest

In children, secondary cardiopulmonary arrests, caused by either respiratory or circulatory failure, are more frequent than primary arrests caused by arrhythmias.^{466–471} So-called asphyxial arrests or respiratory arrests are also more common in young adulthood (e.g., trauma, drowning, poisoning).^{472,473} The outcome from cardiopulmonary arrests in children is poor; identification of the antecedent stages of cardiac or respiratory failure is a priority, as effective early intervention may be life saving. The order of assessment and intervention for any seriously ill or injured child follows the ABCDE principles outlined previously for adults. Summoning a paediatric RRT or MET may reduce the risk of respiratory and/or cardiac arrest in hospitalised children outside the intensive care setting.^{202,474–478}

Management of respiratory and circulatory failure

In children, there are many causes of respiratory and circulatory failure and they may develop gradually or suddenly. Both may be initially compensated but will normally decompensate without adequate treatment. Untreated decompensated respiratory or circulatory failure will lead to cardiopulmonary arrest. Hence, the aim of paediatric life support is early and effective intervention in children with respiratory and circulatory failure to prevent progression to full arrest.

Airway and breathing

- Open the airway and ensure adequate ventilation and oxygenation. Deliver high-flow oxygen.
- Establish respiratory monitoring (first line—pulse oximetry/SpO₂).
- Achieving adequate ventilation and oxygenation may require use of airway adjuncts, bag-mask ventilation (BMV), use of a laryngeal mask airway (LMA), securing a definitive airway by tracheal intubation and positive pressure ventilation.
- Very rarely, a surgical airway may be required.

Rapid-sequence induction and intubation. The child who is in cardiopulmonary arrest and deep coma does not require seda-

Table 1.3

General recommendation for cuffed and uncuffed tracheal tube sizes (internal diameter in mm).

	Uncuffed	Cuffed
Neonates		
Premature	Gestational age in weeks/10	Not used
Full term	3.5	Not usually used
Infants	3.5–4.0	3.0–3.5
Child 1–2 years	4.0–4.5	3.5–4.0
Child >2 years	Age/4 + 4	Age/4 + 3.5

tion or analgesia to be intubated; otherwise, intubation must be preceded by oxygenation (gentle BMV is sometimes required to avoid hypoxia), rapid sedation, analgesia and the use of neuromuscular blocking drugs to minimize intubation complications and failure.⁴⁷⁹ The intubator must be experienced and familiar with drugs used for rapid-sequence induction. The use of cricoid pressure may prevent or limit regurgitation of gastric contents,^{480,481} but it may distort the airway and make laryngoscopy and intubation more difficult.⁴⁸² Cricoid pressure should not be used if either intubation or oxygenation is compromised.

A general recommendation for tracheal tube internal diameters (ID) for different ages is shown in Table 1.3.^{483–488} This is a guide only and tubes one size larger and smaller should always be available. Tracheal tube size can also be estimated from the length of the child's body as measured by resuscitation tapes.⁴⁸⁹

Uncuffed tracheal tubes have been used traditionally in children up to 8 years of age but cuffed tubes may offer advantages in certain circumstances e.g., when lung compliance is poor, airway resistance is high or if there is a large air leak from the glottis.^{483,490,491} The use of cuffed tubes also makes it more likely that the correct tube size will be chosen on the first attempt.^{483,484,492} As excessive cuff pressure may lead to ischaemic damage to the surrounding laryngeal tissue and stenosis, cuff inflation pressure should be monitored and maintained at less than 25 cm H₂O.⁴⁹³

Displaced, misplaced or obstructed tubes occur frequently in the intubated child and are associated with increased risk of death.^{281,494} No single technique is 100% reliable for distinguishing oesophageal from tracheal intubation.^{495–497} Assessment of the correct tracheal tube position is made by:

- laryngoscopic observation of the tube passing beyond the vocal cords;

- detection of end-tidal CO₂ if the child has a perfusing rhythm (this may also be seen with effective CPR, but it is not completely reliable);
- observation of symmetrical chest wall movement during positive pressure ventilation;
- observation of mist in the tube during the expiratory phase of ventilation;
- absence of gastric distension;
- equal air entry heard on bilateral auscultation in the axillae and apices of the chest;
- absence of air entry into the stomach on auscultation;
- improvement or stabilisation of SpO₂ in the expected range (delayed sign!);
- heart rate moving closer to the age-expected value (or remaining within the normal range) (delayed sign!).

If the child is in cardiopulmonary arrest and exhaled CO₂ is not detected despite adequate chest compressions, or if there is any doubt, confirm tracheal tube position by direct laryngoscopy.

Breathing. Give oxygen at the highest concentration (i.e., 100%) during initial resuscitation. Once circulation is restored, give sufficient oxygen to maintain an arterial oxygen saturation (SaO₂) in the range of 94–98%.^{498,499}

Healthcare providers commonly provide excessive ventilation during CPR and this may be harmful. Hyperventilation causes increased intra-thoracic pressure, decreased cerebral and coronary perfusion, and poorer survival rates in animals and adults.^{224,225,286,500–503} Although normoventilation is the objective during resuscitation, it is difficult to know the precise minute volume that is being delivered. A simple guide to deliver an acceptable tidal volume is to achieve modest chest wall rise. Once the airway is protected by tracheal intubation, continue positive pressure ventilation at 10–12 breaths min⁻¹ without interrupting chest compressions. When circulation is restored, or if the child still has a perfusing rhythm, ventilate at 12–20 breaths min⁻¹ to achieve a normal arterial carbon dioxide tension (PaCO₂).

Monitoring end-tidal CO₂ (ETCO₂) with a colorimetric detector or capnometer confirms tracheal tube placement in the child weighing more than 2 kg, and may be used in pre- and in-hospital settings, as well as during any transportation of the child.^{504–507} A colour change or the presence of a capnographic waveform for more than four ventilated breaths indicates that the tube is in the tracheobronchial tree both in the presence of a perfusing rhythm and during cardiopulmonary arrest. Capnography does not rule out intubation of a bronchus. The absence of exhaled CO₂ during cardiopulmonary arrest does not guarantee tube misplacement since a low or absent end tidal CO₂ may reflect low or absent pulmonary blood flow.^{235,508–510} Capnography may also provide information on the efficiency of chest compressions and can give an early indication of ROSC.^{511,512} Efforts should be made to improve chest compression quality if the ETCO₂ remains below 15 mm Hg (2 kPa). Current evidence does not support the use of a threshold ETCO₂ value as an indicator for the discontinuation of resuscitation efforts.

The self-inflating bulb or aspirating syringe (oesophageal detector device, ODD) may be used for the secondary confirmation of tracheal tube placement in children with a perfusing rhythm.^{513,514} There are no studies on the use of the ODD in children who are in cardiopulmonary arrest.

Clinical evaluation of the oxygen saturation of arterial blood (SaO₂) is unreliable; therefore, monitor the child's peripheral oxygen saturation continuously by pulse oximetry (SpO₂).

Circulation

- Establish cardiac monitoring [first line—pulse oximetry (SpO₂), ECG and non-invasive blood pressure (NIBP)].
- Secure vascular access. This may be by peripheral IV or IO cannulation. If already in situ, a central intravenous catheter should be used.
- Give a fluid bolus (20 ml kg⁻¹) and/or drugs (e.g., inotropes, vaso-pressors, anti-arrhythmics) as required.
- Isotonic crystalloids are recommended as initial resuscitation fluid in infants and children with any type of shock, including septic shock.^{515–518}
- Assess and re-assess the child continuously, commencing each time with the airway before proceeding to breathing and then the circulation.
- During treatment, capnography, invasive monitoring of arterial blood pressure, blood gas analysis, cardiac output monitoring, echocardiography and central venous oxygen saturation (ScvO₂) may be useful to guide the management of respiratory and/or circulatory failure.

Vascular access. Venous access can be difficult to establish during resuscitation of an infant or child: if attempts at establishing IV access are unsuccessful after one minute, insert an IO needle instead.^{519,520} Intraosseous or IV access is much preferred to the tracheal route for giving drugs.⁵²¹

Adrenaline. The recommended IV/IO dose of adrenaline in children for the first and for subsequent doses is 10 µg kg⁻¹. The maximum single dose is 1 mg. If needed, give further doses of adrenaline every 3–5 min. Intratracheal adrenaline is no longer recommended,^{522–525} but if this route is ever used, the dose is ten times this (100 µg kg⁻¹).

Advanced management of cardiopulmonary arrest

1. When a child becomes unresponsive, without signs of life (no breathing, cough or any detectable movement), start CPR immediately.
2. Provide BMV with 100% oxygen.
3. Commence monitoring. Send for a manual defibrillator or an AED to identify and treat shockable rhythms as quickly as possible (Fig. 1.13).

ABC

Commence and continue with basic life support

Oxygenate and ventilate with BMV

Provide positive pressure ventilation with a high inspired oxygen concentration

Give five rescue breaths followed by external chest compression and positive pressure ventilation in the ratio of 15:2

Avoid rescuer fatigue by frequently changing the rescuer performing chest compressions

Establish cardiac monitoring

Assess cardiac rhythm and signs of life

(±Check for a central pulse for no more than 10 s)

Non-shockable—asystole, PEA

- Give adrenaline IV or IO (10 µg kg⁻¹) and repeat every 3–5 min.
- Identify and treat any reversible causes (4Hs & 4Ts).

Shockable—VF/pulseless VT

Attempt defibrillation immediately (4 J kg⁻¹):

Paediatric Advanced Life Support

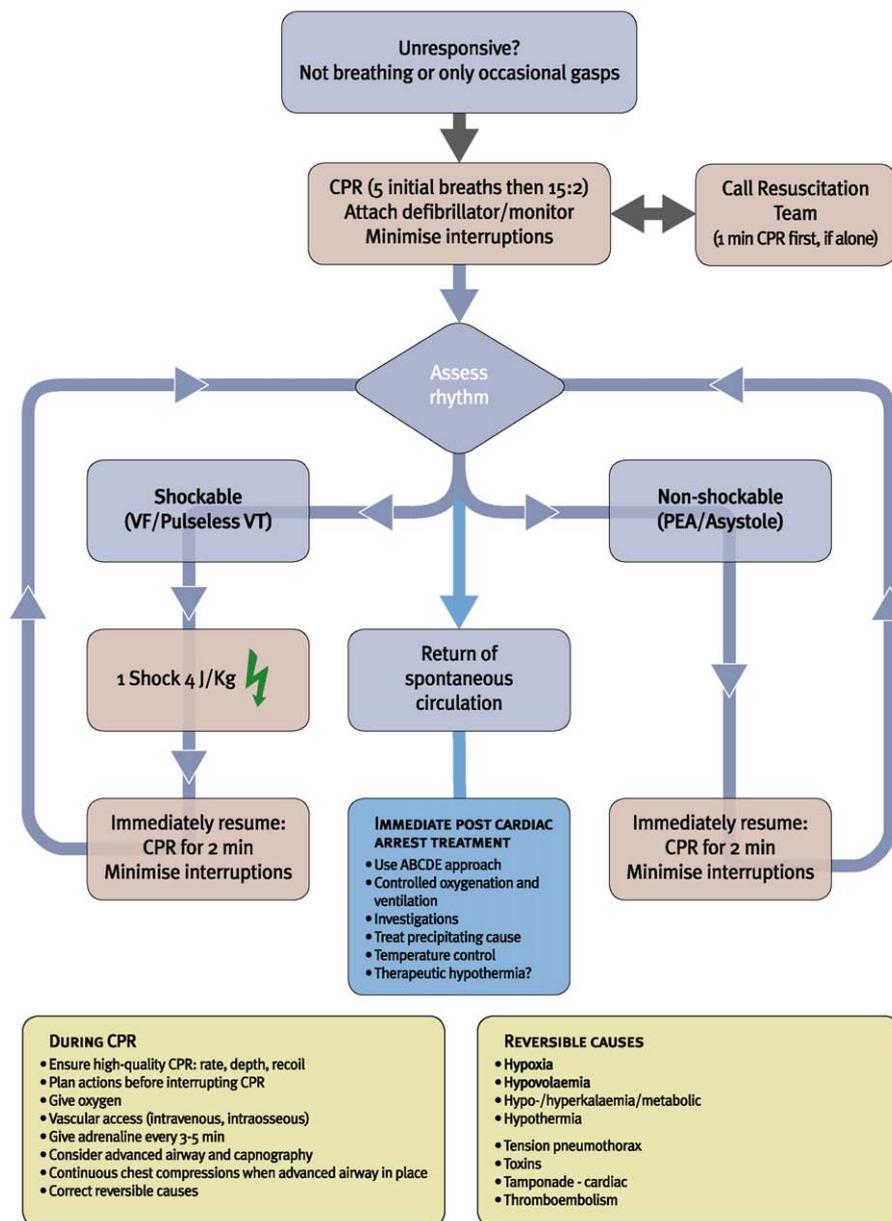


Fig. 1.13. Paediatric advanced life support algorithm. © 2010 ERC.

- 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. Minimise the delay between stopping chest compressions and delivery of the shock—even 5–10 s delay will reduce the chances of the shock being successful.^{71,110}
 - Give one shock.
 - Resume CPR as soon as possible without reassessing the rhythm.
 - After 2 min, check briefly the cardiac rhythm on the monitor
 - Give second shock (4 J kg^{-1}) if still in VF/pulseless VT
 - Give CPR for 2 min as soon as possible without reassessing the rhythm.
 - Pause briefly to assess the rhythm; if still in VF/pulseless VT give a third shock at 4 J kg^{-1} .
 - Give adrenaline $10 \mu\text{g kg}^{-1}$ and amiodarone 5 mg kg^{-1} after the third shock once CPR has been resumed.
 - Give adrenaline every alternate cycle (i.e., every 3–5 min during CPR)
 - Give a second dose of amiodarone 5 mg kg^{-1} if still in VF/pulseless VT after the fifth shock.⁵²⁶
- If the child remains in VF/pulseless VT, continue to alternate shocks of 4 J kg^{-1} with 2 min of CPR. If signs of life become evident, check the monitor for an organised rhythm; if this is present, check for signs of life and a central pulse and evaluate the haemodynamics of the child (blood pressure, peripheral pulse, capillary refill time). Identify and treat any reversible causes (4Hs & 4Ts) remembering that the first 2Hs (hypoxia and hypovolaemia) have the highest prevalence in critically ill or injured children.
- If defibrillation was successful but VF/pulseless VT recurs, resume CPR, give amiodarone and defibrillate again at the dose that was effective previously. Start a continuous infusion of amiodarone.

Echocardiography may be used to identify potentially treatable causes of cardiac arrest in children. Cardiac activity can be rapidly visualised⁵²⁷ and pericardial tamponade diagnosed.²⁶⁸ However, appropriately skilled operators must be available and its use should be balanced against the interruption to chest compressions during examination.

Arrhythmias

Unstable arrhythmias. Check for signs of life and the central pulse of any child with an arrhythmia; if signs of life are absent, treat as for cardiopulmonary arrest. If the child has signs of life and a central pulse, evaluate the haemodynamic status. Whenever the haemodynamic status is compromised, the first steps are:

1. Open the airway.
2. Give oxygen and assist ventilation as necessary.
3. Attach ECG monitor or defibrillator and assess the cardiac rhythm.
4. Evaluate if the rhythm is slow or fast for the child's age.
5. Evaluate if the rhythm is regular or irregular.
6. Measure QRS complex (narrow complexes: <0.08 s duration; wide complexes: >0.08 s).
7. The treatment options are dependent on the child's haemodynamic stability.

Bradycardia is caused commonly by hypoxia, acidosis and/or severe hypotension; it may progress to cardiopulmonary arrest. Give 100% oxygen, and positive pressure ventilation if required, to any child presenting with bradyarrhythmia and circulatory failure. If a poorly perfused child has a heart rate <60 beats min⁻¹, and they do not respond rapidly to ventilation with oxygen, start chest compressions and give adrenaline. If the bradycardia is caused by vagal stimulation (such as after passing a nasogastric tube), atropine may be effective. Cardiac pacing (either transvenous or external) is generally not useful during resuscitation. It may be considered in cases of AV block or sinus node dysfunction unresponsive to oxygenation, ventilation, chest compressions and other medications; pacing is not effective in asystole or arrhythmias caused by hypoxia or ischaemia.⁵²⁸

If SVT is the likely rhythm, vagal manoeuvres (Valsalva or diving reflex) may be used in haemodynamically stable children. They can also be used in haemodynamically unstable children, but only if they do not delay chemical (e.g., adenosine) or electrical cardioversion.⁵²⁹ If the child is unstable with a depressed conscious level, attempt synchronised electrical cardioversion immediately. Electrical cardioversion (synchronised with R wave) is also indicated when vascular access is not available, or when adenosine has failed to convert the rhythm. The first energy dose for electrical cardioversion of SVT is 0.5–1 J kg⁻¹ and the second dose is 2 J kg⁻¹.

In children, wide-QRS complex tachycardia is uncommon and more likely to be supraventricular than ventricular in origin.⁵³⁰ Nevertheless, in haemodynamically unstable children, it must be considered to be VT until proven otherwise. Synchronised cardioversion is the treatment of choice for unstable VT with a pulse. Consider anti-arrhythmic therapy if a second cardioversion attempt is unsuccessful or if VT recurs.

Stable arrhythmias. While maintaining the child's airway, breathing and circulation, contact an expert before initiating therapy. Depending on the child's clinical history, presentation and ECG diagnosis, a child with stable, wide-QRS complex tachycardia may be treated for SVT and be given vagal manoeuvres or adenosine. Amiodarone may be considered as a treatment option if this fails or if the diagnosis of VT is confirmed on an ECG.

Special circumstances

Channelopathy

When sudden unexplained cardiac arrest occurs in children and young adults, obtain a complete past medical and family history (including a history of syncopal episodes, seizures, unexplained accidents/drownings, or sudden death) and review any available previous ECGs. All infants, children, and young adults with sudden, unexpected death should, if possible, have an unrestricted, complete autopsy, performed preferably by pathologists with training and expertise in cardiovascular pathology.^{531–540} Consideration should be given to preservation and genetic analysis of tissue to determine the presence of a channelopathy. Refer families of patients whose cause of death is not found on autopsy to a health care provider/centre with expertise in cardiac rhythm disturbances.

Single ventricle post-stage 1 repair

The incidence of cardiac arrest in infants following single ventricle stage 1 repair is approximately 20%, with a survival to discharge of 33%.⁵⁴¹ There is no evidence that anything other than routine resuscitative protocols should be followed. Diagnosis of the pre-arrest state is difficult but it may be assisted by monitoring the oxygen extraction (superior vena caval ScvO₂) or near infra-red spectroscopy (cerebral and splanchnic circulations).^{542–544} Treatment of high systemic vascular resistance with alpha-adrenergic receptor blockade may improve systemic oxygen delivery,⁵⁴⁵ reduce the incidence of cardiovascular collapse,⁵⁴⁶ and improve survival.⁵⁴⁷

Single ventricle post-Fontan

Children in the pre-arrest state who have Fontan or hemi-Fontan anatomy may benefit from increased oxygenation and an improved cardiac output by instituting negative pressure ventilation.^{548,549} Extracorporeal membrane oxygenation (ECMO) may be useful rescue for children with failing Fontan circulations but no recommendation can be made in favour or against ECMO in those with hemi-Fontan physiology or for rescue during resuscitation.⁵⁵⁰

Pulmonary hypertension

There is an increased risk of cardiac arrest in children with pulmonary hypertension.^{551,552} Follow routine resuscitation protocols in these patients with emphasis on high FiO₂ and alkalosis/hyperventilation because this may be as effective as inhaled nitric oxide in reducing pulmonary vascular resistance.⁵⁵³ Resuscitation is most likely to be successful in patients with a reversible cause who are treated with intravenous epoprostenol or inhaled nitric oxide.⁵⁵⁴ If routine medications that reduce pulmonary artery pressure have been stopped, they should be restarted and the use of aerosolised epoprostenol or inhaled nitric oxide considered.⁵⁵⁵ Right ventricular support devices may improve survival.^{556–559}

Post-arrest management

The principles of post-cardiac arrest management and treatment of the post-cardiac arrest syndrome in children are similar to those of adults.

Temperature control and management

Hypothermia is common in the child following cardiopulmonary resuscitation.³⁵⁰ Central hypothermia (32–34 °C) may be beneficial, whereas fever may be detrimental to the injured brain. Mild hypothermia has an acceptable safety profile in adults^{355,356} and neonates.^{560–565} While it may improve neurological outcome in children, an observational study neither supports nor refutes the use of therapeutic hypothermia in paediatric cardiac arrest.⁵⁶⁶

A child who regains a spontaneous circulation, but remains comatose after cardiopulmonary arrest, may benefit from being cooled to a core temperature of 32–34 °C for at least 24 h. The successfully resuscitated child with hypothermia and ROSC should not be actively rewarmed unless the core temperature is below 32 °C. Following a period of mild hypothermia, rewarm the child slowly at 0.25–0.5 °C h⁻¹.

These guidelines are based on evidence from the use of therapeutic hypothermia in neonates and adults. At the time of writing, there are ongoing, prospective, multicentre trials of therapeutic hypothermia in children following in- and out-of-hospital cardiac arrest. (www.clinicaltrials.gov; NCT00880087 and NCT00878644)

Fever is common following cardiopulmonary resuscitation and is associated with a poor neurological outcome,^{346,348,349} the risk increasing for each degree of body temperature greater than 37 °C.³⁴⁹ There are limited experimental data suggesting that the treatment of fever with antipyretics and/or physical cooling reduces neuronal damage.^{567,568} Antipyretics and accepted drugs to treat fever are safe; therefore, use them to treat fever aggressively.

Glucose control

Both hyper- and hypo-glycaemia may impair outcome of critically ill adults and children and should be avoided, but tight glucose control may also be harmful. Although there is insufficient evidence to support or refute a specific glucose management strategy in children with ROSC after cardiac arrest,^{3,569,570} it is appropriate to monitor blood glucose and avoid hypoglycaemia as well as sustained hyperglycaemia.

Resuscitation of babies at birth

Preparation

Relatively few babies need any resuscitation at birth. Of those that do need help, the overwhelming majority will require only assisted lung aeration. A small minority may need a brief period of chest compressions in addition to lung aeration. Of 100,000 babies born in Sweden in one year, only 10 per 1000 (1%) babies of 2.5 kg or more appeared to need resuscitation at delivery.⁵⁷¹ Of those babies receiving resuscitation, 8 per 1000 responded to mask inflation and only 2 per 1000 appeared to need intubation. The same study tried to assess the unexpected need for resuscitation at birth and found that for low risk babies, i.e., those born after 32 weeks gestation and following an apparently normal labour, about 2 per 1000 (0.2%) appeared to need resuscitation at delivery. Of these, 90% responded to mask inflation alone while the remaining 10% appeared not to respond to mask inflation and therefore were intubated at birth (Fig. 1.14).

Resuscitation or specialist help at birth is more likely to be needed by babies with intrapartum evidence of significant fetal compromise, babies delivering before 35 weeks gestation, babies delivering vaginally by the breech, and multiple pregnancies. Although it is often possible to predict the need for resuscitation or stabilisation before a baby is born, this is not always the case. Therefore, personnel trained in newborn life support should be easily available at every delivery and, should there be any need for intervention, the care of the baby should be their sole responsibility. One person experienced in tracheal intubation of the newborn should ideally be in attendance for deliveries associated with a high risk of requiring neonatal resuscitation. Local guidelines indicating who should attend deliveries should be developed, based on current practice and clinical audit.

An organised educational programme in the standards and skills required for resuscitation of the newborn is therefore essential for any institution in which deliveries occur.

Planned home deliveries

Recommendations as to who should attend a planned home delivery vary from country to country, but the decision to undergo a planned home delivery, once agreed with medical and midwifery staff, should not compromise the standard of initial resuscitation at birth. There will inevitably be some limitations to resuscitation of a newborn baby in the home, because of the distance from further assistance, and this must be made clear to the mother at the time plans for home delivery are made. Ideally, two trained professionals should be present at all home deliveries; one of these must be fully trained and experienced in providing mask ventilation and chest compressions in the newborn.

Equipment and environment

Unlike adult cardiopulmonary resuscitation (CPR), resuscitation at birth is often a predictable event. It is therefore possible to prepare the environment and the equipment before delivery of the baby. Resuscitation should ideally take place in a warm, well-lit, draught free area with a flat resuscitation surface placed below a radiant heater, with other resuscitation equipment immediately available. All equipment must be checked frequently.

When a birth takes place in a non-designated delivery area, the recommended minimum set of equipment includes a device for safe assisted lung aeration of an appropriate size for the newborn, warm dry towels and blankets, a sterile instrument for cutting the umbilical cord and clean gloves for the attendant and assistants. It may also be helpful to have a suction device with a suitably sized suction catheter and a tongue depressor (or laryngoscope) to enable the oropharynx to be examined. Unexpected deliveries outside hospital are most likely to involve emergency services who should plan for such events.

Temperature control

Naked, wet, newborn babies cannot maintain their body temperature in a room that feels comfortably warm for adults. Compromised babies are particularly vulnerable.⁵⁷² Exposure of the newborn to cold stress will lower arterial oxygen tension⁵⁷³ and increase metabolic acidosis.⁵⁷⁴ Prevent heat loss:

- Protect the baby from draughts.
- Keep the delivery room warm. For babies less than 28 weeks gestation the delivery room temperature should be 26 °C.^{575,576}
- Dry the term baby immediately after delivery. Cover the head and body of the baby, apart from the face, with a warm towel to prevent further heat loss. Alternatively, place the baby skin to skin with mother and cover both with a towel.
- If the baby needs resuscitation then place the baby on a warm surface under a preheated radiant warmer.
- In very preterm babies (especially below 28 weeks) drying and wrapping may not be sufficient. A more effective method of keeping these babies warm is to cover the head and body of the baby (apart from the face) with plastic wrapping, without drying the baby beforehand, and then to place the baby so covered under radiant heat.

Initial assessment

The Apgar score was proposed as a “simple, common, clear classification or grading of newborn infants” to be used “as a basis for discussion and comparison of the results of obstetric practices, types of maternal pain relief and the effects of resuscitation” (our emphasis).⁵⁷⁷ It was not designed to be assembled and ascribed in order to then identify babies in need of resuscitation.⁵⁷⁸ However, individual components of the score, namely respiratory rate,

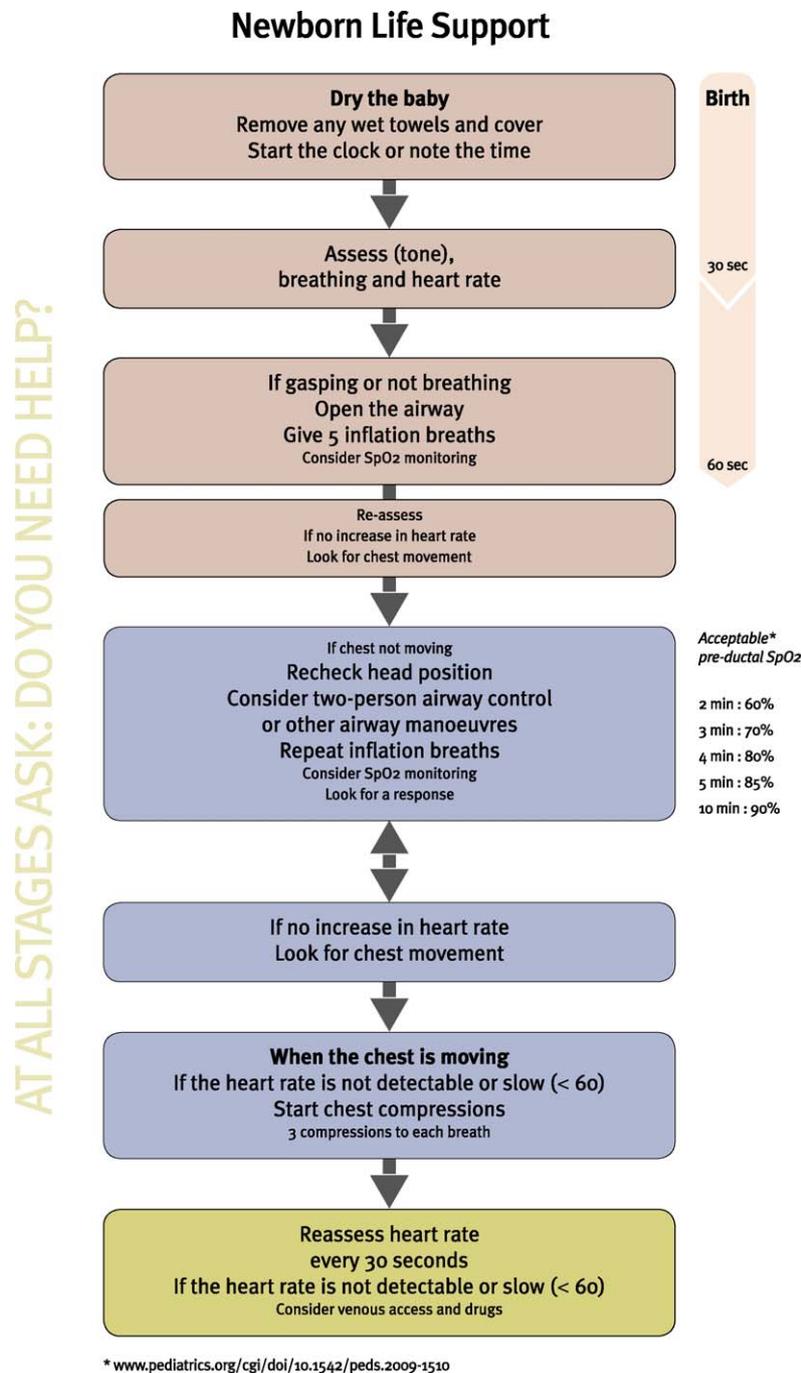


Fig. 1.14. Newborn life support algorithm. © 2010 ERC.

heart rate and tone, if assessed rapidly, can identify babies needing resuscitation.⁵⁷⁷ Furthermore, repeated assessment particularly of heart rate and, to a lesser extent breathing, can indicate whether the baby is responding or whether further efforts are needed.

Breathing

Check whether the baby is breathing. If so, evaluate the rate, depth and symmetry of breathing together with any evidence of an abnormal breathing pattern such as gasping or grunting.

Heart rate

This is assessed best by listening to the apex beat with a stethoscope. Feeling the pulse in the base of the umbilical cord is often

effective but can be misleading, cord pulsation is only reliable if found to be more than 100 beats min^{-1} .⁵⁷⁹ For babies requiring resuscitation and/or continued respiratory support, a modern pulse oximeter can give an accurate heart rate.⁵⁸⁰

Colour

Colour is a poor means of judging oxygenation,⁵⁸¹ which is better assessed using pulse oximetry if possible. A healthy baby is born blue but starts to become pink within 30 s of the onset of effective breathing. Peripheral cyanosis is common and does not, by itself, indicate hypoxemia. Persistent pallor despite ventilation may indicate significant acidosis or rarely hypovolaemia. Although colour is a poor method of judging oxygenation, it should not be ignored: if a baby appears blue check oxygenation with a pulse oximeter.

Tone

A very floppy baby is likely to be unconscious and will need ventilatory support.

Tactile stimulation

Drying the baby usually produces enough stimulation to induce effective breathing. Avoid more vigorous methods of stimulation. If the baby fails to establish spontaneous and effective breaths following a brief period of stimulation, further support will be required.

Classification according to initial assessment

On the basis of the initial assessment, the baby can be placed into one of three groups:

1. Vigorous breathing or crying

Good tone

Heart rate higher than 100 min⁻¹

This baby requires no intervention other than drying, wrapping in a warm towel and, where appropriate, handing to the mother. The baby will remain warm through skin-to-skin contact with mother under a cover, and may be put to the breast at this stage.

2. Breathing inadequately or apnoeic

Normal or reduced tone

Heart rate less than 100 min⁻¹

Dry and wrap. This baby may improve with mask inflation but if this does not increase the heart rate adequately, may also require chest compressions.

3. Breathing inadequately or apnoeic

Floppy

Low or undetectable heart rate

Often pale suggesting poor perfusion

Dry and wrap. This baby will then require immediate airway control, lung inflation and ventilation. Once this has been successfully accomplished the baby may also need chest compressions, and perhaps drugs.

There remains a very rare group of babies who, though breathing adequately and with a good heart rate, remain hypoxaemic. This group includes a range of possible diagnoses such as diaphragmatic hernia, surfactant deficiency, congenital pneumonia, pneumothorax, or cyanotic congenital heart disease.

Newborn life support

Commence newborn life support if assessment shows that the baby has failed to establish adequate regular normal breathing, or has a heart rate of less than 100 min⁻¹. Opening the airway and aerating the lungs is usually all that is necessary. Furthermore, more complex interventions will be futile unless these two first steps have been successfully completed.

Airway

Place the baby on his back with the head in a neutral position. A 2 cm thickness of the blanket or towel placed under the baby's shoulders may be helpful in maintaining proper head position. In floppy babies application of jaw thrust or the use of an appropriately sized oropharyngeal airway may be helpful in opening the airway.

Suction is needed only if the airway is obstructed and is best done under direct vision. Aggressive pharyngeal suction can delay the onset of spontaneous breathing and cause laryngeal spasm and vagal bradycardia.⁵⁸² The presence of thick meconium in a non-vigorous baby is the only indication for considering immediate suction of the oropharynx. Connect a 12–14 FG suction catheter, or a Yankauer sucker, to a suction source not exceeding minus 100 mm Hg.

Breathing

After initial steps at birth, if breathing efforts are absent or inadequate, lung aeration is the priority. In term babies, begin resuscitation with air. The primary measure of adequate initial lung inflation is a prompt improvement in heart rate; assess chest wall movement if heart rate does not improve.

For the first few breaths maintain the initial inflation pressure for 2–3 s. This will help lung expansion. Most babies needing resuscitation at birth will respond with a rapid increase in heart rate within 30 s of lung inflation. If the heart rate increases but the baby is not breathing adequately, ventilate at a rate of about 30 breaths min⁻¹ allowing approximately one second for each inflation, until there is adequate spontaneous breathing.

Adequate passive ventilation is usually indicated by either a rapidly increasing heart rate or a heart rate that is maintained faster than 100 beats min⁻¹. If the baby does not respond in this way the most likely cause is inadequate airway control or inadequate ventilation. Without adequate lung aeration, chest compressions will be ineffective; therefore, confirm lung aeration before progressing to circulatory support. Some practitioners will ensure airway control by tracheal intubation, but this requires training and experience. If this skill is not available and the heart rate is decreasing, re-evaluate the airway position and deliver inflation breaths while summoning a colleague with intubation skills. Continue ventilatory support until the baby has established normal regular breathing.

Circulatory support

Circulatory support with chest compressions is effective only if the lungs have first been successfully inflated. Give chest compressions if the heart rate is less than 60 beats min⁻¹ despite adequate ventilation. The most effective technique for providing chest compressions is to place the two thumbs side by side over the lower third of the sternum just below an imaginary line joining the nipples, with the fingers encircling the torso and supporting the back.^{583–586} An alternative way to find the correct position of the thumbs is to identify the xiphisternum and then to place the thumbs on the sternum one finger's breadth above this point. The sternum is compressed to a depth of approximately one-third of the anterior–posterior diameter of the chest allowing the chest wall to return to its relaxed position between compressions.⁵⁸⁷

Use a CV ratio of 3:1, aiming to achieve approximately 120 events min⁻¹, i.e., approximately 90 compressions and 30 breaths. Check the heart rate after about 30 s and periodically thereafter. Discontinue chest compressions when the spontaneous heart rate is faster than 60 beats min⁻¹.

Drugs

Drugs are rarely indicated in resuscitation of the newly born infant. Bradycardia in the newborn infant is usually caused by inadequate lung inflation or profound hypoxia, and establishing adequate ventilation is the most important step to correct it. However, if the heart rate remains less than 60 beats min⁻¹ despite adequate ventilation and chest compressions, it is reasonable to consider the use of drugs. These are best given via an umbilical venous catheter.

Adrenaline

Despite the lack of human data it is reasonable to use adrenaline when adequate ventilation and chest compressions have failed to increase the heart rate above 60 beats min⁻¹. If adrenaline is used, give 10–30 µg kg⁻¹ *intravenously* as soon as possible. The tracheal route is not recommended but if it is used, it is highly likely that doses of 50–100 µg kg⁻¹ will be required. Neither the safety nor

the efficacy of these higher tracheal doses has been studied. Do not give these high doses intravenously.

Bicarbonate

There are insufficient data to recommend routine use of bicarbonate in resuscitation of the newly born. The hyperosmolarity and carbon dioxide-generating properties of sodium bicarbonate may impair myocardial and cerebral function. Use of sodium bicarbonate is discouraged during brief CPR. If it is used during prolonged arrests unresponsive to other therapy, it should be given only after adequate ventilation and circulation is established with CPR. A dose of 1–2 mmol kg⁻¹ may be given by slow intravenous injection after adequate ventilation and perfusion have been established.

Fluids

If there has been suspected blood loss or the infant appears to be in shock (pale, poor perfusion, weak pulse) and has not responded adequately to other resuscitative measures then consider giving fluid.⁵⁸⁸ This is a rare event. In the absence of suitable blood (i.e., irradiated and leucocyte-depleted group O Rh-negative blood), isotonic crystalloid rather than albumin is the solution of choice for restoring intravascular volume. Give a bolus of 10 ml kg⁻¹ initially. If successful it may need to be repeated to maintain an improvement.

Stopping resuscitation

Local and national committees will determine the indications for stopping resuscitation. If the heart rate of a newly born baby is not detectable and remains undetectable for 10 min, it is then appropriate to consider stopping resuscitation. In cases where the heart rate is less than 60 min⁻¹ at birth and does not improve after 10 or 15 min of continuous and apparently adequate resuscitative efforts, the choice is much less clear. In this situation there is insufficient evidence about outcome to enable firm guidance on whether to withhold or to continue resuscitation.

Communication with the parents

It is important that the team caring for the newborn baby informs the parents of the baby's progress. At delivery, adhere to local plans for routine care and, if possible, hand the baby to the mother at the earliest opportunity. If resuscitation is required inform the parents of the procedures undertaken and why they were required. Record carefully all discussions and decisions in the mother's notes prior to delivery and in the baby's records after birth.

Cardiac arrest in special circumstances

Electrolyte abnormalities

Life-threatening arrhythmias are associated most commonly with potassium disorders, particularly hyperkalaemia, and less commonly with disorders of serum calcium and magnesium. In some cases therapy for life-threatening electrolyte disorders should start before laboratory results become available. There is little or no evidence for the treatment of electrolyte abnormalities during cardiac arrest. Guidance during cardiac arrest is based on the strategies used in the non-arrest patient. There are no major changes in the treatment of these disorders since the International Guidelines 2005.⁵⁸⁹

Poisoning

Poisoning rarely causes cardiac arrest, but is a leading cause of death in victims younger than 40 years of age.⁵⁹⁰ Poisoning by therapeutic or recreational drugs and by household products are the main reasons for hospital admission and poison centre calls. Inappropriate drug dosing, drug interactions and other medication errors can also cause harm. Accidental poisoning is commonest in children. Homicidal poisoning is uncommon. Industrial accidents, warfare or terrorism can also cause exposure to harmful substances.

Prevention of cardiac arrest

Assess and treat the victim using the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach. Airway obstruction and respiratory arrest secondary to a decreased conscious level is a common cause of death after self-poisoning.⁵⁹¹ Pulmonary aspiration of gastric contents can occur after poisoning with central nervous system depressants. Early tracheal intubation of unconscious patients by a trained person decreases the risk of aspiration. Drug-induced hypotension usually responds to fluid infusion, but occasionally vasopressor support (e.g., noradrenaline infusion) is required. A long period of coma in a single position can cause pressure sores and rhabdomyolysis. Measure electrolytes (particularly potassium), blood glucose and arterial blood gases. Monitor temperature because thermoregulation is impaired. Both hypothermia and hyperthermia (hyperpyrexia) can occur after overdose of some drugs. Retain samples of blood and urine for analysis. Patients with severe poisoning should be cared for in a critical care setting. Interventions such as decontamination, enhanced elimination and antidotes may be indicated and are usually second line interventions.⁵⁹² Alcohol excess is often associated with self-poisoning.

Modifications to basic and advanced life support

- Have a high index of personal safety where there is a suspicious cause or unexpected cardiac arrest. This is especially so when more than one casualty collapses simultaneously.
- Avoid mouth-to-mouth ventilation in the presence of chemicals such as cyanide, hydrogen sulphide, corrosives and organophosphates.
- Treat life-threatening tachyarrhythmias with cardioversion according to the peri-arrest arrhythmia guidelines (see *Advanced life support*).⁶ This includes correction of electrolyte and acid-base abnormalities.
- Try to identify the poison(s). Relatives, friends and ambulance crews can provide useful information. Examination of the patient may reveal diagnostic clues such as odours, needle marks, pupil abnormalities, and signs of corrosion in the mouth.
- Measure the patient's temperature because hypo- or hyperthermia may occur after drug overdose (see Sections 8d and 8e).
- Be prepared to continue resuscitation for a prolonged period, particularly in young patients, as the poison may be metabolized or excreted during extended life support measures.
- Alternative approaches that may be effective in severely poisoned patients include: higher doses of medication than in standard protocols; non-standard drug therapies; prolonged CPR.
- Consult regional or national poisons centres for information on treatment of the poisoned patient. The International Programme on Chemical Safety (IPCS) lists poison centres on its website: <http://www.who.int/ipcs/poisons/centre/en/>
- On-line databases for information on toxicology and hazardous chemicals: (<http://toxnet.nlm.nih.gov/>)

Drowning

The World Health Organisation (WHO) estimates that, worldwide, drowning accounts for approximately 450,000 deaths each year and drowning is a common cause of accidental death in Europe. After drowning the duration of hypoxia is the most critical factor in determining the victim's outcome; therefore, oxygenation, ventilation, and perfusion should be restored as rapidly as possible. Immediate resuscitation at the scene is essential for survival and neurological recovery after a drowning incident. This will require provision of CPR by a bystander and immediate activation of the EMS system. Victims who have spontaneous circulation and breathing when they reach hospital usually recover with good outcomes. Research into drowning is limited in comparison with primary cardiac arrest and there is a need for further research in this area.⁵⁹³ The guidelines described in detail in Section 8 of the ERC Guidelines are intended for healthcare professionals and certain groups of lay responders that have a special interest in the care of the drowning victim e.g., lifeguards.¹⁰

Accidental hypothermia

Accidental hypothermia exists when the body core temperature unintentionally drops below 35 °C. Hypothermia can be classified arbitrarily as mild (35–32 °C), moderate (32–28 °C) or severe (less than 28 °C).⁵⁹⁴ In a hypothermic patient, no signs of life alone is unreliable for declaring death. In the pre-hospital setting, resuscitation should be withheld only if the cause of a cardiac arrest is clearly attributable to a lethal injury, fatal illness, prolonged asphyxia, or if the chest is incompressible. All the principles of prevention, basic and advanced life support apply to the hypothermic patient. Use the same ventilation and chest compression rates as for a normothermic patient. Hypothermia can cause stiffness of the chest wall, making ventilation and chest compressions more difficult.

The hypothermic heart may be unresponsive to cardioactive drugs, attempted electrical pacing and defibrillation. Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drugs given repeatedly.⁵⁹⁵ Withhold adrenaline and other CPR drugs until the patient has been warmed to a temperature higher than approximately 30 °C. Once 30 °C has been reached, the intervals between drug doses should be doubled when compared with normothermia intervals. As normothermia is approached (over 35 °C), standard drug protocols should be used.

As the body core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation followed by VF and finally asystole.⁵⁹⁶ Once in hospital, severely hypothermic victims in cardiac arrest should be rewarmed with active internal methods. Arrhythmias other than VF tend to revert spontaneously as the core temperature increases, and usually do not require immediate treatment. Bradycardia may be physiological in severe hypothermia, and cardiac pacing is not indicated unless bradycardia associated with haemodynamic compromise persists after re-warming. The temperature at which defibrillation should first be attempted and how often it should be tried in the severely hypothermic patient has not been established. AEDs may be used on these patients. If VF is detected, give a shock at the maximum energy setting; if VF/VT persists after three shocks, delay further defibrillation attempts until the core temperature is above 30 °C.⁵⁹⁷ If an AED is used, follow the AED prompts while re-warming the patient. CPR and re-warming may have to be continued for several hours to facilitate successful defibrillation.⁵⁹⁷

Rewarming may be passive, active external, or active internal. Passive re-warming is appropriate in conscious victims with mild hypothermia who are still able to shiver. Hypothermic victims with an altered consciousness should be taken to a hospital capable of active external and internal re-warming. In a hypothermic patient

with apnoea and cardiac arrest, extracorporeal re-warming is the preferred method of active internal re-warming because it provides sufficient circulation and oxygenation while the core body temperature is increased by 8–12 °C h⁻¹.⁵⁹⁸

During re-warming, patients will require large volumes of fluids as vasodilation causes expansion of the intravascular space. Continuous haemodynamic monitoring and warm IV fluids are essential. Avoid hyperthermia during and after re-warming. Although there are no formal studies, once ROSC has been achieved use standard strategies for post-resuscitation care, including mild hypothermia if appropriate.

Hyperthermia

Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds that normally maintained by homeostatic mechanisms. Hyperthermia may be exogenous, caused by environmental conditions, or secondary to endogenous heat production.

Environment-related hyperthermia occurs where heat, usually in the form of radiant energy, is absorbed by the body at a rate faster than can be lost by thermoregulatory mechanisms. Hyperthermia occurs along a continuum of heat-related conditions, starting with heat stress, progressing to heat exhaustion, to heat stroke (HS) and finally multiorgan dysfunction and cardiac arrest in some instances.⁵⁹⁹

Heat stroke is a systemic inflammatory response with a core temperature above 40.6 °C, accompanied by mental state change and varying levels of organ dysfunction. There are two forms of HS: classic non-exertional heat stroke (CHS) occurs during high environmental temperatures and often affects the elderly during heat waves⁶⁰⁰; Exertional heat stroke (EHS) occurs during strenuous physical exercise in high environmental temperatures and/or high humidity usually effects healthy young adults.⁶⁰¹ Mortality from heat stroke ranges between 10% and 50%.⁶⁰²

The mainstay of treatment is supportive therapy based on optimizing the ABCDEs and rapidly cooling the patient.^{603–605} Start cooling before the patient reaches hospital. Aim to reduce the core temperature rapidly to approximately 39 °C. Patients with severe heat stroke need to be managed in a critical-care setting.

There are no specific studies on cardiac arrest in hyperthermia. If cardiac arrest occurs, follow standard procedures for basic and advanced life support and cool the patient. Cooling techniques similar to those used to induce therapeutic hypothermia should be used. There are no data on the effects of hyperthermia on defibrillation threshold; therefore, attempt defibrillation according to current guidelines, while continuing to cool the patient. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest.^{606,607} The risk of unfavourable neurological outcome increases for each degree of body temperature >37 °C.³⁴⁹

Asthma

The worldwide prevalence of asthma symptoms ranges from 1% to 18% of the population with a high prevalence in some European countries (United Kingdom, Ireland and Scandinavia).⁶⁰⁸ Annual worldwide deaths from asthma have been estimated at 250,000. National and international guidance for the management of asthma already exists.^{608,609} This guidance focuses on the treatment of patients with near-fatal asthma and cardiac arrest.

Causes of asthma-related cardiac arrest

Cardiac arrest in a person with asthma is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden. Cardiac arrest in those with asthma has been linked to:

- severe bronchospasm and mucous plugging leading to asphyxia (this condition causes the vast majority of asthma-related deaths);
- cardiac arrhythmias caused by hypoxia, which is the commonest cause of asthma-related arrhythmia.⁶¹⁰ Arrhythmias can also be caused by stimulant drugs (e.g., beta-adrenergic agonists, aminophylline) or electrolyte abnormalities;
- dynamic hyperinflation, i.e., auto-positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure;
- tension pneumothorax (often bilateral).

Key interventions to prevent arrest

The patient with severe asthma requires aggressive medical management to prevent deterioration. Base assessment and treatment on an ABCDE approach. Patients with SaO₂ <92% or with features of life-threatening asthma are at risk of hypercapnic respiratory failure and require arterial blood gas measurement. Experienced clinicians should treat these high-risk patients in a critical-care area. The specific drugs and the treatment sequence will vary according to local practice but are described in detail in Section 8f of the ERC Guidelines.¹⁰

Treatment of cardiac arrest caused by asthma

Give basic life support according to standard guidelines. Ventilation will be difficult because of increased airway resistance; try to avoid gastric inflation. Modifications to standard ALS guidelines include considering the need for early tracheal intubation. The very high airways resistance means that there is a significant risk of gastric inflation and hypoventilation of the lungs when attempting to ventilate a severe asthmatic without a tracheal tube. During cardiac arrest this risk is even higher, because the lower oesophageal sphincter pressure is substantially less than normal.⁶¹¹

Respiratory rates of 8–10 breaths min⁻¹ and tidal volume required for a normal chest rise during CPR should not cause dynamic hyperinflation of the lungs (gas trapping). Tidal volume depends on inspiratory time and inspiratory flow. Lung emptying depends on expiratory time and expiratory flow. In mechanically ventilated severe asthmatics, increasing the expiratory time (achieved by reducing the respiratory rate) provides only moderate gains in terms of reduced gas trapping when a minute volume of less than 10 l min⁻¹ is used.⁶¹²

There is limited evidence from case reports of unexpected ROSC in patients with suspected gas trapping when the tracheal tube is disconnected.^{613–617} If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest wall and/or a period of apnoea (disconnection from the tracheal tube) may relieve gas trapping if dynamic hyperinflation occurs. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.¹⁵ Dynamic hyperinflation increases transthoracic impedance.⁶¹⁸ Consider higher shock energies for defibrillation if initial defibrillation attempts fail.¹⁴

There is no good evidence for the use of open-chest cardiac compressions in patients with asthma-associated cardiac arrest. Working through the four Hs and four Ts will identify potentially reversible causes of asthma-related cardiac arrest. Tension pneumothorax can be difficult to diagnose in cardiac arrest; it may be indicated by unilateral expansion of the chest wall, shifting of the trachea and subcutaneous emphysema. Pleural ultrasound in skilled hands is faster and more sensitive than chest X-ray for the detection of pneumothorax.⁶¹⁹ Always consider bilateral pneumothoraces in asthma-related cardiac arrest.

Extracorporeal life support (ECLS) can ensure both organ perfusion and gas exchange in case of otherwise untreatable respiratory and circulatory failure. Cases of successful treatment of asthma-related cardiac arrest in adults using ECLS have been reported^{620,621}; however, the role of ECLS in cardiac arrest caused by asthma has never been investigated in controlled studies.

Anaphylaxis

Anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction. This is characterised by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.^{622,623} Anaphylaxis usually involves the release of inflammatory mediators from mast cells and, or basophils triggered by an allergen interacting with cell-bound immunoglobulin E (IgE). Non-IgE-mediated or non-immune release of mediators can also occur. Histamine and other inflammatory mediator release are responsible for the vasodilatation, oedema and increased capillary permeability.

Anaphylaxis is the likely diagnosis if a patient who is exposed to a trigger (allergen) develops a sudden illness (usually within minutes) with rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.

Use an ABCDE approach to recognise and treat anaphylaxis. Adrenaline should be given to all patients with life-threatening features. The intramuscular (IM) route is the best for most rescuers who have to give adrenaline to treat anaphylaxis. Use the following doses:

>12 years and adults	500 µg IM
>6–12 years	300 µg IM
>6 months–6 years	150 µg IM
<6 months	150 µg IM

Intravenous adrenaline should be used only by those experienced in the use and titration of vasopressors in their normal clinical practice (e.g., anaesthetists, emergency physicians, intensive care doctors). In adults, titrate IV adrenaline using 50 µg boluses according to response. Initially, give the highest concentration of oxygen possible using a mask with an oxygen reservoir.⁴²⁷ Give a rapid IV fluid challenge (20 ml kg⁻¹) in a child or 500–1000 ml in an adult) and monitor the response; give further doses as necessary. Other therapy (steroids, antihistamines, etc) for the treatment of life-threatening asthma is detailed in Section 8g. If cardiac arrest occurs, start CPR immediately and follow current guidelines. Prolonged CPR may be necessary. Rescuers should ensure that help is on its way as early advanced life support (ALS) is essential.

Measurement of mast cell tryptase will help confirm a diagnosis of anaphylaxis. Ideally, take three samples: initial sample as soon as feasible after resuscitation has started; second sample at 1–2 h after the start of symptoms, third sample either at 24 h or in convalescence. All patients presenting with anaphylaxis should be referred to an allergy clinic to identify the cause, and thereby reduce the risk of future reactions and prepare the patient to manage future episodes themselves.

Cardiac arrest following cardiac surgery

Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase, with a reported incidence of 0.7–2.9%.^{624–632} It is usually preceded by physiological deterioration,⁶³³ although it can occur suddenly in stable patients.⁶³⁰ There are usually specific causes of cardiac arrest, such as tamponade, hypovolaemia, myocardial ischaemia, tension pneumothorax, or pacing failure. These are all potentially reversible and

if treated promptly cardiac arrest after cardiac surgery has a relatively high survival rate. Key to the successful resuscitation of cardiac arrest in these patients is recognition of the need to perform emergency re-sternotomy early, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective.

Starting CPR

Start external chest compressions immediately in all patients who collapse without an output. Consider reversible causes: hypoxia – check tube position, ventilate with 100% oxygen; tension pneumothorax – clinical examination, thoracic ultrasound; hypovolaemia, pacing failure. In asystole, secondary to a loss of cardiac pacing, chest compressions may be delayed momentarily as long as the surgically inserted temporary pacing wires can be connected rapidly and pacing re-established (DDD at 100 min⁻¹ at maximum amplitude). The effectiveness of compressions may be verified by looking at the arterial trace, aiming to achieve a systolic blood pressure of at least 80 mm Hg at a rate of 100 min⁻¹.

Defibrillation

There is concern that external chest compressions can cause sternal disruption or cardiac damage.^{634–637} In the post-cardiac surgery ICU, a witnessed and monitored VF/VT cardiac arrest should be treated immediately with up to three quick successive (stacked) defibrillation attempts. Three failed shocks in the post-cardiac surgery setting should trigger the need for emergency re-sternotomy. Further defibrillation is attempted as indicated in the universal algorithm and should be performed with internal paddles at 20J if re-sternotomy has been performed.

Emergency drugs

Use adrenaline very cautiously and titrate to effect (intravenous doses of up to 100 µg in adults). Give amiodarone 300 mg after the 3rd failed defibrillation attempt but do not delay re-sternotomy.

Emergency re-sternotomy

This is an integral part of resuscitation after cardiac surgery, once all other reversible causes have been excluded. Once adequate airway and ventilation has been established, and if three attempts at defibrillation have failed in VF/VT, undertake re-sternotomy without delay. Emergency re-sternotomy is also indicated in asystole or PEA, when other treatments have failed.

Internal defibrillation

Internal defibrillation using paddles applied directly across the ventricles requires considerably less energy than that used for external defibrillation. Use 20J in cardiac arrest, but 5J if the patient has been placed on cardiopulmonary bypass. Continuing cardiac compressions using the internal paddles while charging the defibrillator and delivering the shock during the decompression phase of compressions may improve shock success.^{638,639}

Traumatic cardiorespiratory arrest

Cardiac arrest caused by trauma has a very high mortality, with an overall survival of just 5.6% (range 0–17%).^{640–646} For reasons that are unclear, reported survival rates in the last 5 years are better than reported previously. In those who survive (and where

data are available) neurological outcome is good in only 1.6% of those sustaining traumatic cardiorespiratory arrest (TCRA).

Comotio cordis

Comotio cordis is actual or near cardiac arrest caused by a blunt impact to the chest wall over the heart.^{647–651} A blow to the chest during the vulnerable phase of the cardiac cycle may cause malignant arrhythmias (usually ventricular fibrillation). Comotio cordis occurs mostly during sports (most commonly baseball) and recreational activities and victims are usually young males (mean age 14 years). The overall survival rate from comotio cordis is 15%, but 25% if resuscitation is started within 3 min.⁶⁵¹

Signs of life and initial ECG activity

There are no reliable predictors of survival for TCRA. One study reported that the presence of reactive pupils and sinus rhythm correlate significantly with survival.⁶⁵² In a study of penetrating trauma, pupil reactivity, respiratory activity and sinus rhythm were correlated with survival but were unreliable.⁶⁴⁶ Three studies reported no survivors in patients presenting with asystole or agonal rhythms.^{642,646,653} Another reported no survivors among those with PEA after blunt trauma.⁶⁵⁴ Based on these studies, the American College of Surgeons and the National Association of EMS physicians produced pre-hospital guidelines on withholding resuscitation.⁶⁵⁵

Treatment

Survival from TCRA is correlated with duration of CPR and pre-hospital time.^{644,656–660} Undertake only essential lifesaving interventions on scene and, if the patient has signs of life, transfer rapidly to the nearest appropriate hospital. Consider on scene thoracotomy for appropriate patients.^{661,662} Do not delay for unproven interventions such as spinal immobilization.⁶⁶³ Treat reversible causes: hypoxaemia (oxygenation, ventilation); compressible haemorrhage (pressure, pressure dressings, tourniquets, novel haemostatic agents); non-compressible haemorrhage (splints, intravenous fluid); tension pneumothorax (chest decompression); cardiac tamponade (immediate thoracotomy). Chest compressions may not be effective in hypovolaemic cardiac arrest, but most survivors do not have hypovolaemia and in this subgroup standard advanced life support may be lifesaving. Standard CPR should not delay the treatment of reversible causes (e.g., thoracotomy for cardiac tamponade).

Resuscitative thoracotomy

If physicians with appropriate skills are on scene, prehospital resuscitative thoracotomy may be indicated for selected patients with cardiac arrest associated with penetrating chest injury.

Emergency department thoracotomy (EDT) is best applied to patients with penetrating cardiac injuries who arrive at a trauma centre after a short on scene and transport time with witnessed signs of life or ECG activity (estimated survival rate 31%).⁶⁶⁴ After blunt trauma, EDT should be limited to those with vital signs on arrival and a witnessed cardiac arrest (estimated survival rate 1.6%).

Ultrasound

Ultrasound is a valuable tool for the evaluation of the compromised trauma patient. Haemoperitoneum, haemo- or pneumothorax and cardiac tamponade can be diagnosed reliably in minutes even in the pre-hospital phase.⁶⁶⁵ Pre-hospital ultrasound is now available, although its benefits are yet to be proven.⁶⁶⁶

Cardiac arrest associated with pregnancy

Mortality related to pregnancy in developed countries is rare, occurring in an estimated 1:30,000 deliveries.⁶⁶⁷ The fetus must always be considered when an adverse cardiovascular event occurs in a pregnant woman. Resuscitation guidelines for pregnancy are based largely on case series, extrapolation from non-pregnant arrests, manikin studies and expert opinion based on the physiology of pregnancy and changes that occur in normal labour. Studies tend to address causes in developed countries, whereas the most pregnancy-related deaths occur in developing countries. There were an estimated 342,900 maternal deaths (death during pregnancy, childbirth, or in the 42 days after delivery) worldwide in 2008.⁶⁶⁸

Causes of cardiac arrest in pregnant women include: cardiac disease; pulmonary embolism; psychiatric disorders; hypertensive disorders of pregnancy; sepsis; haemorrhage; amniotic fluid embolism; and ectopic pregnancy.⁶⁶⁹ Pregnant women can also sustain cardiac arrest from the same causes as women of the same age group.

Modifications to BLS guidelines for cardiac arrest during pregnancy

After 20 weeks' gestation, the pregnant woman's uterus can press down against the inferior vena cava and the aorta, impeding venous return and cardiac output. Uterine obstruction of venous return can cause pre-arrest hypotension or shock and, in the critically ill patient, may precipitate arrest.^{670,671} After cardiac arrest, the compromise in venous return and cardiac output by the gravid uterus limits the effectiveness of chest compressions.

The key steps for BLS in a pregnant patient are:

- Call for expert help early (including an obstetrician and neonatologist).
- Start basic life support according to standard guidelines. Ensure good quality chest compressions with minimal interruptions.
- Manually displace the uterus to the left to remove caval compression.
- Add left lateral tilt if this is feasible—the optimal angle of tilt is unknown. Aim for between 15 and 30°. The angle of tilt needs to allow good quality chest compressions and if needed allow caesarean delivery of the fetus (see below).

Modifications to advanced life support

There is a greater potential for gastro-oesophageal sphincter insufficiency and risk of pulmonary aspiration of gastric contents. Early tracheal intubation with correctly applied cricoid pressure decreases this risk. Tracheal intubation will make ventilation of the lungs easier in the presence of increased intra-abdominal pressure. A tracheal tube 0.5–1 mm internal diameter (ID) smaller than that used for a non-pregnant woman of similar size may be necessary because of maternal airway narrowing from oedema and swelling.⁶⁷² There is no change in transthoracic impedance during pregnancy, suggesting that standard shock energies for defibrillation attempts should be used in pregnant patients.⁶⁷³

Rescuers should attempt to identify common and reversible causes of cardiac arrest in pregnancy during resuscitation attempts. The 4Hs and 4Ts approach helps identify all the common causes of cardiac arrest in pregnancy. Pregnant patients are at risk of all the other causes of cardiac arrest for their age group (e.g., ana-phylaxis, drug overdose, trauma). Consider the use of abdominal ultrasound by a skilled operator to detect pregnancy and possible causes during cardiac arrest in pregnancy; however, do not delay other treatments.

If immediate resuscitation attempts fail

Consider the need for an emergency hysterotomy or caesarean section as soon as a pregnant woman goes into cardiac arrest. In some circumstances immediate resuscitation attempts will restore a perfusing rhythm; in early pregnancy this may enable the pregnancy to proceed to term. When initial resuscitation attempts fail, delivery of the fetus may improve the chances of successful resuscitation of the mother and fetus.^{674–676}

- At gestational age <20 weeks, urgent caesarean delivery need not be considered, because a gravid uterus of this size is unlikely to significantly compromise maternal cardiac output.
- At gestational age approximately 20–23 weeks, initiate emergency hysterotomy to enable successful resuscitation of the mother, not survival of the delivered infant, which is unlikely at this gestational age.
- At gestational age approximately ≥24–25 weeks, initiate emergency hysterotomy to save the life of both the mother and the infant.

The best survival rate for infants over 24–25 weeks' gestation occurs when delivery of the infant is achieved within 5 min after the mother's cardiac arrest. This requires that rescuers commence the hysterotomy at about 4 min after cardiac arrest.

Electrocution

Electrical injury is a relatively infrequent but potentially devastating multisystem injury with high morbidity and mortality, causing 0.54 deaths per 100,000 people each year. Most electrical injuries in adults occur in the workplace and are associated generally with high voltage, whereas children are at risk primarily at home, where the voltage is lower (220 V in Europe, Australia and Asia; 110 V in the USA and Canada).⁶⁷⁷ Electrocution from lightning strikes is rare, but worldwide it causes 1000 deaths each year.⁶⁷⁸

Electric shock injuries are caused by the direct effects of current on cell membranes and vascular smooth muscle. Respiratory arrest may be caused by paralysis of the central respiratory control system or the respiratory muscles. Current may precipitate VF if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon).⁶⁷⁹ Electrical current may also cause myocardial ischaemia because of coronary artery spasm. Asystole may be primary, or secondary to asphyxia following respiratory arrest.

Lightning strikes deliver as much as 300 kV over a few milliseconds. In those who survive the initial shock, extensive catecholamine release or autonomic stimulation may occur, causing hypertension, tachycardia, non-specific ECG changes (including prolongation of the QT interval and transient T-wave inversion), and myocardial necrosis. Mortality from lightning injuries is as high as 30%, with up to 70% of survivors sustaining significant morbidity.^{680–682}

Resuscitation

Ensure that any power source is switched off and do not approach the casualty until it is safe. Start standard basic and advanced life support without delay.

- Airway management may be difficult if there are electrical burns around the face and neck. Early tracheal intubation is needed in these cases, as extensive soft-tissue oedema may develop causing airway obstruction. Head and spine trauma can occur after

electrocution. Immobilize the spine until evaluation can be performed.

- Muscular paralysis, especially after high voltage, may persist for several hours⁶⁸¹; ventilatory support is required during this period.
- VF is the commonest initial arrhythmia after high-voltage AC shock; treat with prompt attempted defibrillation. Asystole is more common after DC shock; use standard protocols for this and other arrhythmias.
- Remove smoldering clothing and shoes to prevent further thermal injury.
- Vigorous fluid therapy is required if there is significant tissue destruction. Maintain a good urine output to enhance the excretion of myoglobin, potassium and other products of tissue damage.⁶⁸³
- Consider early surgical intervention in patients with severe thermal injuries.
- Maintain spinal immobilization if there is a likelihood of head or neck trauma.^{684,685}
- Conduct a thorough secondary survey to exclude traumatic injuries caused by tetanic muscular contraction or by the person being thrown.^{685,686}
- Electrocution can cause severe, deep soft-tissue injury with relatively minor skin wounds, because current tends to follow neurovascular bundles; look carefully for features of compartment syndrome, which will necessitate fasciotomy.

Principles of education in resuscitation

Survival from cardiac arrest is determined by the quality of the scientific evidence behind the guidelines, the effectiveness of education and the resources for implementation of the guidelines.⁶⁸⁷ An additional factor is how readily guidelines can be applied in clinical practice and the effect of human factors on putting the theory into practice.⁶⁸⁸ Implementation of Guidelines 2010 is likely to be more successful with a carefully planned, comprehensive implementation strategy that includes education. Delays in providing training materials and freeing staff for training were cited as reasons for delays in the implementation of the 2005 guidelines.^{689,690}

Key educational recommendations

The key issues identified by the Education, Implementation and Teams (EIT) task force of ILCOR during the Guidelines 2010 evidence evaluation process are¹⁹:

- Educational interventions should be evaluated to ensure that they reliably achieve the learning objectives. The aim is to ensure that learners acquire and retain the skills and knowledge that will enable them to act correctly in actual cardiac arrests and improve patient outcomes.
- Short video/computer self-instruction courses, with minimal or no instructor coaching, combined with hands-on practice can be considered as an effective alternative to instructor-led basic life support (CPR and AED) courses.
- Ideally all citizens should be trained in standard CPR that includes compressions and ventilations. There are circumstances however where training in compression-only CPR is appropriate (e.g., opportunistic training with very limited time). Those trained in compression-only CPR should be encouraged to learn standard CPR.
- Basic and advanced life support knowledge and skills deteriorate in as little as three to 6 months. The use of frequent assessments will identify those individuals who require refresher training to help maintain their knowledge and skills.

- CPR prompt or feedback devices improve CPR skill acquisition and retention and should be considered during CPR training for laypeople and healthcare professionals.
- An increased emphasis on non-technical skills (NTS) such as leadership, teamwork, task management and structured communication will help improve the performance of CPR and patient care.
- Team briefings to plan for resuscitation attempts, and debriefings based on performance during simulated or actual resuscitation attempts should be used to help improve resuscitation team and individual performance.
- Research about the impact of resuscitation training on actual patient outcomes is limited. Although manikin studies are useful, researchers should be encouraged to study and report the impact of educational interventions on actual patient outcomes.

Who and how to train

Ideally all citizens should have some knowledge of CPR. There is insufficient evidence for or against the use of training interventions that focus on high-risk populations. However, training can reduce family member and, or patient anxiety, improve emotional adjustment and empowers individuals to feel that they would be able to start CPR.¹⁹

People who require resuscitation training range from laypeople, those without formal healthcare training but with a role that places a duty of care upon them (e.g., lifeguards, first aiders), and healthcare professionals working in a variety of settings including the community, emergency medical systems (EMS), general hospital wards and critical care areas.

Training should be tailored to the needs of different types of learners and learning styles to ensure acquisition and retention of resuscitation knowledge and skills. Those who are expected to perform CPR regularly need to have knowledge of current guidelines and be able to use them effectively as part of a multi-professional team. These individuals require more complex training including both technical and non-technical skills (e.g., teamwork, leadership, structured communication skills).^{691,692} They are divided arbitrarily into basic level and advanced level training interventions whereas in truth this is a continuum.

Basic level and AED training

Bystander CPR and early defibrillation saves lives. Many factors decrease the willingness of bystanders to start CPR, including panic, fear of disease, harming the victim or performing CPR incorrectly.^{693–708} Providing CPR training to laypeople increases willingness to perform CPR.^{696,702–704,709–714}

CPR training and performing CPR during an actual cardiac arrest is safe in most circumstances. Individuals undertaking CPR training should be advised of the nature and extent of the physical activity required during the training program. Learners who develop significant symptoms (e.g., chest pain, severe shortness of breath) during CPR training should be advised to stop. Rescuers who develop significant symptoms during actual CPR should consider stopping CPR (see Basic life support guidelines for further information about risks to the rescuer).⁴

Basic life support and AED curriculum

The curriculum for basic life support and AED training should be tailored to the target audience and kept as simple as possible. The following should be considered as core elements of the basic life support and AED curriculum^{13,19}:

- Personal and environmental risks before starting CPR.
- Recognition of cardiac arrest by assessment of responsiveness, opening of the airway and assessment of breathing.^{4,13}
- Recognition of gasping or abnormal breathing as a sign of cardiac arrest in unconscious unresponsive individuals.^{69,715}
- Good quality chest compressions (including adherence to rate, depth, full recoil and minimizing hands-off time) and rescue breathing.
- Feedback/prompts (including from devices) during CPR training should be considered to improve skill acquisition and retention during basic life support training.⁷¹⁶
- All basic life support and AED training should aim to teach standard CPR including rescue breathing/ventilations. Chest compression-only CPR training has potential advantages over chest compression and ventilation in certain specific situations.^{694,699,702,707,708,711,717,718} An approach to teaching CPR is suggested below.

Standard CPR versus chest compression-only CPR teaching

There is controversy about which CPR skills different types of rescuers should be taught. Compression-only CPR is easier and quicker to teach especially when trying to teach a large number of individuals who would not otherwise access CPR training. In many situations however, standard CPR (which includes ventilation/rescuer breathing) is better, for example in children,⁸⁴ asphyxial arrests, and when bystander CPR is required for more than a few minutes.¹³ A simplified, education-based approach is therefore suggested:

- Ideally, full CPR skills (compressions and ventilation using a 30:2 ratio) should be taught to all citizens.
- When training is time-limited or opportunistic (e.g., EMS telephone instructions to a bystander, mass events, publicity campaigns, YouTube 'viral' videos, or the individual does not wish to train), training should focus on chest compression-only CPR.
- For those trained in compression-only CPR, subsequent training should include training in ventilation as well as chest compressions. Ideally these individuals should be trained in compression-only CPR and then offered training in chest compressions with ventilation at the same training session.
- Those laypersons with a duty of care, such as first aid workers, lifeguards, and child minders, should be taught how to do chest compressions and ventilations.
- For children, rescuers should be encouraged to use whichever adult sequence they have been taught, as outcome is worse if they do nothing. Non-specialists who wish to learn paediatric resuscitation because they have responsibility for children (e.g., parents, teachers, school nurses, lifeguards etc), should be taught that it is preferable to modify adult basic life support and give five initial breaths followed by approximately one minute of CPR before they go for help, if there is no-one to go for them. Chest compression depth for children is at least 1/3 of the A-P diameter of the chest.⁸

Citizen-CPR training should be promoted for all. However being untrained should not be a barrier to performing chest compression-only CPR, preferably with dispatcher telephone advice.

Basic life support and AED training methods

There are numerous methods to deliver basic life support and AED training. Traditional, instructor-led training courses remain the most frequently used method for basic life support and AED training.⁷¹⁹ When compared with traditional instructor-led training, well designed self-instruction programmes (e.g., video, DVD,

computer driven) with minimal or no instructor coaching can be effective alternatives to instructor-led courses for laypeople and healthcare providers learning basic life support and AED skills.^{720–734} It is essential that courses include hands-on practice as part of the programme. The use of CPR prompt/feedback devices may be considered during CPR training for laypeople and healthcare professionals.⁷¹⁶

Duration and frequency of instructor-led basic life support and AED training courses

The optimal duration of instructor-led basic life support and AED training courses has not been determined and is likely to vary according to the characteristics of the participants (e.g., lay or healthcare; previous training; age), the curriculum, the ratio of instructors to participants, the amount of hands-on training and the use of end of course assessments.

Most studies show that CPR skills such as calling for help, chest compressions and ventilations decay within 3–6 months after initial training.^{722,725,735–740} AED skills are retained for longer than basic life support skills alone.^{736,741,742}

Advanced level training

Advanced level training curriculum

Advanced level training is usually for healthcare providers. Curricula should be tailored to match individual learning needs, patient case mix and the individual's role within the healthcare system's response to cardiac arrest. Team training and rhythm recognition skills will be essential to minimize hands-off time when using the 2010 manual defibrillation strategy that includes charging during chest compressions.^{117,743}

Core elements for advanced life support curricula should include:

- Cardiac arrest prevention.^{192,744}
- Good quality chest compressions including adherence to rate, depth, full recoil and minimising hands-off time, and ventilation using basic skills (e.g., pocket mask, bag mask).
- Defibrillation including charging during compressions for manual defibrillation.
- Advanced life support algorithms.
- Non-technical skills (e.g., leadership and team training, communication).

Advanced level training methods

A variety of methods (such as reading manuals, pretests and e-learning can be used to prepare candidates before attending a life support course^{745–753}

Simulation and realistic training techniques

Simulation training is an essential part of resuscitation training. There is large variation in how simulation can be and is used for resuscitation training.⁷⁵⁴ The lack of consistent definitions (e.g., high vs. low fidelity simulation) makes comparisons of studies of different types of simulation training difficult.

Advanced life support training intervals

Knowledge and skill retention declines rapidly after initial resuscitation training. Refresher training is invariably required to maintain knowledge and skills; however, the optimal frequency for refresher training is unclear. Most studies show

that advanced life support skills and knowledge decayed when tested at three to 6 months after training,^{737,755–762} two studies suggested seven to 12 months,^{763,764} and one study 18 months.⁷⁶⁵

The ethics of resuscitation and end-of-life decisions

Several considerations are required to ensure that the decisions to attempt or withhold resuscitation attempts are appropriate, and that patients are treated with dignity. These decisions are complex and may be influenced by individual, international and local cultural, legal, traditional, religious, social and economic factors.⁷⁶⁶

The 2010 ERC Guidelines include the following topics relating to ethics and end-of-life decisions.

- Key principles of ethics.
- Sudden cardiac arrest in a global perspective.
- Outcome and prognostication.
- When to start and when to stop resuscitation attempts.
- Advance directives and do-not-attempt-resuscitation orders.
- Family presence during resuscitation.
- Organ procurement
- Research in resuscitation and informed content.
- Research and training on the recently dead.

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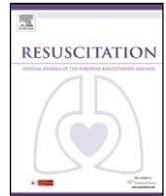
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European Resuscitation Council Guidelines for Resuscitation 2010 Section 2. Adult basic life support and use of automated external defibrillators

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Basic life support (BLS) refers to maintaining airway patency and supporting breathing and the circulation, without the use of equipment other than a protective device.¹ This section contains the guidelines for adult BLS and for the use of an automated external defibrillator (AED). It also includes recognition of sudden cardiac arrest, the recovery position and management of choking (foreign-body airway obstruction). Guidelines for the use of manual defibrillators and starting in-hospital resuscitation are found in Sections 3 and 4.^{2,3}

Summary of changes since 2005 Guidelines

Many of the recommendations made in the ERC Guidelines 2005 remain unchanged, either because no new studies have been published or because new evidence since 2005 has merely strengthened the evidence that was already available. Examples of this are the general design of the BLS and AED algorithms, the way the need for cardiopulmonary resuscitation (CPR) is recognised, the use of AEDs (including the shock protocols), the 30:2 ratio of compressions and ventilations, and the recognition and management of a choking victim. In contrast, new evidence has been published since 2005 that necessitates changes to some components of the 2010 Guidelines. The 2010 changes in comparison with the 2005 Guidelines are summarised here:

- Dispatchers should be trained to interrogate callers with strict protocols to elicit information. This information should focus on the recognition of unresponsiveness and the quality of breathing. In combination with unresponsiveness, absence of breathing or any abnormality of breathing should start a dispatch protocol of suspected cardiac arrest. The importance of gasping as sign of cardiac arrest should result in increased emphasis on its recognition during training and dispatch interrogation.
- All rescuers, trained or not, should provide chest compressions to victims of cardiac arrest. A strong emphasis on delivering high quality chest compressions remains essential. The aim should be to push to a depth of at least 5 cm at a rate of at least 100 compressions per minute, to allow full chest recoil, and to minimise interruptions in chest compressions. Trained rescuers should also provide ventilations with a compression–ventilation ratio of 30:2. Telephone-guided CPR is encouraged for untrained rescuers who should be told to deliver uninterrupted chest compressions only.
- In order to maintain high-quality CPR, feedback to rescuers is important. The use of prompt/feedback devices during CPR will enable immediate feedback to rescuers, and the data stored in rescue equipment can be used to monitor the quality of CPR performance and provide feedback to professional rescuers during debriefing sessions.
- When rescuers apply an AED, the analysis of the heart rhythm and delivery of a shock should not be delayed for a period of CPR; however, CPR should be given with minimal interruptions before application of the AED and during its use.
- Further development of AED programmes is encouraged—there is a need for further deployment of AEDs in both public and residential areas.

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Introduction

Sudden cardiac arrest (SCA) is a leading cause of death in Europe. Depending on the way SCA is defined, it affects about 350,000–700,000 individuals a year.^{4,5} On initial heart-rhythm analysis, about 25–30% of SCA victims have ventricular fibrillation (VF), a percentage that has declined over the last 20 years.^{6–10} It is likely that many more victims have VF or rapid ventricular tachycardia (VT) at the time of collapse but, by the time the first electrocardiogram (ECG) is recorded by ambulance personnel, their rhythm has deteriorated to asystole.^{11,12} When the rhythm is recorded soon after collapse, in particular by an on-site AED, the proportion of victims in VF can be as high as 59%¹³ to 65%.¹⁴ Many victims of SCA can survive if bystanders act immediately while VF is still present, but successful resuscitation is much less likely once the rhythm has deteriorated to asystole.

The recommended treatment for VF cardiac arrest is immediate bystander CPR (combined chest compression and rescue breathing) and early electrical defibrillation. Most cardiac arrests of non-cardiac origin are from respiratory causes such as drowning (among them many children) and asphyxia. In many areas in the world drowning is a major cause of death (see http://www.who.int/water_sanitation_health/diseases/drowning/en/), and rescue breaths are even more critical for successful resuscitation of these victims.

The chain of survival

The following concept of the Chain of Survival summarises the vital steps needed for successful resuscitation (Fig. 2.1). Most of these links apply to victims of both primary cardiac and asphyxial arrest.¹⁵

1. *Early recognition of cardiac arrest:* This includes recognition of the cardiac origin of chest pain; recognition that cardiac arrest has occurred; and rapid activation of the ambulance service by telephoning 112 or the local emergency number. Recognizing cardiac chest pain is particularly important, since the probability of cardiac arrest occurring as a consequence of acute myocardial ischaemia is at least 21–33% in the first hour after onset of symptoms.^{16,17} When a call to the ambulance service is made before a victim collapses, arrival of the ambulance is significantly sooner after collapse and survival tends to be higher.¹⁸
2. *Early bystander CPR:* Immediate CPR can double or triple survival from VF SCA.^{18–21} Performing chest-compression-only CPR is better than giving no CPR at all.^{22,23} When a caller has not been trained in CPR, the ambulance dispatcher should strongly

encourage him to give chest compression-only CPR while awaiting the arrival of professional help.^{24–27}

3. *Early defibrillation:* CPR plus defibrillation within 3–5 min of collapse can produce survival rates as high as 49–75%.^{28–35} Each minute of delay before defibrillation reduces the probability of survival to discharge by 10–12%.^{19,36}
4. *Early advanced life support and standardised post-resuscitation care:* The quality of treatment during the post-resuscitation phase affects outcome.^{37–39} Therapeutic hypothermia is now an established therapy that greatly contributes to improved survival with good neurological outcome.^{40–42}

In most communities, the median time from ambulance call to ambulance arrival (response interval) is 5–8 min,^{13,14} or 11 min to a first shock.⁴³ During this time the victim's survival is dependent on bystanders who initiate BLS and use an AED for defibrillation.

Victims of cardiac arrest need immediate CPR. This provides a small but critical blood flow to the heart and brain. It also increases the likelihood that a defibrillatory shock will terminate VF and enable the heart to resume an effective rhythm and cardiac output. Chest compression is especially important if a shock cannot be delivered sooner than the first few minutes after collapse.⁴⁴ After defibrillation, if the heart is still viable, its pacemaker activity resumes and produces an organised rhythm followed by mechanical contraction. In the first few minutes after successful termination of VF, the heart rhythm may be slow, and the force of contractions weak; chest compressions must be continued until adequate cardiac function returns.⁴⁵

Lay rescuers can be trained to use automated external defibrillators (AEDs), which are increasingly available in public places. An AED uses voice prompts to guide the rescuer, analyses the cardiac rhythm and instructs the rescuer to deliver a shock if VF or rapid ventricular tachycardia (VT) is detected. AEDs are extremely accurate and will deliver a shock only when VF (or rapid VT) is present.⁴⁶ AED function and operation are discussed in Section 3.

Several studies have shown the benefit on survival of immediate CPR, and the detrimental effect of delay before defibrillation. For every minute delay in defibrillation, survival from witnessed VF decreases by 10–12%.^{19,36} When bystander CPR is provided, the decline in survival is more gradual and averages 3–4% per minute.^{12,36,47} Overall, bystander CPR doubles or triples survival from witnessed cardiac arrest.^{19,47,48}

Adult BLS sequence

Throughout this section, the male gender implies both males and females.

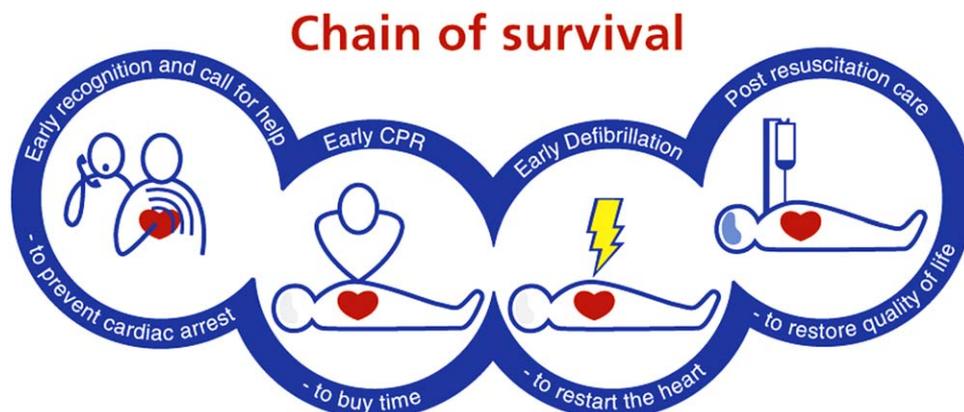


Fig. 2.1. Chain of survival.

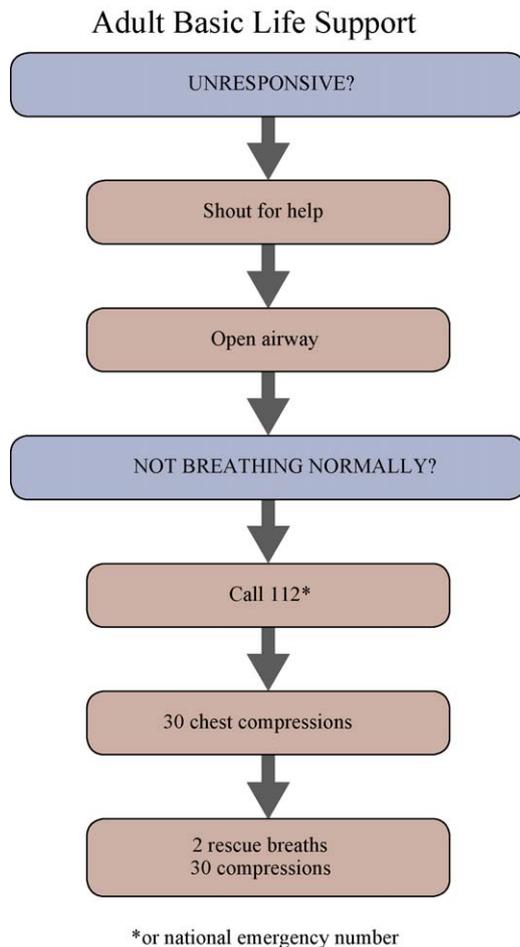


Fig. 2.2. Adult basic life support algorithm.

BLS consists of the following sequence of actions (Fig. 2.2).

1. Make sure you, the victim and any bystanders are safe.
2. Check the victim for a response (Fig. 2.3).
 - gently shake his shoulders and ask loudly: “Are you all right?”
- 3a. If he responds
 - leave him in the position in which you find him, provided there is no further danger;
 - try to find out what is wrong with him and get help if needed;
 - reassess him regularly.
- 3b. If he does *not* respond
 - shout for help (Fig. 2.4)
 - turn the victim onto his back and then open the airway using head tilt and chin lift (Fig. 2.5);
 - place your hand on his forehead and gently tilt his head back;
 - with your fingertips under the point of the victim’s chin, lift the chin to open the airway.
4. Keeping the airway open, look, listen and feel for breathing (Fig. 2.6).
 - look for chest movement;
 - listen at the victim’s mouth for breath sounds;
 - feel for air on your cheek;
 - decide if breathing is normal, not normal or absent

In the first few minutes after cardiac arrest, a victim may be barely breathing, or taking infrequent, slow and noisy gasps. Do not confuse this with normal breathing. Look, listen and feel for *no more* than 10 s to determine whether the victim is breathing normally. If you have any doubt whether breathing is normal, act as if it is not normal.



Fig. 2.3. Check the victim for a response.

- 5a. If he is breathing normally
 - turn him into the recovery position (see below);
 - send or go for help—call 112 or local emergency number for an ambulance;
 - continue to assess that breathing remains normal.
- 5b. If the breathing is *not* normal or absent
 - send someone for help and to find and bring an AED if available; or if you are on your own, use your mobile telephone to alert the ambulance service—leave the victim only when there is no other option.



Fig. 2.4. Shout for help.



Fig. 2.5. Head tilt and chin lift.

- start chest compression as follows:
 - kneel by the side of the victim;
 - place the heel of one hand in the centre of the victim's chest; (which is the lower half of the victim's breastbone (sternum)) (Fig. 2.7);
 - place the heel of your other hand on top of the first hand (Fig. 2.8);
 - interlock the fingers of your hands and ensure that pressure is not applied over the victim's ribs. Keep your arms straight (Fig. 2.9). Do not apply any pressure over the upper abdomen or the bottom end of the bony sternum (breastbone);
 - position yourself vertically above the victim's chest and press down on the sternum at least 5 cm (but not exceeding 6 cm) (Fig. 2.10);
 - after each compression, release all the pressure on the chest without losing contact between your hands and the



Fig. 2.6. Look, listen and feel for normal breathing.

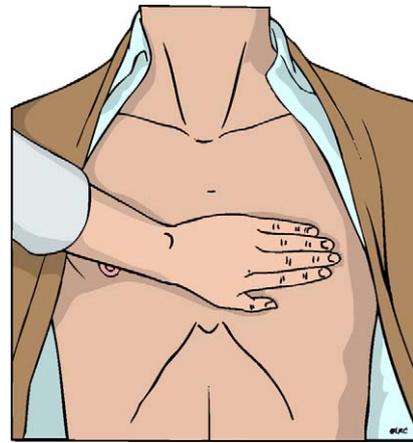


Fig. 2.7. Place the heel of one hand in the centre of the victim's chest.

sternum; repeat at a rate of at least 100 min^{-1} (but not exceeding 120 min^{-1});

- compression and release should take equal amounts of time.

6a. Combine chest compression with rescue breaths.

- After 30 compressions open the airway again using head tilt and chin lift (Fig. 2.5).
- Pinch the soft part of the nose closed, using the index finger and thumb of your hand on the forehead.
- Allow the mouth to open, but maintain chin lift.
- Take a normal breath and place your lips around his mouth, making sure that you have a good seal.
- Blow steadily into the mouth while watching for the chest to rise (Fig. 2.11), taking about 1 s as in normal breathing; this is an effective rescue breath.
- Maintaining head tilt and chin lift, take your mouth away from the victim and watch for the chest to fall as air comes out (Fig. 2.12).
- Take another normal breath and blow into the victim's mouth once more to achieve a total of two effective rescue breaths. The two breaths should not take more than 5 s in all. Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions.
- Continue with chest compressions and rescue breaths in a ratio of 30:2.
- Stop to recheck the victim only if he starts to wake up: to move, open eyes and to breathe normally. Otherwise, do not interrupt resuscitation.

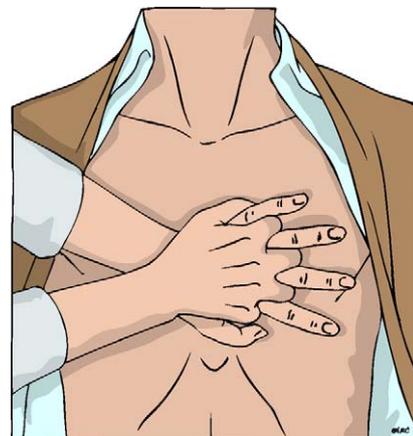


Fig. 2.8. Place the heel of your other hand on top of the first hand.



Fig. 2.9. Interlock the fingers of your hands. Keep your arms straight.

If your initial rescue breath does not make the chest rise as in normal breathing, then before your next attempt:

- look into the victim's mouth and remove any obstruction;
- recheck that there is adequate head tilt and chin lift;
- do not attempt more than two breaths each time before returning to chest compressions.

If there is more than one rescuer present, another rescuer should take over delivering CPR every 2 min to prevent fatigue. Ensure that interruption of chest compressions is minimal during the changeover of rescuers. For this purpose, and to count 30 compressions at the required rate, it may be helpful for the rescuer performing chest compressions to count out loud. Experienced rescuers could do combined two-rescuer CPR and in that situation they should exchange roles/places every 2 min.

6b. Chest-compression-only CPR may be used as follows:

- If you are not trained, or are unwilling to give rescue breaths, give chest compressions only.
- If only chest compressions are given, these should be continuous, at a rate of at least 100 min^{-1} (but not exceeding 120 min^{-1}).

7. Do not interrupt resuscitation until:

- professional help arrives and takes over; or
- the victim starts to wake up: to move, open eyes and to breathe normally; or
- you become exhausted.

Opening the airway

The jaw thrust is not recommended for lay rescuers because it is difficult to learn and perform and may itself cause spinal



Fig. 2.10. Press down on the sternum at least 5 cm.

movement.⁴⁹ Therefore, the lay rescuer should open the airway using a head-tilt-chin-lift manoeuvre for both injured and non-injured victims.

Recognition of cardiorespiratory arrest

Checking the carotid pulse (or any other pulse) is an inaccurate method of confirming the presence or absence of circulation, both for lay rescuers and for professionals.^{50–52} There is, however, no evidence that checking for movement, breathing or coughing (“signs of a circulation”) is diagnostically superior. Healthcare



Fig. 2.11. Blow steadily into his mouth whilst watching for his chest to rise.



Fig. 2.12. Take your mouth away from the victim and watch for his chest to fall as air comes out.

professionals, as well as lay rescuers, have difficulty determining the presence or absence of adequate or normal breathing in unresponsive victims.^{53,54} This may be because the airway is not open or because the victim is making occasional (agonal) gasps. When bystanders are asked by ambulance dispatchers over the telephone if breathing is present, they often misinterpret agonal gasps as normal breathing. This incorrect information can result in the bystander withholding CPR from a cardiac arrest victim.⁵⁵ Agonal gasps are present in up to 40% of cardiac arrest victims in the first minutes after onset, and are associated with higher survival if recognised as a sign of cardiac arrest.⁵⁶ Bystanders describe agonal gasps as barely breathing, heavy or laboured breathing, or noisy or gasping breathing.⁵⁷ Laypeople should, therefore, be taught to begin CPR if the victim is unconscious (unresponsive) and not breathing normally. It should be emphasised during training that agonal gasps occur commonly in the first few minutes after SCA, and that they are an indication to start CPR immediately; they should not be confused with normal breathing.

Adequate description of the victim is also of critical importance during communication with the ambulance dispatch centre. It is important for the dispatcher that the caller can see the victim, but in a small minority of cases the caller is not at the scene.⁵⁸ Information about a victim's breathing is most important, but the description of breathing by callers varies considerably. If the nature of the victim's breathing is not described or actively asked for by the dispatcher, recognition that the victim has had a cardiac arrest is much less likely than if the breathing is described as abnormal or absent.⁵⁹ If, when a caller describes an unconscious victim with absent or abnormal breathing, the dispatcher always responded as for cardiac arrest, cases of cardiac arrest would not be missed.⁶⁰

Confirming the absence of a past medical history of seizures can increase the likelihood of recognizing cardiac arrest among victims presenting with seizure activity.^{59,61} Asking about regularity of breathing can also help to recognise cardiac arrest among callers reporting seizure activity.

An experienced dispatcher can improve the survival rate significantly: if the dispatcher takes very few cardiac arrest calls per year, the survival rate is much lower than if he takes more than nine calls a year (22% versus 39%).⁵⁸ The accuracy of identification of cardiac arrest by dispatchers varies from approximately 50% to over 80%. If the dispatcher recognises cardiac arrest, survival is more likely because appropriate measures can be taken (e.g. telephone-instructed CPR or appropriate ambulance response).^{25,60}

Initial rescue breaths

In primary (non-asphyxial) cardiac arrest the arterial blood is not moving and remains saturated with oxygen for several minutes.⁶² If CPR is initiated within a few minutes, the blood oxygen content remains adequate, and myocardial and cerebral oxygen delivery is limited more by the reduced cardiac output than by a lack of oxygen in the lungs and arterial blood. Initially, therefore, ventilation is less important than chest compressions.^{63,64}

In adults needing CPR, there is a high a-priori probability of a primary cardiac cause. To emphasise the priority of chest compressions, it is recommended that CPR should start with chest compression rather than initial ventilations. Time should not be spent checking the mouth for foreign bodies unless attempted rescue breathing fails to make the chest rise.

Ventilation

During CPR, the purpose of ventilation is to maintain adequate oxygenation and to remove CO₂. The optimal tidal volume, respiratory rate and inspired oxygen concentration to achieve this, however, are not fully known. The current recommendations are based on the following evidence:

1. During CPR, blood flow to the lungs is substantially reduced, so an adequate ventilation–perfusion ratio can be maintained with lower tidal volumes and respiratory rates than normal.⁶⁵
2. Hyperventilation is harmful because it increases intrathoracic pressure, which decreases venous return to the heart and reduces cardiac output. Survival is consequently reduced.⁶⁶
3. Interruptions in chest compression (for example, to check the heart rhythm or for a pulse check) have a detrimental effect on survival.⁶⁷
4. When the airway is unprotected, a tidal volume of 1 l produces significantly more gastric distension than a tidal volume of 500 ml.⁶⁸
5. Low minute-ventilation (lower than normal tidal volume and respiratory rate) can maintain effective oxygenation and ventilation during CPR.^{69–72} During adult CPR, tidal volumes of approximately 500–600 ml (6–7 ml kg⁻¹) are recommended.

The current recommendations are, therefore, for rescuers to give each rescue breath over about 1 s, with enough volume to make the victim's chest rise, but to avoid rapid or forceful breaths. The time taken to give two breaths should not exceed 5 s. These recommendations apply to all forms of ventilation during CPR, including mouth-to-mouth and bag-mask ventilation with and without supplementary oxygen.

Mouth-to-nose ventilation is an acceptable alternative to mouth-to-mouth ventilation.⁷³ It may be considered if the victim's mouth is seriously injured or cannot be opened, the rescuer is assisting a victim in the water, or a mouth-to-mouth seal is difficult to achieve.

There is no published evidence on the safety, effectiveness or feasibility of mouth-to-tracheostomy ventilation, but it may be used for a victim with a tracheostomy tube or tracheal stoma who requires rescue breathing.

To use bag-mask ventilation requires considerable practice and skill.^{74,75} It can be used by properly trained and experienced rescuers who perform two-rescuer CPR.

Chest compression

Chest compressions produce blood flow by increasing the intrathoracic pressure and by directly compressing the heart. Although chest compressions, performed properly, can produce

systolic arterial pressure peaks of 60–80 mm Hg, diastolic pressure remains low and mean arterial pressure in the carotid artery seldom exceeds 40 mm Hg.⁷⁶ Chest compressions generate a small but critical amount of blood flow to the brain and myocardium and increase the likelihood that defibrillation will be successful.

Since the 2005 Guidelines were published, chest compression prompt/feedback devices have generated new data from victims in cardiac arrest that supplement animal and manikin studies.^{77–81} Recommendations based on this evidence are:

1. Each time compressions are resumed, place your hands without delay 'in the centre of the chest'.
2. Compress the chest at a rate of at least 100 min⁻¹.
3. Ensure that the full compression depth of at least 5 cm (for an adult) is achieved.
4. Allow the chest to recoil completely after each compression, i.e. do not lean on the chest during the relaxation phase of the chest compression.
5. Take approximately the same amount of time for compression as relaxation.
6. Minimise interruptions in chest compression in order to ensure the victim receives at least 60 compressions in each minute.
7. Do not rely on feeling the carotid or other pulse as a gauge of effective arterial flow during chest compressions.^{50,82}

Hand position

For adults receiving chest compressions, rescuers should place their hands on the lower half of the sternum. It is recommended that this location be taught in a simplified way, such as, "place the heel of your hand in the centre of the chest with the other hand on top." This instruction should be accompanied by demonstrating placing the hands on the lower half of the sternum on a manikin. Use of the inter nipple line as a landmark for hand placement is not reliable.^{83,84}

Compression rate

There is a positive relationship between the number of compressions actually delivered per minute and the chance of successful resuscitation.⁸¹ While the compression rate (the speed at which the 30 consecutive compressions are given) should be at least 100 min⁻¹, the actual number of compressions delivered during each minute of CPR will be lower due to interruptions to deliver rescue breaths and allow AED analysis, etc. In one out-of-hospital study, rescuers recorded compression rates of 100–120 min⁻¹ but the mean number of compressions was reduced to 64 min⁻¹ by frequent interruptions.⁷⁹ At least 60 compressions should be delivered each minute.

Compression depth

Fear of doing harm, fatigue and limited muscle strength frequently result in rescuers compressing the chest less deeply than recommended. There is evidence that a compression depth of 5 cm and over results in a higher rate of return of spontaneous circulation (ROSC), and a higher percentage of victims admitted alive to hospital, than a compression depth of 4 cm or below.^{77,78} There is no direct evidence that damage from chest compression is related to compression depth, nor has an upper limit of compression depth been established in studies. Nevertheless, it is recommended that, even in large adults, chest compression depth should not exceed 6 cm.

CPR should be performed on a firm surface when possible. Air-filled mattresses should be routinely deflated during CPR.⁸⁵ There is no evidence for or against the use of backboards,^{86,87} but if one

is used, care should be taken to avoid interruption in CPR and dislodging intravenous lines or other tubes during board placement.

Chest decompression

Allowing complete recoil of the chest after each compression results in better venous return to the chest and may improve the effectiveness of CPR.^{88,89} The optimal method of achieving this goal, without compromising other aspects of chest compression technique such as compression depth, has not, however, been established.

Feedback on compression technique

Rescuers can be assisted to achieve the recommended compression rate and depth by prompt/feedback devices that are either built into the AED or manual defibrillator, or are stand-alone devices. The use of such prompt/feedback devices, as part of an overall strategy to improve the quality of CPR, may be beneficial. Rescuers should be aware that the accuracy of devices that measure compression depth varies according to the stiffness of the support surface upon which CPR is being performed (e.g. floor/mattress), and may overestimate compression depth.⁸⁷ Further studies are needed to determine if these devices improve victim outcomes.

Compression–ventilation ratio

Animal data supported an increase in the ratio of compression to ventilation to >15:2.^{90–92} A mathematical model suggests that a ratio of 30:2 provides the best compromise between blood flow and oxygen delivery.^{93,94} A ratio of 30 compressions to 2 ventilations was recommended in the Guidelines 2005 for the single rescuer attempting resuscitation of an adult or child out of hospital, an exception being that a trained healthcare professional should use a ratio of 15:2 for a child. This decreased the number of interruptions in compression and the no-flow fraction,^{95,96} and reduced the likelihood of hyperventilation.^{66,97} Direct evidence that survival rates have increased from this change, however, is lacking. Likewise, there is no new evidence that would suggest a change in the recommended compression to ventilation ratio of 30:2.

Compression-only CPR

Some healthcare professionals as well as lay rescuers indicate that they would be reluctant to perform mouth-to-mouth ventilation, especially in unknown victims of cardiac arrest.^{98,99} Animal studies have shown that chest-compression-only CPR may be as effective as combined ventilation and compression in the first few minutes after non-asphyxial arrest.^{63,100} If the airway is open, occasional gasps and passive chest recoil may provide some air exchange, but this may result in ventilation of the dead space only.^{56,101–103} Animal and mathematical model studies of chest-compression-only CPR have shown that arterial oxygen stores deplete in 2–4 min.^{92,104}

In adults, the outcome of chest compression without ventilation is significantly better than the outcome of giving no CPR at all in non-asphyxial arrest.^{22,23} Several studies of human cardiac arrest have suggested equivalence of chest-compression-only CPR and chest compressions combined with rescue breaths, but none excluded the possibility that chest-compression-only is inferior to chest compressions combined with ventilations.^{23,105} One study suggested superiority of chest-compression-only CPR.²² All these studies have significant limitations because they were based on retrospective database analyses, where the type of BLS was not controlled and did not include CPR according to Guidelines

2005 (30:2 compressions to ventilation ratio). Chest compression alone may be sufficient only in the first few minutes after collapse. Professional help can be expected on average 8 min or later after a call for help, and chest compression only will result in insufficient CPR in many cases. Chest-compression-only CPR is not as effective as conventional CPR for cardiac arrests of non-cardiac origin (e.g., drowning or suffocation) in adults and children.^{106,107}

Chest compression combined with rescue breaths is, therefore, the method of choice for CPR delivered by both trained lay rescuers and professionals. Laypeople should be encouraged to perform compression-only CPR if they are unable or unwilling to provide rescue breaths, or when instructed during an emergency call to an ambulance dispatcher centre.^{26,27}

CPR in confined spaces

Over-the-head CPR for single rescuers and straddle-CPR for two rescuers may be considered for resuscitation in confined spaces.^{108,109}

Risks to the victim during CPR

Many rescuers, concerned that delivering chest compressions to a victim who is not in cardiac arrest will cause serious complications, do not initiate CPR. In a study of dispatch-assisted bystander CPR, however, where non-arrest victims received chest compressions, 12% experienced discomfort but only 2% suffered a fracture: no victims suffered visceral organ injury.¹¹⁰ Bystander CPR extremely rarely leads to serious harm in victims who are eventually found not to be in cardiac arrest. Rescuers should not, therefore, be reluctant to initiate CPR because of concern of causing harm.

Risks to the rescuer during training and during real-life CPR

Physical effects

Observational studies of training or actual CPR performance have described rare occurrences of muscle strain, back symptoms, shortness of breath, hyperventilation, and case reports of pneumothorax, chest pain, myocardial infarction and nerve injury.^{111,112} The incidence of these events is very low, and CPR training and actual performance is safe in most circumstances.¹¹³ Individuals undertaking CPR training should be advised of the nature and extent of the physical activity required during the training programme. Learners and rescuers who develop significant symptoms (e.g. chest pain or severe shortness of breath) during CPR training should be advised to stop.

Rescuer fatigue

Several manikin studies have found that chest compression depth can decrease as little as 2 min after starting chest compressions. An in-hospital patient study showed that, even while using real-time feedback, the mean depth of compression deteriorated between 1.5 and 3 min after starting CPR.¹¹⁴ It is therefore recommended that rescuers change about every 2 min to prevent a decrease in compression quality due to rescuer fatigue. Changing rescuers should not interrupt chest compressions.

Risks during defibrillation

A large randomised trial of public access defibrillation showed that AEDs can be used safely by laypeople and first responders.¹¹⁵ A systematic review identified eight papers that reported a total

of 29 adverse events associated with defibrillation.¹¹⁶ The causes included accidental or intentional defibrillator misuse, device malfunction and accidental discharge during training or maintenance procedures. Four single-case reports described shocks to rescuers from discharging implantable cardioverter defibrillators (ICDs), in one case resulting in a peripheral nerve injury. There are no reports of harm to rescuers from attempting defibrillation in wet environments.

Injury to the rescuer from defibrillation is extremely rare. Nevertheless, rescuers should not continue manual chest compressions during shock delivery. Victims should not be touched during ICD discharge. Direct contact between the rescuer and the victim should be avoided when defibrillation is carried out in wet environments.

Psychological effects

One large, prospective trial of public access defibrillation reported a few adverse psychological effects associated with CPR or AED use that required intervention.¹¹³ Two large, retrospective, questionnaire-based reports relating to performance of CPR by a bystander reported that nearly all respondents regarded their intervention as a positive experience.^{117,118} The rare occurrences of adverse psychological effects in rescuers after CPR should be recognised and managed appropriately.

Disease transmission

There are only very few cases reported where performing CPR has been linked to disease transmission, implicating *Salmonella infantis*, *Staphylococcus aureus*, severe acute respiratory syndrome (SARS), meningococcal meningitis, *Helicobacter pylori*, Herpes simplex virus, cutaneous tuberculosis, stomatitis, tracheitis, *Shigella* and *Streptococcus pyogenes*. One report described herpes simplex virus infection as a result of training in CPR. One systematic review found that in the absence of high-risk activities, such as intravenous cannulation, there were no reports of transmission of hepatitis B, hepatitis C, human immunodeficiency virus (HIV) or cytomegalovirus during either training or actual CPR.¹¹⁹

The risk of disease transmission during training and actual CPR performance is extremely low. Wearing gloves during CPR is reasonable, but CPR should not be delayed or withheld if gloves are not available. Rescuers should take appropriate safety precautions if a victim is known to have a serious infection (e.g. HIV, tuberculosis, hepatitis B virus or SARS).

Barrier devices

No human studies have addressed the safety, effectiveness or feasibility of using barrier devices (such as a face shield or face mask) to prevent victim contact during rescuer breathing. Two studies showed that barrier devices decreased transmission of bacteria in controlled laboratory settings.^{120,121} Because the risk of disease transmission is very low, initiating rescue breathing without a barrier device is reasonable. If the victim is known to have a serious infection (e.g. HIV, tuberculosis, hepatitis B virus, or SARS) a barrier device is recommended.

Recovery position

There are several variations of the recovery position, each with its own advantages. No single position is perfect for all victims.^{122,123} The position should be stable, near to a true lateral position with the head dependent, and with no pressure on the



Fig. 2.13. Place the arm nearest to you out at right angles to his body, elbow bent with the hand palm uppermost.



Fig. 2.14. Bring the far arm across the chest, and hold the back of the hand against the victim's cheek nearest to you.

chest to impair breathing.¹²⁴The ERC recommends the following sequence of actions to place a victim in the recovery position:

- Kneel beside the victim and make sure that both legs are straight.
- Place the arm nearest to you out at right angles to the body, elbow bent with the hand palm uppermost (Fig. 2.13).
- Bring the far arm across the chest, and hold the back of the hand against the victim's cheek nearest to you (Fig. 2.14).
- With your other hand, grasp the far leg just above the knee and pull it up, keeping the foot on the ground (Fig. 2.15).
- Keeping the hand pressed against the cheek, pull on the far leg to roll the victim towards you onto his side.
- Adjust the upper leg so that both hip and knee are bent at right angles.
- Tilt the head back to make sure the airway remains open.
- Adjust the hand under the cheek, if necessary, to keep the head tilted and facing downwards to allow liquid material to drain from the mouth (Fig. 2.16).
- Check breathing regularly.

If the victim has to be kept in the recovery position for more than 30 min, turn him to the opposite side to relieve the pressure on the lower arm.



Fig. 2.15. With your other hand, grasp the far leg just above the knee and pull it up, keeping the foot on the ground.



Fig. 2.16. The recovery position completed. Keep the head tilted to keep the airway open. Keep the face downward to allow fluids to go out.

Foreign-body airway obstruction (choking)

Foreign-body airway obstruction (FBAO) is an uncommon but potentially treatable cause of accidental death.¹²⁵ As most choking events are associated with eating, they are commonly witnessed. Thus, there is often the opportunity for early intervention while the victim is still responsive.

Recognition

Because recognition of airway obstruction is the key to successful outcome, it is important not to confuse this emergency with fainting, myocardial infarction, seizure or other conditions that may cause sudden respiratory distress, cyanosis or loss of consciousness. Foreign bodies may cause either mild or severe airway obstruction. The signs and symptoms enabling differentiation between mild and severe airway obstruction are summarised in Table 2.1. It is important to ask the conscious victim "Are you choking?"

Table 2.1
Differentiation between mild and severe foreign body airway obstruction (FBAO).^a

Sign	Mild obstruction	Severe obstruction
"Are you choking?"	"Yes"	Unable to speak, may nod
Other signs	Can speak, cough, breathe	Cannot breathe/wheezy breathing/silent attempts to cough/unconsciousness

^a General signs of FBAO: attack occurs while eating; victim may clutch his neck.

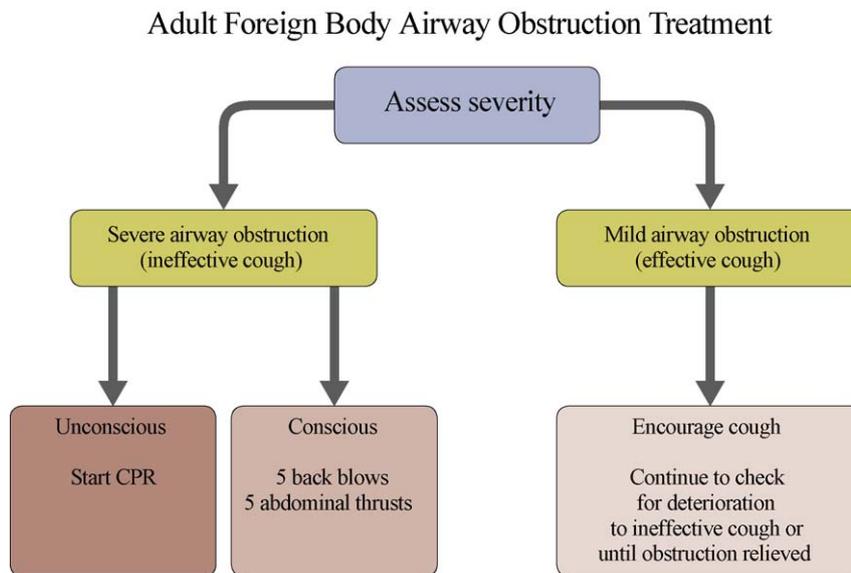


Fig. 2.17. Adult foreign body airway obstruction treatment algorithm.

Adult foreign-body airway obstruction (choking) sequence (this sequence is also suitable for use in children over the age of 1 year) (Fig. 2.17)

1. If the victim shows signs of mild airway obstruction:
 - Encourage continued coughing but do nothing else.
2. If the victim shows signs of severe airway obstruction and is conscious:
 - Apply five back blows as follows:
 - stand to the side and slightly behind the victim;
 - support the chest with one hand and lean the victim well forwards so that when the obstructing object is dislodged it comes out of the mouth rather than goes further down the airway;
 - give five sharp blows between the shoulder blades with the heel of your other hand.
 - If five back blows fail to relieve the airway obstruction, give five abdominal thrusts as follows:
 - stand behind the victim and put both arms round the upper part of the abdomen;
 - lean the victim forwards;
 - clench your fist and place it between the umbilicus (navel) and the ribcage;
 - grasp this hand with your other hand and pull sharply inwards and upwards;
 - repeat five times.
 - If the obstruction is still not relieved, continue alternating five back blows with five abdominal thrusts.
3. If the victim at any time becomes unconscious:
 - support the victim carefully to the ground;
 - immediately activate the ambulance service;
 - begin CPR with chest compressions.

Foreign-body airway obstruction causing mild airway obstruction

Coughing generates high and sustained airway pressures and may expel the foreign body. Aggressive treatment, with back blows, abdominal thrusts and chest compression, may cause potentially serious complications and could worsen the airway obstruction. It should be reserved for victims who have signs of severe airway obstruction. Victims with mild airway obstruction should remain

under continuous observation until they improve, as severe airway obstruction may subsequently develop.

Foreign-body airway obstruction with severe airway obstruction

The clinical data on choking are largely retrospective and anecdotal. For conscious adults and children over 1 year with a complete FBAO, case reports demonstrate the effectiveness of back blows or “slaps”, abdominal thrusts and chest thrusts.¹²⁶ Approximately 50% of episodes of airway obstruction are not relieved by a single technique.¹²⁷ The likelihood of success is increased when combinations of back blows or slaps, and abdominal and chest thrusts are used.¹²⁶

A randomised trial in cadavers¹²⁸ and two prospective studies in anaesthetised volunteers^{129,130} showed that higher airway pressures can be generated using chest thrusts compared with abdominal thrusts. Since chest thrusts are virtually identical to chest compressions, rescuers should be taught to start CPR if a victim of known or suspected FBAO becomes unconscious. The purpose of the chest compressions is primarily to attempt to remove the airway obstruction in the unconscious and supine victim, and only secondarily to promote circulation. Therefore, chest compressions are required even when a professional rescuer still feels a pulse. If the obstruction is not relieved, progressive bradycardia and asystole will occur. During CPR for choking, each time the airway is opened the victim’s mouth should be quickly checked for any foreign body that has been partly expelled. During CPR in other cases, therefore, a routine check of the mouth for foreign bodies is not necessary.

The finger sweep

No studies have evaluated the routine use of a finger sweep to clear the airway in the absence of visible airway obstruction,^{131–133} and four case reports have documented harm to the victim^{131,134} or rescuer¹²⁶ during this manoeuvre. Blind finger sweeps should, therefore, be avoided, and solid material in the airway removed manually only if it can be seen.

Aftercare and referral for medical review

Following successful treatment for FBAO, foreign material may nevertheless remain in the upper or lower respiratory tract and

cause complications later. Victims with a persistent cough, difficulty swallowing or the sensation of an object being still stuck in the throat should, therefore, be referred for a medical opinion. Abdominal thrusts and chest compressions can potentially cause serious internal injuries, and all victims successfully treated with these measures should be examined afterwards for injury.

Resuscitation of children (see also Section 6)^{134a} and victims of drowning (see also Section 8c)^{134b}

For victims of primary cardiac arrest who receive chest-compression-only CPR, oxygen stores become depleted about 2–4 min after initiation of CPR.^{92,104} The combination of chest compressions with ventilation then becomes critically important. After collapse from asphyxial arrest, a combination of chest compressions with ventilations is important immediately after the start of resuscitation. Previous guidelines have tried to address this difference in pathophysiology, and have recommended that victims of identifiable asphyxia (drowning, intoxication) and children should receive 1 min of CPR before the lone rescuer leaves the victim to get

help. The majority of cases of SCA out of hospital, however, occur in adults, and although the rate of VF as the first recorded rhythm has declined over recent years, the cause of adult cardiac arrest remains VF in most cases (59%) when documented in the earliest phase by an AED.¹³ In children, VF is much less common as the primary cardiac arrest rhythm (approximately 7%).¹³⁵ These additional recommendations, therefore, added to the complexity of the guidelines while affecting only a minority of victims.

It is important to be aware that many children do not receive resuscitation because potential rescuers fear causing harm if they are not specifically trained in resuscitation for children. This fear is unfounded; it is far better to use the adult BLS sequence for resuscitation of a child than to do nothing. For ease of teaching and retention laypeople should be taught that the adult sequence may also be used for children who are not responsive and not breathing or not breathing normally.

The following minor modifications to the adult sequence will make it even more suitable for use in children.

- Give 5 initial rescue breaths before starting chest compressions (adult sequence of actions, 5b).

Automated External Defibrillation Algorithm

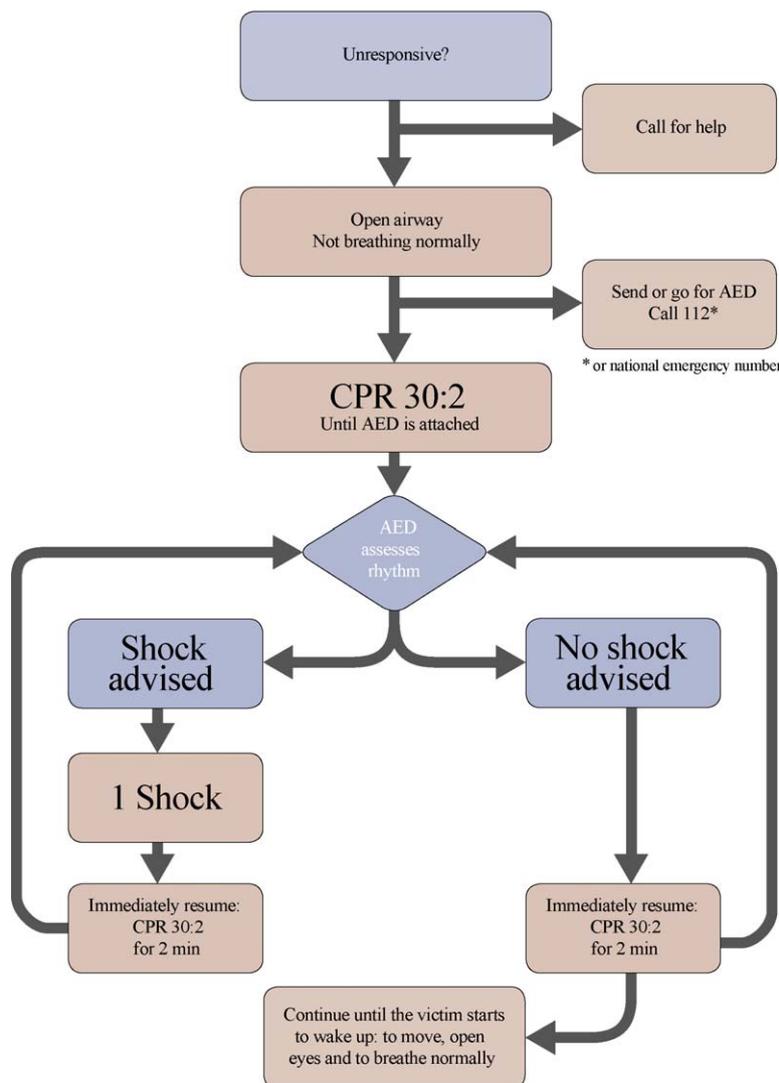


Fig. 2.18. Algorithm for use of an automated external defibrillator. © 2010 ERC.

- A lone rescuer should perform CPR for approximately 1 min before going for help.
- Compress the chest by at least one third of its depth; use 2 fingers for an infant under 1 year; use 1 or 2 hands for a child over 1 year as needed to achieve an adequate depth of compression.

The same modifications of 5 initial breaths and 1 min of CPR by the lone rescuer before getting help, may improve outcome for victims of drowning. This modification should be taught only to those who have a specific duty of care to potential drowning victims (e.g. lifeguards). Drowning is easily identified. It can be difficult, on the other hand, for a layperson to determine whether cardiorespiratory arrest is a direct result of trauma or intoxication. These victims should, therefore, be managed according to the standard BLS protocols.

Use of an automated external defibrillator

Section 3 discusses the guidelines for defibrillation using both automated external defibrillators (AEDs) and manual defibrillators. AEDs are safe and effective when used by laypeople, and make it possible to defibrillate many minutes before professional help arrives. Rescuers should continue CPR with minimal interruption of chest compressions while applying an AED and during its use. Rescuers should concentrate on following the voice prompts immediately they are received, in particular, resuming CPR as soon as instructed.

Standard AEDs are suitable for use in children older than 8 years. For children between 1 and 8 years paediatric pads should be used, together with an attenuator or a paediatric mode if available; if these are not available, the AED should be used as it is. Use of AEDs is not recommended for children <1 year. There are, however, a few case reports describing the use of AEDs in children aged <1 year.^{136,137} The incidence of shockable rhythms in infants is very low except when there is cardiac disease^{135,138,139}; in these rare cases, if an AED is the only defibrillator available its use should be considered (preferably with dose attenuator).

Sequence for use of an AED

See Fig. 2.18

1. Make sure you, the victim, and any bystanders are safe.
2. Follow the Adult BLS sequence (steps 1–5).
 - if the victim is unresponsive and not breathing normally, send someone for help and to find and bring an AED if available;
 - if you are on your own, use your mobile telephone to alert the ambulance service—leave the victim only when there is no other option.
3. Start CPR according to the adult BLS sequence. If you are on your own and the AED is in your immediate vicinity, start by applying the AED.
4. As soon as the AED arrives
 - switch on the AED and attach the electrode pads on the victim's bare chest (Fig. 2.19);
 - if more than one rescuer is present, CPR should be continued while electrode pads are being attached to the chest;
 - follow the spoken/visual directions immediately;
 - ensure that nobody is touching the victim while the AED is analysing the rhythm (Fig. 2.20).
- 5a. If a shock is indicated
 - ensure that nobody is touching the victim (Fig. 2.21);
 - push shock button as directed (fully automatic AEDs will deliver the shock automatically);
 - immediately restart CPR 30:2 (Fig. 2.22);
 - continue as directed by the voice/visual prompts.



Fig. 2.19. Attaching the electrode pads. Place the first electrode pad in the mid-axillary line just below the armpit. Place the second electrode just below the right collarbone (clavicle). © 2010 ERC.

- 5b. If no shock is indicated
 - immediately resume CPR, using a ratio of 30 compressions to 2 rescue breaths;
 - continue as directed by the voice/visual prompts.
6. Continue to follow the AED prompts until
 - professional help arrives and takes over;
 - the victim starts to wake up: moves, open eyes and breathes normally;
 - you become exhausted.

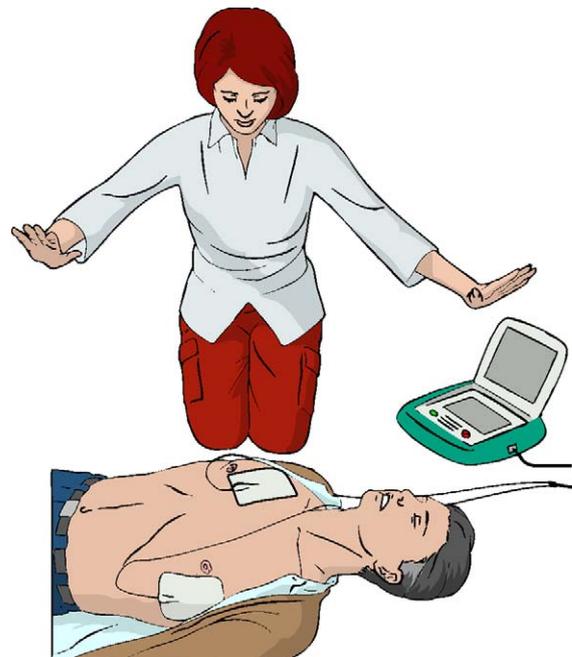


Fig. 2.20. While the AED analyses the heart rhythm, nobody should touch the victim. © 2010 ERC.

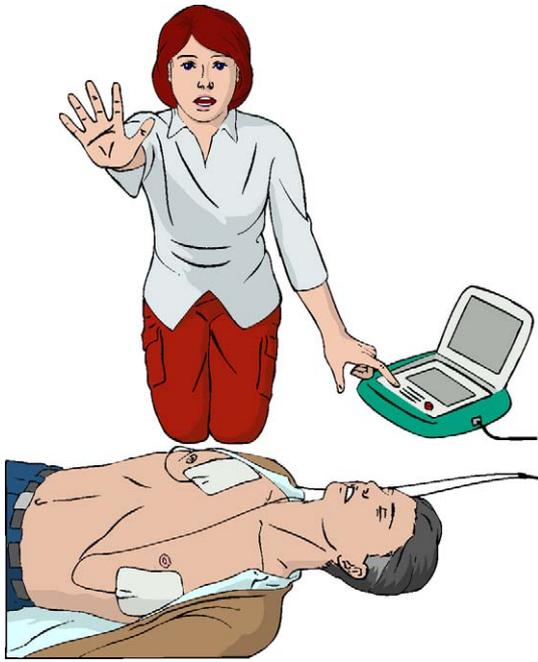


Fig. 2.21. When the shock button is pressed, make sure that nobody touches the victim. © 2010 ERC.



Fig. 2.22. After the shock the AED will prompt you to start CPR. Do not wait—start CPR immediately and alternate 30 chest compressions with 2 rescue breaths. © 2010 ERC.

CPR before defibrillation

The importance of immediate defibrillation, as soon as an AED becomes available, has always been emphasised in guidelines and during teaching, and is considered to have a major impact on survival from ventricular fibrillation. This concept has been challenged, because evidence has suggested that a period of chest compression before defibrillation may improve survival when the time between calling for the ambulance and its arrival exceeds 5 min.^{140,141} Two recent clinical studies^{142,143} and a recent animal study¹⁴⁴ did not confirm this survival benefit. For this reason, a pre-

specified period of CPR, as a routine before rhythm analysis and shock delivery, is not recommended. High-quality CPR, however, must continue while the defibrillation pads are being applied and the defibrillator is being prepared. The importance of early delivery of minimally interrupted chest compression is emphasised. Given the lack of convincing data either supporting or refuting this strategy, it is reasonable for emergency medical services that have already implemented a specified period of chest compression before defibrillation to continue this practice.

Voice prompts

In several places, the sequence of actions states “follow the voice/visual prompts”. Voice prompts are usually programmable, and it is recommended that they be set in accordance with the sequence of shocks and timings for CPR given in Section 2. These should include at least:

1. a single shock only, when a shockable rhythm is detected;
2. no rhythm check, or check for breathing or a pulse, after the shock;
3. a voice prompt for immediate resumption of CPR after the shock (giving chest compressions in the presence of a spontaneous circulation is not harmful);
4. a period of 2 min of CPR before a next prompt to re-analyse the rhythm.

The shock sequence and energy levels are discussed in Section 3.²

Fully automatic AEDs

Having detected a shockable rhythm, a fully automatic AED will deliver a shock without further input from the rescuer. One manikin study has shown that untrained nursing students commit fewer safety errors using a fully automatic AED rather than a (semi-) automated AED.¹⁴⁵ There are no human data to determine whether these findings can be applied to clinical use.

Public access defibrillation programmes

AED programmes should be actively considered for implementation in non-hospital settings. This refers to public places like airports,³² sport facilities, offices, in casinos³⁵ and on aircraft,³³ where cardiac arrests are usually witnessed and trained rescuers are quickly on scene. Lay rescuer AED programmes with very rapid response times, and uncontrolled studies using police officers as first responders,^{146,147} have achieved reported survival rates as high as 49–74%. These programmes will be successful only if enough trained rescuers and AEDs are available.

The full potential of AEDs has not yet been achieved, because they are mostly used in public settings, yet 60–80% of cardiac arrests occur at home. Public access defibrillation (PAD) and first responder AED programmes may increase the number of victims who receive bystander CPR and early defibrillation, thus improving survival from out-of-hospital SCA.¹⁴⁸ Recent data from nationwide studies in Japan and the USA^{13,43} showed that when an AED was available, victims were defibrillated much sooner and with a better chance of survival. However, an AED delivered a shock in only 3.7% and 5% of all VF cardiac arrests, respectively. There was a clear inverse relationship in the Japanese study between the number of AEDs available per square km and the interval between collapse and the first shock, and a positive relationship with survival. In both studies, AED shocks still occurred predominantly in a public rather than a residential setting. Dispatched first responders like

police and fire fighters will, in general, have longer response times, but have the potential to reach the whole population.

When implementing an AED programme, community and programme leaders should consider factors such as the strategic location of AEDs, development of a team with responsibility for monitoring and maintaining the devices, training and retraining programmes for the individuals who are likely to use the AED, and identification of a group of volunteer individuals who are committed to using the AED for victims of cardiac arrest.¹⁴⁹

The logistic problem for first responder programmes is that the rescuer needs to arrive, not just earlier than the traditional ambulance, but within 5–6 min of the initial call, to enable attempted defibrillation in the electrical or circulatory phase of cardiac arrest.⁴⁴ With longer delays, the survival benefits decrease:^{36,47} a few minutes' gain in time will have little impact when a first responder arrives more than 10 min after the call,^{14,150} or when a first responder does not improve on an already short ambulance response time.¹⁵¹ However, small reductions in response intervals achieved by first-responder programmes that impact on many residential victims may be more cost-effective than the larger reductions in response interval achieved by PAD programmes that have an impact on fewer cardiac arrest victims.^{152,153}

Programmes that make AEDs publicly available in residential areas have not yet been evaluated. The acquisition of an AED for individual use at home, even for those considered at high risk of sudden cardiac arrest, has proved not to be effective.¹⁵⁴

Universal AED signage

When a collapse occurs, and an AED must be found rapidly, simple and clear signage indicating the location of, and the fastest way to an AED is important. ILCOR has designed an AED sign that may be recognised worldwide and is recommended for indicating the location of an AED (Fig. 2.23). More detailed information on design and application of this AED sign can be found at: <https://www.erc.edu/index.php/newsitem/en/nid=204/>



Fig. 2.23. Universal ILCOR signage to indicate presence of an AED. This sign can be combined with arrows to indicate the direction of the nearest AED.

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European Resuscitation Council Guidelines for Resuscitation 2010 Section 3. Electrical therapies: Automated external defibrillators, defibrillation, cardioversion and pacing

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Summary of changes since 2005 Guidelines

The most important changes in the 2010 European Resuscitation Council (ERC) guidelines for electrical therapies include:

- The importance of early, uninterrupted chest compressions is emphasised throughout these guidelines.
- Much greater emphasis on minimising the duration of the pre-shock and post-shock pauses. The continuation of compressions during charging of the defibrillator is recommended.
- Immediate resumption of chest compressions following defibrillation is also emphasised; in combination with continuation of compressions during defibrillator charging, the delivery of defibrillation should be achievable with an interruption in chest compressions of no more than 5 s.
- Safety of the rescuer remains paramount, but there is recognition in these guidelines that the risk of harm to a rescuer from a defibrillator is very small, particularly if the rescuer is wearing gloves. The focus is now on a rapid safety check to minimise the pre-shock pause.
- When treating out-of-hospital cardiac arrest, emergency medical services (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 no longer recommended. For some emergency medical services that 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.
- The use of up to three-stacked shocks may be considered if ventricular fibrillation/pulseless ventricular tachycardia (VF/VT)

occurs during cardiac catheterisation or in the early post-operative period following cardiac surgery. This three-shock strategy may also be considered for an initial, witnessed VF/VT cardiac arrest when the patient is already connected to a manual defibrillator.

- Electrode pastes and gels can spread between the two paddles, creating the potential for a spark and should not be used

Introduction

The chapter presents guidelines for defibrillation using both automated external defibrillators (AEDs) and manual defibrillators. There are only a few differences from the 2005 ERC Guidelines. All healthcare providers and lay responders can use AEDs as an integral component of basic life support. Manual defibrillation is used as part of advanced life support (ALS) therapy. Synchronised cardioversion and pacing options are included on many defibrillators and are also discussed in this chapter.

Defibrillation is the passage of an electrical current across the myocardium of sufficient magnitude to depolarise a critical mass of myocardium and enable restoration of coordinated electrical activity. Defibrillation is defined as the termination of fibrillation or, more precisely, the absence of VF/VT at 5 s after shock delivery; however, the goal of attempted defibrillation is to restore an organised rhythm and a spontaneous circulation.

Defibrillator technology is advancing rapidly. AED interaction with the rescuer through voice prompts is now established and future technology may enable more specific instructions to be given by voice prompt. The evolving ability of defibrillators to assess the rhythm whilst CPR is in progress is an important advance and enables rescuers to assess the rhythm without interrupting external chest compressions. In the future, waveform analysis may also enable the defibrillator to calculate the optimal time at which to give a shock.

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A vital link in the Chain of Survival

Defibrillation is a key link in the Chain of Survival and is one of the few interventions that have been shown to improve outcome from VF/VT cardiac arrest. The previous guidelines published in 2005 rightly emphasized the importance of early defibrillation with minimum delay.^{1,2}

The probability of successful defibrillation and subsequent survival to hospital discharge declines rapidly with time^{3,4} and the ability to deliver early defibrillation is one of the most important factors in determining survival from cardiac arrest. For every minute delay in defibrillation, in the absence of bystander CPR, survival from witnessed VF decreases by 10–12%.^{4,5} EMS systems do not generally have the capability to deliver defibrillation through traditional paramedic responders within the first few minutes of a call and the alternative use of trained lay responders to deliver prompt defibrillation using AEDs is now widespread. EMS systems that have reduced time to defibrillation following cardiac arrest using trained lay responders have reported greatly improved survival to hospital discharge rates,^{6–9} some as high as 75% if defibrillation is performed within 3 min of collapse.¹⁰ This concept has also been extended to in-hospital cardiac arrests where staff, other than doctors, are also being trained to defibrillate using an AED before arrival of the cardiac arrest team.¹¹

When bystander CPR is provided, the fall in survival is more gradual and averages 3–4% per minute from collapse to defibrillation^{3,4,12}; bystander CPR can double^{3,4,13} or triple¹⁴ survival from witnessed out-of-hospital cardiac arrest. Resuscitation instructions given by the ambulance service before the arrival of trained help increase the quantity and quality of bystander CPR^{15,16} and use of video instructions by phone may improve performance further.^{17,18}

All healthcare providers with a duty to perform CPR should be trained, equipped, and encouraged to perform defibrillation and CPR. Early defibrillation should be available throughout all hospitals, outpatient medical facilities and public areas of mass gathering (see Section 2).¹⁹ Those trained in the use of an AED should also be trained to deliver high-quality CPR before arrival of ALS providers so that the effectiveness of early defibrillation can be optimised.

Automated external defibrillators

Automated external defibrillators are sophisticated, reliable computerised devices that use voice and visual prompts to guide lay rescuers and healthcare professionals to safely attempt defibrillation in cardiac arrest victims. Some AEDs combine guidance for defibrillation with guidance for the delivery of optimal chest compressions. Use of AEDs by lay or non-healthcare rescuers is covered in Section 2.¹⁹

In many situations, an AED is used to provide initial defibrillation but is subsequently swapped for a manual defibrillator on arrival of EMS personnel. If such a swap is done without considering the phase the AED cycle is in, the next shock may be delayed, which may compromise outcome.²⁰ For this reason, EMS personnel should leave the AED connected while securing airway and IV access. The AED should be left attached for the next rhythm analysis and, if indicated, a shock delivered before the AED is swapped for a manual defibrillator.

Currently many manufacturers use product-specific electrode to defibrillator connectors, which necessitates the defibrillation pads also being removed and replaced with a pair compatible with the new defibrillator. Manufacturers are encouraged to collaborate and develop a universal connector that enables all defibrillation pads to be compatible with all defibrillators. This will have significant patient benefit and minimise unnecessary waste.

In-hospital use of AEDs

At the time of the 2010 Consensus on CPR Science Conference there were no published randomised trials comparing in-hospital use of AEDs with manual defibrillators. Two lower level studies of adults with in-hospital cardiac arrest from shockable rhythms showed higher survival to hospital discharge rates when defibrillation was provided through an AED programme than with manual defibrillation alone.^{21,22} One retrospective study²³ demonstrated no improvements in survival to hospital discharge for in-hospital adult cardiac arrest when using an AED compared with manual defibrillation. In this study, patients in the AED group with initial asystole or pulseless electrical activity (PEA) had a lower survival to hospital discharge rate compared with those in the manual defibrillator group (15% versus 23%; $p=0.04$). A manikin study showed that use of an AED significantly increased the likelihood of delivering three shocks but increased the time to deliver the shocks when compared with manual defibrillators.²⁴ In contrast, a study of mock arrests in simulated patients showed that use of monitoring leads and fully automated defibrillators reduced time to defibrillation when compared with manual defibrillators.²⁵

Delayed defibrillation may occur when patients sustain cardiac arrest in unmonitored hospital beds and in outpatient departments.²⁶ In these areas several minutes may elapse before resuscitation teams arrive with a defibrillator and deliver shocks. Despite limited evidence, AEDs should be considered for the hospital setting as a way to facilitate early defibrillation (a goal of <3 min from collapse), especially in areas where healthcare providers have no rhythm recognition skills or where they use defibrillators infrequently. An effective system for training and retraining should be in place.¹¹ Enough healthcare providers should be trained to enable achievement of the goal of providing the first shock within 3 min of collapse anywhere in the hospital. Hospitals should monitor collapse-to-first shock intervals and monitor resuscitation outcomes.

Shock in manual versus semi-automatic mode

Many AEDs can be operated in both manual and semi-automatic mode but few studies have compared these two options. The semi-automatic mode has been shown to reduce time to first shock when used both in-hospital²⁷ and pre-hospital²⁸ settings, and results in higher VF conversion rates,²⁸ and delivery of fewer inappropriate shocks.²⁹ Conversely, semi-automatic modes result in less time spent performing chest compressions^{29,30} mainly because of a longer pre-shock pause associated with automated rhythm analysis. Despite these differences, no overall difference in return of spontaneous circulation (ROSC), survival, or discharge rate from hospital has been demonstrated in any study.^{23,27,28} The defibrillation mode that affords the best outcome will depend on the system, skills, training and ECG recognition skills of rescuers. A shorter pre-shock pause and lower total hands-off-ratio increases vital organ perfusion and the probability of ROSC.^{31–33} With manual defibrillators and some AEDs it is possible to perform chest compressions during charging and thereby reduce the pre-shock pause to less than 5 s. Trained individuals may deliver defibrillation in manual mode but frequent team training and ECG recognition skills are essential.

Automated rhythm analysis

Automated external defibrillators have microprocessors that analyse several features of the ECG, including frequency and amplitude. Developing technology should soon enable AEDs to provide information about frequency and depth of chest compressions dur-

ing CPR that may improve basic life support (BLS) performance by all rescuers.^{34,35}

Automated external defibrillators have been tested extensively against libraries of recorded cardiac rhythms and in many trials in adults^{36,37} and children.^{38,39} They are extremely accurate in rhythm analysis. Although most AEDs are not designed to deliver synchronised shocks, all AEDs will recommend shocks for VT if the rate and R-wave morphology and duration exceeds preset values. Most AEDs require a 'hands-off' period while the device analyses the rhythm. This 'hands-off' period results in interruption to chest compressions for varying but significant periods of time⁴⁰; a factor shown to have significant adverse impact on outcome from cardiac arrest.⁴¹ Manufacturers of these devices should make every effort to develop software that minimises this analysis period to ensure that interruptions to external chest compressions are kept to a minimum.

Strategies before defibrillation

Minimising the pre-shock pause

The delay between stopping chest compressions and delivery of the shock (the pre-shock pause) must be kept to an absolute minimum; even 5–10 s delay will reduce the chances of the shock being successful.^{31,32,42} The pre-shock pause can easily be reduced to less than 5 s by continuing compressions during charging of the defibrillator and by having an efficient team coordinated by a leader who communicates effectively. The safety check to ensure that nobody is in contact with the patient at the moment of defibrillation should be undertaken rapidly but efficiently. The negligible risk of a rescuer receiving an accidental shock is minimised even further if all rescuers wear gloves.⁴³ The post-shock pause is minimised by resuming chest compressions immediately after shock delivery (see below). The entire process of defibrillation should be achievable with no more than a 5 s interruption to chest compression.

Safe use of oxygen during defibrillation

In an oxygen-enriched atmosphere, sparking from poorly applied defibrillator paddles can cause a fire.^{44–49} There are several reports of fires being caused in this way and most have resulted in significant burns to the patient. There are no case reports of fires caused by sparking where defibrillation was delivered using adhesive pads. In two manikin studies the oxygen concentration in the zone of defibrillation was not increased when ventilation devices (bag-valve device, self-inflating bag, modern intensive care unit ventilator) were left attached to a tracheal tube or the oxygen source was vented at least 1 m behind the patient's mouth.^{50,51} One study described higher oxygen concentrations and longer washout periods when oxygen is administered in confined spaces without adequate ventilation.⁵²

The risk of fire during attempted defibrillation can be minimised by taking the following precautions:

- Take off any oxygen mask or nasal cannulae and place them at least 1 m away from the patient's chest.
- Leave the ventilation bag connected to the tracheal tube or supra-glottic airway device. Alternatively, disconnect any bag-valve device from the tracheal tube or supra-glottic airway device and remove it at least 1 m from the patient's chest during defibrillation.
- If the patient is connected to a ventilator, for example in the operating room or critical care unit, leave the ventilator tubing (breathing circuit) connected to the tracheal tube unless chest

compressions prevent the ventilator from delivering adequate tidal volumes. In this case, the ventilator is usually substituted by a ventilation bag, which can itself be left connected or detached and removed to a distance of at least 1 m. If the ventilator tubing is disconnected, ensure it is kept at least 1 m from the patient or, better still, switch the ventilator off; modern ventilators generate massive oxygen flows when disconnected. During normal use, when connected to a tracheal tube, oxygen from a ventilator in the critical care unit will be vented from the main ventilator housing well away from the defibrillation zone. Patients in the critical care unit may be dependent on positive end expiratory pressure (PEEP) to maintain adequate oxygenation; during cardioversion, when the spontaneous circulation potentially enables blood to remain well oxygenated, it is particularly appropriate to leave the critically ill patient connected to the ventilator during shock delivery.

- Minimise the risk of sparks during defibrillation. Self-adhesive defibrillation pads are less likely to cause sparks than manual paddles.

Some early versions of the LUCAS external chest compression device are driven by high flow rates of oxygen which discharges waste gas over the patient's chest. High ambient levels of oxygen over the chest have been documented using this device, particularly in relatively confined spaces such as the back of the ambulance and caution should be used when defibrillating patients while using the oxygen-powered model.⁵²

The technique for electrode contact with the chest

Optimal defibrillation technique aims to deliver current across the fibrillating myocardium in the presence of minimal transthoracic impedance. Transthoracic impedance varies considerably with body mass, but is approximately 70–80 Ω in adults.^{53,54} The techniques described below aim to place external electrodes (paddles or self-adhesive pads) in an optimal position using techniques that minimise transthoracic impedance.

Shaving the chest

Patients with a hairy chest have poor electrode-to-skin electrical contact and air trapping beneath the electrode. This causes high impedance, reduced defibrillation efficacy, risk of arcing (sparks) from electrode-to-skin and electrode to electrode and is more likely to cause burns to the patient's chest. Rapid shaving of the area of intended electrode placement may be necessary, but do not delay defibrillation if a shaver is not immediately available. Shaving the chest *per se* may reduce transthoracic impedance slightly and has been recommended for elective DC cardioversion with monophasic defibrillators,⁵⁵ although the efficacy of biphasic impedance-compensated waveforms may not be so susceptible to higher transthoracic impedance.⁵⁶

Paddle force

If using paddles, apply them firmly to the chest wall. This reduces transthoracic impedance by improving electrical contact at the electrode–skin interface and reducing thoracic volume.⁵⁷ The defibrillator operator should always press firmly on handheld electrode paddles, the optimal force being 8 kg in adult and 5 kg in children 1–8 years using adult paddles.⁵⁸ Eight kilogram force may be attainable only by the strongest members of the cardiac arrest team and therefore it is recommended that these individuals apply the paddles during defibrillation. Unlike self-adhesive pads, manual paddles have a bare metal plate that requires a conductive material placed between the metal and patient's skin to improve electrical

contact. Use of bare-metal paddles alone creates high transthoracic impedance and is likely to increase the risk of arcing and worsen cutaneous burns from defibrillation.

Electrode position

No human studies have evaluated the electrode position as a determinant of ROSC or survival from VF/VT cardiac arrest. Transmyocardial current during defibrillation is likely to be maximal when the electrodes are placed so that the area of the heart that is fibrillating lies directly between them (i.e. ventricles in VF/VT, atria in AF). Therefore, the optimal electrode position may not be the same for ventricular and atrial arrhythmias.

More patients are presenting with implantable medical devices (e.g., permanent pacemaker, implantable cardioverter defibrillator (ICD)). Medic Alert bracelets are recommended for these patients. These devices may be damaged during defibrillation if current is discharged through electrodes placed directly over the device.^{59,60} Place the electrode away from the device (at least 8 cm)⁵⁹ or use an alternative electrode position (anterior-lateral, anterior-posterior) as described below.

Transdermal drug patches may prevent good electrode contact, causing arcing and burns if the electrode is placed directly over the patch during defibrillation.^{61,62} Remove medication patches and wipe the area before applying the electrode.

Placement for ventricular arrhythmias and cardiac arrest

Place electrodes (either pads or paddles) in the conventional sternal-apical position. The right (sternal) electrode is placed to the right of the sternum, below the clavicle. The apical paddle is placed in the left mid-axillary line, approximately level with the V6 ECG electrode or female breast. This position should be clear of any breast tissue. It is important that this electrode is placed sufficiently laterally. Other acceptable pad positions include

- Placement of each electrode on the lateral chest walls, one on the right and the other on the left side (bi-axillary).
- One electrode in the standard apical position and the other on the right upper back.
- One electrode anteriorly, over the left precordium, and the other electrode posteriorly to the heart just inferior to the left scapula.

It does not matter which electrode (apex/sternum) is placed in either position.

Transthoracic impedance has been shown to be minimised when the apical electrode is not placed over the female breast.⁶³ Asymmetrically shaped apical electrodes have a lower impedance when placed longitudinally rather than transversely.⁶⁴

Placement for atrial arrhythmias

Atrial fibrillation is maintained by functional re-entry circuits anchored in the left atrium. As the left atrium is located posteriorly in the thorax, electrode positions that result in a more posterior current pathway may theoretically be more effective for atrial arrhythmias. Although some studies have shown that antero-posterior electrode placement is more effective than the traditional antero-apical position in elective cardioversion of atrial fibrillation,^{65,66} the majority have failed to demonstrate any clear advantage of any specific electrode position.^{67,68} Efficacy of cardioversion may be less dependent on electrode position when using biphasic impedance-compensated waveforms.⁵⁶ The following electrode positions all appear safe and effective for cardioversion of atrial arrhythmias:

- Traditional antero-apical position.
- Antero-posterior position (one electrode anteriorly, over the left precordium, and the other electrode posteriorly to the heart just inferior to the left scapula).

Respiratory phase

Transthoracic impedance varies during respiration, being minimal at end-expiration. If possible, defibrillation should be attempted at this phase of the respiratory cycle. Positive end expiratory pressure (PEEP) increases transthoracic impedance and should be minimised during defibrillation. Auto-PEEP (gas trapping) may be particularly high in asthmatics and may necessitate higher than usual energy levels for defibrillation.⁶⁹

Electrode size

The Association for the Advancement of Medical Instrumentation recommends a minimum electrode size of for individual electrodes and the sum of the electrode areas should be a minimum of 150 cm².⁷⁰ Larger electrodes have lower impedance, but excessively large electrodes may result in less transmyocardial current flow.⁷¹

For adult defibrillation, both handheld paddle electrodes and self-adhesive pad electrodes 8–12 cm in diameter are used and function well. Defibrillation success may be higher with electrodes of 12 cm diameter compared with those of 8 cm diameter.^{54,72}

Standard AEDs are suitable for use in children over the age of 8 years. In children between 1 and 8 years use paediatric pads with an attenuator to reduce delivered energy or a paediatric mode if they are available; if not, use the unmodified machine, taking care to ensure that the adult pads do not overlap. Use of AEDs is not recommended in children less than 1 year.

Coupling agents

If using manual paddles, disposable gel pads should be used to reduce impedance at the electrode–skin interface. Electrode pastes and gels can spread between the two paddles, creating the potential for a spark and should not be used. Do not use bare electrodes without gel pads because the resultant high transthoracic impedance may impair the effectiveness of defibrillation, increase the severity of any cutaneous burns and risk arcing with subsequent fire or explosion.

Pads versus paddles

Self-adhesive defibrillation pads have practical benefits over paddles for routine monitoring and defibrillation.^{73–77} They are safe and effective and are preferable to standard defibrillation paddles.⁷² Consideration should be given to use of self-adhesive pads in peri-arrest situations and in clinical situations where patient access is difficult. They have a similar transthoracic impedance⁷¹ (and therefore efficacy)^{78,79} to manual paddles and enable the operator to defibrillate the patient from a safe distance rather than leaning over the patient as occurs with paddles. When used for initial monitoring of a rhythm, both pads and paddles enable quicker delivery of the first shock compared with standard ECG electrodes, but pads are quicker than paddles.⁸⁰

When gel pads are used with paddles, the electrolyte gel becomes polarised and thus is a poor conductor after defibrillation. This can cause spurious asystole that may persist for 3–4 min when used to monitor the rhythm; a phenomenon not reported with self-adhesive pads.^{74,81} When using a gel pad/paddle combination confirm a diagnosis of asystole with independent ECG electrodes rather than the paddles.

Fibrillation waveform analysis

It is possible to predict, with varying reliability, the success of defibrillation from the fibrillation waveform.^{82–101} If optimal defibrillation waveforms and the optimal timing of shock delivery can be determined in prospective studies, it should be possible to prevent the delivery of unsuccessful high energy shocks and minimise myocardial injury. This technology is under active development and investigation but current sensitivity and specificity is insufficient to enable introduction of VF waveform analysis into clinical practice.

CPR versus defibrillation as the initial treatment

A number of studies have examined whether a period of CPR prior to defibrillation is beneficial, particularly in patients with an unwitnessed arrest or prolonged collapse without resuscitation. A review of evidence for the 2005 guidelines resulted in the recommendation that it was reasonable for EMS personnel to give a period of about 2 min of CPR (i.e. about five cycles at 30:2) before defibrillation in patients with prolonged collapse (>5 min).¹ This recommendation was based on clinical studies where response times exceeded 4–5 min, a period of 1.5–3 min of CPR by paramedics or EMS physicians before shock delivery improved ROSC, survival to hospital discharge^{102,103} and one year survival¹⁰³ for adults with out-of-hospital VF/VT compared with immediate defibrillation. In some animal studies of VF lasting at least 5 min, CPR before defibrillation improved haemodynamics and survival.^{103–106} A recent ischaemic swine model of cardiac arrest showed a decreased survival after pre-shock CPR.¹⁰⁷

In contrast, two randomized controlled trials, a period of 1.5–3 min of CPR by EMS personnel before defibrillation did not improve ROSC or survival to hospital discharge in patients with out-of-hospital VF/VT, regardless of EMS response interval.^{108,109} Four other studies have also failed to demonstrate significant improvements in overall ROSC or survival to hospital discharge with an initial period of CPR,^{102,103,110,111} although one did show a higher rate of favourable neurological outcome at 30 days and one year after cardiac arrest.¹¹⁰

The duration of collapse is frequently difficult to estimate accurately and there is evidence that performing chest compressions while retrieving and charging a defibrillator improves the probability of survival.¹¹² For these reasons, in any cardiac arrest they have not witnessed, 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 EMS systems 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.

In hospital environments, settings with an AED on-site and available (including lay responders), or EMS-witnessed events, defibrillation should be performed as soon as the defibrillator is available. Chest compressions should be performed until just before the defibrillation attempt (see Section 4 advanced life support).¹¹³

The importance of early, uninterrupted chest compressions is emphasised throughout these guidelines. In practice, it is often difficult to ascertain the exact time of collapse and, in any case, CPR should be started as soon as possible. The rescuer providing chest compressions should interrupt chest compressions only for ventilations, rhythm analysis and shock delivery, and should resume chest compressions as soon as a shock is delivered. When two rescuers are present, the rescuer operating the AED should apply the electrodes whilst CPR is in progress. Interrupt CPR only when it is

necessary to assess the rhythm and deliver a shock. The AED operator should be prepared to deliver a shock as soon as analysis is complete and the shock is advised, ensuring no rescuer is in contact with the victim.

Delivery of defibrillation

One-shock versus three-stacked shock sequence

A major change in the 2005 guidelines was the recommendation to give single rather than three-stacked shocks. This was because animal studies had shown that relatively short interruptions in external chest compression to deliver rescue breaths^{114,115} or perform rhythm analysis³³ were associated with post-resuscitation myocardial dysfunction and reduced survival. Interruptions in external chest compression also reduced the chances of converting VF to another rhythm.³² Analysis of CPR performance during out-of-hospital^{34,116} and in-hospital³⁵ cardiac arrest also showed that significant interruptions were common, with chest compressions comprising no more than 51–76%^{34,35} of total CPR time.

With first shock efficacy of biphasic waveforms generally exceeding 90%,^{117–120} failure to cardiovert VF successfully is more likely to suggest the need for a period of CPR rather than a further shock. Even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation and the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.⁴⁰

Subsequent studies have shown a significantly lower hands-off-ratio with the one-shock protocol¹²¹ and some,^{41,122,123} but not all,^{121,124} have suggested a significant survival benefit from this single-shock strategy. However, all studies except one¹²⁴ were before-after studies and all introduced multiple changes in the protocol, making it difficult to attribute a possible survival benefit to one of the changes.

When defibrillation is warranted, give a single shock and resume chest compressions immediately following the shock. Do not delay CPR for rhythm reanalysis or a pulse check immediately after a shock. Continue CPR (30 compressions:2 ventilations) for 2 min until rhythm reanalysis is undertaken and another shock given (if indicated) (see Section 4 advanced life support).¹¹³ This single-shock strategy is applicable to both monophasic and biphasic defibrillators.

If VF/VT occurs during cardiac catheterisation or in the early post-operative period following cardiac surgery (when chest compressions could disrupt vascular sutures), consider delivering up to three-stacked shocks before starting chest compressions (see Section 8 special circumstances).¹²⁵ 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.

Waveforms

Historically, defibrillators delivering a monophasic pulse had been the standard of care until the 1990s. Monophasic defibrillators deliver current that is unipolar (i.e. one direction of current flow) (Fig. 3.1). Monophasic defibrillators were particularly susceptible to waveform modification depending on transthoracic impedance. Small patients with minimal transthoracic impedance received considerably greater transmural current than larger patients,

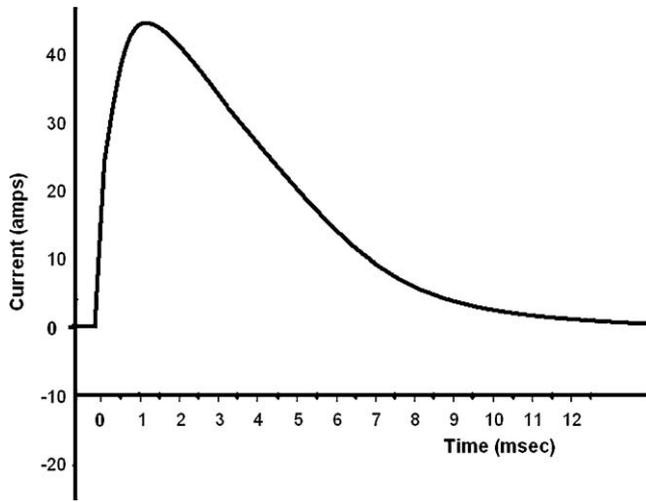


Fig. 3.1. Monophasic damped sinusoidal waveform (MDS).

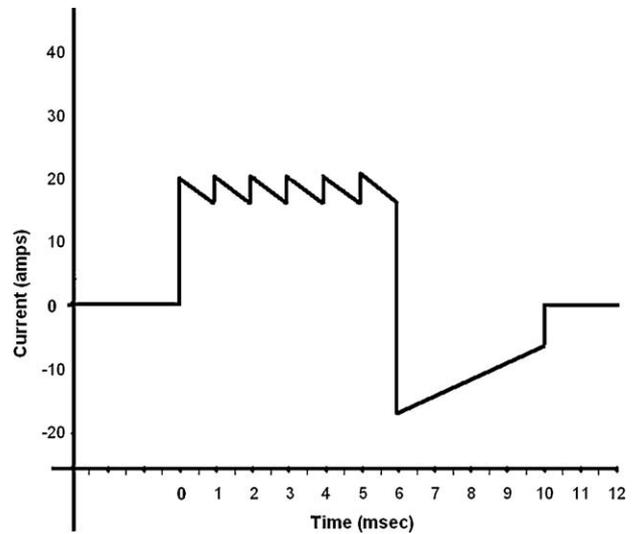


Fig. 3.3. Rectilinear biphasic waveform (RLB).

where not only was the current less, but the waveform lengthened to the extent that its efficacy was reduced.

Monophasic defibrillators are no longer manufactured, and although many will remain in use for several years, biphasic defibrillators have now superseded them. Biphasic defibrillators deliver current that flows in a positive direction for a specified duration before reversing and flowing in a negative direction for the remaining milliseconds of the electrical discharge. There are two main types of biphasic waveform: the biphasic truncated exponential (BTE) (Fig. 3.2) and rectilinear biphasic (RLB) (Fig. 3.3). Biphasic defibrillators compensate for the wide variations in transthoracic impedance by electronically adjusting the waveform magnitude and duration to ensure optimal current delivery to the myocardium, irrespective of the patient's size.

A pulsed biphasic waveform has recently been described in which the current rapidly oscillates between baseline and a positive value before inverting in a negative pattern. This waveform is also in clinical use. It may have a similar efficacy as other biphasic waveforms, but the single clinical study of this waveform was not performed with an impedance compensating device.^{126,127} There are several other different biphasic waveforms, all with no clinical evidence of superiority for any individual waveform compared with another.

All manual defibrillators and AEDs that allow manual override of energy levels should be labelled to indicate their waveform (monophasic or biphasic) and recommended energy levels for attempted defibrillation of VF/VT.

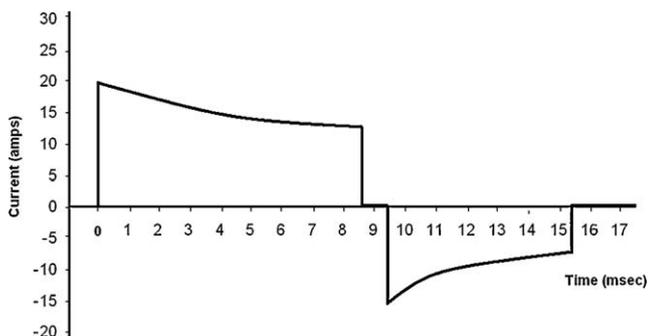


Fig. 3.2. Biphasic truncated exponential waveform (BTE).

Monophasic versus biphasic defibrillation

Although biphasic waveforms are more effective at terminating ventricular arrhythmias at lower energy levels, have demonstrated greater first shock efficacy than monophasic waveforms, and have greater first shock efficacy for long duration VF/VT,^{128–130} no randomised studies have demonstrated superiority in terms of neurologically intact survival to hospital discharge.

Some,^{119,128–133} but not all,¹³⁴ studies suggest the biphasic waveform improves short-term outcomes of VF termination compared with monophasic defibrillation.

Biphasic waveforms have been shown to be superior to monophasic waveforms for elective cardioversion of atrial fibrillation, with greater overall success rates, using less cumulative energy and reducing the severity of cutaneous burns,^{135–138} and are the waveform of choice for this procedure.

Multiphasic versus biphasic defibrillation

A number of multiphasic waveforms (e.g. triphasic, quadriphasic, multiphasic) have also been trialled in animal studies. Animal data suggest that multiphasic waveforms may defibrillate at lower energies and induce less post-shock myocardial dysfunction.^{139–141} These results are limited by studies of short duration of VF (approximately 30 s) and lack of human studies for validation. At present, there are no human studies comparing a multiphasic waveform with biphasic waveforms for defibrillation and no defibrillator currently available uses multiphasic waveforms.

Energy levels

Defibrillation requires the delivery of sufficient electrical energy to defibrillate a critical mass of myocardium, abolish the wavefronts of VF and enable restoration of spontaneous synchronized electrical activity in the form of an organised rhythm. The optimal energy for defibrillation is that which achieves defibrillation whilst causing the minimum of myocardial damage.¹⁴² Selection of an appropriate energy level also reduces the number of repetitive shocks, which in turn limits myocardial damage.¹⁴³

Optimal energy levels for both monophasic and biphasic waveforms are unknown. The recommendations for energy levels are based on a consensus following careful review of the current literature. Although energy levels are selected for defibrillation, it is the transmural current flow that achieves defibrilla-

tion. Current correlates well with successful defibrillation and cardioversion.¹⁴⁴ The optimal current for defibrillation using a monophasic waveform is in the range of 30–40 A. Indirect evidence from measurements during cardioversion for atrial fibrillation suggests that the current during defibrillation using biphasic waveforms is in the range of 15–20 A.¹³⁷ Future technology may enable defibrillators to discharge according to transthoracic current; a strategy that may lead to greater consistency in shock success. Peak current amplitude, average current and phase duration all need to be studied to determine optimal values and manufacturers are encouraged to explore further this move from energy-based to current-based defibrillation.

First shock

Monophasic defibrillators

There are no new published studies looking at the optimal energy levels for monophasic waveforms since publication of the 2005 guidelines. First shock efficacy for long duration cardiac arrest using monophasic defibrillation has been reported as 54–63% for a 200 J monophasic truncated exponential (MTE) waveform^{129,145} and 77–91% using a 200 J monophasic damped sinusoidal (MDS) waveform.^{128–130,145} Because of the lower efficacy of this waveform, the recommended initial energy level for the first shock using a monophasic defibrillator is 360 J. Although higher energy levels risk a greater degree of myocardial injury, the benefits of earlier conversion to a perfusing rhythm are paramount. Atrioventricular block is more common with higher monophasic energy levels, but is generally transient and has been shown not to affect survival to hospital discharge.¹⁴⁶ Only one of 27 animal studies demonstrated harm caused by attempted defibrillation using high energy shocks.¹⁴⁷

Biphasic defibrillators

Relatively few studies have been published in the past 5 years on which to refine the 2005 guidelines. There is no evidence that one biphasic waveform or device is more effective than another. First shock efficacy of the BTE waveform using 150–200 J has been reported as 86–98%.^{128,129,145,148,149} First shock efficacy of the RLB waveform using 120 J is up to 85% (data not published in the paper but supplied by personnel communication).¹³⁰ First shock efficacy of a new pulsed biphasic waveform at 130 J showed a first shock success rate of 90%.¹²⁶ Two studies have suggested equivalence with lower and higher starting energy biphasic defibrillation.^{150,151} Although human studies have not shown harm (raised biomarkers, ECG changes, ejection fraction) from any biphasic waveform up to 360 J,^{150,152} several animal studies have suggested the potential for harm with higher energy levels.^{153–156}

The initial biphasic shock should be no lower than 120 J for RLB waveforms and 150 J for BTE waveforms. Ideally, the initial biphasic shock energy should be at least 150 J for all waveforms.

Manufacturers should display the effective waveform dose range on the face of the biphasic defibrillator; older monophasic defibrillators should also be marked clearly with the appropriate dose range. If the rescuer is unaware of the recommended energy settings of the defibrillator, use the highest setting for all shocks.

Second and subsequent shocks

The 2005 guidelines recommended either a fixed or escalating energy strategy for defibrillation. Subsequent to these recommendations, several studies have demonstrated that although an escalating strategy reduces the number of shocks required to restore an organised rhythm compared with fixed-dose biphasic defibrillation, and may be needed for successful defibrillation,^{157,158} rates of ROSC or survival to hospital discharge are not significantly

different between strategies.^{150,151} Conversely, a fixed-dose biphasic protocol demonstrated high cardioversion rates (>90%) with a three-shock fixed dose protocol but the small number of cases did not exclude a significant lower ROSC rate for recurrent VF.¹⁵⁹ Several in-hospital studies using an escalating shock energy strategy have demonstrated improvement in cardioversion rates (compared with fixed dose protocols) in non-arrest rhythms with the same level of energy selected for both biphasic and monophasic waveforms.^{135,137,160–163}

Monophasic defibrillators

Because the initial shock has been unsuccessful at 360 J, second and subsequent shocks should all be delivered at 360 J.

Biphasic defibrillators

There is no evidence to support either a fixed or escalating energy protocol. Both strategies are acceptable; however, if the first shock is not successful and the defibrillator is capable of delivering shocks of higher energy it is reasonable to increase the energy for subsequent shocks.

Recurrent ventricular fibrillation

If a shockable rhythm recurs after successful defibrillation with ROSC, give the next shock with the energy level that had previously been successful.

Other related defibrillation topics

Defibrillation of children

Cardiac arrest is less common in children. Common causes of VF in children include trauma, congenital heart disease, long QT interval, drug overdose and hypothermia.^{164–166} Ventricular fibrillation is relatively rare compared with adult cardiac arrest, occurring in 7–15% of paediatric and adolescent arrests.^{166–171} Rapid defibrillation of these patients may improve outcome.^{171,172}

The optimal energy level, waveform and shock sequence is unknown but as with adults, biphasic shocks appear to be at least as effective as, and less harmful than, monophasic shocks.^{173–175} The upper limit for safe defibrillation is unknown, but doses in excess of the previously recommended maximum of 4 J kg⁻¹ (as high as 9 J kg⁻¹) have defibrillated children effectively without significant adverse effects.^{38,176,177}

The recommended energy levels for manual monophasic defibrillation are 4 J kg⁻¹ for the initial shock and subsequent shocks. The same energy levels are recommended for manual biphasic defibrillation.¹⁷⁸ As with adults, if a shockable rhythm recurs, use the energy level for defibrillation that had previously been successful.

For defibrillation of children above the age of 8 years, an AED with standard electrodes is used and standard energy settings accepted. For defibrillation of children between 1 and 8 years, special paediatric electrodes and energy attenuators are recommended; these reduce the delivered energy to a level that approaches that of the energy recommended for manual defibrillators. When these electrodes are not available, an AED with standard electrodes should be used. For defibrillation of children below 1 year of age, an AED, is not recommended; however, there are a few case reports describing the use of AEDs in children aged less than 1 year.^{179,180} The incidence of shockable rhythms in infants is very low except when there is cardiac disease^{167,181,182}; in these rare cases, if an AED is the only defibrillator available, its use should be considered (preferably with dose attenuator).

Cardioversion

If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave of the electrocardiogram rather than with the T wave: VF can be induced if a shock is delivered during the relative refractory portion of the cardiac cycle.¹⁸³ Synchronisation can be difficult in VT because of the wide-complex and variable forms of ventricular arrhythmia. Inspect the synchronisation marker carefully for consistent recognition of the R wave. If needed, choose another lead and/or adjust the amplitude. If synchronisation fails, give unsynchronised shocks to the unstable patient in VT to avoid prolonged delay in restoring sinus rhythm. Ventricular fibrillation or pulseless VT requires unsynchronised shocks. Conscious patients must be anaesthetised or sedated before attempting synchronised cardioversion.

Atrial fibrillation

Optimal electrode position has been discussed previously, but anterolateral and antero-posterior are both acceptable positions. Biphasic waveforms are more effective than monophasic waveforms for cardioversion of AF^{135–138}; and cause less severe skin burns.¹⁸⁴ When available, a biphasic defibrillator should be used in preference to a monophasic defibrillator. Differences in biphasic waveforms themselves have not been established.

Monophasic waveforms

A study of electrical cardioversion for atrial fibrillation indicated that 360 J monophasic damped sinusoidal (MDS) shocks were more effective than 100 or 200 J MDS shocks.¹⁸⁵ Although a first shock of 360 J reduces overall energy requirements for cardioversion,¹⁸⁵ 360 J may cause greater myocardial damage and skin burns than occurs with lower monophasic energy levels and this must be taken into consideration. Commence synchronised cardioversion of atrial fibrillation using an initial energy level of 200 J, increasing in a stepwise manner as necessary.

Biphasic waveforms

More data are needed before specific recommendations can be made for optimal biphasic energy levels. Commencing at high energy levels has not shown to result in more successful cardioversion rates compared to lower energy levels.^{135,186–191} An initial synchronised shock of 120–150 J, escalating if necessary is a reasonable strategy based on current data.

Atrial flutter and paroxysmal supraventricular tachycardia

Atrial flutter and paroxysmal SVT generally require less energy than atrial fibrillation for cardioversion.¹⁹⁰ Give an initial shock of 100 J monophasic or 70–120 J biphasic. Give subsequent shocks using stepwise increases in energy.¹⁴⁴

Ventricular tachycardia

The energy required for cardioversion of VT depends on the morphological characteristics and rate of the arrhythmia.¹⁹² Ventricular tachycardia with a pulse responds well to cardioversion using initial monophasic energies of 200 J. Use biphasic energy levels of 120–150 J for the initial shock. Consider stepwise increases if the first shock fails to achieve sinus rhythm.¹⁹²

Pacing

Consider pacing in patients with symptomatic bradycardia refractory to anti-cholinergic drugs or other second line therapy (see Section 4).¹¹³ Immediate pacing is indicated especially when the block is at or below the His-Purkinje level. If transthoracic pacing is ineffective, consider transvenous pacing. Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because this will likely respond to cardiac pacing. The use of epicardial wires to pace the myocardium following cardiac surgery is effective and discussed elsewhere. Do not attempt pacing for asystole unless P waves are present; it does not increase short or long-term survival in- or out-of-hospital.^{193–201} For haemodynamically unstable, conscious patients with bradyarrhythmias, percussion pacing as a bridge to electrical pacing may be attempted, although its effectiveness has not been established.

Implantable cardioverter defibrillators

Implantable cardioverter defibrillators (ICDs) are becoming increasingly common as the devices are implanted more frequently as the population ages. They are implanted because a patient is considered to be at risk from, or has had, a life-threatening shockable arrhythmia and are usually embedded under the pectoral muscle below the left clavicle (in a similar position to pacemakers, from which they cannot be immediately distinguished). On sensing a shockable rhythm, an ICD will discharge approximately 40 J through an internal pacing wire embedded in the right ventricle. On detecting VF/VT, ICD devices will discharge no more than eight times, but may reset if they detect a new period of VF/VT. Patients with fractured ICD leads may suffer repeated internal defibrillation as the electrical noise is mistaken for a shockable rhythm; in these circumstances, the patient is likely to be conscious, with the ECG showing a relatively normal rate. A magnet placed over the ICD will disable the defibrillation function in these circumstances.

Discharge of an ICD may cause pectoral muscle contraction in the patient, and shocks to the rescuer have been documented.²⁰² In view of the low energy levels discharged by ICDs, it is unlikely that any harm will come to the rescuer, but the wearing of gloves and minimising contact with the patient whilst the device is discharging is prudent. Cardioverter and pacing function should always be re-evaluated following external defibrillation, both to check the device itself and to check pacing/defibrillation thresholds of the device leads.

Pacemaker spikes generated by devices programmed to unipolar pacing may confuse AED software and emergency personnel, and may prevent the detection of VF.²⁰³ The diagnostic algorithms of modern AEDs are insensitive to such spikes.

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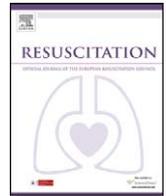
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European Resuscitation Council Guidelines for Resuscitation 2010 Section 4. Adult advanced life support

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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 (SaO₂) can be monitored reliably (by pulse oximetry and/or arterial blood gas analysis), inspired oxygen is titrated to achieve a SaO₂ 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 multi-professional 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 hospitals²⁴ 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 h¹³⁶ or to hospital at weekends¹³⁷ 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 (SpO₂).^{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 10s 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.

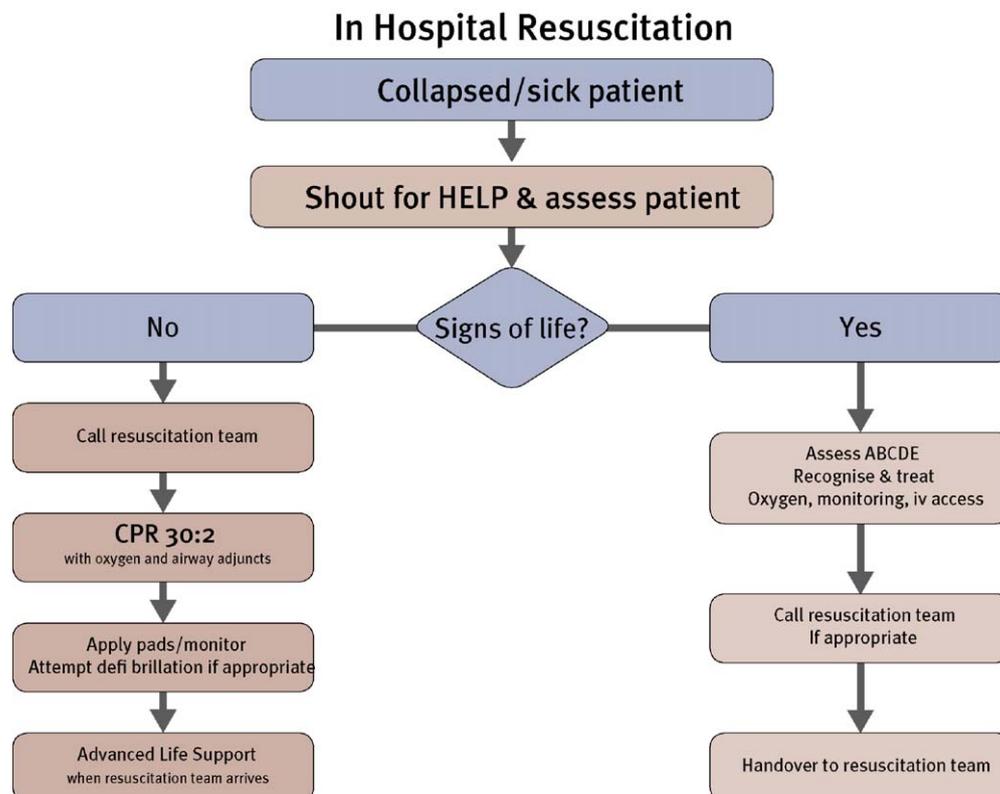


Fig. 4.1. Algorithm for the treatment of in-hospital cardiac arrest. © 2010 ERC.

- 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 (SpO₂)) can be achieved, titrate the inspired oxygen concentration to achieve a SpO₂ 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 ETCO₂ monitoring can be used to indicate the quality of CPR: although an optimal target for ETCO₂ 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

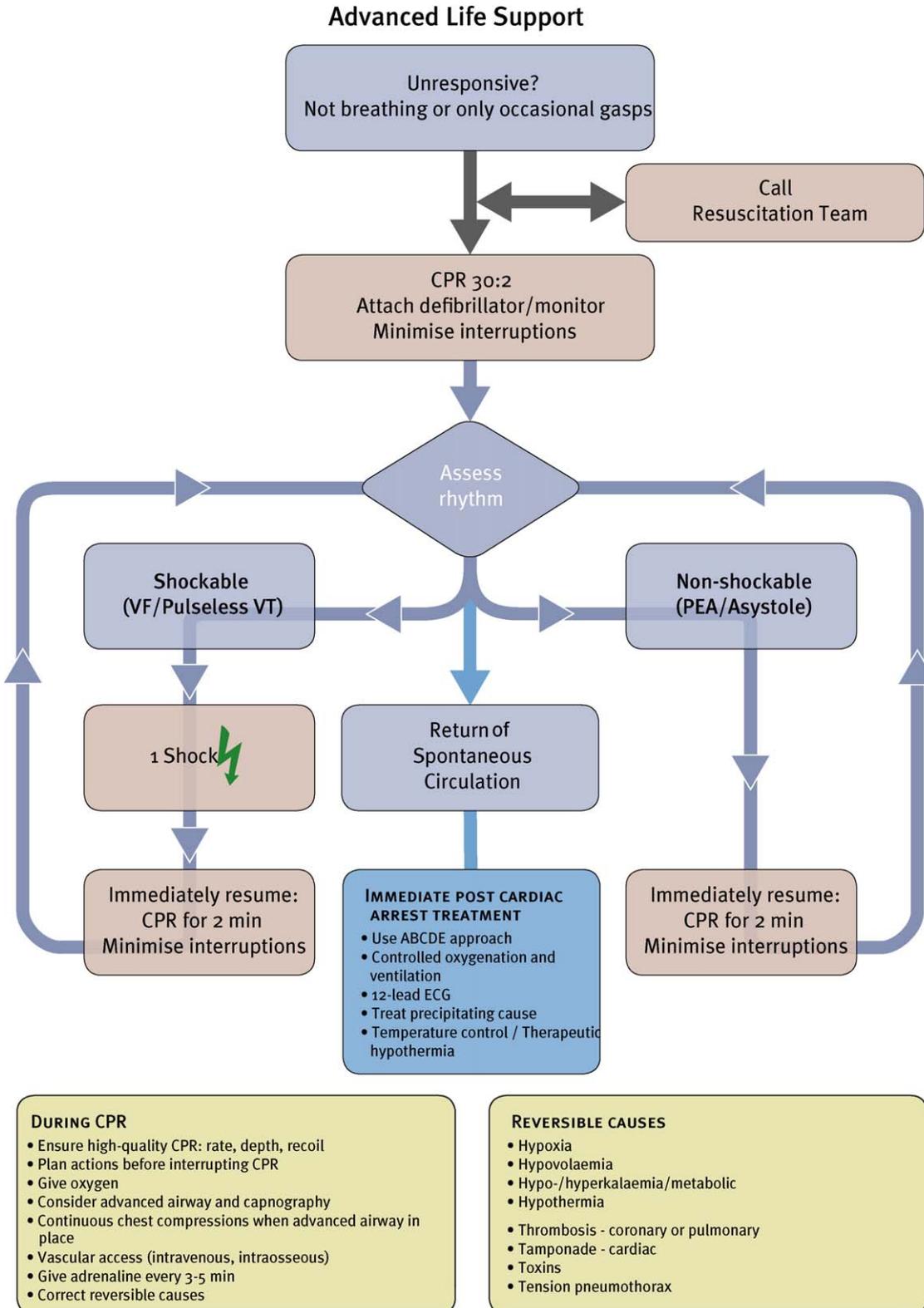


Fig. 4.2. Advanced life support cardiac arrest algorithm. © 2010 ERC.

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 150–200J biphasic).
- Minimise the delay between stopping chest compressions and delivery of the shock (the pre-shock 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 established²⁵³ 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 chest compressions and may be a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. Two prospective human studies have shown that a significant increase in end-tidal CO₂ 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 placebo²⁸⁵ 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 tissues²⁹²; 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 health-care 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.

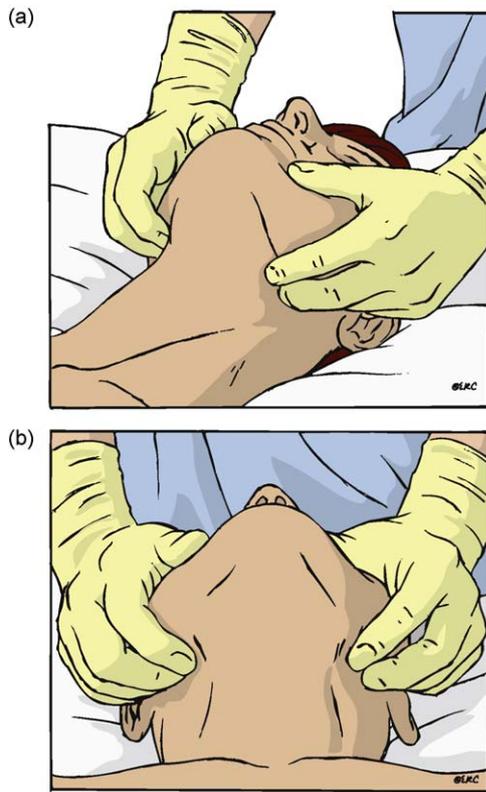


Fig. 4.4. Jaw thrust.

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).

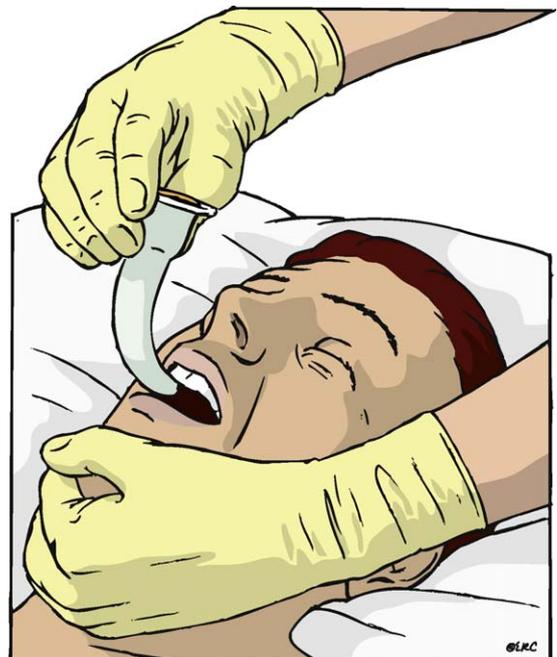


Fig. 4.5. Insertion of oropharyngeal airway.

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 data³²⁸ 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 resuscitator 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,



Fig. 4.6. Mouth-to-mask ventilation.

give two ventilations after each sequence of 30 chest compressions.

Self-inflating bag

The self-inflating bag can be connected to a facemask, tracheal tube or 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.

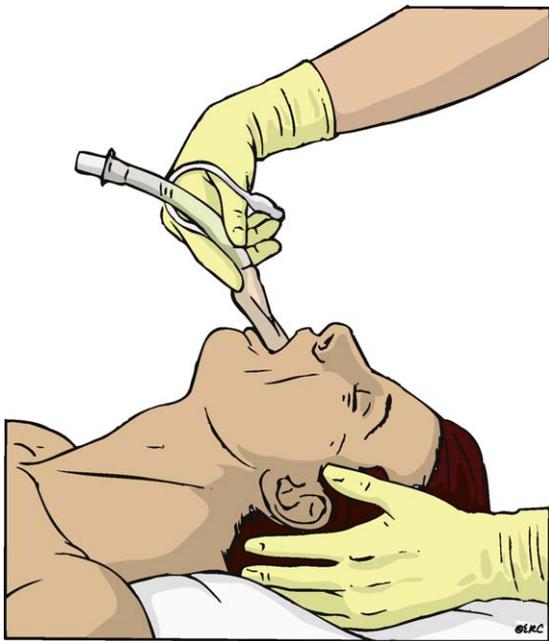


Fig. 4.8. Insertion of a laryngeal mask airway.

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 OHCA.³⁸⁴ A disposable version of the laryngeal tube (LT-D) is available and was inserted successfully by paramedics in 92 OHCA (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 butadiene 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 H₂O 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

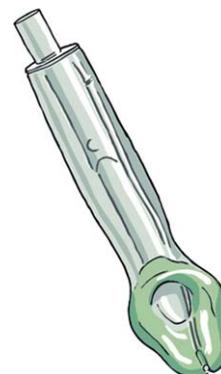


Fig. 4.10. I-gel. © 2010 ERC.

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 H₂O 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 trials⁴⁰⁰: 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 than 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 compressions⁴⁰²; 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 (CO₂) detector devices measure the concentration of exhaled carbon dioxide from the lungs. The persistence of exhaled CO₂ 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 are three types of carbon dioxide detector device:

1. Disposable colorimetric end-tidal carbon dioxide (ETCO₂) detectors use a litmus paper to detect CO₂, and these devices generally give readings of purple (ETCO₂ < 0.5%), tan (ETCO₂ 0.5–2%) and yellow (ETCO₂ > 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 CO₂ 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 ETCO₂ devices generally measure ETCO₂ 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 CO₂ 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 CO₂ 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 ventilation⁴³² and oesophageal 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-analysis⁴⁴² 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 placebo²⁸⁵ 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 amiodarone²⁸⁶ 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 Ca²⁺) 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 state²⁹²; 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 study⁴⁸⁷ and meta-analysis⁴⁸⁸ 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 study⁵⁰⁸ 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 fluid⁵⁰⁹ 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 CPR⁵³⁰ 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™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 OHCA⁵⁹⁷ and improved hemodynamics following failed resuscitation from in-hospital cardiac arrest.⁵⁹¹ Evidence from both clinical^{594,598} and simulation⁵⁹⁹ 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 scans⁶⁰⁰ 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, SpO₂). 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⁺, Mg²⁺, Ca²⁺). 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

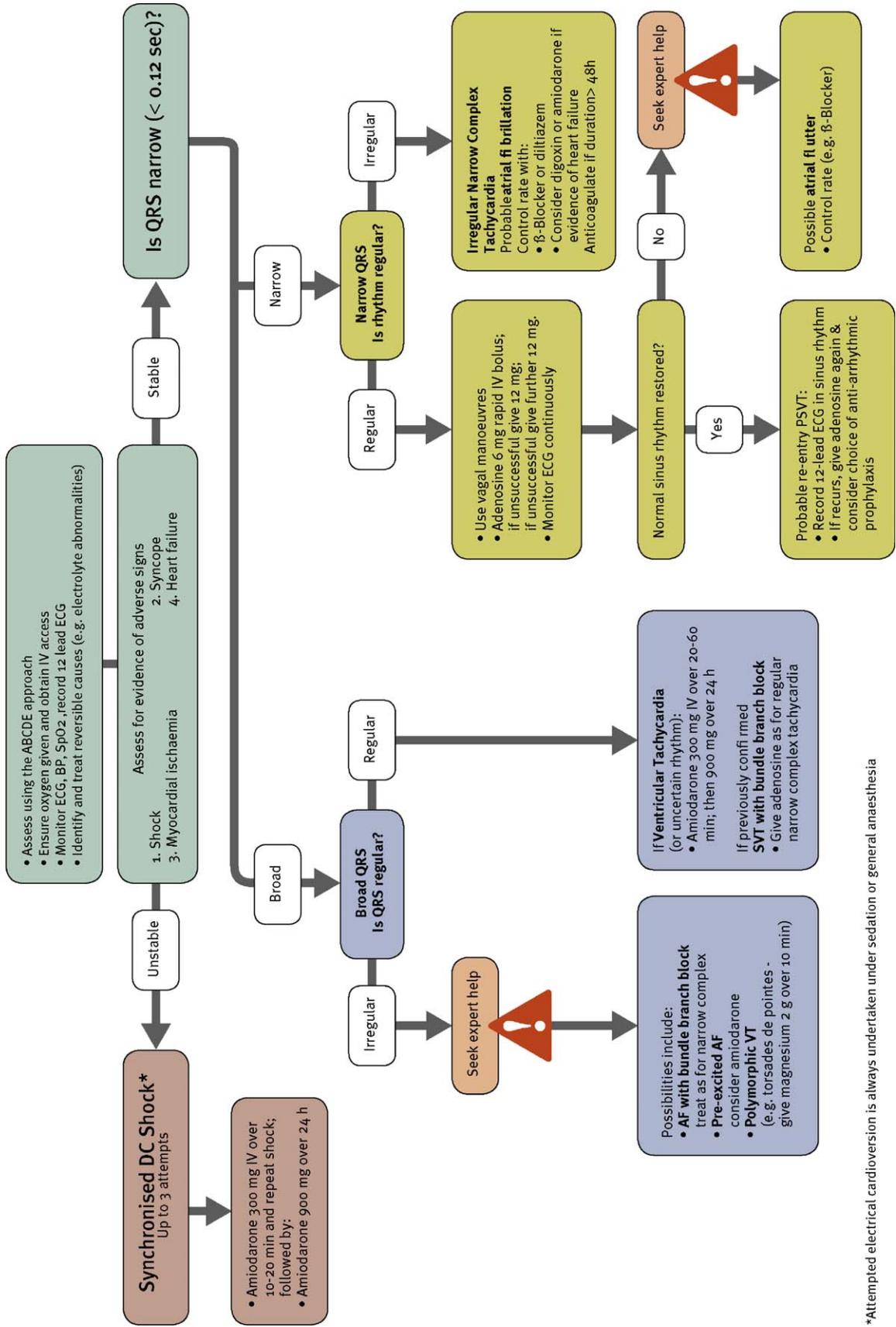
If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronised with the R wave of the ECG rather than with the T wave.⁶⁰⁴ By avoiding the relative refractory period in this way, the risk of inducing ventricular fibrillation is minimised. Conscious 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.

Tachycardia Algorithm (with pulse)



*Attempted electrical cardioversion is always undertaken under sedation or general anaesthesia

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 alternative 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 manoeuvres⁶⁰⁹: 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 $\mu\text{g min}^{-1}$ starting dose), adrenaline (2–10 $\mu\text{g min}^{-1}$) and dopamine (2–10 $\mu\text{g kg}^{-1} \text{min}^{-1}$). 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

Bradycardia Algorithm

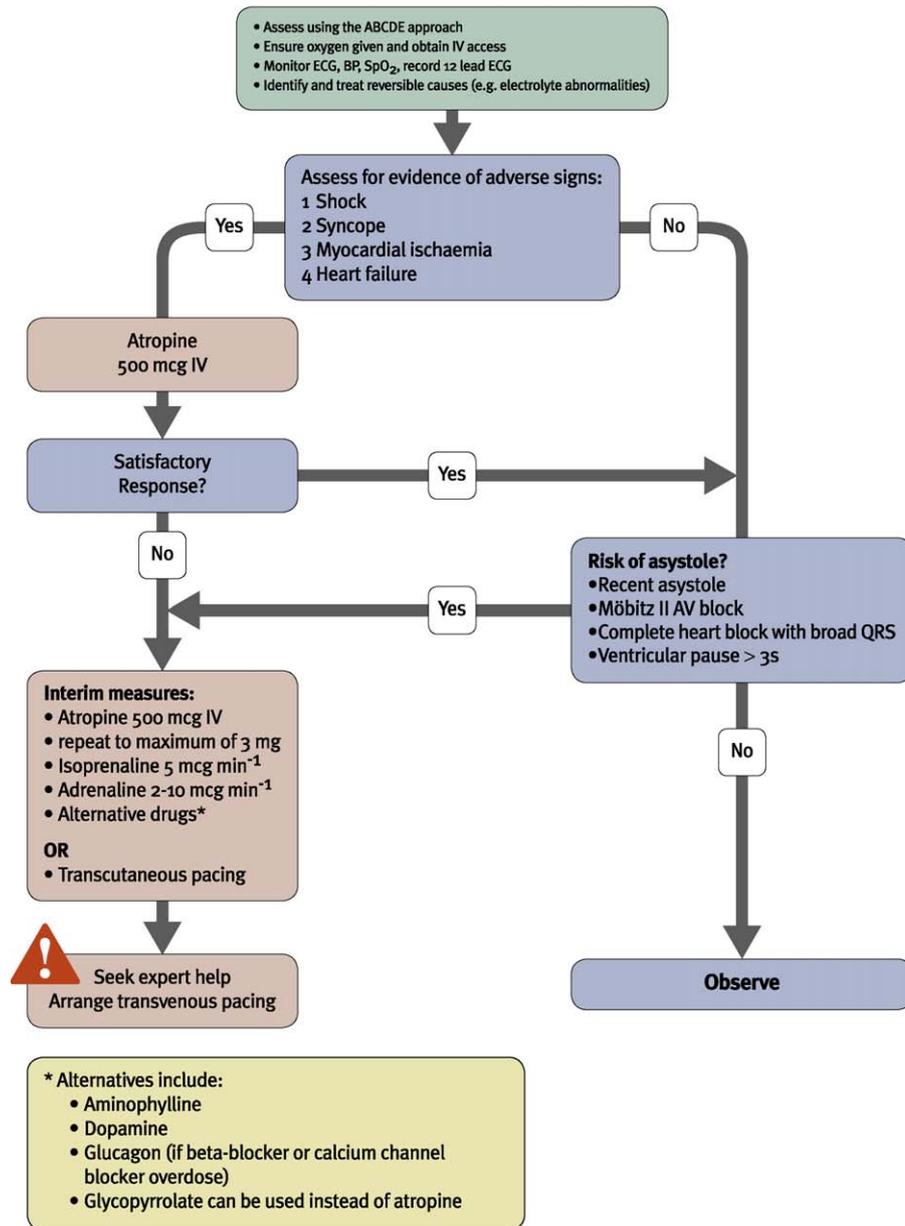


Fig. 4.12. Bradycardia algorithm. © 2010 ERC.

pacings 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 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 $\mu\text{g kg}^{-1}$, followed by a second dose of 350 $\mu\text{g kg}^{-1}$, 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 (β_1) is 5 mg given over 5 min, repeated if necessary after 10 min. Metoprolol (β_1) is given in doses of 2–5 mg at 5-min intervals to a total of 15 mg. Propranolol (β_1 and β_2 effects), 100 $\mu\text{g kg}^{-1}$, is given slowly in three equal doses at 2–3-min intervals.

Intravenous esmolol is a short-acting (half-life of 2–9 min) β_1 -selective beta-blocker. It is given as an intravenous loading dose of 500 $\mu\text{g kg}^{-1}$ over 1 min, followed by an infusion of 50–200 $\mu\text{g kg}^{-1} \text{ min}^{-1}$.

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 (FiO₂) 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 PCO₂ after resuscitation from cardiac arrest, but it is reasonable to adjust ventilation to achieve normocarbia and to monitor this using the end-tidal PCO₂ 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 Hg⁶²⁹ 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-fold⁶⁸¹ 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 ≤ 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 ≥ 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 (CMRO₂) by about 6% for each 1 °C reduction in temperature⁷⁰³ 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 trial⁷⁰⁴ and a pseudo-randomised trial⁶⁶⁹ 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 to 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 carefully⁷¹⁵:

- 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, hypomagnesemia 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 been 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 diazepam⁴⁵²) 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 haemofiltration⁷⁶³ 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 with²⁴⁵ and without⁶⁴⁰ 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 μ V, 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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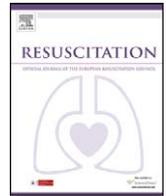
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European Resuscitation Council Guidelines for Resuscitation 2010 Section 5. Initial management of acute coronary syndromes

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Summary of main changes since 2005 Guidelines

Changes in the management of acute coronary syndrome since the 2005 guidelines include:

Definitions

The term non-ST-elevation myocardial infarction-acute coronary syndrome (non-STEMI-ACS) has been introduced for both NSTEMI and unstable angina pectoris because the differential diagnosis is dependent on biomarkers that may be detectable only after hours, whereas decisions on treatment are dependent on the clinical signs at presentation.

Chest pain units and decision rules for early discharge

- History, clinical examinations, biomarkers, ECG criteria and risk scores are unreliable for the identification of patients who may be safely discharged early.
- The role of chest pain observation units (CPUs) is to identify, by using repeated clinical examinations, ECG and biomarker testing, those patients who require admission for invasive procedures. This may include provocative testing and, in selected patients, imaging procedures as cardiac computed tomography, magnetic resonance imaging, etc.

Symptomatic treatment

- Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided.
- Nitrates should not be used for diagnostic purposes.
- Supplementary oxygen to be given only to those patients with hypoxaemia, breathlessness or pulmonary congestion. Hyperoxaemia may be harmful in uncomplicated infarction.

Causal treatment

- Guidelines for treatment with acetyl salicylic acid (ASA) have been made more liberal and it may now be given by bystanders with or without dispatchers assistance.
- Revised guidance for new antiplatelet and antithrombin treatment for patients with ST elevation myocardial infarction (STEMI) and non-STEMI-ACS based on therapeutic strategy.
- Gp IIb/IIIa inhibitors before angiography/percutaneous coronary intervention (PCI) are discouraged.

Reperfusion strategy in STEMI

- Primary PCI (PPCI) is the preferred reperfusion strategy provided it is performed in a timely manner by an experienced team.
- A nearby hospital may be bypassed by emergency medical services (EMS) provided PPCI can be achieved without too much delay.
- The acceptable delay between start of fibrinolysis and first balloon inflation varies widely between about 45 and 180 min depending on infarct localisation, age of the patient, and duration of symptoms.
- 'Rescue PCI' should be undertaken if fibrinolysis fails.
- The strategy of routine PCI immediately after fibrinolysis ('facilitated PCI') is discouraged.
- Patients with successful fibrinolysis but not in a PCI-capable hospital should be transferred for angiography and eventual PCI, performed optimally 6–24 h after fibrinolysis (the 'pharmacoinvasive' approach).
- Angiography and, if necessary, PCI may be reasonable in patients with return of spontaneous circulation (ROSC) after cardiac arrest and may be part of a standardised post-cardiac arrest protocol.
- To achieve these goals, the creation of networks including EMS, non-PCI capable hospitals and PCI hospitals is useful.

Primary and secondary prevention

- Recommendations for the use of beta-blockers are more restricted: there is no evidence for routine intravenous beta-blockers except in specific circumstances such as for the

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treatment of tachyarrhythmias. Otherwise, beta-blockers should be started in low doses only after the patient is stabilised.

- Guidelines on the use of prophylactic anti-arrhythmics, angiotensin converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs) and statins are unchanged.

Introduction

The incidence of acute ST-elevation myocardial infarction (AMI) is decreasing in many European countries [1]; however, the incidence of non-STEMI acute coronary syndrome (non-STEMI ACS) is increasing [2,3]. Although in-hospital mortality from STEMI has been reduced significantly by modern reperfusion therapy and improved secondary prophylaxis, the overall 28-day mortality is virtually unchanged because about two thirds of those who die do so before hospital arrival, mostly from lethal arrhythmias triggered by ischaemia [4]. Thus, the best chance of improving survival from an ischaemic attack is reducing the delay from symptom onset to first medical contact and targeted treatment started in the early out-of-hospital phase.

The term acute coronary syndrome (ACS) encompasses three different entities of the acute manifestation of coronary heart disease (Fig. 5.1): STEMI, NSTEMI and unstable angina pectoris (UAP). Non-ST-elevation myocardial infarction and UAP are usually combined in the term non-STEMI-ACS. The common pathophysiology of ACS is a ruptured or eroded atherosclerotic plaque [5]. Electrocardiographic (ECG) characteristics (absence or presence of ST elevation) differentiate STEMI from non-STEMI-ACS. The latter may present with ST-segment depression, nonspecific ST-segment wave abnormalities, or even a normal ECG. In the absence of ST elevation, an increase in the plasma concentration of cardiac biomarkers, particularly troponin T or I as the most specific markers of myocardial cell necrosis, indicates NSTEMI.

Acute coronary syndromes are the commonest cause of malignant arrhythmias leading to sudden cardiac death. The therapeutic goals are to treat acute life-threatening conditions, such as ventricular fibrillation (VF) or extreme bradycardia, and to preserve left ventricular function and prevent heart failure by minimising the extent of myocardial damage. The current guidelines address

the first hours after onset of symptoms. Out-of-hospital treatment and initial therapy in the emergency department (ED) may vary according to local capabilities, resources and regulations. The data supporting out-of-hospital treatment are often extrapolated from studies of initial treatment after hospital admission; there are few high-quality out-of-hospital studies. Comprehensive guidelines for the diagnosis and treatment of ACS with and without ST elevation have been published by the European Society of Cardiology and the American College of Cardiology/American Heart Association. The current recommendations are in line with these guidelines [6,7].

Diagnosis and risk stratification in acute coronary syndromes

Since early treatment offers the greatest benefits, and myocardial ischaemia is the leading precipitant of sudden cardiac death, it is essential that the public is aware of the typical symptoms associated with ACS. Several groups of patients however, are less likely to seek prompt medical care when symptoms of an ACS appear. Thus significant delays in the initiation of treatment/reperfusion have been reported in women, the elderly, people belonging to ethnic and racial minorities or low socioeconomic classes, and those living alone [8].

Patients at risk, and their families, should be able to recognize characteristic symptoms such as chest pain, which may radiate into other areas of the upper body, often accompanied by other symptoms including dyspnoea, sweating, nausea or vomiting and syncope. They should understand the importance of early activation of the emergency medical service (EMS) system and, ideally, should be trained in basic life support (BLS). Optimal strategies for increasing layperson awareness of the various ACS presentations and improvement of ACS recognition in vulnerable populations remain to be determined.

Moreover, EMS dispatchers must be trained to recognize ACS symptoms and to ask targeted questions. When an ACS is suspected, an EMS crew trained in advanced life support (ALS) and capable of making the diagnosis and starting treatment should be alerted.

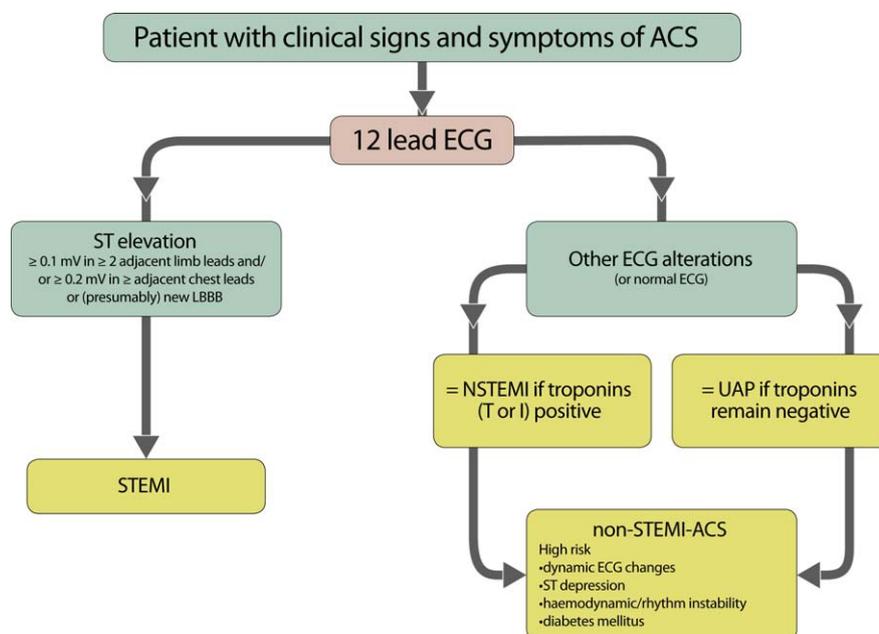


Fig. 5.1. Definitions of acute coronary syndromes (ACS) (STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; UAP, unstable angina pectoris).

Given the high urgency for emergency revascularisation in STEMI and other high-risk patients, specific systems of care can be implemented to improve STEMI recognition and shorten time to treatment.

The sensitivity, specificity, and clinical impact of various diagnostic strategies have been evaluated for ACS. Information from clinical presentation, ECG, biomarker testing and imaging techniques should all be taken into account in order to establish the diagnosis and at the same time estimate the risk so that optimal decisions for patient admission and therapy/reperfusion are made.

Signs and symptoms of ACS

Typically ACS appears with symptoms such as radiating chest pain, shortness of breath and sweating; however, atypical symptoms or unusual presentations may occur in the elderly, in females, and in diabetics [9,10]. None of these signs and symptoms of ACS can be used alone for the diagnosis of ACS. A reduction in chest pain after nitroglycerin administration can be misleading and is not recommended as a diagnostic manoeuvre [11]. Symptoms may be more intense and last longer in patients with STEMI but are not reliable for discriminating between STEMI and non-STEMI-ACS.

The patient's history should be evaluated carefully during first contact with healthcare providers. It may provide the first clues for the presence of an ACS, trigger subsequent investigations and, in combination with information from other diagnostic tests, can help in making triage and therapeutic decisions in the out-of-hospital setting and the emergency department (ED).

12-lead ECG

A 12-lead ECG is the key investigation for assessment of an ACS. In case of STEMI, it indicates the need for immediate reperfusion therapy (i.e. primary percutaneous coronary intervention (PCI) or prehospital fibrinolysis). When an ACS is suspected, a 12-lead ECG should be acquired and interpreted as soon as possible after first patient contact, to facilitate earlier diagnosis and triage. Prehospital or ED ECG yields useful diagnostic information when interpreted by trained health care providers [12].

Recording of a 12-lead ECG out-of-hospital enables advanced notification to the receiving facility and expedites treatment decisions after hospital arrival: in many studies, the time from hospital admission to initiating reperfusion therapy is reduced by 10–60 min [13,14]. Trained EMS personnel (emergency physicians, paramedics and nurses) can identify STEMI, defined by ST elevation of ≥ 0.1 mV elevation in at least two adjacent limb leads or >0.2 mV in two adjacent precordial leads, with a high specificity and sensitivity comparable to diagnostic accuracy in the hospital [15–17]. It is thus reasonable that paramedics and nurses be trained to diagnose STEMI without direct medical consultation, as long as there is strict concurrent provision of medically directed quality assurance.

If interpretation of the prehospital ECG is not available on-site, computer interpretation [18,19] or field transmission of the ECG is reasonable. Recording and transmission of diagnostic quality ECGs to the hospital usually takes less than 5 min. When used for the evaluation of patients with suspected ACS, computer interpretation of the ECG may increase the specificity of diagnosis of STEMI, especially for clinicians inexperienced in reading ECGs. The benefit of computer interpretation; however, is dependent on the accuracy of the ECG report. Incorrect reports may mislead inexperienced ECG readers. Thus computer-assisted ECG interpretation should not replace, but may be used as an adjunct to, interpretation by an experienced clinician.

Biomarkers

In the absence of ST elevation on the ECG, the presence of a suggestive history and elevated concentrations of biomarkers (troponin T and troponin I, CK, CK-MB, myoglobin) characterise non-STEMI and distinguish it from STEMI and unstable angina respectively. Measurement of a cardiac-specific troponin is preferable. Elevated concentrations of troponin are particularly helpful in identifying patients at increased risk of adverse outcome [20].

Cardiac biomarker testing should be part of the initial evaluation of all patients presenting to the ED with symptoms suggestive of cardiac ischaemia [21]. However, the delay in release of biomarkers from damaged myocardium prevents their use in diagnosing myocardial infarction in the first 4–6 h after the onset of symptoms [22]. For patients who present within 6 h of symptom onset, and have an initial negative cardiac troponin, biomarkers should be re-measured between 6 and 12 h after symptom onset. In order to use the measured biomarker optimally, clinicians should be familiar with the sensitivity, precision and institutional norms of the assay, and also the release kinetics and clearance. Highly sensitive (ultra-sensitive) cardiac troponin assays have been developed. They can increase sensitivity for the diagnosis of MI in patients with symptoms suspicious of cardiac ischaemia [23]. If the highly sensitive cardiac troponin assays are unavailable, multi-marker evaluation with CK-MB or myoglobin in conjunction with troponin may be considered to improve the sensitivity of diagnosing AMI.

There is no evidence to support the use of troponin point-of-care testing (POCT) in isolation as a primary test in the prehospital setting to evaluate patients with symptoms suspicious of cardiac ischaemia [23]. In the ED, use of point-of-care troponin assays may help to shorten time to treatment and length of ED stay [24]. Until further randomised control trials are performed, other serum assays should not be considered first-line steps in the diagnosis and management of patients presenting with ACS symptoms [25].

Decision rules for early discharge

Attempts have been made to combine evidence from history, physical examination serial ECGs and serial biomarker measurement in order to form clinical decision rules that would help triage of ED patients with suspected ACS.

None of these rules is adequate and appropriate to identify ED chest pain patients with suspected ACS who can be safely discharged from the ED [26].

Likewise, the scoring systems for risk stratification of patients with ACS that have been validated in the inpatient environment (e.g. Thrombolysis in Myocardial Infarction (TIMI) score, Global Registry of Acute Coronary Events (GRACE) score, Fast Revascularisation in Instability in Coronary Disease (FRISC) score or Goldman Criteria) should not be used to identify low-risk patients suitable for discharge from the ED.

A subgroup of patients younger than 40 years with non-classical presentations and lacking significant past medical history, who have normal serial biomarkers and 12-lead ECGs, have a very low short-term event rate.

Chest pain observation protocols

In patients suspected of an ACS the combination of an unremarkable past history and physical examination with negative initial ECG and biomarkers cannot be used to exclude ACS reliably. Therefore a follow up period is mandatory in order to reach a diagnosis and make therapeutic decisions.

Chest pain observation protocols are rapid systems for assessment of patients with suspected ACS. They should generally include a history and physical examination, followed by a period of obser-

vation, during which serial electrocardiography and cardiac marker measurements are performed. Patient evaluation should be complemented by either a non-invasive evaluation for anatomical coronary disease or provocative testing for inducible myocardial ischaemia at some point after AMI is excluded. These protocols may be used to improve accuracy in identifying patients requiring inpatient admission or further diagnostic testing while maintaining patient safety, reducing length of stay and reducing costs [27].

In patients presenting to the ED with a history suggestive of ACS, but normal initial workup, chest pain (observation) units may represent a safe and effective strategy for evaluating patients. They may be recommended as a means to reduce length of stay, hospital admissions and healthcare costs, improve diagnostic accuracy and improve quality of life [28]. There is no direct evidence demonstrating that chest pain units or observation protocols reduce adverse cardiovascular outcomes, particularly mortality, for patients presenting with possible ACS.

Imaging techniques

Effective screening of patients with suspected ACS, but with negative ECG and negative cardiac biomarkers, remains challenging. Non-invasive imaging techniques (CT angiography [29], cardiac magnetic resonance, myocardial perfusion imaging [30], and echocardiography [31]) have been evaluated as means of screening these low-risk patients and identifying subgroups that can be discharged home safely.

Although there are no large multicentre trials, existing evidence indicates that these diagnostic modalities enable early and accu-

rate diagnosis with a reduction in length of stay and costs without increasing cardiac events. Both the exposure to radiation and iodinated contrast should be considered when using multi-detector computer tomography (MDCT) and myocardial perfusion imaging.

Treatment of acute coronary syndromes—symptoms

Nitrates

Glyceryl trinitrate is an effective treatment for ischaemic chest pain and has beneficial haemodynamic effects, such as dilation of the venous capacitance vessels, dilation of the coronary arteries and, to a minor extent, the peripheral arteries. Glyceryl trinitrate may be considered if the systolic blood pressure is above 90 mm Hg and the patient has ongoing ischaemic chest pain (Fig. 5.2). Glyceryl trinitrate can also be useful in the treatment of acute pulmonary congestion. Nitrates should not be used in patients with hypotension (systolic blood pressure ≤ 90 mm Hg), particularly if combined with bradycardia, and in patients with inferior infarction and suspected right ventricular involvement. Use of nitrates under these circumstances can decrease the blood pressure and cardiac output.

Analgesia

Morphine is the analgesic of choice for nitrate-refractory pain and also has calming effects on the patient making sedatives unnecessary in most cases. Since morphine is a dilator of venous capacitance vessels, it may have additional benefit in patients with pulmonary congestion. Give morphine in initial doses of 3–5 mg

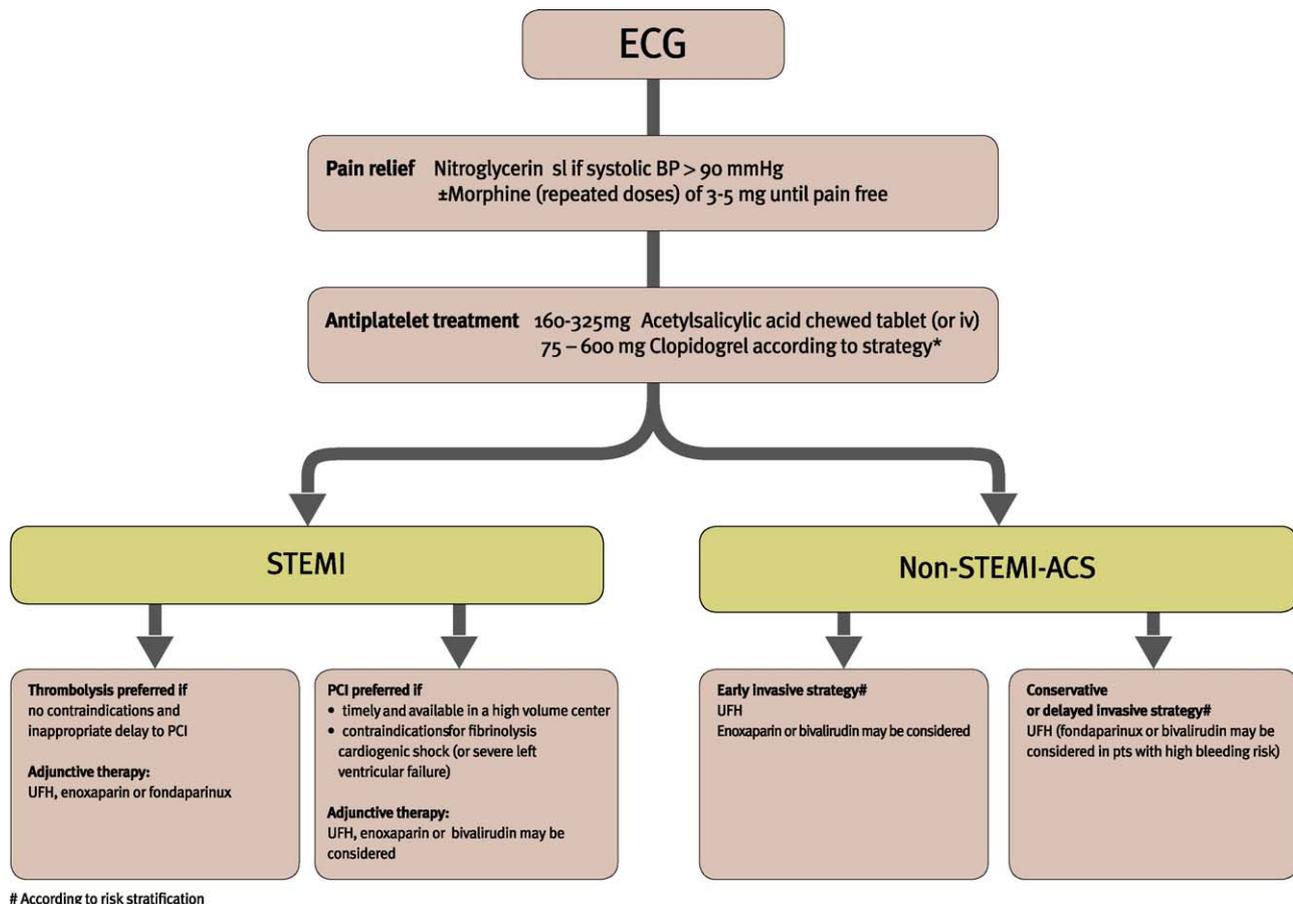


Fig. 5.2. Treatment algorithm for acute coronary syndromes (BP, blood pressure; PCI, percutaneous coronary intervention; UFH, unfractionated heparin). *Prasugrel, 60 mg loading dose, may be chosen as an alternative to clopidogrel in patients with STEMI and planned PPCI provided there is no history of stroke or transient ischaemic attack. At the time of writing, ticagrelor has not yet been approved as an alternative to clopidogrel.

intravenously and repeat every few minutes until the patient is pain-free. Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided for analgesia because of their pro-thrombotic effects [32].

Oxygen

Monitoring of the arterial oxygen saturation with pulse oximetry (SpO₂) will help to determine the need for supplemental oxygen. These patients do not need supplemental oxygen unless they are hypoxaemic. Limited data suggest that high-flow oxygen may be harmful in patients with uncomplicated myocardial infarction [33–35]. Aim to achieve an oxygen saturation of 94–98%, or 88–92% if the patient is at risk of hypercapnic respiratory failure [36].

Treatment of acute coronary syndromes—cause

Inhibitors of platelet aggregation

Inhibition of platelet aggregation is of primary importance for initial treatment of coronary syndromes as well as for secondary prevention, since platelet activation and aggregation is the key process initiating an ACS.

Acetylsalicylic acid (ASA)

Large randomised controlled trials indicate decreased mortality when ASA (75–325 mg) is given to hospitalised patients with ACS. A few studies have suggested reduced mortality if ASA is given earlier [37,38]. Therefore, give ASA as soon as possible to all patients with suspected ACS unless the patient has a known true allergy to ASA. ASA may be given by the first healthcare provider, bystander or by dispatcher assistance according to local protocols. The initial dose of chewable ASA is 160–325 mg. Other forms of ASA (soluble, IV) may be as effective as chewed tablets.

Adenosine diphosphate (ADP)-receptor inhibitors

Thienopyridines (clopidogrel, prasugrel) and the cyclo-pentyl-triazolo-pyrimidine, ticagrelor, inhibit the ADP-receptor irreversibly, which further reduces platelet aggregation in addition to that produced by ASA. In contrast to clopidogrel, metabolism of prasugrel and of ticagrelor is independent of a genetically determined variability of drug metabolism and activation. Therefore prasugrel and ticagrelor lead to a more reliable and stronger inhibition of platelet aggregation.

A large randomised study comparing a loading dose of 300 mg clopidogrel followed by 75 mg daily with prasugrel (loading dose 60 mg, followed by 10 mg daily) in patients with ACS resulted in fewer major adverse cardiac events (MACE) with prasugrel; however, the bleeding rate was higher. Bleeding risk was increased markedly in patients weighing less than 60 kg and those older than 75 years [39]. A significantly increased intracranial bleeding rate was observed in patients with a history of transient ischaemic attack (TIA) and/or stroke. In another study, ticagrelor proved to be superior to clopidogrel with respect to MACE [40]. At the time of writing, ticagrelor has not yet been approved as an alternative to clopidogrel.

ADP-receptor inhibitors in NON-STEMI ACS

Clopidogrel. If given in addition to heparin and ASA in high-risk non-STEMI-ACS patients, clopidogrel improves outcome [41,42]. Even if there is no large scale study investigating pre-treatment with clopidogrel, compared with peri-interventional application

– either with a 300 or 600 mg loading dose – do not postpone treatment until angiography/PCI is undertaken because the highest event rates are observed in the early phase of the syndrome. In unselected patients undergoing PCI, pre-treatment with a higher loading dose of clopidogrel resulted in better outcome [43].

Therefore, clopidogrel should be given as early as possible in addition to ASA and an antithrombin to all patients presenting with non-STEMI ACS. If a conservative approach is selected, give a loading dose of 300 mg; with a planned PCI strategy, an initial dose of 600 mg may be preferred.

Prasugrel. Prasugrel (60 mg loading dose) may be given instead of clopidogrel to patients with high-risk non-STEMI ACS and planned PCI at angiography, provided coronary stenoses are suitable for PCI. Contraindications (history of TIA/stroke) and the benefit – risk relation in patients with high bleeding risk (weight <60 kg, age >75 years) should be considered.

ADP-receptor inhibitors in STEMI

Clopidogrel. Although there is no large study on the use of clopidogrel for pre-treatment of patients presenting with STEMI and planned PCI, it is likely that this strategy is beneficial. Since platelet inhibition is more profound with a higher dose, a 600 mg loading dose given as soon as possible is recommended for patients presenting with STEMI and planned PCI.

Two large randomised trials studied clopidogrel compared with placebo in patients with STEMI treated conservatively or with fibrinolysis [44,45]. One study included patients up to 75 years of age, treated with fibrinolysis, ASA, an antithrombin and a loading dose of 300 mg clopidogrel [45]. Treatment with clopidogrel resulted in fewer occluded culprit coronary arteries at angiography and fewer re-infarctions, without an increased bleeding risk. The other study investigated STEMI patients without age limits to be treated conservatively or with fibrinolysis. In this trial, clopidogrel (no loading, 75 mg daily) compared with placebo resulted in fewer deaths and a reduction of the combined endpoint of death and stroke [44]. Therefore patients with STEMI treated with fibrinolysis should be treated with clopidogrel (300 mg loading dose up to an age of 75 years and 75 mg without loading dose if >75 years of age) in addition to ASA and an antithrombin.

Prasugrel. Prasugrel with a loading dose of 60 mg may be given in addition to ASA and an antithrombin to patients presenting with STEMI with planned PCI. Contraindications (history of TIA/stroke), and relation of bleeding risk vs benefit in patients with a body weight <60 kg or aged >75 years should be taken into account. There is no data on prehospital treatment with prasugrel and no data on prasugrel if used in the context of fibrinolysis.

Glycoprotein (Gp) IIB/IIIa inhibitors

Gp IIB/IIIa receptor inhibition is the common final link of platelet aggregation. Eptifibatid and tirofiban lead to reversible inhibition, while abciximab leads to irreversible inhibition of the Gp IIB/IIIa receptor. Older studies from the pre-stent era mostly support the use of this class of drugs [46,47]. Newer studies mostly document neutral or worsened outcomes [48–51]. Finally in most supporting, as well as neutral or opposing studies, bleeding occurred in more patients treated with Gp IIB/IIIa receptor blockers. There are insufficient data to support routine pre-treatment with Gp IIB/IIIa inhibitors in patients with STEMI or non-STEMI-ACS. For high-risk patients with non-STEMI-ACS, in-hospital upstream treatment with eptifibatid or tirofiban may be acceptable whereas abciximab may be given only in the context of PCI [47,52]. Newer alternatives for antiplatelet treatment should be considered because of

the increased bleeding risk with Gp IIB/IIIA inhibitors when used with heparins.

Antithrombins

Unfractionated heparin (UFH) is an indirect inhibitor of thrombin, which in combination with ASA is used as an adjunct with fibrinolytic therapy or primary PCI (PPCI) and is an important part of treatment of unstable angina and STEMI. Limitations of unfractionated heparin include its unpredictable anticoagulant effect in individual patients, the need to give it intravenously and the need to monitor aPTT. Moreover, heparin can induce thrombocytopenia. Since publication of the 2005 ERC guidelines on ACS, large randomised trials have been performed testing several alternative antithrombins for the treatment of patients with ACS. In comparison with UFH, these alternatives have a more specific factor Xa activity (low molecular weight heparins [LMWH], fondaparinux) or are direct thrombin inhibitors (bivalirudin). With these newer antithrombins, in general, there is no need to monitor the coagulation system and there is a reduced risk of thrombocytopenia.

Antithrombins in non-STEMI-ACS

In comparison with UFH, enoxaparin reduces the combined endpoint of mortality, myocardial infarction and the need for urgent revascularisation, if given within the first 24–36 h of onset of symptoms of non-STEMI-ACS [53,54]. Although enoxaparin causes more minor bleeding than UFH, the incidence of serious bleeding is not increased.

Bleeding worsens the prognosis of patients with ACS [55]. Fondaparinux and bivalirudin cause less bleeding than UFH [56–59]. In most of the trials on patients presenting with non-STEMI-ACS, the UFH alternatives were given only after hospital admission; it may be invalid to extrapolate the results of these studies to the prehospital or ED setting. For patients with a planned initial conservative approach, fondaparinux and enoxaparin are reasonable alternatives to UFH. There are insufficient data to recommend any LMWH other than enoxaparin. For patients with an increased bleeding risk consider giving fondaparinux or bivalirudin. For patients with a planned invasive approach, enoxaparin or bivalirudin are reasonable alternatives to UFH. In one study, catheter thrombi were observed in patients undergoing PCI who had received fondaparinux – additional UFH was required [56]. Since enoxaparin and fondaparinux may accumulate in patients with renal impairment, dose adjustment is necessary; bivalirudin or UFH are alternatives in this situation. Bleeding risk may be increased by switching the anticoagulant; therefore, the initial agent should be maintained with the exception of fondaparinux where additional UFH is necessary for patients undergoing PCI [60].

Antithrombins in STEMI

Antithrombins for patients to be treated with fibrinolysis

Enoxaparin. Several randomised studies of patients with STEMI undergoing fibrinolysis have shown that additional treatment with enoxaparin instead of UFH produced better clinical outcomes (irrespective of the fibrinolytic used) but a slightly increased bleeding rate in elderly (≥ 75 years) and low weight patients (BW < 60 kg) [61–63]. Reduced doses of enoxaparin in elderly and low weight patients maintained the improved outcome while reducing the bleeding rate [64]. It is also reasonable to give enoxaparin instead of UFH for prehospital treatment.

Dosing of enoxaparin: In patients <75 years, give an initial bolus of 30 mg IV followed by 1 mg kg⁻¹ SC every 12 h (first SC dose shortly after the IV bolus). Treat patients ≥ 75 years

with 0.75 mg kg⁻¹ SC every 12 h without an initial IV dose. Patients with known impaired renal function (creatinine clearance <30 ml⁻¹ min⁻¹) may be given 1 mg kg⁻¹ enoxaparin SC once daily or may be treated with UFH. There are insufficient data to recommend other LMWH.

Fondaparinux. Several studies show superiority or neutral outcome when fondaparinux was compared with UFH as an adjunct for fibrinolysis in STEMI patients [56]. Fondaparinux (initially 2.5 mg SC followed by 2.5 mg SC daily) may be considered specifically with non-fibrin-specific fibrinolytics (i.e. streptokinase) in patients with a plasma creatinine concentration <3 mg dl⁻¹ (250 μ m l⁻¹).

Bivalirudin. There are insufficient data to recommend bivalirudin instead of UFH in STEMI patients to be treated with fibrinolysis. Since bleeding risk may be increased by switching the anticoagulants, the initial agent should be maintained, with the exception of fondaparinux, where additional UFH is necessary if an invasive procedure is planned [60].

Antithrombins for STEMI patients to be treated with primary PCI (PPCI)

There is a paucity of studies on prehospital or ED initiation of antithrombin treatment for patients with STEMI and planned PPCI. Therefore treatment recommendations for these settings have to be extrapolated from in-hospital investigations, until the more specific results of ongoing studies are available.

Enoxaparin. Several registries and smaller studies documented favourable or neutral outcome when enoxaparin was compared with UFH for contemporary PPCI (i.e. broad use of thienopyridines and/or Gp IIB/IIIA receptor blockers) [65,66]. Therefore, enoxaparin is a safe and effective alternative to UFH. There are insufficient data to recommend any LMWH other than enoxaparin for PPCI in STEMI. Switching from UFH to enoxaparin or vice versa may lead to an increased bleeding risk and therefore should be avoided [60]. Dose adjustment of enoxaparin is necessary for patients with renal impairment.

Fondaparinux. When compared with UFH, fondaparinux resulted in similar clinical outcomes but less bleeding when used in the context of PPCI [56]; however, thrombus formation on catheters required treatment with additional UFH. Even if fondaparinux reduces the bleeding risk compared with UFH in STEMI patients undergoing PPCI, the use of the two agents is not recommended over UFH alone. The dose of fondaparinux requires adjustment in patients with renal impairment.

Bivalirudin. Two large randomised studies documented less bleeding and a reduction in short and long term mortality when bivalirudin was compared with UFH plus Gp IIB/IIIA receptor blockers in patients with STEMI and planned PCI [67–69]. Several other studies and case series showed also better or neutral results and less bleeding when bivalirudin was compared with UFH; therefore, bivalirudin is a safe alternative to UFH. However, a slightly increased rate of stent thrombosis was observed within the first 24 h after PCI [67].

Strategies and systems of care

Several systematic strategies to improve quality of out-of-hospital care for patients with ACS have been investigated. These strategies are principally intended to promptly identify patients with STEMI in order to shorten the delay to reperfusion treatment. Also triage criteria have been developed to select high-risk patients

with non-STEMI-ACS for transport to tertiary care centres offering 24/7 PCI services. In this context, several specific decisions have to be made during initial care beyond the basic diagnostic steps necessary for clinical evaluation of the patient and interpretation of a 12-lead ECG. These decisions relate to:

- (1) Reperfusion strategy in patients with STEMI i.e. PPCI vs (pre-) hospital fibrinolysis.
- (2) Bypassing a closer but non-PCI capable hospital and taking measures to shorten the delay to intervention if PPCI is the chosen strategy.
- (3) Procedures in special situations e.g. for patients successfully resuscitated from non-traumatic cardiac arrest, patients with shock or patients with non-STEMI ACS who are unstable or have signs of very high risk.

Reperfusion strategy in patients presenting with STEMI

Reperfusion therapy in patients with STEMI is the most important advance in the treatment of myocardial infarction in the last 25 years. For patients presenting with STEMI within 12 h of symptom onset, reperfusion should be initiated as soon as possible independent of the method chosen [7,70–72]. Reperfusion may be achieved with fibrinolysis, with PPCI, or a combination of both. Efficacy of reperfusion therapy is profoundly dependent on the duration of symptoms. Fibrinolysis is effective specifically in the first 2–3 h after symptom onset; PPCI is less time sensitive [73].

Fibrinolysis

A meta-analysis of six trials involving 6434 patients documented a 17% decrease in mortality among patients treated with out-of-hospital fibrinolysis compared with in-hospital fibrinolysis [74]. An effective and safe system for out-of-hospital fibrinolytic therapy requires adequate facilities for the diagnosis and treatment of STEMI and its complications. Ideally, there should be a capability of communicating with experienced hospital doctors (e.g. emergency physicians or cardiologists). The average time gained with out-of-hospital fibrinolysis was 60 min, and the results were independent of the experience of the provider. Thus, giving fibrinolytics out-of-hospital to patients with STEMI or signs and symptoms of an ACS with presumed new LBBB is beneficial. Fibrinolytic therapy can be given safely by trained paramedics, nurses or physicians using an established protocol [75–80]. The efficacy is greatest within the first 3 h of the onset of symptoms [74]. Patients with symptoms of ACS and ECG evidence of STEMI (or presumably new LBBB or true posterior infarction) presenting directly to the ED should be given fibrinolytic therapy as soon as possible unless there is timely access to PPCI.

Risks of fibrinolytic therapy

Healthcare professionals who give fibrinolytic therapy must be aware of its contraindications (Table 5.1) and risks. Patients with large AMIs (e.g. indicated by extensive ECG changes) are likely to gain most from fibrinolytic therapy. Benefits of fibrinolytic therapy are less impressive in inferior wall infarctions than in anterior infarctions. Older patients have an absolute higher risk of death, but the absolute benefit of fibrinolytic therapy is similar to that of younger patients. Patients over 75 years have an increased risk of intracranial bleeding from fibrinolysis; thus, the absolute benefit of fibrinolysis is reduced by this complication. The risk of intracranial bleeding is increased in patients with a systolic blood pressure of over 180 mm Hg; this degree of hypertension is a relative contraindication to fibrinolytic therapy. The risk of intracranial bleeding is also depending on the use of antithrombin and antiplatelet therapy.

Table 5.1
Contraindications for fibrinolysis.^a

Absolute contraindications
Haemorrhagic stroke or stroke of unknown origin at any time
Ischaemic stroke in the preceding 6 months
Central nervous system damage or neoplasms
Recent major trauma/surgery/head injury (within the preceding 3 weeks)
Gastro-intestinal bleeding within the last month
Known bleeding disorder
Aortic dissection
Relative contraindications
Transient ischaemic attack in preceding 6 months
Oral anticoagulant therapy
Pregnancy within 1-week post-partum
Non-compressible punctures
Traumatic resuscitation
Refractory hypertension (systole. blood pressure >180 mm Hg)
Advanced liver disease
Infective endocarditis
Active peptic ulcer

^a According to the guidelines of the European Society of Cardiology.

Primary percutaneous intervention

Coronary angioplasty with or without stent placement has become the first-line treatment for patients with STEMI, because it has been shown to be superior to fibrinolysis in the combined endpoints of death, stroke and reinfarction in several studies and meta-analyses [81,82]. This improvement was found when PPCI was undertaken by a skilled person in a high-volume centre with a limited delay to first balloon inflation after first medical contact [83]. Therefore PPCI performed at a high-volume centre shortly after first medical contact (FMC), by an experienced operator who maintains an appropriate expert status, is the preferred treatment as it improves morbidity and mortality as compared with immediate fibrinolysis.

Fibrinolysis vs primary PCI

Primary PCI has been limited by access to catheter laboratory facilities, appropriately skilled clinicians and delay to first balloon inflation. Fibrinolysis therapy is a widely available reperfusion strategy. Both treatment strategies are well established and have been the subject of large randomised multicentre trials over the last decades. Over this time both therapies have evolved significantly and the body of evidence is heterogeneous. In the randomised studies comparing PPCI with fibrinolytic therapy, the typical delay from decision to the beginning of treatment with either PPCI or fibrinolytic therapy was less than 60 min. Several reports and registries comparing fibrinolytic (including prehospital administration) therapy with PPCI showed a trend of improved survival if fibrinolytic therapy was initiated within 2 h of onset of symptoms and was combined with rescue or delayed PCI [84–86]. In registries that reflect standard practice more realistically the acceptable PPCI related delay (i.e. the diagnosis to balloon interval minus the diagnosis to needle interval) to maintain the superiority of PPCI over fibrinolysis varied considerably between 45 and >180 min depending on the patients' conditions (i.e. age, localisation of infarction, and duration of symptoms) [87]. Moreover there are few data for benefit of PPCI over fibrinolysis in specific subgroups such as patients post-CABG, with renal failure or with diabetes [88,89]. Time delay to PCI may be significantly shortened by improving the systems of care [13,90–93], e.g.

- Prehospital ECG registration
- ECG transmission to the receiving hospital
- Arranging single call activation of the catheterization laboratory

- Requiring the catheterization laboratory to be ready within 20 min
- Having an attending cardiologists always at the hospital
- Providing real-time data feedback
- Fostering senior management commitment
- Encouraging a team-based approach

If PPCI cannot be accomplished within an adequate timeframe, independent of the need for emergent transfer, then immediate fibrinolysis should be considered unless there is a contraindication. For those patients with a contraindication to fibrinolysis, PCI should still be pursued despite the delay, rather than not providing reperfusion therapy at all. For those STEMI patients presenting in shock, primary PCI (or coronary artery bypass surgery) is the preferred reperfusion treatment. Fibrinolysis should only be considered if there is a substantial delay to PCI.

Triage and inter-facility transfer for primary PCI

The risk of death, reinfarction or stroke is reduced if patients with STEMI are transferred promptly from community hospitals to tertiary care facilities for PPCI [82,94,95]. It is less clear whether immediate fibrinolytic therapy (in- or out-of-hospital) or transfer for PPCI is superior for younger patients presenting with anterior infarction and within a short duration of <2–3 h [87]. Transfer of STEMI patients for PPCI is reasonable for those presenting more than 3 h but less than 12 h after the onset of symptoms, provided that the transfer can be achieved rapidly.

Combination of fibrinolysis and percutaneous coronary intervention

Fibrinolysis and PCI may be used in a variety of combinations to restore coronary blood flow and myocardial perfusion. There are several ways in which the two therapies can be combined. There is some lack of uniformity in the nomenclature used to describe PCI in these regimens. Facilitated PCI is used to describe PCI performed immediately after fibrinolysis, a pharmaco-invasive strategy refers to PCI performed routinely 3–24 h after fibrinolysis, and rescue PCI is defined as PCI performed for a failed reperfusion (as evidenced by <50% resolution of ST-segment elevation at 60–90 min after completion of fibrinolytic treatment). These strategies are distinct from a routine PCI approach where the angiography and intervention is performed several days after successful fibrinolysis.

Several studies and meta-analyses demonstrate worse outcome with routine PCI performed immediately or as early as possible after fibrinolysis [48,95]. Therefore routine facilitated PCI is not recommended even if there may be some specific subgroups of patients which may benefit from this procedure [96]. It is reasonable to perform angiography and PCI when necessary in patients with failed fibrinolysis according to clinical signs and/or insufficient ST-segment resolution [97].

In case of clinically successful fibrinolysis (evidenced by clinical signs and ST-segment resolution >50%), angiography delayed by several hours after fibrinolysis (the 'pharmaco-invasive' approach) has been shown to improve outcome. This strategy includes early transfer for angiography and PCI if necessary after fibrinolytic treatment [98,99].

Special situations

Cardiogenic shock

Cardiogenic shock (and to some extent severe left ventricular failure) is one of the complications of ACS and has a mortality of more than 50%. Cardiogenic shock in STEMI is not a contraindication to fibrinolytic therapy, but PCI is the treatment of choice. Early

revascularisation (i.e. PPCI, PCI early after fibrinolysis) is indicated for those patients who develop shock within 36 h after symptom onset of AMI and are suitable for revascularisation [100].

Suspect right ventricular infarction in patients with inferior infarction, clinical shock and clear lung fields. ST-segment elevation ≥ 1 mm in lead V4R is a useful indicator of right ventricular infarction. These patients have an in-hospital mortality of up to 30% and many benefit greatly from reperfusion therapy. Avoid nitrates and other vasodilators, and treat hypotension with intravenous fluid.

Reperfusion after successful CPR

Coronary heart disease is the most frequent cause of out-of-hospital cardiac arrest. Many of these patients will have an acute coronary occlusion with signs of STEMI on the ECG, but cardiac arrest due to ischaemic heart disease can also occur in the absence of these findings. Several case series have shown that angiography and, if necessary, PCI is feasible in patients with return of spontaneous circulation (ROSC) after cardiac arrest. In many patients coronary artery occlusion or high degree stenoses can be identified and treated. Fibrinolysis may be an alternative in patients with ECG signs of STEMI [101]. Therefore in patients with STEMI or new LBBB on ECG following ROSC after out-of-hospital cardiac arrest, immediate angiography and percutaneous intervention or fibrinolysis should be considered [102,103]. It is reasonable to perform immediate angiography and PCI in selected patients despite the lack of ST elevation on the ECG or prior clinical findings such as chest pain. It is reasonable to include reperfusion treatment in a standardized post-cardiac arrest protocol as part of a strategy to improve outcome [104]. Reperfusion treatment should not preclude other therapeutic strategies including therapeutic hypothermia.

Primary and secondary prevention

Preventive interventions in patients presenting with an ACS should be initiated early after hospital admission and should be continued if already in place. Preventive measures improve prognosis by reducing the number of major adverse cardiac events. Prevention with drugs encompasses beta-blockers, angiotensin converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARB) and statins, as well as basic treatment with ASA and, if indicated, thienopyridines.

Beta-blockers

Several studies, undertaken mainly in the pre-reperfusion era, indicate a decreased mortality, incidence of reinfarction and cardiac rupture as well as a lower incidence of ventricular fibrillation and supraventricular arrhythmia in patients treated early with a beta-blocker [105]. Intravenous beta-blockade may also reduce mortality in patients undergoing PPCI who are not on oral beta-blockers.

Beta-blocker studies are very heterogeneous with respect to time of start of treatment. There is paucity of data on administration in the prehospital or ED settings. Moreover, recent studies indicate an increased risk of cardiogenic shock in patients with STEMI, even if the rate of severe tachyarrhythmia is reduced by beta-blockade [106].

There is no evidence to support routine intravenous beta-blockers in the prehospital or initial ED settings. It may be indicated in special situations such as severe hypertension or tachyarrhythmias in the absence of contraindications. It is reasonable to start oral beta-blockers at low doses only after the patient is stabilized.

Anti-arrhythmics

There is no evidence to support the use of anti-arrhythmic prophylaxis after ACS. Ventricular fibrillation (VF) accounts for most of the early deaths from ACS; the incidence of VF is highest in the first hours after onset of symptoms. This explains why numerous studies have been performed with the aim of demonstrating the prophylactic effect of antiarrhythmic therapy [107]. The effects of antiarrhythmic drugs (lidocaine, magnesium, disopyramide, mexiletine, verapamil, sotalol, and tocainamide) given prophylactically to patients with ACS have been studied. Prophylaxis with lidocaine reduces the incidence of VF but may increase mortality [108]. Routine treatment with magnesium in patients with AMI does not improve mortality. Arrhythmia prophylaxis using disopyramide, mexiletine, verapamil, or other anti-arrhythmics given within the first hours of an ACS does not improve mortality. Therefore prophylactic anti-arrhythmics are not recommended.

Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers

Oral ACE inhibitors reduce mortality when given to patients with AMI with or without early reperfusion therapy. The beneficial effects are most pronounced in patients presenting with anterior infarction, pulmonary congestion or left ventricular ejection fraction <40%. Do not give ACE inhibitors if the systolic blood pressure is less than 100 mm Hg on admission or if there is a known contraindication to these drugs. A trend towards higher mortality has been documented if an intravenous ACE inhibitor is started within the first 24 h after onset of symptoms. Therefore, give an oral ACE inhibitor within 24 h after symptom onset in patients with AMI regardless of whether early reperfusion therapy is planned, particularly in those patients with anterior infarction, pulmonary congestion or a left ventricular ejection fraction below 40%. Do not give intravenous ACE inhibitors within 24 h of onset of symptoms. Give an angiotensin receptor blocker (ARB) to patients intolerant of ACE inhibitors [109,110].

Statins

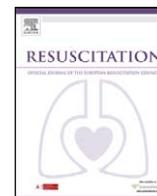
Statins reduce the incidence of major adverse cardiovascular events when given early within the first days after onset an ACS [111,112]. Initiation of statin therapy should be considered within 24 h of onset of symptoms of ACS unless contraindicated (target LDL cholesterol values <80 mg dl⁻¹ [2.1 mmol l⁻¹]). If patients are already receiving statin therapy, it should be not interrupted [113].

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European Resuscitation Council Guidelines for Resuscitation 2010 Section 6. Paediatric life support

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Introduction

These guidelines on paediatric life support are based on two main principles: (1) the incidence of critical illness, particularly cardiopulmonary arrest, and injury in children is much lower than in adults; (2) most paediatric emergencies are served primarily by providers who are not paediatric specialists and who have limited paediatric emergency medical experience. Therefore, guidelines on paediatric life support must incorporate the best available scientific evidence but must also be simple and feasible. Finally, international guidelines need to acknowledge the variation in national and local emergency medical infrastructures and allow flexibility when necessary.

The process

The European Resuscitation Council (ERC) published guidelines for paediatric life support (PLS) in 1994, 1998, 2000 and 2005.^{1–5} The latter two were based on the International Consensus on Science published by the International Liaison Committee on Resuscitation (ILCOR).^{6–8} This process was repeated in 2009/2010, and the resulting Consensus on Science with Treatment Recommendations (CoSTR) was published simultaneously in Resuscitation, Circulation and Pediatrics.^{9,10} The PLS Working Party of the ERC has developed the ERC PLS Guidelines based on the 2010 CoSTR and supporting scientific literature. The guidelines for resuscitation of babies at birth are now covered in Section 7.¹¹

Summary of changes since the 2005 Guidelines

Guideline changes have been made in response to convincing new scientific evidence and to simplify teaching and retention. As before, there remains a paucity of good-quality evidence on paediatric resuscitation. Therefore to facilitate and support dissemination and implementation of the PLS Guidelines, changes have been made only if there is new, high-level scientific evidence or to ensure consistency with the adult guidelines. The feasibility of applying the same guidance for all adults and children remains a major topic of study. Major changes in these new guidelines include:

Recognition of cardiac arrest

Healthcare providers cannot reliably determine the presence or absence of a pulse in less than 10 s in infants or children.^{12,13} Therefore pulse palpation cannot be the sole determinant of cardiac arrest and the need for chest compressions. If the victim is unresponsive, not breathing normally, and there are no signs of life, lay rescuers should begin CPR. Healthcare providers should look for signs of life and if they are confident in the technique, they may add pulse palpation for diagnosing cardiac arrest and decide whether they should begin chest compressions or not. The decision to begin CPR must be taken in less than 10 s. According to the child's age, carotid (children), brachial (infants) or femoral pulse (children and infants) checks may be used.^{14,15}

Compression ventilation ratios

The compression ventilation (CV) ratio used for children should be based on whether one, or more than one rescuer is present.¹⁶ Lay rescuers, who usually learn only single-rescuer techniques, should

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be taught to use a ratio of 30 compressions to 2 ventilations which is the same as the adult guidelines and enables anyone trained in basic life support (BLS) to resuscitate children with minimal additional information. Rescuers with a duty to respond should learn and use a 15:2 CV ratio as this has been validated in animal and manikin studies.^{17–21} This latter group, who would normally be healthcare professionals, should receive enhanced training targeted specifically at the resuscitation of children. For them, simplicity would be lost if a different ratio was taught for the scenario when one or two or more rescuers were present. However, those with a duty to respond can use the 30:2 ratio if they are alone, particularly if they are not achieving an adequate number of compressions because of difficulty in the transition between ventilation and compression. Ventilation remains a very important component of CPR in asphyxial arrests.²² Rescuers who are unable or unwilling to provide mouth-to-mouth ventilation should be encouraged to perform at least compression-only CPR.

CPR quality

The compression technique for infants includes two-finger compression for single rescuers and the two-thumb encircling technique for two or more rescuers.^{23–27} For older children, a one- or two-hand technique can be used, according to rescuer preference.²⁸ The emphasis is on achieving an adequate depth of compression: at least 1/3 of the anterior-posterior chest diameter in all children (i.e., approximately 4 cm in infants and approximately 5 cm in children). Subsequent complete release should also be emphasised. Chest compressions must be performed with minimal interruptions to minimise no-flow time. For both infants and children, the compression rate should be at least 100 but not greater than 120 min⁻¹.

Defibrillation

Automated external defibrillators

Case reports indicate that automated external defibrillators (AEDs) are safe and successful when used in children older than 1 year of age.^{29,30} Automated external defibrillators are capable of identifying arrhythmias in children accurately; in particular, they are extremely unlikely to advise a shock inappropriately.^{31–33} Consequently, the use of AEDs is indicated in all children aged greater than 1 year.³⁴ Nevertheless, if there is any possibility that an AED may need to be used in children, the purchaser should check that the performance of the particular model has been tested against paediatric arrhythmias. Many manufacturers now supply purpose-made paediatric pads or software, which typically attenuate the output of the machine to 50–75 J³⁵ and these are recommended for children aged 1–8 years.^{36,37} If an attenuated shock or a manually adjustable machine is not available, an unmodified adult AED may be used in children older than 1 year.³⁸ The evidence to support a recommendation for the use of AEDs in children aged less than 1 year is limited to case reports.^{39,40} The incidence of shockable rhythms in infants is very low except when they suffer from cardiac disease.^{41–43} In these rare cases, the risk/benefit ratio may be favourable and use of an AED (preferably with dose attenuator) should be considered.

Manual defibrillators

The treatment recommendation for paediatric ventricular fibrillation (VF) or paediatric pulseless ventricular tachycardia (VT) remains immediate defibrillation. In adult advanced life support (ALS), the recommendation is to give a single shock and then resume CPR immediately without checking for a pulse or re-

assessing the rhythm (see Section 4).^{44–47} To reduce the no-flow time, chest compressions should be continued while applying and charging the paddles or self-adhesive pads (if the size of the child's chest allows this). Chest compressions should be briefly paused once the defibrillator is charged to deliver the shock. The ideal energy dose for safe and effective defibrillation in children is unknown, but animal models and small paediatric case series show that doses larger than 4 J kg⁻¹ defibrillate effectively with negligible side effects.^{29,37,48,49} Clinical studies in children indicate that doses of 2 J kg⁻¹ are insufficient in most cases.^{13,42,50} Biphasic shocks are at least as effective and produce less post-shock myocardial dysfunction than monophasic shocks.^{36,37,49,51–53}

Therefore, for simplicity and consistency with adult BLS and ALS guidance, a single-shock strategy using a non-escalating dose of 4 J kg⁻¹ (preferably biphasic but monophasic is acceptable) is recommended for defibrillation in children. Use the largest size paddles or pads that fit on the infant or child's chest in the antero-lateral or antero-posterior position without the pads/paddles touching each other.¹³

Airway

Cuffed tracheal tubes

Cuffed tracheal tubes can be used safely in infants and young children. The size should be selected by applying a validated formula.

Cricoid pressure

The safety and value of using cricoid pressure during tracheal intubation is not clear. Therefore, the application of cricoid pressure should be modified or discontinued if it impedes ventilation or the speed or ease of intubation.

Capnometry

Monitoring exhaled carbon dioxide (CO₂), ideally by capnography, is helpful to confirm correct tracheal tube position and recommended during CPR to help assess and optimize its quality.

Titration of oxygen

Based on increasing evidence of potential harm from hyperoxaemia after cardiac arrest, once spontaneous circulation is restored, inspired oxygen should be titrated to limit the risk of hyperoxaemia.

Rapid response systems

Implementation of a rapid response system in a paediatric in-patient setting may reduce rates of cardiac and respiratory arrest and in-hospital mortality.

New topics

New topics in the 2010 guidelines include channelopathies (i.e., the importance of autopsy and subsequent family testing) and several new special circumstances: trauma, single ventricle pre- and post-1st stage repair, post-Fontan circulation, and pulmonary hypertension.

Terminology

In the following text the masculine includes the feminine and child refers to both infants and children unless noted otherwise.

The term *newly born* refers to a neonate immediately after delivery. A *neonate* is a child within 4 weeks of age. An *infant* is a child under 1 year of age, and the term *child* refers to children between 1 year and onset of puberty. From puberty children are referred to as *adolescents* for whom the adult guidelines apply. Furthermore, it is necessary to differentiate between infants and older children, as there are some important differences with respect to diagnostic and interventional techniques between these two groups. The onset of puberty, which is the physiological end of childhood, is the most logical landmark for the upper age limit for use of paediatric guidance. If rescuers believe the victim to be a child they should use the paediatric guidelines. If a misjudgement is made and the victim turns out to be a young adult, little harm will accrue, as studies of aetiology have shown that the paediatric pattern of cardiac arrest continues into early adulthood.⁵⁴

A. Paediatric basic life support

Sequence of actions

Rescuers who have been taught adult BLS and have no specific knowledge of paediatric resuscitation may use the adult sequence, as outcome is worse if they do nothing. Non-specialists who wish to learn paediatric resuscitation because they have responsibility for children (e.g., teachers, school nurses, lifeguards), should be taught that it is preferable to modify adult BLS and perform five initial breaths followed by approximately 1 min of CPR before they go for help (see adult BLS guideline).

The following sequence is to be followed by *those with a duty to respond to paediatric emergencies* (usually health professional teams) (Fig. 6.1).

1. Ensure the safety of rescuer and child.
2. Check the child's responsiveness:
 - Gently stimulate the child and ask loudly: are you all right?
- 3A. If the child responds by answering or moving:
 - Leave the child in the position in which you find him (provided he is not in further danger).
 - Check his condition and get help if needed.
 - Re-assess him regularly.
- 3B. If the child does not respond:
 - Shout for help.
 - Turn carefully the child on his back.
 - Open the child's airway by tilting the head and lifting the chin.
 - Place your hand on his forehead and gently tilt his head back.
 - At the same time, with your fingertip(s) under the point of the child's chin, lift the chin. Do not push on the soft tissues under the chin as this may obstruct the airway.
 - If you still have difficulty in opening the airway, try a jaw thrust: place the first two fingers of each hand behind each side of the child's mandible and push the jaw forward.

Have a low threshold for suspecting an injury to the neck; if so, try to open the airway by jaw thrust alone. If jaw thrust alone does not enable adequate airway patency, add head tilt a small amount at a time until the airway is open.

4. Keeping the airway open, look, listen and feel for normal breathing by putting your face close to the child's face and looking along the chest:
 - **Look** for chest movements.
 - **Listen** at the child's nose and mouth for breath sounds.
 - **Feel** for air movement on your cheek.

Paediatric basic life support

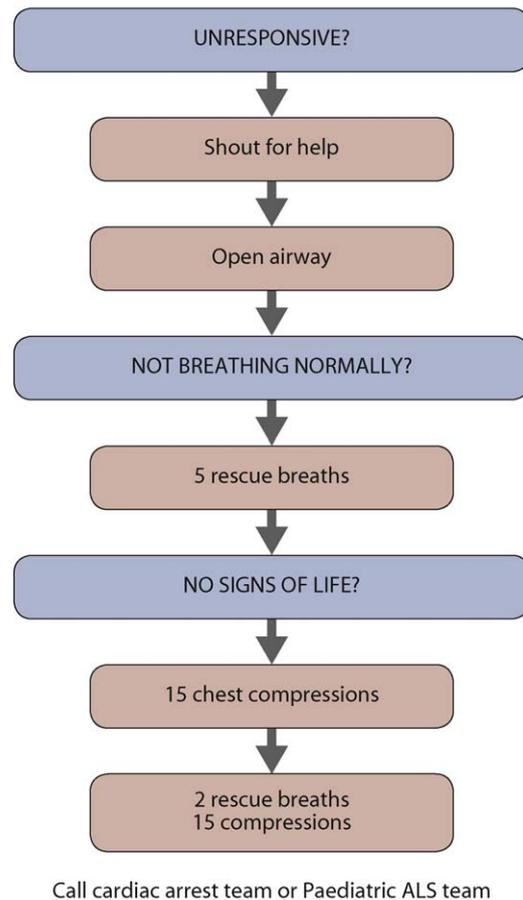


Fig. 6.1. Paediatric basic life support algorithm for those with a duty to respond to paediatric emergencies.

In the first few minutes after a cardiac arrest a child may be taking slow infrequent gasps. Look, listen and feel for no more than 10 s before deciding – if you have any doubt whether breathing is normal, act as if it is not normal:

- 5A. If the child is breathing normally:
 - Turn the child on his side into the recovery position (see below).
 - Send or go for help – call the local emergency number for an ambulance.
 - Check for continued breathing.
- 5B. If breathing is not normal or absent:
 - Carefully remove any obvious airway obstruction.
 - Give five initial rescue breaths.
 - While performing the rescue breaths note any gag or cough response to your action. These responses or their absence will form part of your assessment of 'signs of life', which will be described later.

Rescue breaths for a child over 1 year of age (Fig. 6.2):

- Ensure head tilt and chin lift.
- Pinch the soft part of the nose closed with the index finger and thumb of your hand on his forehead.
- Allow the mouth to open, but maintain chin lift.
- Take a breath and place your lips around the mouth, making sure that you have a good seal.



Fig. 6.2. Mouth-to-mouth ventilation – child.

- Blow steadily into the mouth over about 1–1.5 s watching for chest rise.
- Maintain head tilt and chin lift, take your mouth away from the victim and watch for his chest to fall as air comes out.
- Take another breath and repeat this sequence five times. Identify effectiveness by seeing that the child's chest has risen and fallen in a similar fashion to the movement produced by a normal breath.

Rescue breaths for an infant (Fig. 6.3):

- Ensure a neutral position of the head (as an infant's head is usually flexed when supine, this may require some extension) and a chin lift.
- Take a breath and cover the mouth and nose of the infant with your mouth, making sure you have a good seal. If the nose and mouth cannot be covered in the older infant, the rescuer may attempt to seal only the infant's nose or mouth with his mouth (if the nose is used, close the lips to prevent air escape).
- Blow steadily into the infant's mouth and nose over 1–1.5 s, sufficient to make the chest visibly rise.
- Maintain head position and chin lift, take your mouth away from the victim and watch for his chest to fall as air comes out.
- Take another breath and repeat this sequence five times.



Fig. 6.3. Mouth-to-mouth and nose ventilation – infant.

For both infants and children, if you have difficulty achieving an effective breath, the airway may be obstructed:

- Open the child's mouth and remove any visible obstruction. Do not perform a blind finger sweep.
- Ensure that there is adequate head tilt and chin lift but also that the neck is not over extended.
- If head tilt and chin lift has not opened the airway, try the jaw thrust method.
- Make up to five attempts to achieve effective breaths, if still unsuccessful, move on to chest compressions.

6. Assess the child's circulation.

Take no more than 10 s to:

- Look for signs of life – this includes any movement, coughing or normal breathing (not abnormal gasps or infrequent, irregular breaths).

If you check the pulse, ensure you take no more than 10 s.

In a child over 1 year – feel for the carotid pulse in the neck.

In an infant – feel for the brachial pulse on the inner aspect of the upper arm.

The femoral pulse in the groin, which is half way between the anterior superior iliac spine and the symphysis pubis, can also be used in infant and children.

- If you are confident that you can detect signs of life within 10 s:
 - Continue rescue breathing, if necessary, until the child starts breathing effectively on his own.
 - Turn the child on to his side (into the recovery position) if he remains unconscious.
 - Re-assess the child frequently.
- If there are no signs of life, unless you are CERTAIN you can feel a definite pulse of greater than 60 beats min^{-1} within 10 s:
 - Start chest compressions.
 - Combine rescue breathing and chest compressions:

Chest compressions:

For all children, compress the lower half of the sternum: To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Compress the sternum one finger's breadth above this; the compression should be sufficient to depress the sternum by at least one third of the depth of the chest. Don't be afraid to push too hard: "Push Hard and Fast". Release the pressure completely and repeat at a rate of at least 100 min^{-1} (but not exceeding 120 min^{-1}). After 15 compressions, tilt the head, lift the chin, and give two effective breaths. Continue compressions and breaths in a ratio of 15:2. The best method for compression varies slightly between infants and children.

Chest compression in infants (Fig. 6.4): The lone rescuer compresses the sternum with the tips of two fingers. If there are two or more rescuers, use the encircling technique. Place both thumbs flat side by side on the lower half of the sternum (as above) with the tips pointing towards the infant's head. Spread the rest of both hands with the fingers together to encircle the lower part of the infant's rib cage with the tips of the fingers supporting the infant's back. For both methods, depress the lower sternum by at least one third of the depth of the infant's chest.

Chest compression in children over 1 year of age (Figs. 6.5 and 6.6): Place the heel of one hand over the lower half of the sternum (as above). Lift the fingers to ensure that pressure is not applied over the child's ribs. Position yourself vertically above the victim's chest and, with your arm straight, compress the sternum to depress it by at least one third of the depth of the chest. In larger children or for small rescuers, this is achieved most easily by using both hands with the fingers interlocked.

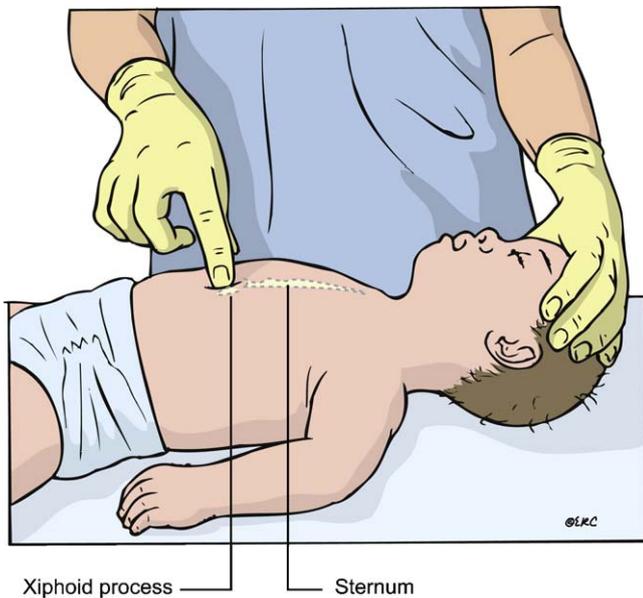


Fig. 6.4. Chest compression – infant.

8. Do not interrupt resuscitation until:

- The child shows signs of life (starts to wake up, to move, opens eyes and to breathe normally or a definite pulse of greater than 60 min^{-1} is palpated).
- Further qualified help arrives and takes over.
- You become exhausted.

When to call for assistance

It is vital for rescuers to get help as quickly as possible when a child collapses.

- When more than one rescuer is available, one starts resuscitation while another rescuer goes for assistance.
- If only one rescuer is present, undertake resuscitation for about 1 min before going for assistance. To minimise interruption in CPR, it may be possible to carry an infant or small child whilst summoning help.



Fig. 6.5. Chest compression with one hand – child.



Fig. 6.6. Chest compression with two hands – child.

- The only exception to performing 1 min of CPR before going for help is in the case of a child with a witnessed, sudden collapse when the rescuer is alone. In this case, cardiac arrest is likely to be caused by an arrhythmia and the child will need defibrillation. Seek help immediately if there is no one to go for you.

Recovery position

An unconscious child whose airway is clear, and who is breathing normally, should be turned on his side into the recovery position.

There are several recovery positions; they all aim to prevent airway obstruction and reduce the likelihood of fluids such as saliva, secretions or vomit from entering into the upper airway.

There are important principles to be followed.

- Place the child in as near true lateral position as possible, with his mouth dependent, which should enable the free drainage of fluid.
- The position should be stable. In an infant, this may require a small pillow or a rolled-up blanket to be placed along his back to maintain the position, so preventing the infant from rolling into either the supine or prone position.
- Avoid any pressure on the child's chest that may impair breathing.
- It should be possible to turn the child onto his side and back again to the recovery position easily and safely, taking into consideration the possibility of cervical spine injury by in-line cervical stabilisation techniques.

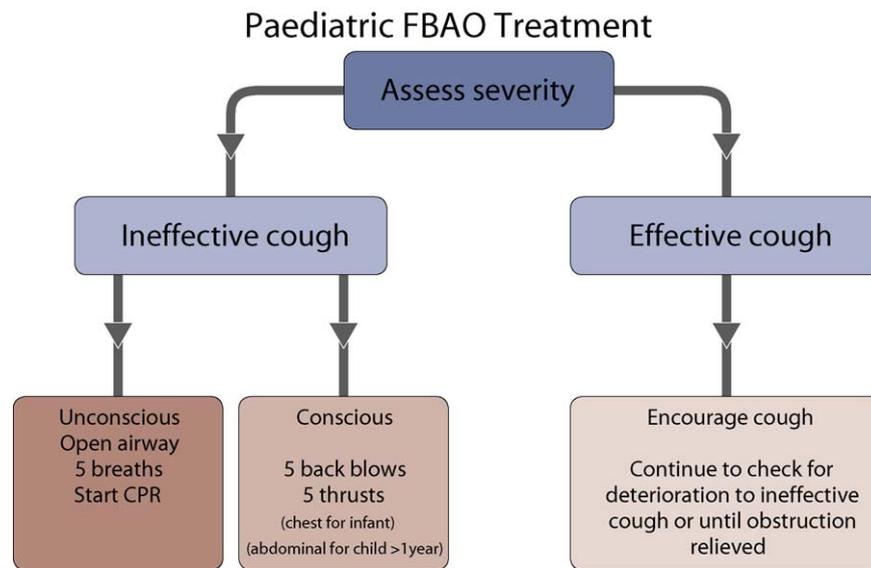


Fig. 6.7. Paediatric foreign body airway obstruction algorithm.

- Regularly change side to avoid pressure points (i.e., every 30 min).
- The adult recovery position is suitable for use in children.

Foreign body airway obstruction

No new evidence on this subject was presented during the 2010 Consensus Conference. Back blows, chest thrusts and abdominal thrusts all increase intrathoracic pressure and can expel foreign bodies from the airway. In half of the episodes more than one technique is needed to relieve the obstruction.⁵⁵ There are no data to indicate which measure should be used first or in which order they should be applied. If one is unsuccessful, try the others in rotation until the object is cleared.

The foreign body airway obstruction (FBAO) algorithm for children was simplified and aligned with the adult version in 2005 guidelines; this continues to be the recommended sequence for managing FBAO (Fig. 6.7).

The most significant difference from the adult algorithm is that abdominal thrusts should not be used for infants. Although abdominal thrusts have caused injuries in all age groups, the risk is particularly high in infants and very young children. This is because of the horizontal position of the ribs, which leaves the upper abdominal viscera much more exposed to trauma. For this reason, the guidelines for the treatment of FBAO are different between infants and children.

Recognition of foreign body airway obstruction

When a foreign body enters the airway the child reacts immediately by coughing in an attempt to expel it. A spontaneous cough is likely to be more effective and safer than any manoeuvre a rescuer might perform. However, if coughing is absent or ineffective and the object completely obstructs the airway, the child will rapidly become asphyxiated. Active interventions to relieve FBAO are therefore required only when coughing becomes ineffective, but they then need to be commenced rapidly and confidently. The majority of choking events in infants and children occur during play or eating episodes, when a carer is usually present; thus, the events are frequently witnessed and interventions are usually initiated when the child is conscious.

Foreign body airway obstruction is characterised by the sudden onset of respiratory distress associated with coughing, gagging

or stridor (Table 6.1). Similar signs and symptoms may be associated with other causes of airway obstruction such as laryngitis or epiglottitis; these conditions are managed differently to that of FBAO. Suspect FBAO if the onset was very sudden and there are no other signs of illness; there may be clues to alert the rescuer, e.g., a history of eating or playing with small items immediately before the onset of symptoms.

Relief of FBAO (Fig. 6.7)

1. Safety and summoning assistance

Safety is paramount: rescuers must not place themselves in danger and should consider the safest treatment of the choking child.

If the child is coughing effectively, no external manoeuvre is necessary. Encourage the child to cough, and monitor continually.

If the child's coughing is (or is becoming) ineffective, shout for help immediately and determine the child's conscious level.

2. Conscious child with FBAO

If the child is still conscious but has absent or ineffective coughing, give back blows.

If back blows do not relieve the FBAO, give chest thrusts to infants or abdominal thrusts to children. These manoeuvres create an artificial cough, increasing intrathoracic pressure and dislodging the foreign body.

Table 6.1 Sign of foreign body airway obstruction.

General signs of FBAO	
Witnessed episode	
Coughing/choking	
Sudden onset	
Recent history of playing with/eating small objects	
Ineffective coughing	Effective cough
Unable to vocalise	Crying or verbal response to questions
Quiet or silent cough	Loud cough
Unable to breathe	Able to take a breath before coughing
Cyanosis	Fully responsive
Decreasing level of consciousness	

Back blows in infants.

- Support the infant in a head downward, prone position, to enable gravity to assist removal of the foreign body.
- A seated or kneeling rescuer should be able to support the infant safely across their lap.
- Support the infant's head by placing the thumb of one hand, at the angle of the lower jaw, and one or two fingers from the same hand, at the same point on the other side of the jaw.
- Do not compress the soft tissues under the infant's jaw, as this will exacerbate the airway obstruction.
- Deliver up to five sharp back blows with the heel of one hand in the middle of the back between the shoulder blades.
- The aim is to relieve the obstruction with each blow rather than to give all five.

Back blows in children over 1 year.

- Back blows are more effective if the child is positioned head down.
- A small child may be placed across the rescuer's lap as with the infant.
- If this is not possible, support the child in a forward leaning position and deliver the back blows from behind.

If back blows fail to dislodge the object, and the child is still conscious, use chest thrusts for infants or abdominal thrusts for children. Do not use abdominal thrusts (Heimlich manoeuvre) in infants.

Chest thrusts for infants.

- Turn the infant into a head downward supine position. This is achieved safely by placing the free arm along the infant's back and encircling the occiput with the hand.
- Support the infant down your arm, which is placed down (or across) your thigh.
- Identify the landmark for chest compressions (on the lower half of the sternum, approximately a finger's breadth above the xiphisternum).
- Give five chest thrusts; these are similar to chest compressions but sharper and delivered at a slower rate.

Abdominal thrusts for children over 1 year.

- Stand or kneel behind the child; place your arms under the child's arms and encircle his torso.
- Clench your fist and place it between the umbilicus and xiphisternum.
- Grasp this hand with the other hand and pull sharply inwards and upwards.
- Repeat up to five times.
- Ensure that pressure is not applied to the xiphoid process or the lower rib cage – this may cause abdominal trauma.

Following the chest or abdominal thrusts, re-assess the child. If the object has not been expelled and the victim is still conscious, continue the sequence of back blows and chest (for infant) or abdominal (for children) thrusts. Call out, or send, for help if it is still not available. Do not leave the child at this stage.

If the object is expelled successfully, assess the child's clinical condition. It is possible that part of the object may remain in the respiratory tract and cause complications. If there is any doubt, seek medical assistance. Abdominal thrusts may cause internal injuries and all victims treated with abdominal thrusts should be examined by a doctor.⁵

3. Unconscious child with FBAO

If the child with FBAO is, or becomes, unconscious, place him on a firm, flat surface. Call out, or send, for help if it is still not available. Do not leave the child at this stage; proceed as follows:

Airway opening. Open the mouth and look for any obvious object. If one is seen, make an attempt to remove it with a single finger sweep. Do not attempt blind or repeated finger sweeps – these can impact the object more deeply into the pharynx and cause injury.

Rescue breaths. Open the airway using a head tilt/chin lift and attempt five rescue breaths. Assess the effectiveness of each breath: if a breath does not make the chest rise, reposition the head before making the next attempt.

Chest compressions and CPR.

- Attempt five rescue breaths and if there is no response (moving, coughing, spontaneous breaths) proceed to chest compressions without further assessment of the circulation.
- Follow the sequence for single rescuer CPR (step 7B above) for approximately a minute before summoning the EMS (if this has not already been done by someone else).
- When the airway is opened for attempted delivery of rescue breaths, look to see if the foreign body can be seen in the mouth.
- If an object is seen, attempt to remove it with a single finger sweep.
- If it appears the obstruction has been relieved, open and check the airway as above; deliver rescue breaths if the child is not breathing.
- If the child regains consciousness and exhibits spontaneous effective breathing, place him in a safe position on his side (recovery position) and monitor breathing and conscious level whilst awaiting the arrival of the EMS.

B. Paediatric advanced life support

Prevention of cardiopulmonary arrest

In children, secondary cardiopulmonary arrests, caused by either respiratory or circulatory failure, are more frequent than primary arrests caused by arrhythmias.^{56–61} So-called asphyxial arrests or respiratory arrests are also more common in young adulthood (e.g., trauma, drowning, poisoning).^{62,63} The outcome from cardiopulmonary arrests in children is poor; identification of the antecedent stages of cardiac or respiratory failure is a priority, as effective early intervention may be life saving.

The order of assessment and intervention for any seriously ill or injured child follows the ABC principles.

- A indicates airway (Ac for airway and cervical spine stabilisation for the injured child).
- B indicates breathing.
- C indicates circulation (with haemorrhage control in injured child).

Interventions are made at each step of the assessment as abnormalities are identified. The next step of the assessment is not started until the preceding abnormality has been managed and corrected if possible. Summoning a paediatric rapid response team or medical emergency team may reduce the risk of respiratory and/or cardiac arrest in hospitalised children outside the intensive care setting.^{64–69} This team should include at least one paediatrician with specific knowledge in the field and one specialised nurse, and should be called to evaluate a potentially critically ill child who is

not already in a paediatric intensive care unit (PICU) or paediatric emergency department (ED).

Diagnosing respiratory failure: assessment of A and B

Assessment of a potentially critically ill child starts with assessment of airway (A) and breathing (B). Abnormalities in airway patency or gas exchange in the lungs can lead to respiratory failure.

Signs of respiratory failure include:

- *Respiratory rate* outside the normal range for the child's age – either too fast or too slow.
- Initially increasing *work of breathing*, which may progress to inadequate/decreased work of breathing as the patient tires or compensatory mechanisms fail, additional noises such as stridor, wheeze, grunting, or the loss of breath sounds.
- Decreased *tidal volume* marked by shallow breathing, decreased chest expansion or decreased air entry at auscultation.
- *Hypoxaemia* (without/with supplemental oxygen) generally identified by cyanosis but best evaluated by pulse oximetry.

There may be associated signs in other organ systems that are either affected by inadequate ventilation and oxygenation or act to compensate the respiratory problem. These are detectable in step C of the assessment and include:

- Increasing tachycardia (compensatory mechanism in an attempt to increase oxygen delivery).
- Pallor.
- Bradycardia (ominous indicator of the loss of compensatory mechanisms).
- Alteration in the level of consciousness (a sign that compensatory mechanisms are overwhelmed).

Diagnosing circulatory failure: assessment of C

Circulatory failure (or shock) is characterised by a mismatch between metabolic demand by the tissues and delivery of oxygen and nutrients by the circulation.⁷⁰ Physiological compensatory mechanisms lead to changes in the heart rate, in the systemic vascular resistance (which commonly increases as an adaptive response) and in tissue and organ perfusion. Signs of circulatory failure include:

- Increased *heart rate* (bradycardia is an ominous sign of physiological decompensation).
- Decreased systemic *blood pressure*.
- Decreased *peripheral perfusion* (prolonged capillary refill time, decreased skin temperature, pale or mottled skin).
- Weak or absent peripheral pulses.
- Decreased or increased *intravascular volume*.
- Decreased urine output and metabolic acidosis.

Other systems may be affected, for example:

- Respiratory frequency may be increased initially, in an attempt to improve oxygen delivery, later becoming slow and accompanied by decompensated circulatory failure.
- Level of consciousness may decrease because of poor cerebral perfusion.

Diagnosing cardiopulmonary arrest

Signs of cardiopulmonary arrest include:

- Unresponsiveness to pain (coma).
- Apnoea or gasping respiratory pattern.
- Absent circulation.
- Pallor or deep cyanosis.

Palpation of a pulse is not reliable as the sole determinant of the need for chest compressions.^{71,72} If cardiac arrest is suspected, and in the absence of signs of life, rescuers (lay and professional) should begin CPR unless they are certain they can feel a central pulse within 10 s (infants – brachial or femoral artery; children – carotid or femoral artery). If there is any doubt, start CPR.^{72–75} If personnel skilled in echocardiography are available, this investigation may help to detect cardiac activity and potentially treatable causes for the arrest.⁷⁶ However, echocardiography must not interfere with the performance of chest compressions.

Management of respiratory and circulatory failure

In children, there are many causes of respiratory and circulatory failure and they may develop gradually or suddenly. Both may be initially compensated but will normally decompensate without adequate treatment. Untreated decompensated respiratory or circulatory failure will lead to cardiopulmonary arrest. Hence, the aim of paediatric life support is early and effective intervention in children with respiratory and circulatory failure to prevent progression to full arrest.

Airway and breathing

- Open the airway and ensure adequate ventilation and oxygenation. Deliver high-flow oxygen.
- Establish respiratory monitoring (first line – pulse oximetry/SpO₂).
- Achieving adequate ventilation and oxygenation may require use of airway adjuncts, bag-mask ventilation (BMV), use of a laryngeal mask airway (LMA), securing a definitive airway by tracheal intubation and positive pressure ventilation.
- Very rarely, a surgical airway may be required.

Circulation

- Establish cardiac monitoring (first line – pulse oximetry/SpO₂, electrocardiography/ECG and non-invasive blood pressure/NIBP).
- Secure intravascular access. This may be by peripheral intravenous (IV) or by intraosseous (IO) cannulation. If already in situ, a central intravenous catheter should be used.
- Give a fluid bolus (20 ml kg⁻¹) and/or drugs (e.g., inotropes, vasopressors, anti-arrhythmics) as required.
- Isotonic crystalloids are recommended as initial resuscitation fluid in infants and children with any type of shock, including septic shock.^{77–80}
- Assess and re-assess the child continuously, commencing each time with the airway before proceeding to breathing and then the circulation.
- During treatment, capnography, invasive monitoring of arterial blood pressure, blood gas analysis, cardiac output monitoring, echocardiography and central venous oxygen saturation (ScvO₂) may be useful to guide the treatment of respiratory and/or circulatory failure.

Airway

Open the airway using basic life support techniques. Oropharyngeal and nasopharyngeal airways adjuncts can help maintain the airway. Use the oropharyngeal airway only in the unconscious child, in whom there is no gag reflex. Use the appropriate size (from the incisors to the angle of the mandible), to avoid pushing the tongue backward and obstructing the epiglottis, or directly compressing the glottis. The soft palate in the child can be damaged by insertion of the oropharyngeal airway – avoid this by inserting the oropharyngeal airway with care; do not use any force. The nasopharyngeal airway is usually tolerated better in the conscious or semi-conscious child (who has an effective gag reflex), but should not be used if there is a basal skull fracture or a coagulopathy. The correct insertion depth should be sized from the nostrils to the angle of the mandible but must be re-assessed after insertion. These simple airway adjuncts do not protect the airway from aspiration of secretions, blood or stomach contents.

Laryngeal mask airway (LMA)

Although bag-mask ventilation remains the recommended first line method for achieving airway control and ventilation in children, the LMA is an acceptable airway device for providers trained in its use.^{81,82} It is particularly helpful in airway obstruction caused by supraglottic airway abnormalities or if bag-mask ventilation is not possible. The LMA does not totally protect the airway from aspiration of secretions, blood or stomach contents, and therefore close observation is required. Use of the LMA is associated with a higher incidence of complications in small children compared with adults.^{83,84} Other supraglottic airway devices (e.g., laryngeal tube), which have been used successfully in children's anaesthesia, may also be useful in an emergency but there are few data on the use of these devices in paediatric emergencies.⁸⁵

Tracheal intubation

Tracheal intubation is the most secure and effective way to establish and maintain the airway, prevent gastric distension, protect the lungs against pulmonary aspiration, enable optimal control of the airway pressure and provide positive end expiratory pressure (PEEP). The oral route is preferable during resuscitation. Oral intubation is quicker and simpler, and is associated with fewer complications than nasal placement. In the conscious child, the judicious use of anaesthetics, sedatives and neuromuscular blocking drugs is essential in order to avoid multiple intubation attempts or intubation failure.^{86–95} The anatomy of a child's airway differs significantly from that of an adult; hence, intubation of a child requires special training and experience. Clinical examination and capnography must be used to confirm correct tracheal tube placement. The tracheal tube must be secured and vital signs monitored.⁹⁶ It is also essential to plan an alternative airway management technique in case the trachea cannot be intubated.

There is currently no evidence-based recommendation defining the setting-, patient- and operator-related criteria for prehospital tracheal intubation of children. Prehospital tracheal intubation of children may be considered if:

- (1) the airway and/or breathing is seriously compromised or threatened;
- (2) the mode and duration of transport require the airway to be secured early (e.g., air transport); and
- (3) if the operator is adequately skilled in advanced paediatric airway management including the use of drugs to facilitate tracheal intubation.⁹⁷

Table 6.2

General recommendation for cuffed and uncuffed tracheal tube sizes (internal diameter in mm).

	Uncuffed	Cuffed
Neonates <i>premature</i>	Gestational age in weeks/10	Not used
Neonates <i>Full term</i>	3.5	Not usually used
Infants	3.5–4.0	3.0–3.5
Child 1–2 years	4.0–4.5	3.5–4.0
Child >2 years	Age/4 + 4	Age/4 + 3.5

Rapid-sequence induction and intubation

The child who is in cardiopulmonary arrest and/or deep coma does not require sedation or analgesia to be intubated; otherwise, intubation must be preceded by oxygenation (gentle BMV is sometimes required to avoid hypoxia), rapid sedation, analgesia and the use of neuromuscular blocking drugs to minimise intubation complications and failure.⁹⁸ The intubator must be experienced and familiar with drugs used for rapid-sequence induction. The use of cricoid pressure may prevent or limit regurgitation of gastric contents^{99,100} but it may distort the airway and make laryngoscopy and intubation more difficult.¹⁰¹ Cricoid pressure should not be used if either intubation or oxygenation is compromised.

Tracheal tube sizes

A general recommendation for tracheal tube internal diameters (ID) for different ages is shown in Table 6.2.^{102–107} This is a guide only and tubes one size larger and smaller should always be available. Tracheal tube size can also be estimated from the length of the child's body as measured by resuscitation tapes.¹⁰⁸

Cuffed versus uncuffed tracheal tubes

Uncuffed tracheal tubes have been used traditionally in children up to 8 years of age but cuffed tubes may offer advantages in certain circumstances e.g., when lung compliance is poor, airway resistance is high or if there is a large air leak from the glottis.^{102,109,110} The use of cuffed tubes also makes it more likely that the correct tube size will be chosen on the first attempt.^{102,103,111} The correctly sized cuffed tracheal tube is as safe as an uncuffed tube for infants and children (not for neonates) provided attention is paid to its placement, size and cuff inflation pressure.^{109,110,112} As excessive cuff pressure may lead to ischaemic damage to the surrounding laryngeal tissue and stenosis, cuff inflation pressure should be monitored and maintained at less than 25 cm H₂O.¹¹²

Confirmation of correct tracheal tube placement

Displaced, misplaced or obstructed tubes occur frequently in the intubated child and are associated with increased risk of death.^{113,114} No single technique is 100% reliable for distinguishing oesophageal from tracheal intubation.^{115–117}

Assessment of the correct tracheal tube position is made by:

- laryngoscopic observation of the tube passing beyond the vocal cords;
- detection of end-tidal CO₂ (by colorimetry or capnometry/-graphy) if the child has a perfusing rhythm (this may also be seen with effective CPR, but it is not completely reliable);
- observation of symmetrical chest wall movement during positive pressure ventilation;
- observation of mist in the tube during the expiratory phase of ventilation;
- absence of gastric distension;
- equal air entry heard on bilateral auscultation in the axillae and apices of the chest;
- absence of air entry into the stomach on auscultation;

- improvement or stabilisation of SpO₂ in the expected range (delayed sign!);
- improvement of heart rate towards the age-expected value (or remaining within the normal range) (delayed sign!).

If the child is in cardiopulmonary arrest and exhaled CO₂ is not detected despite adequate chest compressions, or if there is any doubt, confirm tracheal tube position by direct laryngoscopy. After correct placement and confirmation, secure the tracheal tube and re-assess its position. Maintain the child's head in the neutral position. Flexion of the head drives the tube further into the trachea whereas extension may pull it out of the airway.¹¹⁸ Confirm the position of the tracheal tube at the mid-trachea by chest X-ray; the tracheal tube tip should be at the level of the 2nd or 3rd thoracic vertebra.

DOPES is a useful acronym for the causes of sudden deterioration in an intubated child:

- Displacement of the tracheal tube.
- Obstruction of the tracheal tube or of the heat and moisture exchanger (HME).
- Pneumothorax.
- Equipment failure (source of gas, bag-mask, ventilator, etc.).
- Stomach (gastric distension may alter diaphragm mechanics).

Breathing

Oxygenation

Give oxygen at the highest concentration (i.e., 100%) during initial resuscitation. Once circulation is restored, give sufficient oxygen to maintain an arterial oxygen saturation (SaO₂) in the range of 94–98%.^{119,120}

Studies in neonates suggest some advantages of using room air during resuscitation (see Section 7).^{11,121–124} In the older child, there is no evidence of benefit for air instead of oxygen, so use 100% oxygen for initial resuscitation and after return of a spontaneous circulation (ROSC) titrate the fraction inspired oxygen (FiO₂) to achieve a SaO₂ in the range of 94–98%. In smoke inhalation (carbon monoxide poisoning) and severe anaemia however a high FiO₂ should be maintained until the problem has been solved because in these circumstances dissolved oxygen plays an important role in oxygen transport.

Ventilation

Healthcare providers commonly provide excessive ventilation during CPR and this may be harmful. Hyperventilation causes increased intrathoracic pressure, decreased cerebral and coronary perfusion, and poorer survival rates in animals and adults.^{125–131} Although normoventilation is the objective during resuscitation, it is difficult to know the precise minute volume that is being delivered. A simple guide to deliver an acceptable tidal volume is to achieve modest chest wall rise. Use a ratio of 15 chest compressions to 2 ventilations and a compression rate of 100–120 min⁻¹.¹²⁵ Once ROSC has been achieved, provide normal ventilation (rate/volume) based on the victim's age and, as soon as possible, by monitoring end-tidal CO₂ and blood gas values.

Once the airway is protected by tracheal intubation, continue positive pressure ventilation at 10–12 breaths min⁻¹ without interrupting chest compressions. Take care to ensure that lung inflation is adequate during chest compressions. When circulation is restored, or if the child still has a perfusing rhythm, ventilate at 12–20 breaths min⁻¹ to achieve a normal arterial carbon dioxide tension (PaCO₂). Hyperventilation and hypoventilation are harmful.

Bag-mask ventilation (BMV)

Bag-mask ventilation (BMV) is effective and safe for a child requiring assisted ventilation for a short period, i.e., in the prehospital setting or in an emergency department.^{114,132–135} Assess the effectiveness of BMV by observing adequate chest rise, monitoring heart rate and auscultating for breath sounds, and measuring peripheral oxygen saturation (SpO₂). Any healthcare provider with a responsibility for treating children must be able to deliver BMV effectively.

Prolonged ventilation

If prolonged ventilation is required, the benefits of a secured airway probably outweigh the potential risks associated with tracheal intubation. For emergency intubation, both cuffed and uncuffed tracheal tubes are acceptable.

Monitoring of breathing and ventilation

End-tidal CO₂

Monitoring end-tidal CO₂ (ETCO₂) with a colorimetric detector or capnometer confirms tracheal tube placement in the child weighing more than 2 kg, and may be used in pre- and in-hospital settings, as well as during any transportation of the child.^{136–139} A colour change or the presence of a capnographic waveform for more than four ventilated breaths indicates that the tube is in the tracheobronchial tree both in the presence of a perfusing rhythm and during cardiopulmonary arrest. Capnography does not rule out intubation of a bronchus. The absence of exhaled CO₂ during cardiopulmonary arrest does not guarantee tube misplacement since a low or absent ETCO₂ may reflect low or absent pulmonary blood flow.^{140–143}

Capnography may also provide information on the efficiency of chest compressions and can give an early indication of ROSC.^{144,145} Efforts should be made to improve chest compression quality if the ETCO₂ remains below 15 mmHg (2 kPa). Care must be taken when interpreting ETCO₂ values especially after the administration of adrenaline or other vasoconstrictor drugs when there may be a transient decrease in values,^{146–150} or after the use of sodium bicarbonate when there may be a transient increase.¹⁵¹ Current evidence does not support the use of a threshold ETCO₂ value as an indicator for the discontinuation of resuscitation efforts.

Oesophageal detector devices

The self-inflating bulb or aspirating syringe (oesophageal detector device, ODD) may be used for the secondary confirmation of tracheal tube placement in children with a perfusing rhythm.^{152,153} There are no studies on the use of the ODD in children who are in cardiopulmonary arrest.

Pulse oximetry

Clinical evaluation of the oxygen saturation of arterial blood (SaO₂) is unreliable; therefore, monitor the child's peripheral oxygen saturation continuously by pulse oximetry (SpO₂). Pulse oximetry can be unreliable under certain conditions, for example, if the child is in circulatory failure, in cardiopulmonary arrest or has poor peripheral perfusion. Although pulse oximetry is relatively simple, it is a poor guide to tracheal tube displacement. Capnography detects tracheal tube dislodgement more rapidly than pulse oximetry.¹⁵⁴

Circulation

Vascular access

Vascular access is essential to enable drugs and fluids to be given, and blood samples obtained. Venous access can be dif-

difficult to establish during resuscitation of an infant or child. In critically ill children, whenever venous access is not readily attainable intraosseous access should be considered early, especially if the child is in cardiac arrest or decompensated circulatory failure.^{155–157} In any case, in critically ill children, if attempts at establishing intravenous (IV) access are unsuccessful after 1 min, insert an intraosseous (IO) needle instead.^{155,158}

Intraosseous access

Intraosseous access is a rapid, safe, and effective route to give drugs, fluids and blood products.^{159–168} The onset of action and time to achieve adequate plasma drug concentrations are similar to that achieved via the central venous route.^{169,170} Bone marrow samples can be used to cross match for blood type or group¹⁷¹ for chemical analysis^{172,173} and for blood gas measurement (the values are comparable to central venous blood gases if no drug has been injected in the cavity).^{172,174–176} However samples can damage autoanalysers and should be used preferably in cartridge analyser. Flush each drug with a bolus of normal saline to ensure dispersal beyond the marrow cavity, and to achieve faster distribution to the central circulation. Inject large boluses of fluid using manual pressure. Intraosseous access can be maintained until definitive IV access has been established. The benefits of semi-automated IO devices remain to be seen but preliminary experiences show them to be rapid and effective for obtaining circulatory access.^{167,168,177,178}

Intravenous access

Peripheral IV access provides plasma concentrations of drugs and clinical responses equivalent to central or IO access.^{156,157,179–181} Central venous lines provide more secure long-term access but, compared with IO or peripheral IV access, offer no advantages during resuscitation.^{156,179–181}

Tracheal tube access

Intraosseous or IV access should be definitely preferred to the tracheal route for giving drugs.¹⁸² Drugs given via the trachea have highly variable absorption but, for guidance, the following dosages have been recommended:

Adrenaline	100 $\mu\text{g kg}^{-1}$
Lidocaine	2–3 mg kg^{-1}
Atropine	30 $\mu\text{g kg}^{-1}$

The optimal dose of naloxone is not known.

Dilute the drug in 5 ml of normal saline and follow administration with five ventilations.^{183–185} Do not give non-lipid soluble medications (e.g., glucose, bicarbonate, calcium) via the tracheal tube because they will damage the airway mucosa.

Fluids and drugs

Volume expansion is indicated when a child shows signs of circulatory failure in the absence of volume overload.¹⁸⁶ Isotonic crystalloids are recommended as the initial resuscitation fluid for infants and children with any type of circulatory failure.

If systemic perfusion is inadequate, give a bolus of 20 ml kg^{-1} of an isotonic crystalloid even if the systemic blood pressure is normal. Following every bolus, re-assess the child's clinical state, using ABC, to decide whether a further bolus or other treatment is required.

There are insufficient data to make recommendations about the use of hypertonic saline for circulatory failure associated with head injuries or hypovolaemia.^{187,188}

There are also insufficient data to recommend delayed fluid resuscitation in the hypotensive child with blunt trauma.¹⁸⁹ Avoid dextrose containing solutions unless there

is hypoglycaemia.^{190–193} Monitor glucose levels and avoid hypoglycaemia; infants and small children are particularly prone to hypoglycaemia.

Adenosine

Adenosine is an endogenous nucleotide that causes a brief atrioventricular (AV) block and impairs accessory bundle re-entry at the level of the AV node. Adenosine is recommended for the treatment of supraventricular tachycardia (SVT).¹⁹⁴ It is safe because it has a short half-life (10 s); give it intravenously via upper limb or central veins to minimise the time taken to reach the heart. Give adenosine rapidly, followed by a flush of 3–5 ml of normal saline.¹⁹⁵ Adenosine must be used with caution in asthmatics, second or third degree AV block, long QT syndromes and in cardiac transplant recipients.

Adrenaline (epinephrine)

Adrenaline is an endogenous catecholamine with potent α , β_1 and β_2 adrenergic actions. It is placed prominently in the cardiac arrest treatment algorithms for non-shockable and shockable rhythms. Adrenaline induces vasoconstriction, increases diastolic pressure and thereby improves coronary artery perfusion pressure, enhances myocardial contractility, stimulates spontaneous contractions, and increases the amplitude and frequency of VF, so increasing the likelihood of successful defibrillation.

The recommended IV/IO dose of adrenaline in children for the first and for subsequent doses is 10 $\mu\text{g kg}^{-1}$. The maximum single dose is 1 mg. If needed, give further doses of adrenaline every 3–5 min. Intratracheal adrenaline is no longer recommended,^{196–199} but if this route is ever used, the dose is ten times this (100 $\mu\text{g kg}^{-1}$).

The use of higher doses of adrenaline via the IV or IO route is not recommended routinely as it does not improve survival or neurological outcome after cardiopulmonary arrest.^{200–203}

Once spontaneous circulation is restored, a continuous infusion of adrenaline may be required. Its haemodynamic effects are dose related; there is also considerable variability in response between children; therefore, titrate the infusion dose to the desired effect. High infusion rates may cause excessive vasoconstriction, compromising extremity, mesenteric, and renal blood flow. High-dose adrenaline can cause severe hypertension and tachyarrhythmias.²⁰⁴

To avoid tissue damage it is essential to give adrenaline through a secure intravascular line (IV or IO). Adrenaline (and other catecholamines) is inactivated by alkaline solutions and should never be mixed with sodium bicarbonate.²⁰⁵

Amiodarone

Amiodarone is a non-competitive inhibitor of adrenergic receptors: it depresses conduction in myocardial tissue and therefore slows AV conduction, and prolongs the QT interval and the refractory period. Except when given for the treatment of refractory VF/pulseless VT, amiodarone must be injected slowly (over 10–20 min) with systemic blood pressure and ECG monitoring to avoid causing hypotension. This side effect is less common with the aqueous solution.²⁰⁶ Other rare but significant adverse effects are bradycardia and polymorphic VT.²⁰⁷

Atropine

Atropine accelerates sinus and atrial pacemakers by blocking the parasympathetic response. It may also increase AV conduction. Small doses (< 100 μg) may cause paradoxical bradycardia.²⁰⁸ In

bradycardia with poor perfusion that is unresponsive to ventilation and oxygenation, the first line drug is adrenaline, not atropine.

Atropine is recommended for bradycardia caused by increased vagal tone or cholinergic drug toxicity.^{209–212}

Calcium

Calcium is essential for myocardial function^{213,214} but routine use of calcium does not improve the outcome from cardiopulmonary arrest.^{215–217}

Calcium is indicated in the presence of hypocalcaemia, calcium channel blocker overdose, hypermagnesaemia and hyperkalaemia.^{218–220}

Glucose

Data from neonates, children and adults indicate that both hyper- and hypo-glycaemia are associated with poor outcome after cardiopulmonary arrest,^{221–223} but it is uncertain if this is causative or merely an association.²²⁴ Check blood or plasma glucose concentration and monitor closely in any ill or injured child, including after cardiac arrest. Do not give glucose-containing fluids during CPR unless hypoglycaemia is present. Avoid hyper- and hypo-glycaemia following ROSC. Strict glucose control has not shown survival benefits in adults when compared with moderate glucose control^{225,226} and it increases the risk of hypoglycaemia in neonates, children and adults.^{227–231}

Magnesium

There is no evidence for giving magnesium routinely during cardiopulmonary arrest.²³² Magnesium treatment is indicated in the child with documented hypomagnesaemia or with torsades de pointes VT regardless of the cause.²³³

Sodium bicarbonate

Do not give sodium bicarbonate routinely during cardiopulmonary arrest or after ROSC.^{220,234,235} After effective ventilation and chest compressions have been achieved and adrenaline given, sodium bicarbonate may be considered for the child with prolonged cardiopulmonary arrest and/or severe metabolic acidosis. Sodium bicarbonate may also be considered in case of haemodynamic instability and co-existing hyperkalaemia, or in the management of tricyclic antidepressant drug overdose. Excessive quantities of sodium bicarbonate may impair tissue oxygen delivery, produce hypokalaemia, hypernatraemia, hyperosmolality, and inactivate catecholamines.

Lidocaine

Lidocaine is less effective than amiodarone for defibrillation-resistant VF/pulseless VT in adults²³⁶ and therefore is not the first line treatment in defibrillation-resistant VF/pulseless VT in children.

Procainamide

Procainamide slows intra-atrial conduction and prolongs the QRS and QT intervals. It can be used in SVT^{237–239} or VT²⁴⁰ resistant to other medications in the haemodynamically stable child. However, paediatric data are sparse and procainamide should be used cautiously.^{241,242} Procainamide is a potent vasodilator and can cause hypotension: infuse it slowly with careful monitoring.^{243–245}

Vasopressin – terlipressin

Vasopressin is an endogenous hormone that acts at specific receptors, mediating systemic vasoconstriction (via V₁ receptor) and the reabsorption of water in the renal tubule (by the V₂ receptor).²⁴⁶ There is currently insufficient evidence to support or refute the use of vasopressin or terlipressin as an alternative to, or in combination with, adrenaline in any cardiac arrest rhythm in adults or children.^{247–258}

Some studies have reported that terlipressin (a long-acting analogue of vasopressin with comparable effects) improves haemodynamics in children with refractory, vasodilatory septic shock, but its impact on survival is less clear.^{255–257,259,260} Two paediatric series suggested that terlipressin could be effective in refractory cardiac arrest.^{258,261}

These drugs could be used in cardiac arrest refractory to several adrenaline doses.

Defibrillators

Defibrillators are either automatically or manually operated, and may be capable of delivering either monophasic or biphasic shocks. Manual defibrillators capable of delivering the full energy requirements from neonates upwards must be available within hospitals and in other healthcare facilities caring for children at risk of cardiopulmonary arrest. Automated external defibrillators (AEDs) are preset for all variables including the energy dose.

Pad/paddle size for defibrillation

Select the largest possible available paddles to provide good contact with the chest wall. The ideal size is unknown but there should be good separation between the pads.^{13,262,263}

Recommended sizes are:

- 4.5 cm diameter for infants and children weighing <10 kg.
- 8–12 cm diameter for children >10 kg (older than 1 year).

To decrease skin and thoracic impedance, an electrically conducting interface is required between the skin and the paddles. Preformed gel pads or self-adhesive defibrillation electrodes are effective. Do not use ultrasound gel, saline-soaked gauze, alcohol-soaked gauze/pads or ultrasound gel.

Position of the paddles

Apply the paddles firmly to the bare chest in the antero-lateral position, one paddle placed below the right clavicle and the other in the left axilla (Fig. 6.8). If the paddles are too large and there is a danger of charge arcing across the paddles, one should be placed on the upper back, below the left scapula and the other on the front, to the left of the sternum. This is known as the antero-posterior position and is also acceptable.

Optimal paddle force

To decrease transthoracic impedance during defibrillation, apply a force of 3 kg for children weighing < 10 kg and 5 kg for larger children.^{264,265} In practice, this means that the paddles should be applied firmly.

Energy dose in children

The ideal energy dose for safe and effective defibrillation is unknown. Biphasic shocks are at least as effective and produce less post-shock myocardial dysfunction than monophasic

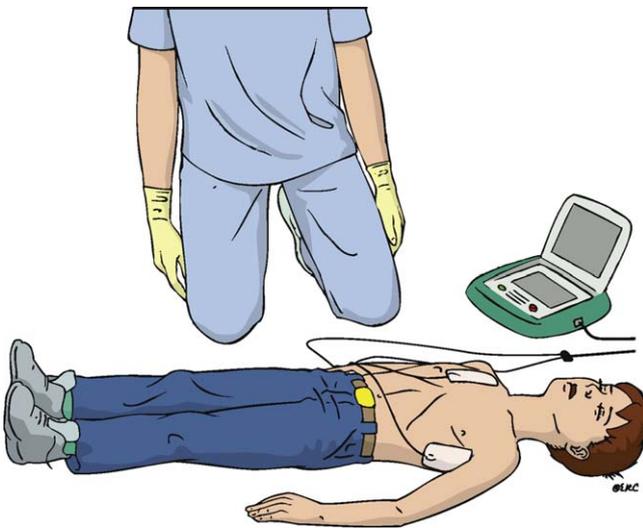


Fig. 6.8. Paddle positions for defibrillation – child.

Shockable – VF/pulseless VT

Attempt defibrillation immediately (4 J kg^{-1}):

- 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. Minimise the delay between stopping chest compressions and delivery of the shock – even 5–10 s delay will reduce the chances of the shock being successful.^{268,269}
- Give one shock.
- Resume CPR as soon as possible without re-assessing the rhythm.
- After 2 min, check briefly the cardiac rhythm on the monitor.
- Give second shock (4 J kg^{-1}) if still in VF/pulseless VT.
- Give CPR for 2 min as soon as possible without re-assessing the rhythm.
- Pause briefly to assess the rhythm; if still in VF/pulseless VT give a third shock at 4 J kg^{-1} .
- Give adrenaline $10\text{ }\mu\text{g kg}^{-1}$ and amiodarone 5 mg kg^{-1} after the third shock once CPR has been resumed.
- Give adrenaline every alternate cycle (i.e., every 3–5 min during CPR).
- Give a second dose of amiodarone 5 mg/kg ²⁷⁰ if still in VF/pulseless VT after the fifth shock.

If the child remains in VF/pulseless VT, continue to alternate shocks of 4 J kg^{-1} with 2 min of CPR. If signs of life become evident, check the monitor for an organised rhythm; if this is present, check for signs of life and a central pulse and evaluate the haemodynamics of the child (blood pressure, peripheral pulse, capillary refill time).

Identify and treat any reversible causes (4 Hs and 4 Ts) remembering that the first 2 Hs (hypoxia and hypovolaemia) have the highest prevalence in critically ill or injured children (Fig. 6.11).

If defibrillation was successful but VF/pulseless VT recurs, resume CPR, give amiodarone and defibrillate again at 4 J kg^{-1} . Start a continuous infusion of amiodarone.

Reversible causes of cardiac arrest

The reversible causes of cardiac arrest can be considered quickly by recalling the 4 Hs and 4 Ts:

- Hypoxia.
- Hypovolaemia.
- Hyper/hypokalaemia.
- Hypothermia.
- Tension pneumothorax.
- Toxic/therapeutic disturbances.
- Tamponade (coronary or pulmonary).
- Thrombosis (coronary or pulmonary).

Sequence of events in cardiopulmonary arrest

1. When a child becomes unresponsive, without signs of life (no breathing, cough or any detectable movement), start CPR immediately.
2. Provide BMV with 100% oxygen.
3. Commence monitoring. Send for a manual defibrillator or an AED to identify and treat shockable rhythms as quickly as possible.

In the less common circumstance of a witnessed sudden collapse, early activation of the emergency services and getting an AED may be more appropriate; start CPR as soon as possible.

shocks.^{36,49,51–53,266} Animal models show better results with paediatric doses of $3\text{--}4\text{ J kg}^{-1}$ than with lower doses,⁴⁹ or adult doses.³⁸ Clinical studies in children indicate that doses of 2 J kg^{-1} are insufficient in most cases.^{12,38,42} Doses larger than 4 J kg^{-1} (as much as 9 J kg^{-1}) have defibrillated children effectively with negligible side effects.^{29,48} When using a manual defibrillator, use 4 J kg^{-1} (preferably biphasic but monophasic waveform is also acceptable) for the first and subsequent shocks.

If no manual defibrillator is available, use an AED that can recognise paediatric shockable rhythms.^{31,32,267} The AED should be equipped with a dose attenuator which decreases the delivered energy to a lower dose more suitable for children aged 1–8 years (50–75 J).^{34,37} If such an AED is not available, use a standard AED and the preset adult energy levels. For children above 8 years, use a standard AED with standard paddles. Although the evidence to support a recommendation for the use of AEDs (preferably with dose attenuator) in children less than 1 year is limited to case reports,^{39,40} it is acceptable if no other option is available.

Advanced management of cardiopulmonary arrest (Fig. 6.9)

ABC

Commence and continue with basic life support
Oxygenate and ventilate with BMV

- Provide positive pressure ventilation with a high inspired oxygen concentration
- Give five rescue breaths followed by external chest compression and positive pressure ventilation in the ratio of 15:2
- Avoid rescuer fatigue by frequently changing the rescuer performing chest compressions
- Establish cardiac monitoring

Assess cardiac rhythm and signs of life
(±check for a central pulse for no more than 10 s)

Non-shockable – asystole, pulseless electrical activity (PEA)

- Give adrenaline IV or IO ($10\text{ }\mu\text{g kg}^{-1}$) and repeat every 3–5 min.
- Identify and treat any reversible causes (4 Hs and 4 Ts) (Fig. 6.10).

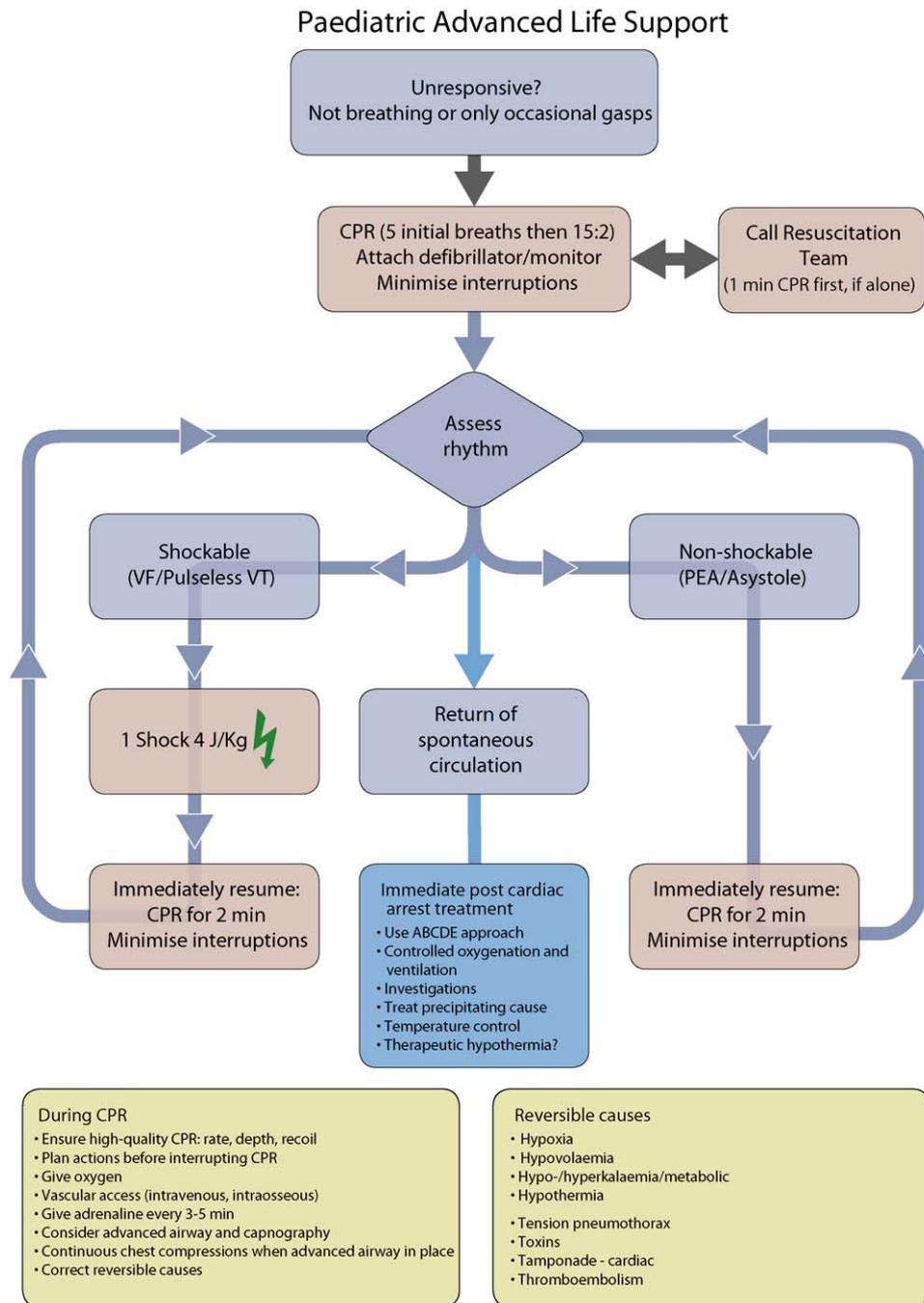


Fig. 6.9. Paediatric advanced life support algorithm.

Cardiac monitoring

Position the cardiac monitor leads or defibrillation paddles as soon as possible to enable differentiation between a shockable and a non-shockable cardiac rhythm. Invasive monitoring of systemic blood pressure may help to improve effectiveness of chest compression²⁷¹ but must not delay the provision of basic or advanced resuscitation.

Shockable rhythms are pulseless VT and VF. These rhythms are more likely after sudden collapse in children with heart disease or adolescents.^{41–43} Non-shockable rhythms are pulseless electrical activity (PEA), bradycardia ($<60 \text{ min}^{-1}$ with no signs of circulation), and asystole. PEA and bradycardia often have wide-QRS complexes.

Echocardiography may be used to identify potentially treatable causes of cardiac arrest in children. Cardiac activity can be rapidly visualised⁷⁶ and pericardial tamponade diagnosed.²⁷² However, appropriately skilled operators must be available and its use should be balanced against the interruption to chest compressions during examination.

Non-shockable rhythms

Most cardiopulmonary arrests in children and adolescents are of respiratory origin.^{54,58,273–275} A period of immediate CPR is therefore mandatory in this age group before searching for an AED or manual defibrillator, as its immediate availability will not improve the outcome of a respiratory arrest.^{17,276}

CARDIAC ARREST: NON SHOCKABLE RHYTHM

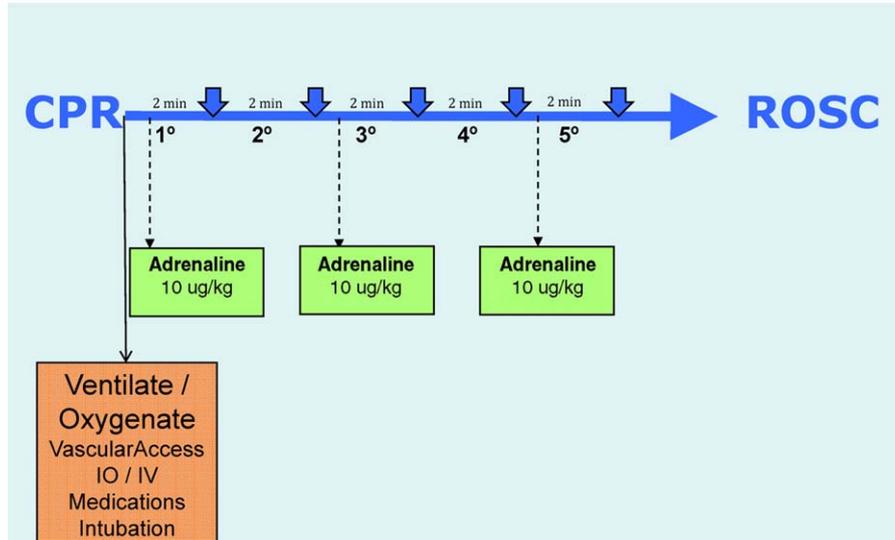


Fig. 6.10. Paediatric algorithm for non-shockable rhythm.

CARDIAC ARREST – SHOCKABLE RHYTHM

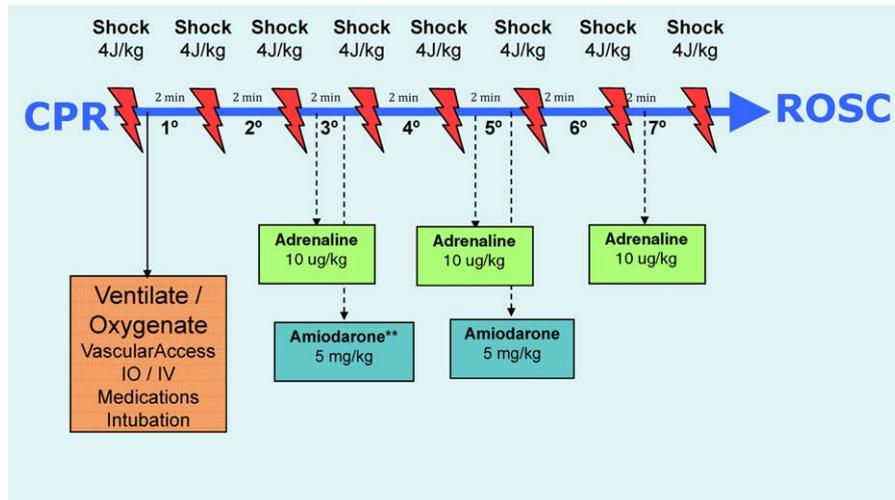


Fig. 6.11. Paediatric algorithm for shockable rhythm.

Bystander CPR is associated with a better neurological outcome in adults and children.^{277–279} The most common ECG patterns in infants, children and adolescents with cardiopulmonary arrest are asystole and PEA. PEA is characterised by organised, wide or narrow complex electrical activity, usually (but not always) at a slow rate, and absent pulses. It commonly follows a period of hypoxia or myocardial ischaemia, but occasionally can have a reversible cause (i.e., one of the 4 Hs and 4 Ts) that led to a sudden impairment of cardiac output.

Shockable rhythms

Primary VF occurs in 3.8–19% of cardiopulmonary arrests in children.^{13,41–43,60,274,275,277} The incidence of VF/pulseless VT increases with age.^{267,280} The primary determinant of survival from VT/pulseless VT cardiopulmonary arrest is the time to defibrillation. Prehospital defibrillation within the first 3 min of witnessed adult VF arrest results in >50% survival. However, the success of defibrillation decreases dramatically the longer the time until defibrillation:

for every minute delay in defibrillation (without any CPR), survival decreases by 7–10%. Survival after more than 12 min of VF in adult victims is <5%.²⁸¹ Cardiopulmonary resuscitation provided before defibrillation for response intervals longer than 5 min improved outcome in some studies,^{282,283} but not in others.²⁸⁴

Secondary VF is present at some point in up to 27% of in-hospital resuscitation events. It has a much poorer prognosis than primary VF.⁴³

Drugs in shockable rhythms

Adrenaline (epinephrine)

Adrenaline is given every 3–5 min by the IV or IO route in preference to the tracheal tube route.

Amiodarone in VF/pulseless VT

Amiodarone is indicated in defibrillation-resistant VF/pulseless VT. Experimental and clinical experience with amiodarone in children is scarce; evidence from adult studies^{236,285,286} demonstrates increased survival to hospital admission, but not to hospital dis-

charge. One paediatric case series demonstrates the effectiveness of amiodarone for life-threatening ventricular arrhythmias.²⁸⁷ Therefore, IV amiodarone has a role in the treatment of defibrillation refractory or recurrent VF/pulseless VT in children.

Extracorporeal life support

Extracorporeal life support should be considered for children with cardiac arrest refractory to conventional CPR, if the arrest occurs in a highly supervised environment and available expertise and equipment to rapidly initiate extracorporeal life support (ECLS).

Arrhythmias

Unstable arrhythmias

Check for signs of life and the central pulse of any child with an arrhythmia; if signs of life are absent, treat as for cardiopulmonary arrest. If the child has signs of life and a central pulse, evaluate the haemodynamic status. Whenever the haemodynamic status is compromised, the first steps are:

1. Open the airway.
2. Give oxygen and assist ventilation as necessary.
3. Attach ECG monitor or defibrillator and assess the cardiac rhythm.
4. Evaluate if the rhythm is slow or fast for the child's age.
5. Evaluate if the rhythm is regular or irregular.
6. Measure QRS complex (narrow complexes: <0.08 s duration; wide complexes: >0.08 s).
7. The treatment options are dependent on the child's haemodynamic stability.

Bradycardia

Bradycardia is caused commonly by hypoxia, acidosis and/or severe hypotension; it may progress to cardiopulmonary arrest. Give 100% oxygen, and positive pressure ventilation if required, to any child presenting with bradyarrhythmia and circulatory failure.

If a poorly perfused child has a heart rate <60 beats min⁻¹, and they do not respond rapidly to ventilation with oxygen, start chest compressions and give adrenaline. If the bradycardia is caused by vagal stimulation (such as after passing a nasogastric tube), atropine may be effective.

Cardiac pacing (either transvenous or external) is generally not useful during resuscitation. It may be considered in cases of AV block or sinus node dysfunction unresponsive to oxygenation, ventilation, chest compressions and other medications; pacing is not effective in asystole or arrhythmias caused by hypoxia or ischaemia.²⁸⁸

Tachycardia

Narrow complex tachycardia

If SVT is the likely rhythm, vagal manoeuvres (Valsalva or diving reflex) may be used in haemodynamically stable children. They can also be used in haemodynamically unstable children, but only if they do not delay chemical or electrical cardioversion.²⁸⁹ If the child is unstable with a depressed conscious level, attempt synchronised electrical cardioversion immediately.

Adenosine is usually effective in converting SVT into sinus rhythm. It is given by rapid, intravenous injection as close as practicable to the heart (see above), and followed immediately by a bolus of normal saline. If the child is too haemodynamically unsta-

ble, omit vagal manoeuvres and adenosine and attempt electrical cardioversion immediately.

Electrical cardioversion (synchronised with R wave) is also indicated when vascular access is not available, or when adenosine has failed to convert the rhythm. The first energy dose for electrical cardioversion of SVT is 0.5–1 J kg⁻¹ and the second dose is 2 J kg⁻¹. If unsuccessful, give amiodarone or procainamide under guidance from a paediatric cardiologist or intensivist before the third attempt. Verapamil may be considered as an alternative therapy in older children but should not be routinely used in infants.

Amiodarone has been shown to be effective in the treatment of SVT in several paediatric studies.^{270,287,290–297} However, since most studies of amiodarone use in narrow complex tachycardias have been for junctional ectopic tachycardia in postoperative children, the applicability of its use in all cases of SVT may be limited. If the child is haemodynamically stable, early consultation with an expert is recommended before giving amiodarone. An expert should also be consulted about alternative treatment strategies because the evidence to support other drugs in the treatment of SVT is limited and inconclusive.^{298,299} If amiodarone is used in this circumstance, avoid rapid administration because hypotension is common.

Wide complex tachycardia

In children, wide-QRS complex tachycardia is uncommon and more likely to be supraventricular than ventricular in origin.³⁰⁰ Nevertheless, in haemodynamically unstable children, it must be considered to be VT until proven otherwise. Ventricular tachycardia occurs most often in the child with underlying heart disease (e.g., after cardiac surgery, cardiomyopathy, myocarditis, electrolyte disorders, prolonged QT interval, central intracardiac catheter). Synchronised cardioversion is the treatment of choice for unstable VT with a pulse. Consider anti-arrhythmic therapy if a second cardioversion attempt is unsuccessful or if VT recurs.

Amiodarone has been shown to be effective in treating paediatric arrhythmias,²⁹¹ although cardiovascular side effects are common.^{270,287,292,297,301}

Stable arrhythmias

Whilst maintaining the child's airway, breathing and circulation, contact an expert before initiating therapy. Depending on the child's clinical history, presentation and ECG diagnosis, a child with stable, wide-QRS complex tachycardia may be treated for SVT and be given vagal manoeuvres or adenosine. Amiodarone may be considered as a treatment option if this fails or if the diagnosis of VT is confirmed on an ECG. Procainamide may also be considered in stable SVT refractory to vagal manoeuvres and adenosine.^{239,302–304} and in stable VT.^{239,240,305,306} Do not give procainamide with amiodarone.

Special circumstances

Channelopathy

When sudden unexplained cardiac arrest occurs in children and young adults, obtain a complete past medical and family history (including a history of syncopal episodes, seizures, unexplained accidents/drownings, or sudden death) and review any available previous ECGs. All infants, children, and young adults with sudden, unexpected death should, if possible, have an unrestricted, complete autopsy, performed preferably by pathologists with training and expertise in cardiovascular pathology.^{307–316} Consideration should be given to preservation and genetic analysis of tissue to determine the presence of a channelopathy. Refer families of

patients whose cause of death is not found on autopsy to a health care provider/centre with expertise in cardiac rhythm disturbances.

Life support for blunt or penetrating trauma

There is a very high mortality associated with cardiac arrest from major (blunt or penetrating) trauma.^{317–320} There is little evidence to support any additional specific interventions that are different from the routine management of cardiac arrest; however, the use of resuscitative thoracotomy may be considered in children with penetrating injuries.^{321–325}

Single ventricle post-stage 1 repair

The incidence of cardiac arrest in infants following single ventricle stage 1 repair is approximately 20%, with a survival to discharge of 33%.³²⁶ There is no evidence that anything other than routine resuscitative protocols should be followed. Diagnosis of the pre-arrest state is difficult but it may be assisted by monitoring the oxygen extraction (superior vena caval ScvO₂) or near infrared spectroscopy (cerebral and splanchnic circulations).^{327–329} Treatment of high systemic vascular resistance with alpha-adrenergic receptor blockade may improve systemic oxygen delivery,³³⁰ reduce the incidence of cardiovascular collapse,³³¹ and improve survival.³³²

Single ventricle post-Fontan

Children in the pre-arrest state who have Fontan or hemi-Fontan anatomy may benefit from increased oxygenation and an improved cardiac output by instituting negative pressure ventilation.^{333,334} Extracorporeal membrane oxygenation (ECMO) may be useful rescue for children with failing Fontan circulations but no recommendation can be made in favour or against ECMO in those with hemi-Fontan physiology or for rescue during resuscitation.³³⁵

Pulmonary hypertension

There is an increased risk of cardiac arrest in children with pulmonary hypertension.^{336,337} Follow routine resuscitation protocols in these patients with emphasis on high FiO₂ and alkalosis/hyperventilation because this may be as effective as inhaled nitric oxide in reducing pulmonary vascular resistance.³³⁸ Resuscitation is most likely to be successful in patients with a reversible cause who are treated with intravenous epoprostenol or inhaled nitric oxide.³³⁹ If routine medications that reduce pulmonary artery pressure have been stopped, they should be restarted and the use of aerosolised epoprostenol or inhaled nitric oxide considered.³⁴⁰ Right ventricular support devices may improve survival.^{341–344}

Post-arrest management

After prolonged, complete, whole-body hypoxia-ischaemia ROSC has been described as an unnatural pathophysiological state, created by successful CPR.³⁴⁵ Post-arrest management must be a multidisciplinary activity and include all the treatments needed for complete neurological recovery. The main goals are to reverse brain injury and myocardial dysfunction, and to treat the systemic ischaemia/reperfusion response and any persistent precipitating pathology.

Myocardial dysfunction

Myocardial dysfunction is common after cardiopulmonary resuscitation.^{345–348} Vasoactive drugs (adrenaline, dobutamine,

dopamine and noradrenaline) may improve the child's post-arrest haemodynamic values but the drugs must be titrated according to the clinical condition.^{349–359}

Temperature control and management

Hypothermia is common in the child following cardiopulmonary resuscitation.³⁶⁰ Central hypothermia (32–34 °C) may be beneficial, whereas fever may be detrimental to the injured brain. Mild hypothermia has an acceptable safety profile in adults^{361,362} and neonates.^{363–368} Whilst it may improve neurological outcome in children, an observational study neither supports nor refutes the use of therapeutic hypothermia in paediatric cardiac arrest.³⁶⁹

A child who regains a spontaneous circulation, but remains comatose after cardiopulmonary arrest, may benefit from being cooled to a core temperature of 32–34 °C for at least 24 h. The successfully resuscitated child with hypothermia and ROSC should not be actively rewarmed unless the core temperature is below 32 °C. Following a period of mild hypothermia, rewarm the child slowly at 0.25–0.5 °C h⁻¹.

There are several methods to induce, monitor and maintain body temperature in children. External and/or internal cooling techniques can be used to initiate cooling.^{370–372} Shivering can be prevented by deep sedation and neuromuscular blockade. Complications can occur and include an increased risk of infection, cardiovascular instability, coagulopathy, hyperglycaemia and electrolyte abnormalities.^{373–375}

These guidelines are based on evidence from the use of therapeutic hypothermia in neonates and adults. At the time of writing, there are ongoing, prospective, multicentre trials of therapeutic hypothermia in children following in- and out-of-hospital cardiac arrest (www.clinicaltrials.gov; NCT00880087 and NCT00878644).

Fever is common following cardiopulmonary resuscitation and is associated with a poor neurological outcome,^{376–378} the risk increasing for each degree of body temperature greater than 37 °C.³⁷⁶ There are limited experimental data suggesting that the treatment of fever with antipyretics and/or physical cooling reduces neuronal damage.^{379,380} Antipyretics and accepted drugs to treat fever are safe; therefore, use them to treat fever aggressively.

Glucose control

Both hyper- and hypo-glycaemia may impair outcome of critically ill adults and children and should be avoided,^{228–230,381–383} but tight glucose control may also be harmful.^{231,384} Although there is insufficient evidence to support or refute a specific glucose management strategy in children with ROSC after cardiac arrest,^{225,226,345} it is appropriate to monitor blood glucose and avoid hypoglycaemia as well as sustained hyperglycaemia.

Prognosis of cardiopulmonary arrest

Although several factors are associated with outcome after cardiopulmonary arrest and resuscitation^{41,60,385–389} there are no simple guidelines to determine when resuscitative efforts become futile.

After 20 min of resuscitation, the resuscitation team leader should consider whether or not to stop.^{273,390–394} The relevant considerations in the decision to continue the resuscitation include the cause of arrest,^{60,395} pre-existing medical conditions, age,^{41,389} site of arrest, whether the arrest was witnessed,^{60,394} the duration of untreated cardiopulmonary arrest ('no flow'), number of doses of adrenaline, the ETCO₂ value, the presence of a shockable rhythm as the first or subsequent rhythm,^{386,387} the

promptness of extracorporeal life support for a reversible disease process,^{396–398} and associated special circumstances (e.g., icy water drowning,^{277,399,400} exposure to toxic drugs).

Parental presence

In some Western societies, the majority of parents prefer to be present during the resuscitation of their child.^{401–410} Parental presence has neither been perceived as disruptive,^{403,411–415} nor stressful for the staff.^{401,403,412} Parents witnessing their child's resuscitation believe their presence to be beneficial to the child.^{401–403,410,414–417} Allowing parents to be at the side of their child helps them to gain a realistic view of the attempted resuscitation and the child's death. Furthermore, they may have the opportunity to say goodbye to their child. Families who are present at their child's death show better adjustment and undergo a better grieving process.^{402–404,414,415,417,418}

Parental presence in the resuscitation room may help healthcare providers maintain their professional behaviour, whilst helping them to see the child as a human being and a family member.⁴¹¹ However in out-of-hospital resuscitation, some EMS providers may feel threatened by the presence of relatives or are concerned that relatives may interfere with their resuscitation efforts.⁴¹⁹ Evidence about parental presence during resuscitation comes from selected countries and can probably not be generalised to all of Europe, where there could be different socio-cultural and ethical considerations.

Family presence guidelines

When relatives are allowed in the resuscitation room, a dedicated member of the resuscitation team should be present with the parents to explain the process in an empathetic manner, ensuring that the parents do not interfere with or distract the resuscitation process. If the presence of the parents is impeding the progress of the resuscitation, they should be sensitively asked to leave. When appropriate, physical contact with the child should be allowed and, wherever possible, the parents should be allowed to be with their dying child at the final moment.^{411,420–423}

The leader of the resuscitation team, not the parents, will decide when to stop the resuscitation; this should be expressed with sensitivity and understanding. After the event, the team should be debriefed, to enable any concerns to be expressed and for the team to reflect on their clinical practice in a supportive environment.

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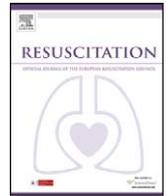
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European Resuscitation Council Guidelines for Resuscitation 2010 Section 7. Resuscitation of babies at birth

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Introduction

The following guidelines for resuscitation at birth have been developed during the process that culminated in the 2010 International Consensus Conference on Emergency Cardiovascular Care (ECC) and Cardiopulmonary Resuscitation (CPR) Science with Treatment Recommendations.^{1,2} They are an extension of the guidelines already published by the ERC³ and take into account recommendations made by other national and international organisations.

Summary of changes since 2005 Guidelines

The following are the main changes that have been made to the guidelines for resuscitation at birth in 2010:

- For uncompromised babies, a delay in cord clamping of at least 1 min from the complete delivery of the infant, is now recommended. As yet there is insufficient evidence to recommend an appropriate time for clamping the cord in babies who are severely compromised at birth.
- For term infants, air should be used for resuscitation at birth. If, despite effective ventilation, oxygenation (ideally guided by oximetry) remains unacceptable, use of a higher concentration of oxygen should be considered.
- Preterm babies less than 32 weeks gestation may not reach the same arterial blood oxygen saturations in air as those achieved by term babies. Therefore blended oxygen and air should be given judiciously and its use guided by pulse oximetry. If a blend of oxygen and air is not available use what is available.
- Preterm babies of less than 28 weeks gestation should be completely covered in a food-grade plastic wrap or bag up to their necks, without drying, immediately after birth. They should then be nursed under a radiant heater and stabilised. They should

remain wrapped until their temperature has been checked after admission. For these infants delivery room temperatures should be at least 26 °C.

- The recommended compression:ventilation ratio for CPR remains at 3:1 for newborn resuscitation.
- Attempts to aspirate meconium from the nose and mouth of the unborn baby, while the head is still on the perineum, are not recommended. If presented with a floppy, apnoeic baby born through meconium it is reasonable to rapidly inspect the oropharynx to remove potential obstructions. If appropriate expertise is available, tracheal intubation and suction may be useful. However, if attempted intubation is prolonged or unsuccessful, start mask ventilation, particularly if there is persistent bradycardia.
- If adrenaline (epinephrine) is given then the intravenous route is recommended using a dose of 10–30 $\mu\text{g kg}^{-1}$. If the tracheal route is used, it is likely that a dose of at least 50–100 $\mu\text{g kg}^{-1}$ will be needed to achieve a similar effect to 10 $\mu\text{g kg}^{-1}$ intravenously.
- Detection of exhaled carbon dioxide in addition to clinical assessment is recommended as the most reliable method to confirm placement of a tracheal tube in neonates with spontaneous circulation.
- Newly born infants born at term or near-term with evolving moderate to severe hypoxic–ischemic encephalopathy should, where possible, be offered therapeutic hypothermia. This does not affect immediate resuscitation but is important for post-resuscitation care.

The guidelines that follow do not define the only way that resuscitation at birth should be achieved; they merely represent a widely accepted view of how resuscitation at birth can be carried out both safely and effectively (Fig. 7.1).

Preparation

Relatively few babies need any resuscitation at birth. Of those that do need help, the overwhelming majority will require only assisted lung aeration. A small minority may need a brief period of chest compressions in addition to lung aeration. Of 100,000 babies born in Sweden in 1 year, only 10 per 1000 (1%) babies of 2.5 kg or more appeared to need resuscitation at delivery.⁴ Of those babies

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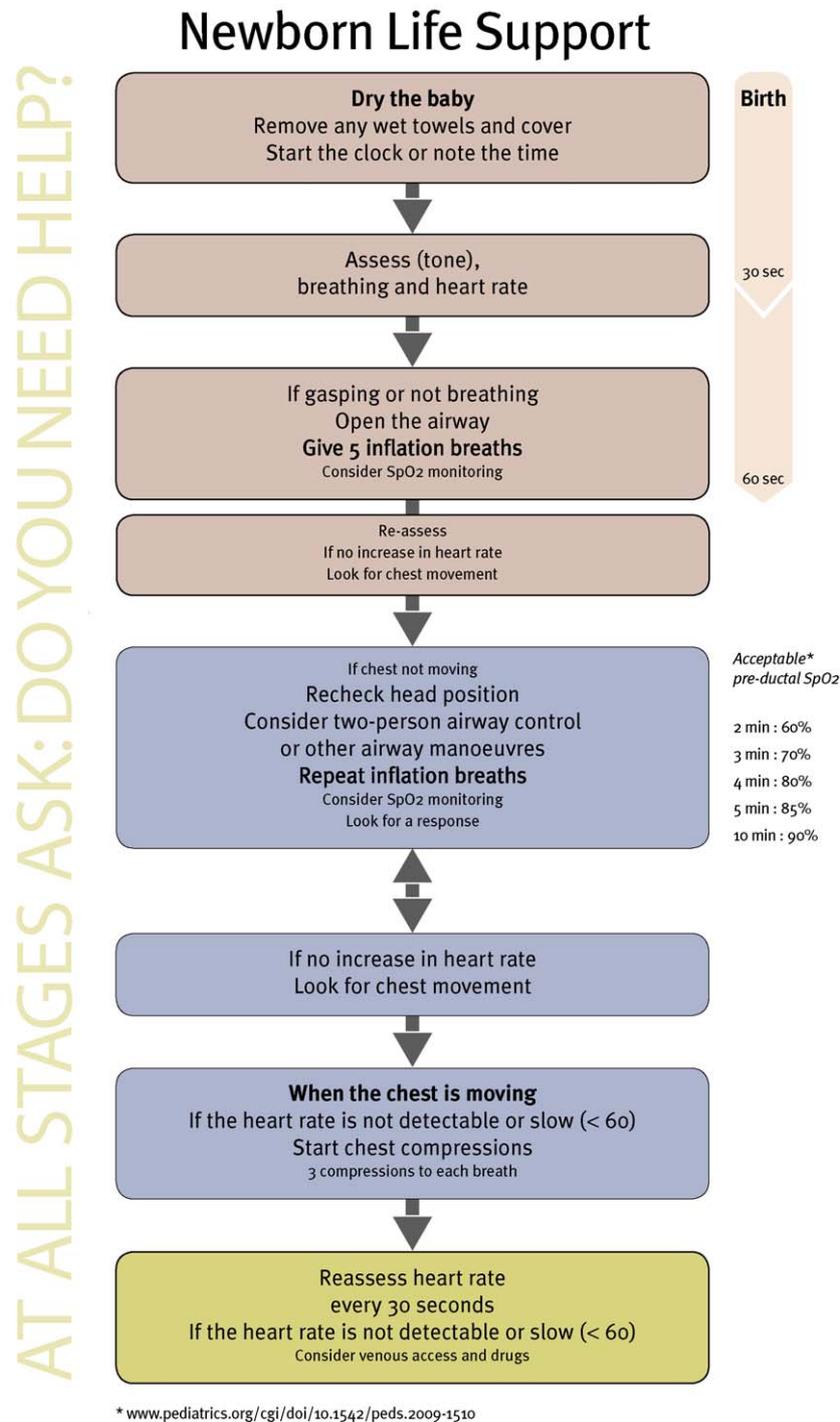


Fig. 7.1. Newborn life support algorithm.

receiving resuscitation, 8 per 1000 responded to mask inflation and only 2 per 1000 appeared to need intubation. The same study tried to assess the unexpected need for resuscitation at birth and found that for low risk babies, i.e. those born after 32 weeks gestation and following an apparently normal labour, about 2 per 1000 (0.2%) appeared to need resuscitation at delivery. Of these, 90% responded to mask inflation alone while the remaining 10% appeared not to respond to mask inflation and therefore were intubated at birth.

Resuscitation or specialist help at birth is more likely to be needed by babies with intrapartum evidence of significant fetal compromise, babies delivering before 35 weeks gestation, babies

delivering vaginally by the breech, and multiple pregnancies. Although it is often possible to predict the need for resuscitation or stabilisation before a baby is born, this is not always the case. Therefore, personnel trained in newborn life support should be easily available at every delivery and, should there be any need for intervention, the care of the baby should be their sole responsibility. One person experienced in tracheal intubation of the newborn should ideally be in attendance for deliveries associated with a high risk of requiring neonatal resuscitation. Local guidelines indicating who should attend deliveries should be developed, based on current practice and clinical audit.

An organised educational programme in the standards and skills required for resuscitation of the newborn is therefore essential for any institution in which deliveries occur.

Planned home deliveries

Recommendations as to who should attend a planned home delivery vary from country to country, but the decision to undergo a planned home delivery, once agreed with medical and midwifery staff, should not compromise the standard of initial resuscitation at birth. There will inevitably be some limitations to resuscitation of a newborn baby in the home, because of the distance from further assistance, and this must be made clear to the mother at the time plans for home delivery are made. Ideally, two trained professionals should be present at all home deliveries; one of these must be fully trained and experienced in providing mask ventilation and chest compressions in the newborn.

Equipment and environment

Unlike adult CPR, resuscitation at birth is often a predictable event. It is therefore possible to prepare the environment and the equipment before delivery of the baby. Resuscitation should ideally take place in a warm, well-lit, draught free area with a flat resuscitation surface placed below a radiant heater, with other resuscitation equipment immediately available. All equipment must be checked frequently.

When a birth takes place in a non-designated delivery area, the recommended minimum set of equipment includes a device for safe assisted lung aeration of an appropriate size for the newborn, warm dry towels and blankets, a sterile instrument for cutting the umbilical cord and clean gloves for the attendant and assistants. It may also be helpful to have a suction device with a suitably sized suction catheter and a tongue depressor (or laryngoscope) to enable the oropharynx to be examined. Unexpected deliveries outside hospital are most likely to involve emergency services who should plan for such events.

Temperature control

Naked, wet, newborn babies cannot maintain their body temperature in a room that feels comfortably warm for adults. Compromised babies are particularly vulnerable.⁵ Exposure of the newborn to cold stress will lower arterial oxygen tension⁶ and increase metabolic acidosis.⁷ Prevent heat loss:

- Protect the baby from draughts.
- Keep the delivery room warm. For babies less than 28 weeks gestation the delivery room temperature should be 26 °C.^{8,9}
- Dry the term baby immediately after delivery. Cover the head and body of the baby, apart from the face, with a warm towel to prevent further heat loss. Alternatively, place the baby skin to skin with mother and cover both with a towel.
- If the baby needs resuscitation then place the baby on a warm surface under a preheated radiant warmer.
- In very preterm babies (especially below 28 weeks) drying and wrapping may not be sufficient. A more effective method of keeping these babies warm is to cover the head and body of the baby (apart from the face) with plastic wrapping, without drying the baby beforehand, and then to place the baby so covered under radiant heat.

Initial assessment

The Apgar score was proposed as a “simple, common, clear classification or grading of newborn infants” to be used “as a basis for discussion and comparison of the results of obstetric practices, types of maternal pain relief and the effects of resuscitation” (our emphasis).¹⁰ It was not designed to be assembled and ascribed in order to then identify babies in need of resuscitation.¹¹ However, individual components of the score, namely respiratory rate, heart rate and tone, if assessed rapidly, can identify babies needing resuscitation (and Virginia Apgar herself found that heart rate was the most important predictor of immediate outcome).¹⁰ Furthermore, repeated assessment particularly of heart rate and, to a lesser extent breathing, can indicate whether the baby is responding or whether further efforts are needed.

Breathing

Check whether the baby is breathing. If so, evaluate the rate, depth and symmetry of breathing together with any evidence of an abnormal breathing pattern such as gasping or grunting.

Heart rate

This is best assessed by listening to the apex beat with a stethoscope. Feeling the pulse in the base of the umbilical cord is often effective but can be misleading, cord pulsation is only reliable if found to be more than 100 beats per minute (bpm).¹² For babies requiring resuscitation and/or continued respiratory support, a modern pulse oximeter can give an accurate heart rate.¹³

Colour

Colour is a poor means of judging oxygenation,¹⁴ which is better assessed using pulse oximetry if possible. A healthy baby is born blue but starts to become pink within 30 s of the onset of effective breathing. Peripheral cyanosis is common and does not, by itself, indicate hypoxemia. Persistent pallor despite ventilation may indicate significant acidosis or rarely hypovolaemia. Although colour is a poor method of judging oxygenation, it should not be ignored: if a baby appears blue check oxygenation with a pulse oximeter.

Tone

A very floppy baby is likely to be unconscious and will need ventilatory support.

Tactile stimulation

Drying the baby usually produces enough stimulation to induce effective breathing. Avoid more vigorous methods of stimulation. If the baby fails to establish spontaneous and effective breaths following a brief period of stimulation, further support will be required.

Classification according to initial assessment

On the basis of the initial assessment, the baby can be placed into one of three groups:

1. Vigorous breathing or crying

Good tone

Heart rate higher than 100 min⁻¹

This baby requires no intervention other than drying, wrapping in a warm towel and, where appropriate, handing to the

mother. The baby will remain warm through skin-to-skin contact with mother under a cover, and may be put to the breast at this stage.

2. **Breathing inadequately or apnoeic**
Normal or reduced tone

Heart rate less than 100 min⁻¹

Dry and wrap. This baby may improve with mask inflation but if this does not increase the heart rate adequately, may also require chest compressions.

3. **Breathing inadequately or apnoeic**
Floppy

Low or undetectable heart rate

Often pale suggesting poor perfusion

Dry and wrap. This baby will then require immediate airway control, lung inflation and ventilation. Once this has been successfully accomplished the baby may also need chest compressions, and perhaps drugs.

There remains a very rare group of babies who, though breathing adequately and with a good heart rate, remain hypoxaemic. This group includes a range of possible diagnoses such as diaphragmatic hernia, surfactant deficiency, congenital pneumonia, pneumothorax, or cyanotic congenital heart disease.

Newborn life support

Commence newborn life support if assessment shows that the baby has failed to establish adequate regular normal breathing, or has a heart rate of less than 100 min⁻¹. Opening the airway and aerating the lungs is usually all that is necessary. Furthermore, more complex interventions will be futile unless these two first steps have been successfully completed.

Airway

Place the baby on his or her back with the head in a neutral position (Fig. 7.2). A 2 cm thickness of the blanket or towel placed under the baby's shoulder may be helpful in maintaining proper head position. In floppy babies application of jaw thrust or the use of an appropriately sized oropharyngeal airway may be helpful in opening the airway.

Suction is needed only if the airway is obstructed. Obstruction may be caused by particulate meconium but can also be caused by blood clots, thick tenacious mucus or vernix even in deliveries where meconium staining is not present. However, aggressive pharyngeal suction can delay the onset of spontaneous breathing and cause laryngeal spasm and vagal bradycardia.¹⁵ The presence of thick meconium in a non-vigorous baby is the only indication for *considering* immediate suction of the oropharynx. If suction is

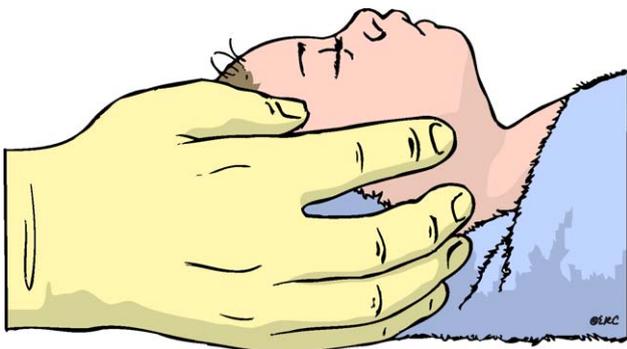


Fig. 7.2. Newborn with head in neutral position.

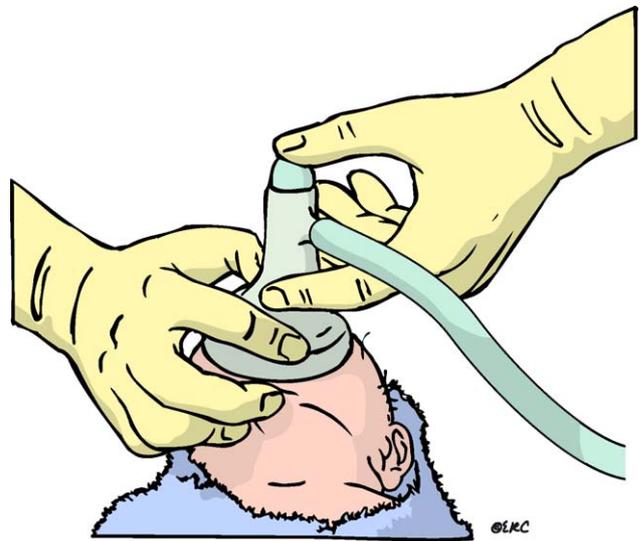


Fig. 7.3. Mask ventilation of newborn.

attempted this is best done under direct vision. Connect a 12–14 FG suction catheter, or a Yankauer sucker, to a suction source not exceeding minus 100 mm Hg.

Breathing

After initial steps at birth, if breathing efforts are absent or inadequate, lung aeration is the priority (Fig. 7.3). In term babies, begin resuscitation with air. The primary measure of adequate initial lung inflation is a prompt improvement in heart rate; assess chest wall movement if heart rate does not improve.

For the first five inflation breaths maintain the initial inflation pressure for 2–3 s. This will help lung expansion. Most babies needing resuscitation at birth will respond with a rapid increase in heart rate within 30 s of lung inflation. If the heart rate increases but the baby is not breathing adequately, ventilate at a rate of about 30 breaths min⁻¹ allowing approximately 1 s for each inflation, until there is adequate spontaneous breathing.

Adequate passive ventilation is usually indicated by either a rapidly increasing heart rate or a heart rate that is maintained faster than 100 min⁻¹. If the baby does not respond in this way the most likely cause is inadequate airway control or inadequate ventilation. Look for passive chest movement in time with inflation efforts; if these are present then lung aeration has been achieved. If these are absent then airway control and lung aeration has not been confirmed. Without adequate lung aeration, chest compressions will be ineffective; therefore, confirm lung aeration before progressing to circulatory support.

Some practitioners will ensure airway control by tracheal intubation, but this requires training and experience. If this skill is not available and the heart rate is decreasing, re-evaluate the airway position and deliver inflation breaths while summoning a colleague with intubation skills.

Continue ventilatory support until the baby has established normal regular breathing.

Circulatory support

Circulatory support with chest compressions is effective only if the lungs have first been successfully inflated. Give chest compressions if the heart rate is less than 60 min⁻¹ despite adequate ventilation.

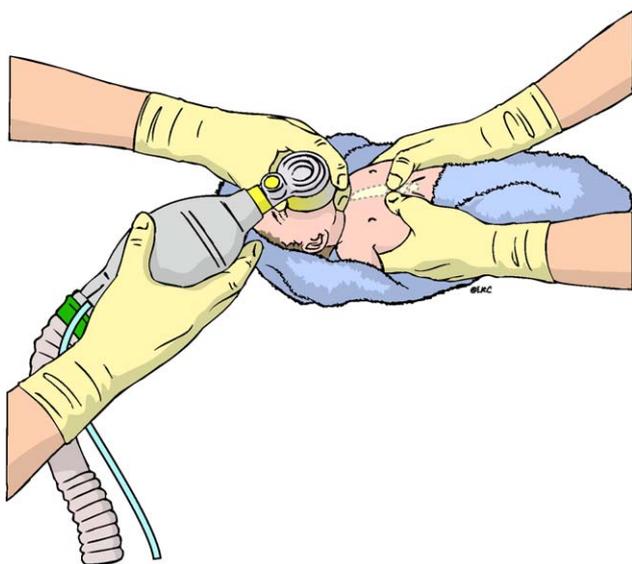


Fig. 7.4. Ventilation and chest compression of newborn.

The most effective technique for providing chest compressions is to place the two thumbs side by side over the lower third of the sternum just below an imaginary line joining the nipples, with the fingers encircling the torso and supporting the back (Fig. 7.4).^{16–19} An alternative way to find the correct position of the thumbs is to identify the xiphisternum and then to place the thumbs on the sternum one finger's breadth above this point. The sternum is compressed to a depth of approximately one-third of the anterior–posterior diameter of the chest allowing the chest wall to return to its relaxed position between compressions.²⁰

Use a ratio of three compressions to one ventilation, aiming to achieve approximately 120 events per minute, i.e. approximately 90 compressions and 30 ventilations. There are theoretical advantages to allowing a relaxation phase that is very slightly longer than the compression phase.²¹ However, the quality of the compressions and breaths are probably more important than the rate.

Check the heart rate after about 30 s and every 30 s thereafter. Discontinue chest compressions when the spontaneous heart rate is faster than 60 min⁻¹.

Drugs

Drugs are rarely indicated in resuscitation of the newly born infant. Bradycardia in the newborn infant is usually caused by inadequate lung inflation or profound hypoxia, and establishing adequate ventilation is the most important step to correct it. However, if the heart rate remains less than 60 min⁻¹ despite adequate ventilation and chest compressions, it is reasonable to consider the use of drugs. These are best given via an umbilical venous catheter (Fig. 7.5).

Adrenaline

Despite the lack of human data it is reasonable to use adrenaline when adequate ventilation and chest compressions have failed to increase the heart rate above 60 min⁻¹. If adrenaline is used, a dose of 10–30 µg kg⁻¹ should be administered *intravenously* as soon as possible.

The tracheal route is not recommended (see below) but if it is used, it is highly likely that doses of 50–100 µg kg⁻¹ will be required. Neither the safety nor the efficacy of these higher tracheal doses has been studied. Do *not* give these high doses intravenously.

Bicarbonate

If effective spontaneous cardiac output is not restored despite adequate ventilation and adequate chest compressions, reversing intracardiac acidosis may improve myocardial function and achieve a spontaneous circulation. There are insufficient data to recommend routine use of bicarbonate in resuscitation of the newly born. The hyperosmolarity and carbon dioxide-generating properties of sodium bicarbonate may impair myocardial and cerebral function. Use of sodium bicarbonate is discouraged during brief CPR. If it is used during prolonged arrests unresponsive to other therapy, it should be given only after adequate ventilation and circulation is established with CPR. A dose of 1–2 mmol kg⁻¹ may be given by slow intravenous injection after adequate ventilation and perfusion have been established.

Fluids

If there has been suspected blood loss or the infant appears to be in shock (pale, poor perfusion, weak pulse) and has not responded adequately to other resuscitative measures then consider giving fluid.²² This is a rare event. In the absence of suitable blood (i.e. irradiated and leucocyte-depleted group O Rh-negative blood), isotonic crystalloid rather than albumin is the solution of choice for restoring intravascular volume. Give a bolus of 10 ml kg⁻¹ initially. If successful it may need to be repeated to maintain an improvement.

Stopping resuscitation

Local and national committees will determine the indications for stopping resuscitation. If the heart rate of a newly born baby is not detectable and remains undetectable for 10 min, it is then appropriate to consider stopping resuscitation. The decision to continue resuscitation efforts when the heart rate has been undetectable for longer than 10 min is often complex and may be influenced by issues such as the presumed aetiology, the gestation of the baby, the potential reversibility of the situation, and the parents' previous expressed feelings about acceptable risk of morbidity.

In cases where the heart rate is less than 60 min⁻¹ at birth and does not improve after 10 or 15 min of continuous and apparently adequate resuscitative efforts, the choice is much less clear. In this situation there is insufficient evidence about outcome to enable firm guidance on whether to withhold or to continue resuscitation.

Communication with the parents

It is important that the team caring for the newborn baby informs the parents of the baby's progress. At delivery, adhere to

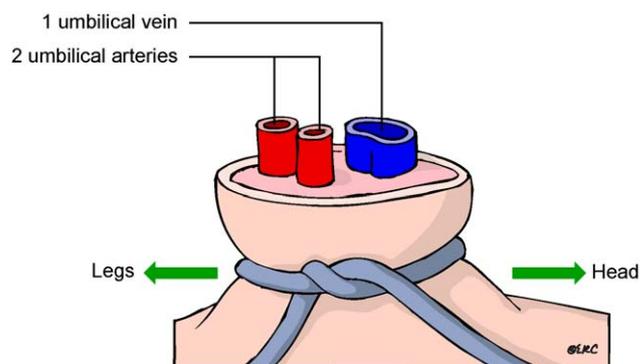


Fig. 7.5. Newborn umbilical cord showing the arteries and veins.

the routine local plan and, if possible, hand the baby to the mother at the earliest opportunity. If resuscitation is required inform the parents of the procedures undertaken and why they were required.

Decisions to discontinue resuscitation should ideally involve senior paediatric staff. Whenever possible, the decision to attempt resuscitation of an extremely preterm baby should be taken in close consultation with the parents and senior paediatric and obstetric staff. Where a difficulty has been foreseen, for example in the case of severe congenital malformation, discuss the options and prognosis with the parents, midwives, obstetricians and birth attendants before delivery.²³ Record carefully all discussions and decisions in the mother's notes prior to delivery and in the baby's records after birth.

Specific questions addressed at the 2010 Consensus Conference on CPR Science

Maintaining normal temperature in preterm infants

Significantly preterm babies are likely to become hypothermic despite careful application of the traditional techniques for keeping them warm (drying, wrapping and placing under radiant heat).²⁴ Several randomised controlled trials and observational studies have shown that placing the preterm baby under radiant heat and then covering the baby with food-grade plastic wrapping without drying them, significantly improves temperature on admission to intensive care compared with traditional techniques.^{25–27} The baby's temperature must be monitored closely because of the small but described risk of inducing hyperthermia with this technique.²⁸ All resuscitation procedures including intubation, chest compression and insertion of lines, can be achieved with the plastic cover in place. Significantly preterm babies maintain their temperature better when the ambient temperature of the delivery room is 26 °C or higher.^{8,9}

Infants born to febrile mothers have a higher incidence of perinatal respiratory depression, neonatal seizures, early mortality, and cerebral palsy.^{28–30} Animal studies indicate that hyperthermia during or following ischaemia is associated with a progression of cerebral injury.^{31,32} Hyperthermia should be avoided.

Meconium

In the past it was hoped that clearing meconium from the airway of babies at birth would reduce the incidence and severity of meconium aspiration syndrome (MAS). However, studies supporting this view were based on a comparison of suctioning on the outcome of a group of babies with the outcome of historical controls.^{33,34} Furthermore other studies failed to find any evidence of benefit from this practice.^{35,36} More recently, a multi-centre randomised controlled trial reported in 2000³⁷ showed that routine elective intubation and suctioning of these infants, if vigorous at birth, did not reduce MAS and a further randomised study published in 2004 showed that suctioning the nose and mouth of such babies on the perineum and before delivery of the shoulders (intrapartum suctioning) was also ineffective.³⁸ Intrapartum suctioning and routine intubation and suctioning of vigorous infants born through meconium-stained liquor are not recommended. There remains the question of what to do with non-vigorous infants in this situation. Observational studies have confirmed that these babies are at increased risk of meconium aspiration syndrome but there have been no randomised studies of the effect of intubation followed by suctioning versus no intubation in this group.

Recommendation: In the absence of randomised, controlled trials, there is insufficient evidence to recommend a change in the current practice of performing direct oropharyngeal and tracheal

suctioning of *non-vigorous babies* with meconium-stained amniotic fluid, if feasible. However, if attempted intubation is prolonged or unsuccessful, mask ventilation should be implemented, particularly if there is persistent bradycardia.

Air or 100% oxygen

For the newly born infant in need of resuscitation at birth, the rapid establishment of pulmonary gas exchange to replace the failure of placental respiration is the key to success. In the past it has seemed reasonable that delivery of a high concentration of oxygen to the tissues at risk of hypoxia might help to reduce the number of cells which were damaged by the anaerobic process. However, in the last 30 years the 'oxygen paradox' – the fact that cell and tissue injury is increased if hypoxic tissue is then exposed to high concentrations of oxygen – has been recognized, the role of free radicals, antioxidants and their link with apoptosis and reperfusion injury has been explored, and the idea of oxidative stress established. In the light of this knowledge it has become increasingly difficult to sustain the idea that exposure to high concentrations of oxygen, however brief, is without risk. Furthermore, randomised studies in asphyxiated newborn babies strongly suggest that air is certainly as effective as 100% oxygen, if not more effective, at least in the short term.³⁹

There is also abundant evidence from animal and human studies that hyperoxaemia alone is damaging to the brain and other organs at the cellular level, particularly after asphyxia. Animal studies suggest that the risk is greatest to the immature brain during the brain growth spurt (mid-pregnancy to 3 years).⁴⁰ These risks include deleterious effects on glial progenitor cells and myelination.⁴¹

Other issues include concerns that pulmonary vascular resistance may take longer to resolve if air is used rather than oxygen for lung inflation at birth. However, though two studies have shown that it may be reduced a little further and a little faster by use of oxygen rather than air, there is a price to pay. Exposure to high concentrations of oxygen at birth results in the creation of increased reactive oxygen species which, in turn, reduces the potential for pulmonary artery vaso-relaxation later in the neonatal course.

There are now numerous reports of oximetry data following delivery. When using technology available from the early 2000 s, a reliable reading can be obtained from >90% of normal term births, approximately 80% of those born preterm, and 80–90% of those apparently requiring resuscitation, within 2 min of birth.⁴² Uncompromised babies born at term at sea level have SaO₂ ~60% during labour,⁴³ which increases to >90% by 10 min.⁴⁴ The 25th percentile is approximately 40% at birth and increases to ~80% at 10 min.⁴⁵ Values are lower in those born by Caesarean section⁴⁶ and those born at altitude.⁴⁷ Those born preterm may take longer to reach >90%.⁴⁵ Those given supplemental oxygen had a higher incidence of SaO₂ >95%, even when a protocol to decrease the FiO₂ was implemented, although the extent of this was restricted by insufficient power and the particular protocols used in the studies.^{48,49}

Recommendation: In term infants receiving resuscitation at birth with positive-pressure ventilation, it is best to begin with air as opposed to 100% oxygen. If, despite effective ventilation, there is no increase in heart rate or oxygenation (guided by oximetry wherever possible) remains unacceptable, use a higher concentration of oxygen.

As many preterm babies less than 32 weeks gestation will not achieve target values for transcutaneous oxygen saturation in air, blended oxygen and air may be given judiciously and ideally guided by pulse oximetry. Both hyperoxaemia and hypoxemia should be avoided. If a blend of oxygen and air is not available, resuscitation should be initiated with air.

Timing of cord clamping

Cine-radiographic studies of babies taking their first breath at delivery showed that those whose cords were clamped prior to this had an immediate decrease in the size of the heart during the subsequent three or four cardiac cycles. The heart then increased in size to almost the same size as the fetal heart. The initial decrease in size could be interpreted as being due to filling of the of the newly-opened pulmonary vascular system during aeration with the subsequent increase in size occurring as a consequence of blood returning to the heart from the lung.⁵⁰ Brady and James drew attention to the occurrence of a bradycardia apparently induced by clamping the cord before the first breath and noted that this did not occur in babies where clamping occurred after breathing was established.⁵¹ Might such early clamping of the cord in a significantly preterm infant, whose ability to inflate his lungs by generating negative intrathoracic pressures is already compromised, either induce or prolong a bradycardia leading to a 'need' for resuscitation?

Studies in term infants clamped late have shown an improvement in iron status and a number of other haematological indices over the next 3–6 months. They have also shown greater use of phototherapy for jaundice in the delayed group but the use of phototherapy was neither controlled nor defined and many would regard this of little consequence.

Studies in preterm infants have consistently shown improved stability in the immediate postnatal period and reduced exposure to blood transfusion in the ensuing weeks. Some studies have suggested a reduced incidence of intraventricular haemorrhage and of late-onset sepsis.⁵² Again some studies report increased jaundice and use of phototherapy but there have been no reports of increased use of exchange transfusion.

Studies have not addressed any effect of delaying cord clamping on babies apparently needing resuscitation at birth because such babies have been excluded.

Recommendation: Delay in umbilical cord clamping for at least 1 min is recommended for newborn infants not requiring resuscitation. A similar delay should be applied to premature babies being stabilised. For babies requiring resuscitation, resuscitative intervention remains the priority.

Initial breaths and assisted ventilation

In term infants, spontaneous or assisted initial inflations create a functional residual capacity (FRC).^{53–59} The optimum pressure, inflation time and flow required to establish an effective FRC has not been determined. Average initial peak inflating pressures of 30–40 cm H₂O (inflation time undefined) usually ventilate unresponsive term infants successfully.^{54,56,57,59} Assisted ventilation rates of 30–60 breaths min⁻¹ are used commonly, but the relative efficacy of various rates has not been investigated.

Where pressure is being monitored, an initial inflation pressure of 20 cm H₂O may be effective, but 30–40 cm H₂O or higher may be required in some term babies. If pressure is not being monitored but merely limited by a non-adjustable 'blow-off' valve, use the minimum inflation required to achieve an increase in heart rate. There is insufficient evidence to recommend an optimum inflation time. In summary, try to provide artificial ventilation at 30–60 breaths min⁻¹ to achieve or maintain a heart rate higher than 100 min⁻¹ promptly.

Assisted ventilation of preterm infants

Animal studies show that preterm lungs are easily damaged by large-volume inflations immediately after birth⁶⁰ and that

maintaining a positive end expiratory pressure (PEEP) immediately after birth protects against lung damage. Positive end expiratory pressure also improves lung compliance and gas exchange.^{61,62}

Both over-inflation and repeated collapse of the alveoli have been shown to cause damage in animal studies. Inflation pressure is measured in an imperfect attempt to limit tidal volume. Ideally tidal volume would be measured, and after lung aeration, limited to between 4 and 8 ml kg⁻¹ to avoid over-distension.⁶³

When ventilating preterm infants, very obvious passive chest wall movement may indicate excessive tidal volumes and should be avoided. Monitoring of pressure may help to provide consistent inflations and avoid high pressures. If positive-pressure ventilation is required, an initial inflation pressure of 20–25 cm H₂O is adequate for most preterm infants.^{64,65} If a prompt increase in heart rate or chest movement is not obtained, higher pressures may be needed. If continued positive-pressure ventilation is required, PEEP may be beneficial. Continuous positive airway pressure (CPAP) in spontaneously breathing preterm infants following resuscitation may also be beneficial.⁶⁵

Devices

Effective ventilation can be achieved with a flow-inflating, a self-inflating bag or with a T-piece mechanical device designed to regulate pressure.^{66–68} The blow-off valves of self-inflating bags are flow-dependent and pressures generated may exceed the value specified by the manufacturer if compressed vigorously.⁶⁹ Target inflation pressures and long inspiratory times are achieved more consistently in mechanical models when using T-piece devices than when using bags,⁷⁰ although the clinical implications are not clear. More training is required to provide an appropriate pressure using flow-inflating bags compared with self-inflating bags.⁷¹ A self-inflating bag, a flow-inflating bag, or a T-piece mechanical device, all designed to regulate pressure or limit pressure applied to the airway, can be used to ventilate a newborn.

Laryngeal mask airways

A number of studies have shown that laryngeal mask airways (LMAs) can be effectively used at birth to ventilate babies weighing over 2000 g, greater than 33 weeks gestation and apparently needing resuscitation. Case reports suggest that laryngeal masks have been successfully used when intubation has been tried and failed – and occasionally vice-versa. There are few data on smaller or less mature babies.

Recommendation: The laryngeal mask airway can be used in resuscitation of the newborn, particularly if facemask ventilation is unsuccessful or tracheal intubation is unsuccessful or not feasible. The laryngeal mask airway may be considered as an alternative to a facemask for positive-pressure ventilation among newborns weighing more than 2000 g or delivered ≥ 34 weeks gestation. There is limited evidence, however, to evaluate its use for newborns weighing <2000 g or delivered <34 weeks gestation. The laryngeal mask airway may be considered as an alternative to tracheal intubation as a secondary airway for resuscitation among newborns weighing more than 2000 g or delivered ≥ 34 weeks gestation.^{72–74} The laryngeal mask airway has not been evaluated in the setting of meconium-stained fluid, during chest compressions, or for the administration of emergency intra-tracheal medications.

Carbon dioxide detection with face mask or LMA ventilation

Colorimetric exhaled CO₂ detectors have been used during mask ventilation of a few preterm infants in the intensive care unit⁷⁵ and

Table 7.1
Oral tracheal tube lengths by gestation.

Gestation (weeks)	Tracheal tube at lips (cm)
23–24	5.5
25–26	6.0
27–29	6.5
30–32	7.0
33–34	7.5
35–37	8.0
38–40	8.5
41–43	9.0

in the delivery room,⁷⁶ and may help to identify airway obstruction. Neither additional benefit above clinical assessment alone, nor risks attributed to their use have been identified. The use of exhaled CO₂ detectors with other interfaces (e.g. nasal airways, laryngeal masks) during PPV in the delivery room has not been reported.

Confirming tracheal tube placement

Tracheal intubation may be considered at several points during neonatal resuscitation:

- When suctioning to remove meconium or other tracheal blockage is required.
- If bag-mask ventilation is ineffective or prolonged.
- When chest compressions are performed.
- Special circumstances (e.g. congenital diaphragmatic hernia or birth weight below 1000 g).

The use and timing of tracheal intubation will depend on the skill and experience of the available resuscitators. Appropriate tube lengths based on gestation are shown in Table 7.1.⁷⁷

Tracheal tube placement must be assessed visually during intubation, and positioning confirmed. Following tracheal intubation and intermittent positive-pressure, a prompt increase in heart rate is a good indication that the tube is in the tracheobronchial tree.⁷⁸ Exhaled CO₂ detection is effective for confirmation of tracheal tube placement in infants, including VLBW infants^{79–82} and neonatal studies suggest that it confirms tracheal intubation in neonates with a cardiac output more rapidly and more accurately than clinical assessment alone.^{81–83} Failure to detect exhaled CO₂ strongly suggests oesophageal intubation^{79,81} but false negative readings have been reported during cardiac arrest⁷⁹ and in VLBW infants despite models suggesting efficacy.⁸⁴ However, neonatal studies have excluded infants in need of extensive resuscitation. There is no comparative information to recommend any one method for detection of exhaled carbon dioxide in the neonatal population. False positives may occur with colorimetric devices contaminated with adrenaline (epinephrine), surfactant and atropine.⁷⁵

Poor or absent pulmonary blood flow or tracheal obstruction may prevent detection of exhaled CO₂ despite correct tracheal tube placement. Tracheal tube placement is identified correctly in nearly all patients who are not in cardiac arrest;⁸⁰ however, in critically ill infants with poor cardiac output, inability to detect exhaled CO₂ despite correct placement may lead to unnecessary extubation. Other clinical indicators of correct tracheal tube placement include evaluation of condensed humidified gas during exhalation and presence or absence of chest movement, but these have not been evaluated systematically in newborn babies.

Recommendation: Detection of exhaled carbon dioxide in addition to clinical assessment is recommended as the most reliable method to confirm tracheal placement in neonates with spontaneous circulation.

Route and dose of adrenaline (epinephrine)

Despite the widespread use of adrenaline during resuscitation, no placebo controlled clinical trials have evaluated its effectiveness, nor has the ideal dose or route of administration been defined.

Neonatal case series or case reports^{85,86} indicate that adrenaline administered by the tracheal route using a wide range of doses (3–250 µg kg⁻¹) may be associated with return of spontaneous circulation (ROSC) or an increase in heart rate. These case series are limited by inconsistent standards for adrenaline administration and are subject to both selection and reporting bias.

One good quality case series indicates that tracheal adrenaline (10 µg kg⁻¹) is likely to be less effective than the same dose administered intravenously.⁸⁷ This is consistent with evidence extrapolated from neonatal animal models indicating that higher doses (50–100 µg kg⁻¹) of adrenaline may be required when given via the tracheal route to achieve the same blood adrenaline concentrations and haemodynamic response as achieved after intravenous administration.^{88,89} Adult animal models demonstrate that blood concentrations of adrenaline are significantly lower following tracheal compared with intravenous administration^{90,91} and that tracheal doses ranging from 50 to 100 µg kg⁻¹ may be required to achieve ROSC.⁹²

Although it has been widely assumed that adrenaline can be given faster by the tracheal route than by the intravenous route, no clinical trials have evaluated this hypothesis. Two studies have reported cases of inappropriately early use of tracheal adrenaline before airway and breathing are established.^{85,86} One case series describing in-hospital paediatric cardiac arrests suggested that survival was higher among infants who received their first dose of adrenaline by the tracheal route; however, the time required for first dose administration using the tracheal and intravenous routes were not provided.⁹³

Paediatric^{94,95} and newborn animal studies⁹⁶ showed no benefit and a trend toward reduced survival and worse neurological status after high-dose intravenous adrenaline (100 mcg kg⁻¹) during resuscitation. This is in contrast to a single paediatric case series using historic controls that indicated a marked improvement in ROSC using high-dose intravenous adrenaline (100 mcg kg⁻¹). However, a meta-analysis of five adult clinical trials indicates that whilst high-dose intravenous adrenaline may increase ROSC, it offers no benefit in survival to hospital discharge.⁹⁷

Recommendation: If adrenaline is administered, give an intravenous dose 10–30 µg kg⁻¹ as soon as possible. Higher intravenous doses should not be given and may be harmful. If intravenous access is *not* available, then it may be reasonable to try tracheal adrenaline. If adrenaline is administered by the tracheal route, it is likely that a larger dose (50–100 µg kg⁻¹) will be required to achieve a similar effect to the 10 µg kg⁻¹ intravenous dose.

Post-resuscitation care

Babies who have required resuscitation may later deteriorate. Once adequate ventilation and circulation are established, the infant should be maintained in or transferred to an environment in which close monitoring and anticipatory care can be provided.

Glucose

Hypoglycaemia was associated with adverse neurological outcome in a neonatal animal model of asphyxia and resuscitation.⁹⁸ Newborn animals that were hypoglycaemic at the time of an anoxic or hypoxic-ischemic insult had larger areas of cerebral infarction and/or decreased survival compared to controls.^{99,100} One clinical study demonstrated an association between hypoglycaemia and poor neurological outcome following perinatal asphyxia.¹⁰¹ In

adults, children and extremely low-birth-weight infants receiving intensive care, hyperglycaemia has been associated with a worse outcome.^{102–104} However, in paediatric patients, hyperglycaemia after hypoxia–ischaemia does not appear to be harmful,¹⁰⁵ which confirms data from animal studies¹⁰⁶ some of which suggest it may be protective.¹⁰⁷ However, the range of blood glucose concentration that is associated with the least brain injury following asphyxia and resuscitation cannot be defined based on available evidence. Infants who require significant resuscitation should be monitored and treated to maintain glucose in the normal range.

Induced hypothermia

Several randomised, controlled, multi-centre trials of induced hypothermia (33.5–34.5°C) of babies born at more than 36 weeks gestational age, with moderate to severe hypoxic–ischemic encephalopathy have shown that cooling significantly reduced death and neuro-developmental disability at 18 months.^{108–111} Systemic and selective head cooling produced similar results.^{109–113} Modest hypothermia may be associated with bradycardia and elevated blood pressure that do not usually require treatment, but a rapid increase in body temperature may cause hypotension.¹¹⁴ Profound hypothermia (core temperature below 33°C) may cause arrhythmia, bleeding, thrombosis, and sepsis, but studies so far have not reported these complications in infants treated with modest hypothermia.^{109,115}

Newly born infants born at term or near-term with evolving moderate to severe hypoxic–ischemic encephalopathy should, where possible, be offered therapeutic hypothermia. Whole body cooling and selective head cooling are both appropriate strategies. Cooling should be initiated and conducted under clearly defined protocols with treatment in neonatal intensive care facilities and with the capabilities for multidisciplinary care. Treatment should be consistent with the protocols used in the randomised clinical trials (i.e. commence within 6 h of birth, continue for 72 h of birth and re-warm over at least 4 h). Animal data would strongly suggest that the effectiveness of cooling is related to early intervention. There is no evidence in human newborns that cooling is effective if started more than 6 h after birth. Carefully monitor for known adverse effects of cooling – thrombocytopenia and hypotension. All treated infants should be followed longitudinally.

Withholding or discontinuing resuscitation

Mortality and morbidity for newborns varies according to region and to availability of resources.¹¹⁶ Social science studies indicate that parents desire a larger role in decisions to resuscitate and to continue life support in severely compromised babies.¹¹⁷ Opinions vary amongst providers, parents and societies about the balance of benefits and disadvantages of using aggressive therapies in such babies.^{118,119}

Withholding resuscitation

It is possible to identify conditions associated with high mortality and poor outcome, where withholding resuscitation may be considered reasonable, particularly when there has been the opportunity for discussion with parents.^{24,120,121}

A consistent and coordinated approach to individual cases by the obstetric and neonatal teams and the parents is an important goal.²³ Withholding resuscitation and discontinuation of life-sustaining treatment during or following resuscitation are considered by many to be ethically equivalent and clinicians should not be hesitant to withdraw support when the possibility of functional survival is highly unlikely. The following

guidelines must be interpreted according to current regional outcomes.

- Where gestation, birth weight, and/or congenital anomalies are associated with almost certain early death, and unacceptably high morbidity is likely among the rare survivors, resuscitation is not indicated.¹²² Examples from the published literature include: extreme prematurity (gestational age less than 23 weeks and/or birthweight less than 400 g), and anomalies such as anencephaly and confirmed Trisomy 13 or 18.
- Resuscitation is nearly always indicated in conditions associated with a high survival rate and acceptable morbidity. This will generally include babies with gestational age of 25 weeks or above (unless there is evidence of fetal compromise such as intrauterine infection or hypoxia–ischaemia) and those with most congenital malformations.
- In conditions associated with uncertain prognosis, where there is borderline survival and a relatively high rate of morbidity, and where the anticipated burden to the child is high, parental desires regarding resuscitation should be supported.

Withdrawing resuscitation efforts

Data from infants without signs of life from birth, lasting at least 10 min or longer, show either high mortality or severe neuro-developmental disability.^{123,124} If faced with a newly born baby with no detectable heart rate which remains undetectable for 10 min, it is appropriate to then consider stopping resuscitation. The decision to continue resuscitation efforts when the infant has no detectable heart rate for longer than 10 min is often complex and may be influenced by issues such as the presumed aetiology of the arrest, the gestation of the baby, the potential reversibility of the situation, and the parents' previous expressed feelings about acceptable risk of morbidity.

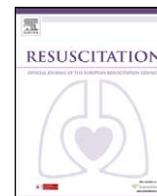
If the heart rate is less than 60 min⁻¹ at birth and persisting after 10 or 15 min the situation is even less clear and a firm recommendation cannot be made.

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European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: Electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution

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8a. Life-threatening electrolyte disorders

Overview

Electrolyte abnormalities can cause cardiac arrhythmias or cardiopulmonary arrest. Life-threatening arrhythmias are associated most commonly with potassium disorders, particularly hyperkalaemia, and less commonly with disorders of serum calcium and magnesium. In some cases therapy for life-threatening electrolyte disorders should start before laboratory results become available.

The electrolyte values for definitions have been chosen as a guide to clinical decision-making. The precise values that trigger treatment decisions will depend on the patient's clinical condition and rate of change of electrolyte values.

There is little or no evidence for the treatment of electrolyte abnormalities during cardiac arrest. Guidance during cardiac arrest is based on the strategies used in the non-arrest patient. There are

no major changes in the treatment of these disorders since Guidelines 2005.¹

Prevention of electrolyte disorders

Identify and treat life-threatening electrolyte abnormalities before cardiac arrest occurs. Remove any precipitating factors (e.g., drugs) and monitor electrolyte values to prevent recurrence of the abnormality. Monitor renal function in patients at risk of electrolyte disorders (e.g., chronic kidney disease, cardiac failure). In haemodialysis patients, review the dialysis prescription regularly to avoid inappropriate electrolyte shifts during treatment.

Potassium disorders

Potassium homeostasis

Extracellular potassium concentration is regulated tightly between 3.5 and 5.0 mmol l⁻¹. A large concentration gradient normally exists between intracellular and extracellular fluid compartments. This potassium gradient across cell membranes contributes to the excitability of nerve and muscle cells, including

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the myocardium. Evaluation of serum potassium must take into consideration the effects of changes in serum pH. When serum pH decreases (acidaemia), serum potassium increases because potassium shifts from the cellular to the vascular space. When serum pH increases (alkalaemia), serum potassium decreases because potassium shifts intracellularly. Anticipate the effects of pH changes on serum potassium during therapy for hyperkalaemia or hypokalaemia.

Hyperkalaemia

This is the most common electrolyte disorder associated with cardiopulmonary arrest. It is usually caused by increased potassium release from cells, impaired excretion by the kidneys or accidental potassium chloride administration.

Definition

There is no universal definition. We have defined hyperkalaemia as a serum potassium concentration higher than 5.5 mmol l^{-1} ; in practice, hyperkalaemia is a continuum. As the potassium concentration increases above this value the risk of adverse events increases and the need for urgent treatment increases. Severe hyperkalaemia has been defined as a serum potassium concentration higher than 6.5 mmol l^{-1} .

Causes

There are several potential causes of hyperkalaemia, including renal failure, drugs (angiotensin converting enzyme inhibitors (ACE-I), angiotensin II receptor antagonists, potassium sparing diuretics, non-steroidal anti-inflammatory drugs (NSAIDs), beta-blockers, trimethoprim), tissue breakdown (rhabdomyolysis, tumour lysis, haemolysis), metabolic acidosis, endocrine disorders (Addison's disease), hyperkalaemic periodic paralysis, or diet (may be sole cause in patients with advanced chronic kidney disease). Abnormal erythrocytes or thrombocytosis may cause a spuriously high potassium concentration.² The risk of hyperkalaemia is even greater when there is a combination of factors such as the concomitant use of ACE-I and NSAIDs or potassium sparing diuretics.

Recognition of hyperkalaemia

Exclude hyperkalaemia in patients with an arrhythmia or cardiac arrest.³ Patients may present with weakness progressing to flaccid paralysis, paraesthesia, or depressed deep tendon reflexes. Alternatively, the clinical picture can be overshadowed by the primary illness causing hyperkalaemia. The first indicator of hyperkalaemia may also be the presence of ECG abnormalities, arrhythmias, cardiopulmonary arrest or sudden death. The effect of hyperkalaemia on the ECG depends on the absolute serum potassium as well as the rate of increase. Most patients will have ECG abnormalities at a serum potassium concentration higher than 6.7 mmol l^{-1} .⁴ The use of a blood gas analyser that measures potassium can reduce delay in recognition.

The ECG changes associated with hyperkalaemia are usually progressive and include:

- first degree heart block (prolonged PR interval) [$>0.2 \text{ s}$];
- flattened or absent P waves;
- tall, peaked (tenting) T waves [T wave larger than R wave in more than 1 lead];
- ST-segment depression;
- S and T wave merging (sine wave pattern);
- widened QRS [$>0.12 \text{ s}$];
- ventricular tachycardia;
- bradycardia;

- cardiac arrest (pulseless electrical activity [PEA], ventricular fibrillation/pulseless ventricular tachycardia [VF/VT], asystole).

Treatment of hyperkalaemia

There are three key treatments for hyperkalaemia⁵:

1. cardiac protection;
2. shifting potassium into cells;
3. removing potassium from the body.

Intravenous calcium salts are not generally indicated in the absence of ECG changes. Monitor effectiveness of treatment, be alert to rebound hyperkalaemia and take steps to prevent recurrence of hyperkalaemia. When hyperkalaemia is strongly suspected, e.g., in the presence of ECG changes, start life-saving treatment even before laboratory results are available. The treatment of hyperkalaemia has been the subject of a Cochrane review.⁶

Patient not in cardiac arrest. Assess ABCDE (Airway, Breathing, Circulation, Disability, Exposure) and correct any abnormalities. Obtain intravenous access, check serum potassium and record an ECG. Treatment is determined according to severity of hyperkalaemia.

Approximate values are provided to guide treatment.

Mild elevation ($5.5\text{--}5.9 \text{ mmol l}^{-1}$):

- Remove potassium from body: potassium exchange resins – calcium resonium 15–30 g OR sodium polystyrene sulfonate (Kayexalate) 15–30 g in 50–100 ml of 20% sorbitol, given either orally or by retention enema (onset in 1–3 h; maximal effect at 6 h).
- Address cause of hyperkalaemia to correct and avoid further rise in serum potassium (e.g., drugs, diet).

Moderate elevation ($6\text{--}6.4 \text{ mmol l}^{-1}$) without ECG changes:

- Shift potassium intracellularly with glucose/insulin: 10 units short-acting insulin and 25 g glucose IV over 15–30 min (onset in 15–30 min; maximal effect at 30–60 min; monitor blood glucose).
- Remove potassium from the body as described above.
- Haemodialysis: consider if oliguric; haemodialysis is more efficient than peritoneal dialysis at removing potassium.

Severe elevation ($\geq 6.5 \text{ mmol l}^{-1}$) without ECG changes. Seek expert help and:

- Use multiple shifting agents.
- Glucose/insulin (see above).
- Salbutamol 5 mg nebulised. Several doses (10–20 mg) may be required (onset in 15–30 min).
- Sodium bicarbonate: 50 mmol IV over 5 min if metabolic acidosis present (onset in 15–30 min). Bicarbonate alone is less effective than glucose plus insulin or nebulised salbutamol; it is best used in conjunction with these medications.^{7,8}
- Use removal strategies above.

Severe elevation ($\geq 6.5 \text{ mmol l}^{-1}$) WITH toxic ECG changes. Seek expert help and:

- Protect the heart first with calcium chloride: 10 ml 10% calcium chloride IV over 2–5 min to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane. This protects the heart by reducing the risk of pulseless VT/VF but does not lower serum potassium (onset in 1–3 min).

- Use multiple shifting agents (see above).
- Use removal strategies.
- Prompt specialist referral is essential.

Patient in cardiac arrest. Modifications to BLS There are no modifications to basic life support in the presence of electrolyte abnormalities.

Modifications to ALS

- Follow the universal algorithm. Hyperkalaemia can be confirmed rapidly using a blood gas analyser if available. Protect the heart first: give 10 ml 10% calcium chloride IV by rapid bolus injection.
- Shift potassium into cells:
 - Glucose/insulin: 10 units short-acting insulin and 25 g glucose IV by rapid injection.
 - Sodium bicarbonate: 50 mmol IV by rapid injection (if severe acidosis or renal failure).
- Remove potassium from body: dialysis: consider this for cardiac arrest induced by hyperkalaemia that is resistant to medical treatment. Several dialysis modes have been used safely and effectively in cardiac arrest, but this may only be available in specialist centres.

Indications for dialysis

Haemodialysis (HD) is the most effective method for removal of potassium from the body. The principle mechanism of action is the diffusion of potassium ions across the membrane down the potassium ion gradient. The typical decline in serum potassium is 1 mmol l^{-1} in the first 60 min, followed by 1 mmol l^{-1} over the next 2 h. The efficacy of HD in decreasing serum potassium concentration can be improved by performing dialysis with a low potassium concentration in the dialysate,⁹ a high blood flow rate¹⁰ or a high dialysate bicarbonate concentration.¹¹

Consider haemodialysis early for hyperkalaemia associated with established renal failure, oliguric acute kidney injury ($<400 \text{ ml day}^{-1}$ urine output) or when there is marked tissue breakdown. Dialysis is also indicated when hyperkalaemia is resistant to medical treatment. Serum potassium frequently rebounds after initial treatment. In unstable patients continuous renal replacement therapy (CRRT) (e.g., continuous veno-veno haemofiltration) is less likely to compromise cardiac output than intermittent haemodialysis. CRRT is now widely available in many intensive care units.

Cardiac arrest in haemodialysis patients

Cardiac arrest is the most common cause of death in haemodialysis patients.¹² Events occurring particularly during haemodialysis treatment, pose several novel considerations.

Initial steps. Call the resuscitation team and seek expert help immediately. Whilst BLS is underway, a trained dialysis nurse should be assigned to the dialysis machine. The conventional practice is to return the patient's blood volume and take off haemodialysis, although this approach is not the most time-efficient.¹³

Defibrillation. A shockable cardiac rhythm (VF/VT) is more common in patients undergoing haemodialysis^{14,15} than in the general population.^{16,17}

The safest method to deliver a shock during dialysis requires further study. Most haemodialysis machine manufacturers recommend disconnection from the dialysis equipment prior to defibrillation.¹⁸ An alternative and rapid disconnect technique for haemodialysis has been described. Disconnection during continuous venovenous haemofiltration is not required.¹³ The use of

automated external defibrillators in dialysis centres can facilitate early defibrillation.¹⁹

Vascular access. In life-threatening circumstances and cardiac arrest, vascular access used for haemodialysis can be used to give drugs.¹³

Potentially reversible causes. All of the standard reversible causes (4 Hs and 4 Ts) apply to dialysis patients. Electrolyte disorders, particularly hyperkalaemia, and fluid overload (e.g., pulmonary oedema) are most common causes.

Hypokalaemia

Hypokalaemia is common in hospital patients.²⁰ Hypokalaemia increases the incidence of arrhythmias, particularly in patients with pre-existing heart disease and in those treated with digoxin.

Definition

Hypokalaemia is defined as a serum potassium $<3.5 \text{ mmol l}^{-1}$. Severe hypokalaemia is defined as a $\text{K}^+ <2.5 \text{ mmol l}^{-1}$ and may be associated with symptoms.

Causes

Causes of hypokalaemia include gastrointestinal loss (diarrhoea), drugs (diuretics, laxatives, steroids), renal losses (renal tubular disorders, diabetes insipidus, dialysis), endocrine disorders (Cushing's Syndrome, hyperaldosteronism), metabolic alkalosis, magnesium depletion, and poor dietary intake. Treatment strategies used for hyperkalaemia may also induce hypokalaemia.

Recognition of hypokalaemia

Exclude hypokalaemia in every patient with an arrhythmia or cardiac arrest. In dialysis patients, hypokalaemia occurs commonly at the end of a haemodialysis session or during treatment with peritoneal dialysis.

As serum potassium concentration decreases, the nerves and muscles are predominantly affected causing fatigue, weakness, leg cramps, constipation. In severe cases ($\text{K}^+ <2.5 \text{ mmol l}^{-1}$), rhabdomyolysis, ascending paralysis and respiratory difficulties may occur.

ECG features of hypokalaemia are:

- U waves;
- T wave flattening;
- ST-segment changes;
- arrhythmias, especially if patient is taking digoxin;
- cardiopulmonary arrest (PEA, pulseless VT/VF, asystole).

Treatment

This depends on the severity of hypokalaemia and the presence of symptoms and ECG abnormalities. Gradual replacement of potassium is preferable, but in an emergency, intravenous potassium is required. The maximum recommended IV dose of potassium is 20 mmol h^{-1} , but more rapid infusion (e.g., 2 mmol min^{-1} for 10 min, followed by 10 mmol over 5–10 min) is indicated for unstable arrhythmias when cardiac arrest is imminent. Continuous ECG monitoring is essential during IV infusion and the dose should be titrated after repeated sampling of serum potassium levels.

Many patients who are potassium deficient are also deficient in magnesium. Magnesium is important for potassium uptake and for the maintenance of intracellular potassium levels, particularly in the myocardium. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases of hypokalaemia.²¹

Table 8.1

Calcium and magnesium disorders with associated clinical presentation, ECG manifestations and recommended treatment.

Disorder	Causes	Presentation	ECG	Treatment
Hypercalcaemia [Calcium] > 2.6 mmol l ⁻¹	Primary or tertiary hyperparathyroidism	Confusion	Short QT interval	Fluid replacement IV
	Malignancy Sarcoidosis Drugs	Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Prolonged QRS Interval Flat T waves AV-block Cardiac arrest	Furosemide 1 mg kg ⁻¹ IV Hydrocortisone 200–300 mg IV Pamidronate 30–90 mg IV Treat underlying cause
Hypocalcaemia [Calcium] < 2.1 mmol l ⁻¹	Chronic renal failure Acute pancreatitis	Paraesthesia Tetany	Prolonged QT interval T wave inversion	Calcium chloride 10% 10–40 ml Magnesium sulphate 50% 4–8 mmol (if necessary)
	Calcium channel blocker overdose	Seizures	Heart block	
	Toxic shock syndrome Rhabdomyolysis Tumour lysis syndrome	AV-block Cardiac arrest	Cardiac arrest	
Hypermagnesaemia [Magnesium] > 1.1 mmol l ⁻¹	Renal failure	Confusion	Prolonged PR and QT intervals	Consider treatment when [Magnesium] > 1.75 mmol l ⁻¹
	Iatrogenic	Weakness Respiratory depression	T wave peaking AV-block	Calcium chloride 10% 5–10 ml repeated if necessary Ventilatory support if necessary
		AV-block Cardiac arrest	Cardiac arrest	Saline diuresis – 0.9% saline with furosemide 1 mg kg ⁻¹ IV Haemodialysis
Hypomagnesaemia [Magnesium] < 0.6 mmol l ⁻¹	GI loss	Tremor	Prolonged PR and QT Intervals	Severe or symptomatic:
	Polyuria	Ataxia	ST-segment depression	2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 15 min.
	Starvation Alcoholism	Nystagmus Seizures	T-wave inversion Flattened P waves	Torsade de pointes: 2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 1–2 min.
	Malabsorption	Arrhythmias – torsade de pointes Cardiac arrest	Increased QRS duration Torsade de pointes	Seizure: 2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 10 min.

Calcium and magnesium disorders

The recognition and management of calcium and magnesium disorders is summarised in Table 8.1.

Summary

Electrolyte abnormalities are among the most common causes of cardiac arrhythmias. Of all the electrolyte abnormalities, hyperkalaemia is most rapidly fatal. A high degree of clinical suspicion and aggressive treatment of underlying electrolyte abnormalities can prevent many patients from progressing to cardiac arrest.

8b. Poisoning

General considerations

Poisoning rarely causes cardiac arrest, but is a leading cause of death in victims younger than 40 years of age.²² Evidence for treatment consists primarily of small case-series, animal studies and case reports. Poisoning by therapeutic or recreational drugs and by household products are the main reasons for hospital admission and poison centre calls. Inappropriate drug dosing, drug interactions and other medication errors can also cause harm. Accidental poisoning is commonest in children. Homicidal poisoning is uncommon. Industrial accidents, warfare or terrorism can also cause exposure to harmful substances.

Prevention of cardiac arrest

Assess using the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach. Airway obstruction and respiratory arrest secondary to a decreased conscious level is a common cause of death after self-poisoning.²³ Pulmonary aspiration of gastric contents can occur after poisoning with central nervous system depressants. Early tracheal intubation of unconscious patients by a trained person decreases the risk of aspiration. Drug-induced hypotension usually responds to fluid infusion, but occasionally vasopressor support (e.g., noradrenaline infusion) is required. A long period of coma in a single position can cause pressure sores and rhabdomyolysis. Measure electrolytes (particularly potassium), blood glucose and arterial blood gases. Monitor temperature because thermoregulation is impaired. Both hypothermia and hyperthermia (hyperpyrexia) can occur after overdose of some drugs. Retain samples of blood and urine for analysis. Patients with severe poisoning should be cared for in a critical-care setting.

Interventions such as decontamination, enhanced elimination and antidotes may be indicated and are usually second line interventions.²⁴ Alcohol excess is often associated with self-poisoning.

Modifications to BLS/ALS

- Have a high index of personal safety where there is a suspicious cause or unexpected cardiac arrest. This is especially so when more than one casualty collapses simultaneously.

- Avoid mouth-to-mouth ventilation in the presence of chemicals such as cyanide, hydrogen sulphide, corrosives and organophosphates.
- Treat life-threatening tachyarrhythmias with cardioversion according to the peri-arrest arrhythmia guidelines (see Section 4, Advanced Life Support).^{24a} This includes correction of electrolyte and acid-base abnormalities.
- Try to identify the poison(s). Relatives, friends and ambulance crews can provide useful information. Examination of the patient may reveal diagnostic clues such as odours, needle marks, pupil abnormalities, and signs of corrosion in the mouth.
- Measure the patient's temperature because hypo- or hyperthermia may occur after drug overdose (see Sections 8d and 8e).
- Be prepared to continue resuscitation for a prolonged period, particularly in young patients, as the poison may be metabolized or excreted during extended life support measures.
- Alternative approaches which may be effective in severely poisoned patients include: higher doses of medication than in standard protocols; non-standard drug therapies; prolonged CPR.
- Consult regional or national poisons centres for information on treatment of the poisoned patient. The International Programme on Chemical Safety (IPCS) lists poison centres on its website: <http://www.who.int/ipcs/poisons/centre/en/>.
- Online databases for information on toxicology and hazardous chemicals: (<http://toxnet.nlm.nih.gov/>).

Specific therapeutic measures

There are few specific therapeutic measures for poisoning that are useful immediately and improve outcomes.^{25–29}

Therapeutic measures include decontamination, multiple-dose activated charcoal, enhancing elimination and the use of specific antidotes. Many of these interventions should be used only based on expert advice. For up-to-date guidance in severe or uncommon poisonings, seek advice from a poisons centre.

Gastrointestinal decontamination

Activated charcoal adsorbs most drugs. Its benefit decreases over time after ingestion. There is no evidence that treatment with activated charcoal improves clinical outcome. Consider giving a single dose of activated charcoal to patients who have ingested a potentially toxic amount of poison (known to be adsorbed by activated charcoal) up to 1 h previously.³⁰ Give it only to patients with an intact or protected airway.

Multiple-dose activated charcoal significantly increases drug elimination, but no controlled study in poisoned patients has shown a reduction in morbidity and mortality and should only be used following expert advice. There is little evidence to support the use of gastric lavage. It should only be considered within 1 h of ingestion of a potentially life-threatening amount of a poison. Even then, clinical benefit has not been confirmed in controlled studies. Gastric lavage is contraindicated if the airway is not protected and if a hydrocarbon with high aspiration potential or a corrosive substance has been ingested.^{27,28}

Volunteer studies show substantial decreases in the bioavailability of ingested drugs but no controlled clinical trials show that whole-bowel irrigation improves the outcome of the poisoned patient. Based on volunteer studies, whole-bowel irrigation may be considered for potentially toxic ingestions of sustained-release or enteric-coated drugs. Its use for the removal of iron, lead, zinc, or packets of illicit drugs is a theoretical option. Whole-bowel irrigation is contraindicated in patients with bowel obstruction, perforation, ileus, and haemodynamic instability.³¹

Laxatives (cathartics) or emetics (e.g., ipecacuanha) have no role in the management of the acutely poisoned patient and are not recommended.^{26,32,33}

Enhancing elimination

Urine alkalisation (urine pH of 7.5 or higher) by intravenous sodium bicarbonate infusion is a first line treatment for moderate-to-severe salicylate poisoning in patients who do not need haemodialysis.²⁵ Urine alkalisation with high urine flow (approximately 600 ml h⁻¹) should also be considered in patients with severe poisoning by the herbicides 2,4-dichlorophenoxyacetic acid and methylchlorophenoxypropionic acid (mecoprop). Hypokalaemia is the most common complication of alkalaemia.

Haemodialysis or haemoperfusion should be considered in specific life-threatening poisonings only. Haemodialysis removes drugs or metabolites that are water soluble, have a low volume of distribution and low plasma protein binding. Haemoperfusion can remove substances that have a high degree of plasma protein binding.

Specific poisonings

These guidelines address only some causes of cardiorespiratory arrest due to acute poisoning.

Benzodiazepines

Patients at risk of cardiac arrest

Overdose of benzodiazepines can cause loss of consciousness, respiratory depression and hypotension. Flumazenil, a competitive antagonist of benzodiazepines, should only be used only for reversal of sedation caused by a single ingestion of any of the benzodiazepines and when there is no history or risk of seizures. Reversal of benzodiazepine intoxication with flumazenil can be associated with significant toxicity (seizure, arrhythmia, hypotension, and withdrawal syndrome) in patients with benzodiazepine dependence or co-ingestion of proconvulsant medications such as tricyclic antidepressants.^{34–36} The routine use of flumazenil in the comatose overdose patient is not recommended.

Modifications to BLS/ALS

There are no specific modifications required for cardiac arrest caused by benzodiazepines.^{36–40}

Opioids

Opioid poisoning causes respiratory depression followed by respiratory insufficiency or respiratory arrest. The respiratory effects of opioids are reversed rapidly by the opiate antagonist naloxone.

Patients at risk of cardiac arrest

In severe respiratory depression caused by opioids, there are fewer adverse events when airway opening, oxygen administration and ventilation are carried out before giving naloxone.^{41–47} The use of naloxone can prevent the need for intubation. The preferred route for giving naloxone depends on the skills of the rescuer: IV, intramuscular (IM), subcutaneous (SC) and intranasal (IN) routes can be used. The non-IV routes can be quicker because time is saved in not having to establish IV access, which can be extremely difficult in an IV drug abuser. The initial doses of naloxone are 400 µg IV,⁴³ 800 µg IM, 800 µg SC,⁴³ or 2 mg IN.^{48,49} Large opioid overdoses may require titration of naloxone to a total dose of 6–10 mg. The duration of action of naloxone is approximately 45–70 min, but respiratory depression can persist for 4–5 h after opioid overdose.

Thus, the clinical effects of naloxone may not last as long as those of a significant opioid overdose. Titrate the dose until the victim is breathing adequately and has protective airway reflexes.

Acute withdrawal from opioids produces a state of sympathetic excess and may cause complications such as pulmonary oedema, ventricular arrhythmia and severe agitation. Use naloxone reversal of opioid intoxication with caution in patients suspected of opioid dependence.

Modifications for ALS

There are no studies supporting the use of naloxone once cardiac arrest associated with opioid toxicity has occurred. Cardiac arrest is usually secondary to a respiratory arrest and associated with severe brain hypoxia. Prognosis is poor.⁴² Giving naloxone is unlikely to be harmful. Once cardiac arrest has occurred, follow standard resuscitation protocols.

Tricyclic antidepressants

This section addresses both tricyclic and related cyclic drugs (e.g., amitriptyline, desipramine, imipramine, nortriptyline, doxepin, and clomipramine). Self-poisoning with tricyclic antidepressants is common and can cause hypotension, seizures, coma and life-threatening arrhythmias. Cardiac toxicity mediated by anticholinergic and Na⁺ channel-blocking effects can produce a wide complex tachycardia (VT). Hypotension is exacerbated by alpha-1 receptor blockade. Anticholinergic effects include mydriasis, fever, dry skin, delirium, tachycardia, ileus, and urinary retention. Most life-threatening problems occur within the first 6 h after ingestion.^{50–52}

Patient at risk of cardiac arrest

A widening QRS complex (>100 ms) and right axis deviation indicates a greater risk of arrhythmias.^{53–55} Sodium bicarbonate should be considered for the treatment of tricyclic-induced ventricular conduction abnormalities.^{56–63} While no study has investigated the optimal target arterial pH with bicarbonate therapy, a pH of 7.45–7.55 has been commonly accepted and seems reasonable.

Intravenous lipid infusions in experimental models of tricyclic toxicity have suggested benefit but there are few human data.^{64,65} Anti-tricyclic antibodies have also been beneficial in experimental models of tricyclic cardiotoxicity.^{66–71} One small human study⁷² provided evidence of safety but clinical benefit has not been shown.

Modifications to BLS/ALS

There are no randomised controlled trials evaluating conventional versus alternative treatments for cardiac arrest caused by tricyclic toxicity. One small case-series of cardiac arrest patients, showed improvement with the use of sodium bicarbonate.⁷³

Cocaine

Sympathetic overstimulation associated with cocaine toxicity can cause agitation, tachycardia, hypertensive crisis, hyperthermia and coronary vasoconstriction causing myocardial ischaemia with angina.

Patients at risk of cardiac arrest

In patients with severe cardiovascular toxicity, alpha blockers (phentolamine),⁷⁴ benzodiazepines (lorazepam, diazepam),^{75,76} calcium channel blockers (verapamil),⁷⁷ morphine,⁷⁸ and sublingual nitroglycerine^{79,80} may be used as needed to control hypertension, tachycardia, myocardial ischaemia and agitation. The evidence for or against the use of beta-blocker drugs,^{81–84} including those beta-blockers with alpha blocking properties (carvedilol

and labetalol),^{85–87} is limited. The best choice of anti-arrhythmic drug for the treatment of cocaine-induced tachyarrhythmias is not known.

Modifications to BLS/ALS

If cardiac arrest occurs, follow standard resuscitation guidelines.⁸⁸

Local anaesthetics

Systemic toxicity of local anaesthetics involves the central nervous system, the cardiovascular system. Severe agitation, loss of consciousness, with or without tonic-clonic convulsions, sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias can all occur. Toxicity can be potentiated in pregnancy, extremes of age, or hypoxaemia. Toxicity typically occurs in the setting of regional anaesthesia, when a bolus of local anaesthetic inadvertently enters an artery or vein.

Patients at risk of cardiac arrest

Evidence for specific treatment is limited to case reports involving cardiac arrest and severe cardiovascular toxicity and animal studies. Patients with both cardiovascular collapse and cardiac arrest attributable to local anaesthetic toxicity may benefit from treatment with intravenous 20% lipid emulsion in addition to standard advanced life support.^{89–103} Give an initial intravenous bolus of 20% lipid emulsion followed by an infusion at 15 ml kg⁻¹ h⁻¹. Give up to three bolus doses of lipid at 5-min intervals and continue the infusion until the patient is stable or has received up to a maximum of 12 ml kg⁻¹ of lipid emulsion.¹⁰⁴

Modifications to BLS/ALS

Standard cardiac arrests drugs (e.g., adrenaline) should be given according to standard guidelines, although animal studies provide inconsistent evidence for their role in local anaesthetic toxicity.^{100,103,105–107}

Beta-blockers

Beta-blocker toxicity causes bradyarrhythmias and negative inotropic effects that are difficult to treat, and can lead to cardiac arrest.

Patients at risk of cardiac arrest

Evidence for treatment is based on case reports and animal studies. Improvement has been reported with glucagon (50–150 µg kg⁻¹),^{108–121} high-dose insulin and glucose,^{122–124} phosphodiesterase inhibitors,^{125,126} calcium salts,¹²⁷ extracorporeal and intra-aortic balloon pump support,^{128–130} and calcium salts.¹³¹

Calcium channel blockers

Calcium channel blocker overdose is emerging as a common cause of prescription drug poisoning deaths.^{22,132} Overdose of short-acting drugs can rapidly progress to cardiac arrest. Overdose by sustained-release formulations can result in delayed onset of arrhythmias, shock, and sudden cardiac collapse. Asymptomatic patients are unlikely to develop symptoms if the interval between the ingestion and the call is greater than 6 h for immediate-release products, 18 h for modified-release products other than verapamil, and 24 h for modified-release verapamil.

Patients at risk of cardiac arrest

Intensive cardiovascular support is needed for managing a massive calcium channel blocker overdose. While calcium chlo-

ride in high doses can overcome some of the adverse effects, it rarely restores normal cardiovascular status. Haemodynamic instability may respond to high doses of insulin given with glucose supplementation and electrolyte monitoring in addition to standard treatments including fluids and inotropic drugs.^{133–148} Other potentially useful treatments include glucagon, vasopressin and phosphodiesterase inhibitors.^{139,149}

Digoxin

Although cases of digoxin poisoning are fewer than those involving calcium channel and beta-blockers, the mortality rate from digoxin is far greater. Other drugs including calcium channel blockers and amiodarone can also cause plasma concentrations of digoxin to rise. Atrioventricular conduction abnormalities and ventricular hyperexcitability due to digoxin toxicity can lead to severe arrhythmias and cardiac arrest.

Patients at risk of cardiac arrest

Standard resuscitation measures and specific antidote therapy with digoxin-specific antibody fragments should be used if there are arrhythmias associated with haemodynamic instability.^{150–163} Antibody-specific therapy may also be effective in poisoning from plants as well as Chinese herbal medications containing digitalis glycosides.^{150,164,165} Digoxin-specific antibody fragments interfere with digoxin immunoassay measurements and can lead to overestimation of plasma digoxin concentrations.

Cyanide

Cyanide is generally considered to be a rare cause of acute poisoning; however, cyanide exposure occurs relatively frequently in patients with smoke inhalation from residential or industrial fires. Its main toxicity results from inactivation of cytochrome oxidase (at cytochrome a3), thus uncoupling mitochondrial oxidative phosphorylation and inhibiting cellular respiration, even in the presence of adequate oxygen supply. Tissues with the highest oxygen needs (brain and heart) are the most severely affected by acute cyanide poisoning.

Patients at risk of cardiac arrest

Patients with severe cardiovascular toxicity (cardiac arrest, cardiovascular instability, metabolic acidosis, or altered mental status) caused by known or suspected cyanide poisoning should receive cyanide antidote therapy in addition to standard resuscitation, including oxygen. Initial therapy should include a cyanide scavenger (either intravenous hydroxocobalamin or a nitrite – i.e., intravenous sodium nitrite and/or inhaled amyl nitrite), followed as soon as possible by intravenous sodium thiosulphate.^{166–175} Hydroxocobalamin and nitrites are equally effective but hydroxocobalamin may be safer because it does not cause methaemoglobin formation or hypotension.

Modifications to BLS/ALS

In case of cardiac arrest caused by cyanide, standard ALS treatment will fail to restore spontaneous circulation as long as cellular respiration is blocked. Antidote treatment is needed for reactivation of cytochrome oxidase.

Carbon monoxide

Carbon monoxide poisoning is common. There were about 25,000 carbon monoxide related hospital admissions reported in the US in 2005.¹⁷⁶ Patients who develop cardiac arrest caused by carbon monoxide rarely survive to hospital discharge, even if

return of spontaneous circulation is achieved; however, hyperbaric oxygen therapy may be considered in these patients as it may reduce the risk of developing persistent or delayed neurological injury.^{177–185} The risks inherent in transporting critically ill post-arrest patients to a hyperbaric facility may be significant, and must be weighed against the possibility of benefit on a case-by-case basis. Patients who develop myocardial injury caused by carbon monoxide have an increased risk of cardiac and all-cause mortality lasting at least 7 years after the event; it is reasonable to recommend cardiology follow-up for these patients.^{186,187}

8c. Drowning

Overview

Drowning is a common cause of accidental death in Europe. After drowning the duration of hypoxia is the most critical factor in determining the victim's outcome; therefore, oxygenation, ventilation, and perfusion should be restored as rapidly as possible. Immediate resuscitation at the scene is essential for survival and neurological recovery after a drowning incident. This will require provision of CPR by a bystander and immediate activation of the EMS system. Victims who have spontaneous circulation and breathing when they reach hospital usually recover with good outcomes. Research into drowning is limited in comparison with primary cardiac arrest and there is a need for further research in this area.¹⁸⁸ These guidelines are intended for healthcare professionals and certain groups of lay responders that have a special interest in the care of the drowning victim, e.g., lifeguards.

Epidemiology

The World Health Organization (WHO) estimates that, worldwide, drowning accounts for approximately 450,000 deaths each year. A further 1.3 million disability-adjusted life-years are lost each year as a result of premature death or disability from drowning¹⁸⁹; 97% of deaths from drowning occur in low- and middle-income countries.¹⁸⁹ In 2006 there were 312 accidental deaths from drowning in the United Kingdom¹⁹⁰ and 3582 in the United States,¹⁹¹ yielding an annual incidence of drowning of 0.56 and 1.2 per 100,000 population, respectively.¹⁹² Death from drowning is more common in young males, and is the leading cause of accidental death in Europe in this group.¹⁸⁹ Factors associated with drowning (e.g., suicide, traffic accidents, alcohol and drug abuse) varies between countries.¹⁹³

Definitions, classifications and reporting

Over 30 different terms have been used to describe the process and outcome from submersion- and immersion-related incidents.¹⁹⁴ The International Liaison Committee on Resuscitation (ILCOR) defines drowning as “a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium. Implicit in this definition is that a liquid/air interface is present at the entrance of the victim's airway, preventing the victim from breathing air. The victim may live or die after this process, but whatever the outcome, he or she has been involved in a drowning incident”.¹⁹⁵ Immersion means to be covered in water or other fluid. For drowning to occur, usually at least the face and airway must be immersed. Submersion implies that the entire body, including the airway, is under the water or other fluid.

ILCOR recommends that the following terms, previously used, should no longer be used: dry and wet drowning, active and passive drowning, silent drowning, secondary drowning and drowned versus near-drowned.¹⁹⁵ The Utstein drowning style should be used

when reporting outcomes from drowning incidents to improve consistency in information between studies.¹⁹⁵

Pathophysiology

The pathophysiology of drowning has been described in detail.^{195,196} In brief, after submersion, the victim initially breathes before developing laryngospasm. During this time the victim frequently swallows large quantities of water. As breath holding/laryngospasm continues, hypoxia and hypercapnia develops. Eventually these reflexes abate and the victim aspirates water into their lungs leading to worsening hypoxaemia. Without rescue and restoration of ventilation the victim will become bradycardic before sustaining a cardiac arrest. The key feature to note in the pathophysiology of drowning is that cardiac arrest occurs as a consequence of hypoxia and correction of hypoxaemia is critical to obtaining a return of spontaneous circulation.

Treatment

Treatment of a drowning victim involves four distinct but inter-related phases. These comprise (i) aquatic rescue, (ii) basic life support, (iii) advanced life support, and (iv) post-resuscitation care. Rescue and resuscitation of the drowning victim almost always involves a multi-professional team approach. The initial rescue from the water is usually undertaken either by bystanders or those with a duty to respond such as trained lifeguards or lifeboat operators. Basic life support is often provided by the initial responders before arrival of the emergency medical services. Resuscitation frequently continues into hospital where, if return of spontaneous circulation is achieved, transfer to critical care often follows. Drowning incidents vary in their complexity from an incident involving a single victim to one that involves several or multiple victims. The emergency response will vary according to the number of victims involved and available resources. If the number of victims outweighs the available resources then a system of triage to determine who to prioritise for treatment is likely to be necessary. The remainder of this section will focus on the management of the individual drowning victim where there are sufficient resources available.

Basic life support

Aquatic rescue and recovery from the water. Always be aware of personal safety and minimize the danger to yourself and the victim at all times. Whenever possible, attempt to save the drowning victim without entry into the water. Talking to the victim, reaching with a rescue aid (e.g., stick or clothing), or throwing a rope or buoyant rescue aid may be effective if the victim is close to dry land. Alternatively, use a boat or other water vehicle to assist with the rescue. Avoid entry into the water whenever possible. If entry into the water is essential, take a buoyant rescue aid or flotation device.¹⁹⁷ It is safer to enter the water with two rescuers than alone. Never dive head first in the water when attempting a rescue. You may lose visual contact with the victim and run the risk of a spinal injury.

Remove all drowning victims from the water by the fastest and safest means available and resuscitate as quickly as possible. The incidence of cervical spine injury in drowning victims is very low (approximately 0.5%).¹⁹⁸ Spinal immobilisation can be difficult to perform in the water and can delay removal from the water and adequate resuscitation of the victim. Poorly applied cervical collars can also cause airway obstruction in unconscious patients.¹⁹⁹ Cervical spine immobilisation is not indicated unless signs of severe injury are apparent or the history is consistent with the possibility of severe injury.²⁰⁰ These circumstances include a history of diving,

water-slide use, signs of trauma or signs of alcohol intoxication. If the victim is pulseless and apnoeic remove them from the water as quickly as possible (even if a back support device is not available), while attempting to limit neck flexion and extension.

Rescue breathing. The first and most important treatment for the drowning victim is alleviation of hypoxaemia. Prompt initiation of rescue breathing or positive pressure ventilation increases survival.^{201–204} If possible supplement rescue breaths/ventilations with oxygen.²⁰⁵ Give five initial ventilations/rescue breaths as soon as possible.

Rescue breathing can be initiated whilst the victim is still in shallow water provided the safety of the rescuer is not compromised. It is likely to be difficult to pinch the victim's nose, so mouth-to-nose ventilation may be used as an alternative to mouth-to-mouth ventilation.

If the victim is in deep water, open their airway and if there is no spontaneous breathing start in-water rescue breathing if trained to do so. In-water resuscitation is possible,²⁰⁶ but should ideally be performed with the support of a buoyant rescue aid.²⁰⁷ Give 10–15 rescue breaths over approximately 1 min.²⁰⁷ If normal breathing does not start spontaneously, and the victim is <5 min from land, continue rescue breaths while towing. If more than an estimated 5 min from land, give further rescue breaths over 1 min, then bring the victim to land as quickly as possible without further attempts at ventilation.²⁰⁷

Chest compression. The victim should be placed on a firm surface before starting chest compressions as compressions are ineffective in the water.^{208,209} Confirm the victim is unresponsive and not breathing normally and then give 30 chest compressions. Continue CPR in a ratio of 30 compressions to 2 ventilations. Most drowning victims will have sustained cardiac arrest secondary to hypoxia. In these patients, compression-only CPR is likely to be less effective and should be avoided.

Automated external defibrillation. Once CPR is in progress, if an AED is available, dry the victim's chest, attach the AED pads and turn the AED on. Deliver shocks according to the AED prompts.

Regurgitation during resuscitation. Although rescue breathing is difficult to perform perfectly on a drowning victim because of the need for very high inflation pressures or the presence of fluid in the airway, every attempt should be made to continue ventilation until advanced life support providers arrive. Regurgitation of stomach contents and swallowed/inhaled water is common during resuscitation from drowning.²¹⁰ If this prevents ventilation completely, turn the victim on their side and remove the regurgitated material using directed suction if possible. Care should be taken if spinal injury is suspected but this should not prevent or delay life-saving interventions such as airway opening, ventilations and chest compressions. Abdominal thrusts can cause regurgitation of gastric contents and other life-threatening injuries and should not be used.²¹¹

Advanced life support

Airway and breathing. Give high-flow oxygen, ideally through an oxygen mask with reservoir bag, during the initial assessment of the spontaneously breathing drowning victim.²⁰⁵ Consider non-invasive ventilation or continuous positive airway pressure if the victim fails to respond to treatment with high-flow oxygen.²¹² Use pulse oximetry and arterial blood gas analysis to titrate the concentration of inspired oxygen. Consider early tracheal intubation and controlled ventilation for victims who fail to respond to these initial measures or who have a reduced level of consciousness. Take

care to ensure optimal preoxygenation before intubation. Use a rapid-sequence induction with cricoid pressure to reduce the risk of aspiration.²¹³ Pulmonary oedema fluid may pour from the airway and may need suctioning to enable a view of the larynx.

After the tracheal tube is confirmed in position, titrate the inspired oxygen concentration to achieve an SaO₂ of 94–98%.²⁰⁵ Set positive end-expiratory pressure (PEEP) to at least 5–10 cm H₂O, however higher PEEP levels (15–20 cm H₂O) may be required if the patients is severely hypoxaemic.²¹⁴

In the event of cardiopulmonary arrest protect the airway of the victim early in the resuscitation attempt, ideally with a cuffed tracheal tube – reduced pulmonary compliance requiring high inflation pressures may limit the use of a supraglottic airway device.

Circulation and defibrillation. Differentiating respiratory from cardiac arrest is particularly important in the drowning victim. Delaying the initiation of chest compressions if the victim is in cardiac arrest will reduce survival.

The typical post-arrest gasping is very difficult to distinguish from the initial respiratory efforts of a spontaneous recovering drowning victim. Palpation of the pulse as the sole indicator of the presence or absence of cardiac arrest is unreliable.²¹⁵ When available additional diagnostic information should be obtained from other monitoring modalities such as ECG trace, end-tidal CO₂, and echocardiography to confirm the diagnosis of cardiac arrest.

If the victim is in cardiac arrest, follow standard advanced life support protocols. If the victims core body temperature is less than 30 °C, limit defibrillation attempts to three, and withhold IV drugs until the core body temperature increases above 30 °C (see Section 8d).

During prolonged immersion, victims may become hypovolaemic from the hydrostatic pressure of the water on the body. Give IV fluid to correct hypovolaemia. After return of spontaneous circulation, use haemodynamic monitoring to guide fluid resuscitation.

Discontinuing resuscitation efforts

Making a decision to discontinue resuscitation efforts on a victim of drowning is notoriously difficult. No single factor can accurately predict good or poor survival with 100% certainty. Decisions made in the field frequently prove later to have been incorrect.²¹⁶ Continue resuscitation unless there is clear evidence that such attempts are futile (e.g., massive traumatic injuries, *rigor mortis*, putrefaction etc), or timely evacuation to a medical facility is not possible. Neurologically intact survival has been reported in several victims submerged for greater than 60 min however these rare case reports almost invariably occur in children submerged in ice-cold water.^{217,218}

Post-resuscitation care

Salt versus fresh water. Much attention has focused in the past on differences between salt-water and fresh-water drowning. Extensive data from animal studies and human case-series have shown that, irrespective of the tonicity of the inhaled fluid, the predominant pathophysiological process is hypoxaemia, driven by surfactant wash-out and dysfunction, alveolar collapse, atelectasis, and intrapulmonary shunting. Small differences in electrolyte disturbance are rarely of any clinical relevance and do not usually require treatment.

Lung injury. Victims of drowning are at risk of developing acute respiratory distress syndrome (ARDS) after submersion.²¹⁹ Although there are no randomised controlled trials undertaken specifically in this population of patients it seems reasonable to include strategies such as protective ventilation that have been

shown to improve survival in patients with ARDS.²²⁰ The severity of lung injury varies from a mild self-limiting illness to refractory hypoxaemia. In severe cases extracorporeal membrane oxygenation has been used with some success.^{221,222} The clinical and cost effectiveness of these interventions has not been formally tested in randomised controlled trials.

Pneumonia is common after drowning. Prophylactic antibiotics have not been shown to be of benefit,²²³ although they may be considered after submersion in grossly contaminated water such as sewage. Give broad-spectrum antibiotics if signs of infection develop subsequently.^{200,224}

Hypothermia after drowning. Victims of submersion may develop primary or secondary hypothermia. If the submersion occurs in icy water (<5 °C or 41 °F), hypothermia may develop rapidly and provide some protection against hypoxia. Such effects, however, have typically been reported after submersion of children in ice-cold water.¹⁸⁹ Hypothermia may also develop as a secondary complication of the submersion and subsequent heat loss through evaporation during attempted resuscitation (see Section 8d).

Case reports describing patients with severe accidental hypothermia have shown that survival is possible after either passive or active warming.²⁰⁰ In contrast, there is evidence of benefit from induced hypothermia for comatose victims resuscitated from pre-hospital cardiac arrests.^{225,226} To date, there is no convincing evidence to guide therapy in this patient group. A pragmatic approach might be to consider rewarming until a core temperature of 32–34 °C is achieved, taking care to avoid hyperthermia (>37 °C) during the subsequent period of intensive care (International Life Saving Federation, 2003).

Other supportive care. Attempts have been made to improve neurological outcome following drowning with the use of barbiturates, intracranial pressure (ICP) monitoring, and steroids. None of these interventions has been shown to alter outcome. In fact, signs of intracranial hypertension serve as a symptom of significant neurological hypoxic injury, and there is no evidence that attempts to alter the ICP will affect outcome.²⁰⁰

Follow-up. Cardiac arrhythmias may cause rapid loss of consciousness leading to drowning if the victim is in water at the time. Take a careful history in survivors of a drowning incident to identify features suggestive of arrhythmic syncope. Symptoms may include syncope (whilst supine position, during exercise, with brief prodromal symptoms, repetitive episodes or associated with palpitations), seizures or a family history of sudden death. The absence of structural heart disease at post-mortem examination does not rule the possibility of sudden cardiac death. Post-mortem genetic analysis has proved helpful in these situations and should be considered if there is uncertainty over the cause of a drowning death.^{227–229}

8d. Accidental hypothermia

Definition

Accidental hypothermia exists when the body core temperature unintentionally drops below 35 °C. Hypothermia can be classified arbitrarily as mild (35–32 °C), moderate (32–28 °C) or severe (less than 28 °C).²³⁰ The Swiss staging system²³¹ based on clinical signs can be used by rescuers at the scene to describe victims: stage I – clearly conscious and shivering; stage II – impaired consciousness without shivering; stage III – unconscious; stage IV – no breathing; and stage V – death due to irreversible hypothermia.

Diagnosis

Accidental hypothermia may be under-diagnosed in countries with a temperate climate. In persons with normal thermoregulation, hypothermia may develop during exposure to cold environments, particularly wet or windy conditions, and in people who have been immobilised, or following immersion in cold water. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia may follow a mild insult. The risk of hypothermia is also increased by drug or alcohol ingestion, exhaustion, illness, injury or neglect especially when there is a decrease in the level of consciousness. Hypothermia may be suspected from the clinical history or a brief external examination of a collapsed patient. A low-reading thermometer is needed to measure the core temperature and confirm the diagnosis. The core temperature measured in the lower third of the oesophagus correlates well with the temperature of the heart. Epitympanic ('tympanic') measurement – using a thermistor technique – is a reliable alternative but may be lower than the oesophageal temperature if the environmental temperature is very cold, the probe is not well insulated, the external auditory canal is blocked or during cardiac arrest when there is no flow in the carotid artery.²³² Widely available 'tympanic' thermometers based on infrared technique do not seal the ear canal and are not designed for low core temperature readings.²³³ In the hospital setting, the method of temperature measurement should be the same throughout resuscitation and rewarming. Use oesophageal, bladder, rectal or tympanic temperature measurements.^{234,235}

Decision to resuscitate

Cooling of the human body decreases cellular oxygen consumption by ~6% per 1 °C decrease in core temperature.²³⁶ At 28 °C oxygen consumption is reduced by ~50% and at 22 °C by ~75%. In some cases, hypothermia can exert a protective effect on the brain and vital organs²³⁷ and intact neurological recovery may be possible even after prolonged cardiac arrest if deep hypothermia develops before asphyxia. Beware of diagnosing death in a hypothermic patient because cold alone may produce a very slow, small-volume, irregular pulse and unrecordable blood pressure. In a hypothermic patient, no signs of life (Swiss hypothermia stage IV) alone is unreliable for declaring death. At 18 °C the brain can tolerate periods of circulatory arrest for ten times longer than at 37 °C. Dilated pupils can be caused by a variety of insults and must not be regarded as a sign of death. Good quality survival has been reported after cardiac arrest and a core temperature of 13.7 °C after immersion in cold water with prolonged CPR.²³⁸ In another case, a severely hypothermic patient was resuscitated successfully after six and a half hours of CPR.²³⁹

In the pre-hospital setting, resuscitation should be withheld only if the cause of a cardiac arrest is clearly attributable to a lethal injury, fatal illness, prolonged asphyxia, or if the chest is incompressible. In all other patients the traditional guiding principle that "no one is dead until warm and dead" should be considered. In remote wilderness areas, the impracticalities of achieving rewarming have to be considered. In the hospital setting involve senior doctors and use clinical judgment to determine when to stop resuscitating a hypothermic arrest victim.

Resuscitation

All the principles of prevention, basic and advanced life support apply to the hypothermic patient. Use the same ventilation and chest compression rates as for a normothermic patient. Hypothermia can cause stiffness of the chest wall, making ventilation and chest compressions more difficult. Do not delay urgent procedures, such as inserting vascular catheters and tracheal intubation. The

advantages of adequate oxygenation and protection from aspiration outweigh the minimal risk of triggering VF by performing tracheal intubation.²⁴⁰

Clear the airway and, if there is no spontaneous respiratory effort, ventilate the patient's lungs with high concentrations of oxygen. Consider careful tracheal intubation when indicated according to advanced life support guidelines. Palpate a central artery, look at the ECG (if available), and look for signs of life for up to 1 min before concluding that there is no cardiac output. Echocardiography or ultrasound with Doppler may be used to establish whether there is a cardiac output or peripheral blood flow. If there is any doubt about whether a pulse is present, start CPR immediately. Once CPR is under way, confirm hypothermia with a low-reading thermometer.

The hypothermic heart may be unresponsive to cardio-active drugs, attempted electrical pacing and defibrillation. Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drugs given repeatedly.²⁴¹ The evidence for the efficacy of drugs in severe hypothermia is limited and based mainly on animal studies. For instance, in severe hypothermic cardiac arrest, adrenaline may be effective in increasing coronary perfusion pressure, but not survival.^{242,243} The efficacy of amiodarone is also reduced.²⁴⁴ For these reasons, withhold adrenaline and other CPR drugs until the patient has been warmed to a temperature higher than approximately 30 °C. Once 30 °C has been reached, the intervals between drug doses should be doubled when compared with normothermia intervals. As normothermia is approached (over 35 °C), standard drug protocols should be used. Remember to rule out other primary causes of cardiorespiratory arrest using the 4 Hs and 4 Ts approach (e.g., drug overdose, hypothyroidism, trauma).

Arrhythmias

As the body core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation followed by VF and finally asystole.²⁴⁵ Once in hospital, severely hypothermic victims in cardiac arrest should be rewarmed with active internal methods. Arrhythmias other than VF tend to revert spontaneously as the core temperature increases, and usually do not require immediate treatment. Bradycardia may be physiological in severe hypothermia, and cardiac pacing is not indicated unless bradycardia associated with haemodynamic compromise persists after rewarming.

The temperature at which defibrillation should first be attempted and how often it should be tried in the severely hypothermic patient has not been established. AEDs may be used on these patients. If VF is detected, give a shock at the maximum energy setting; if VF/VT persists after three shocks, delay further defibrillation attempts until the core temperature is above 30 °C.²⁴⁶ If an AED is used, follow the AED prompts while rewarming the patient. CPR and rewarming may have to be continued for several hours to facilitate successful defibrillation.²⁴⁶

Rewarming

General measures for all victims include removal from the cold environment, prevention of further heat loss and rapid transfer to hospital. In the field, a patient with moderate or severe hypothermia (Swiss stages ≥ II) should be immobilised and handled carefully, oxygenated adequately, monitored (including ECG and core temperature), and the whole body dried and insulated.²⁴¹ Wet clothes should be cut off rather than stripped off; this will avoid excessive movement of the victim. Conscious victims can mobilise as exercise rewarms a person more rapidly than shivering. Exercise can increase any after-drop, i.e., further cooling after removal from a cold environment. Somnolent or comatose victims have a low threshold for developing VF or pulseless VT and should be

immobilised and kept horizontal to avoid an after-drop or cardiovascular collapse. Adequate oxygenation is essential to stabilise the myocardium and all victims should receive supplemental oxygen. If the patient is unconscious, the airway should be protected. Pre hospital, prolonged investigation and treatments should be avoided, as further heat loss is difficult to prevent.

Rewarming may be passive, active external, or active internal. Passive rewarming is appropriate in conscious victims with mild hypothermia who are still able to shiver. This is best achieved by full body insulation with wool blankets, aluminium foil, cap and warm environment. The application of chemical heat packs to the trunk is particularly helpful in moderate and severe hypothermia to prevent further heat loss in the pre hospital setting. If the patient is unconscious and the airway is not secured, insulation should be arranged around the patient in the recovery (lateral decubitus) position. Rewarming in the field with heated intravenous fluids and warm humidified gases is not efficient. Infusing a litre of 40 °C warm fluid to a 70 kg patient at 28 °C elevates the core temperature by only about 0.3 °C.²⁴¹ Intensive active rewarming must not delay transport to a hospital where advanced rewarming techniques, continuous monitoring and observation are available. In general, alert hypothermic and shivering victims without an arrhythmia may be transported to the nearest hospital for passive rewarming and observation. Hypothermic victims with an altered consciousness should be taken to a hospital capable of active external and internal rewarming.

Several active in-hospital rewarming techniques have been described, although in a patient with stable circulation no technique has shown better survival over others. Active external rewarming techniques include forced air rewarming and warmed (up to 42 °C) intravenous fluids. These techniques are effective (rewarming rate 1–1.5 °C h⁻¹) in patients with severe hypothermia and a perfusing rhythm.^{247,248} Even in severe hypothermia no significant after-drop or malignant arrhythmias have been reported. Rewarming with forced air and warm fluid has been widely implemented by clinicians because it is easy and effective. Active internal rewarming techniques include warm humidified gases; gastric, peritoneal, pleural or bladder lavage with warmed fluids (at 40 °C), and extracorporeal rewarming.^{237,249–253}

In a hypothermic patient with apnoea and cardiac arrest, extracorporeal rewarming is the preferred method of active internal rewarming because it provides sufficient circulation and oxygenation while the core body temperature is increased by 8–12 °C h⁻¹.²⁵³ Survivors in one case-series had an average of 65 min of conventional CPR before cardiopulmonary bypass,²⁵⁴ which underlines that continuous CPR is essential. Unfortunately, facilities for extracorporeal rewarming are not always available and a combination of rewarming techniques may have to be used. It is advisable to contact the destination hospital well in advance of arrival to make sure that the unit can accept the patient for extracorporeal rewarming. Extracorporeal membrane oxygenation (ECMO) reduces the risk of intractable cardiorespiratory failure commonly observed after rewarming and may be a preferable extracorporeal rewarming procedure.²⁵⁵

During rewarming, patients will require large volumes of fluids as vasodilation causes expansion of the intravascular space. Continuous haemodynamic monitoring and warm IV fluids are essential. Avoid hyperthermia during and after rewarming. Although there are no formal studies, once ROSC has been achieved use standard strategies for post-resuscitation care, including mild hypothermia if appropriate (Section 4g).^{24a}

Avalanche burial

In Europe and North America, there are about 150 snow avalanche deaths each year. Most are sports-related and involve

skiers, snowboarders and snowmobilers. Death from avalanches is due to asphyxia, trauma and hypothermia. Avalanches occur in areas that are difficult to access by rescuers in a timely manner, and burials frequently involve multiple victims. The decision to initiate full resuscitative measures should be determined by the number of victims and the resources available, and should be informed by the likelihood of survival.²⁵⁶ Avalanche victims are not likely to survive when they are:

- buried >35 min and in cardiac arrest with an obstructed airway on extrication;
- buried initially and in cardiac arrest with an obstructed airway on extrication, and an initial core temperature of <32 °;
- buried initially and in cardiac arrest on extrication with an initial serum potassium of >12 mmol.

Full resuscitative measures, including extracorporeal rewarming, when available, are indicated for all other avalanche victims without evidence of an unsurvivable injury.

8e. Hyperthermia

Definition

Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds that normally maintained by homeostatic mechanisms. Hyperthermia may be exogenous, caused by environmental conditions, or secondary to endogenous heat production.

Environment-related hyperthermia occurs where heat, usually in the form of radiant energy, is absorbed by the body at a rate faster than can be lost by thermoregulatory mechanisms. Hyperthermia occurs along a continuum of heat-related conditions, starting with heat stress, progressing to heat exhaustion, to heat stroke (HS) and finally multiorgan dysfunction and cardiac arrest in some instances.²⁵⁷

Malignant hyperthermia (MH) is a rare disorder of skeletal muscle calcium homeostasis characterised by muscle contracture and life-threatening hypermetabolic crisis following exposure of genetically predisposed individuals to halogenated anaesthetics and depolarizing muscle relaxants.^{258,259}

The key features and treatment of heat stress and heat exhaustion are included in Table 8.2.

Heat stroke

Heat stroke is a systemic inflammatory response with a core temperature above 40.6 °C, accompanied by mental state change and varying levels of organ dysfunction. There are two forms of HS: classic non-exertional heat stroke (CHS) occurs during high environmental temperatures and often effects the elderly during heat waves²⁶⁰; The 2003 heatwave in France was associated with an increased incidence of cardiac arrests in those over 60-years old.²⁶¹ Exertional heat stroke (EHS) occurs during strenuous physical exercise in high environmental temperatures and/or high humidity usually effects healthy young adults.²⁶² Mortality from heat stroke ranges between 10 and 50%.²⁶³

Predisposing factors

The elderly are at increased risk for heat-related illness because of underlying illness, medication use, declining thermoregulatory mechanisms and limited social support. There are several risk factors: lack of acclimatization, dehydration, obesity, alcohol, cardiovascular disease, skin conditions (psoriasis, eczema,

Table 8.2
Heat stress and heat exhaustion.

Condition	Features	Treatment
Heat stress	Normal or mild temperature elevation Heat oedema: swelling of feet and ankles Heat syncope: vasodilation causing hypotension Heat cramps: sodium depletion causing cramps	Rest Elevation of oedematous limbs Cooling Oral rehydration Salt replacement
Heat exhaustion	Systemic reaction to prolonged heat exposure (hours to days) Temperature > 37 °C and < 40 °C Headache, dizziness, nausea, vomiting, tachycardia, hypotension, sweating muscle pain, weakness and cramps Haemoconcentration Hyponatraemia or hypernatraemia May progress rapidly to heat stroke	As above Consider IV fluids and ice packs for severe cases

scleroderma, burn, cystic fibrosis), hyperthyroidism, pheochromocytoma and drugs (anticholinergics, diamorphine, cocaine, amphetamine, phenothiazines, sympathomimetics, calcium channel blockers, beta-blockers).

Clinical presentation

Heat stroke can resemble septic shock and may be caused by similar mechanisms.²⁶⁴ A single centre case-series reported 14 ICU deaths in 22 heat stroke patients admitted to ICU with multiple organ failure.²⁶⁵ Features include:

- core temperature 40.6 °C or more;
- hot, dry skin (sweating is present in about 50% of cases of exertional heat stroke);
- early signs and symptoms, e.g., extreme fatigue, headache, fainting, facial flushing, vomiting and diarrhoea;
- cardiovascular dysfunction including arrhythmias²⁶⁶ and hypotension;
- respiratory dysfunction including ARDS²⁶⁷;
- central nervous system dysfunction including seizures and coma²⁶⁸;
- liver and renal failure²⁶⁹;
- coagulopathy²⁶⁷;
- rhabdomyolysis.²⁷⁰

Other clinical conditions need to be considered, including:

- drug toxicity^{271,272};
- drug withdrawal syndrome;
- serotonin syndrome²⁷³;
- neuroleptic malignant syndrome²⁷⁴;
- sepsis²⁷⁵;
- central nervous system infection;
- endocrine disorders, e.g., thyroid storm, pheochromocytoma.²⁷⁶

Management

The mainstay of treatment is supportive therapy based on optimizing the ABCDEs and rapidly cooling the patient.^{277–279} Start cooling before the patient reaches hospital. Aim to rapidly reduce the core temperature to approximately 39 °C. Patients with severe heat stroke need to be managed in a critical-care setting. Use haemodynamic monitoring to guide fluid therapy. Large volumes of fluid may be required. Correct electrolyte abnormalities as described in Section 8a.

Cooling techniques

Several cooling methods have been described, but there are few formal trials to determine which method is best. Simple cooling techniques include drinking cool fluids, fanning the completely undressed patient and spraying tepid water on the patient. Ice packs over areas where there are large superficial blood vessels (axillae, groins, neck) may also be useful. Surface cooling methods may cause shivering. In cooperative stable patients, immersion in cold water can be effective²⁸⁰; however, this may cause peripheral vasoconstriction, shunt blood away from the periphery and reduce heat dissipation. Immersion is also not practical in the sick-est patients.

Further techniques to cool patients with hyperthermia are similar to those used for therapeutic hypothermia after cardiac arrest (see Section 4g).^{24a} Cold intravenous fluids will decrease body temperature. Gastric, peritoneal,²⁸¹ pleural or bladder lavage with cold water will lower the core temperature. Intravascular cooling techniques include the use of cold IV fluids,²⁸² intravascular cooling catheters^{283,284} and extracorporeal circuits,²⁸⁵ e.g., continuous veno-veno haemofiltration or cardiopulmonary bypass.

Drug therapy in heat stroke

There are no specific drug therapies in heat stroke to lower core temperature. There is no good evidence that antipyretics (e.g., non-steroidal anti-inflammatory drugs or paracetamol) are effective in heat stroke. Diazepam may be useful to treat seizures and facilitate cooling.²⁸⁶ Dantrolene (see below) has not been shown to be beneficial.^{287–289}

Malignant hyperthermia

Malignant hyperthermia is a life-threatening genetic sensitivity of skeletal muscles to volatile anaesthetics and depolarizing neuromuscular blocking drugs, occurring during or after anaesthesia.²⁹⁰ Stop triggering agents immediately; give oxygen, correct acidosis and electrolyte abnormalities. Start active cooling and give dantrolene.²⁹¹

Other drugs such as 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines also cause a condition similar to malignant hyperthermia and the use of dantrolene may be beneficial.²⁹²

Modifications to cardiopulmonary resuscitation and post-resuscitation care

There are no specific studies on cardiac arrest in hyperthermia. If cardiac arrest occurs, follow standard procedures for basic

and advanced life support and cool the patient. Cooling techniques similar to those used to induce therapeutic hypothermia should be used (see Section 4g).^{24a} There are no data on the effects of hyperthermia on defibrillation threshold; therefore, attempt defibrillation according to current guidelines, while continuing to cool the patient. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest.^{293,294} The risk of unfavourable neurological outcome increases for each degree of body temperature >37 °C.²⁹⁵ Provide post-resuscitation care according to normal guidelines.

8f. Asthma

Introduction

Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma.²⁹⁶ The worldwide prevalence of asthma symptoms ranges from 1 to 18% of the population with a high prevalence in some European countries (United Kingdom, Ireland and Scandinavia).²⁹⁶ International differences in asthma symptom prevalence appears to be decreasing in recent years, especially in adolescents.²⁹⁷ The World Health Organisation has estimated that 15 million disability-adjusted life-years (DALYs) are lost annually from asthma, representing 1% of the global disease burden. Annual worldwide deaths from asthma have been estimated at 250,000. The death rate does not appear to be correlated with asthma prevalence.²⁹⁶ National and international guidance for the management of asthma already exists.^{296,298} This guidance focuses on the treatment of patients with near-fatal asthma and cardiac arrest.

Patients at risk of asthma-related cardiac arrest

The risk of near-fatal asthma attacks is not necessarily related to asthma severity.²⁹⁹ Patients most at risk include those with:

- a history of near-fatal asthma requiring intubation and mechanical ventilation;
- a hospitalisation or emergency care for asthma in the past year³⁰⁰;
- low or no use of inhaled corticosteroids³⁰¹;
- an increasing use and dependence of beta-2 agonists³⁰²;
- anxiety, depressive disorders and/or poor compliance with therapy.³⁰³

Causes of cardiac arrest

Cardiac arrest in a person with asthma is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden. Cardiac arrest in those with asthma has been linked to:

- severe bronchospasm and mucous plugging leading to asphyxia (this condition causes the vast majority of asthma-related deaths);
- cardiac arrhythmias caused by hypoxia, which is the commonest cause of asthma-related arrhythmia.³⁰⁴ Arrhythmias can also be caused by stimulant drugs (e.g., beta-adrenergic agonists, aminophylline) or electrolyte abnormalities;
- dynamic hyperinflation, i.e., auto-positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure;
- tension pneumothorax (often bilateral).

Diagnosis

Wheezing is a common physical finding, but severity does not correlate with the degree of airway obstruction. The absence of wheezing may indicate critical airway obstruction, whereas increased wheezing may indicate a positive response to bronchodilator therapy. SaO₂ may not reflect progressive alveolar hypoventilation, particularly if oxygen is being given. The SaO₂ may initially decrease during therapy because beta-agonists cause both bronchodilation and vasodilation and may initially increase intrapulmonary shunting.

Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis,³⁰⁵ pneumonia, foreign bodies, pulmonary embolism, bronchiectasis and subglottic mass.³⁰⁶

The severity of an asthma attack is defined in Table 8.3.

Key interventions to prevent arrest

The patient with severe asthma requires aggressive medical management to prevent deterioration. Base assessment and treatment on an ABCDE approach. Patients with SaO₂ <92% or with features of life-threatening asthma are at risk of hypercapnia and require arterial blood gas measurement. Experienced clinicians should treat these high-risk patients in a critical-care area. The specific drugs and the treatment sequence will vary according to local practice.

Oxygen

Use a concentration of inspired oxygen that will achieve an SaO₂ 94–98%.²⁰⁵ High-flow oxygen by mask is sometimes necessary.

Table 8.3

The severity of asthma.

Asthma	Features
Near-fatal	Raised PaCO ₂ and/or requiring mechanical ventilation with raised inflation pressures
Life-threatening	Any one of: PEF < 33% best or predicted bradycardia SpO ₂ < 92%, dysrhythmia PaO ₂ < 8 kPa, hypotension Normal PaCO ₂ (4.6–6.0 kPa (35–45 mmHg)), exhaustion Silent chest, confusion Cyanosis, coma Feeble respiratory effort
Acute severe	Any one of: PEF 33–50% best or predicted Respiratory rate > 25 min ⁻¹ Heart rate > 110 min ⁻¹ Inability to complete sentences in one breath
Moderate exacerbation	Increasing symptoms PEF > 50–75% best or predicted No features of acute severe asthma
Brittle	Type 1: wide PEF variability (>40% diurnal variation for >50% of the time over a period >150 days) despite intense therapy Type 2: sudden severe attacks on a background of apparently well controlled asthma

PEF, peak expiratory flow.

Nebulised beta-2 agonists

Salbutamol, 5 mg nebulised, is the cornerstone of therapy for acute asthma in most of the world. Repeated doses every 15–20 min are often needed. Severe asthma may necessitate continuous nebulised salbutamol. Nebuliser units that can be driven by high-flow oxygen should be available. The hypoventilation associated with severe or near-fatal asthma may prevent effective delivery of nebulised drugs. If a nebuliser is not immediately available beta-2 agonists can be temporarily administered by repeating activations of a metered dose inhaler via a large volume spacer device.^{298,307} Nebulised adrenaline does not provide additional benefit over and above nebulised beta-2 agonists in acute asthma.³⁰⁸

Intravenous corticosteroids

Early use of systemic corticosteroids for acute asthma in the emergency department significantly reduces hospital admission rates, especially for those patients not receiving concomitant corticosteroid therapy.³⁰⁹ Although there is no difference in clinical effects between oral and IV formulations of corticosteroids,³¹⁰ the IV route is preferable because patients with near-fatal asthma may vomit or be unable to swallow.

Nebulised anticholinergics

Nebulised anticholinergics (ipratropium, 0.5 mg 4–6 hourly) may produce additional bronchodilation in severe asthma or in those who do not respond to beta-agonists.^{311,312}

Nebulised magnesium sulphate

Results of small randomised controlled trials showed that a nebulised isotonic solution of magnesium sulphate (250 mmol l⁻¹) in a volume of 2.5–5 ml in combination with beta-2 agonists is safe and it is associated with both an improvement of pulmonary function tests and a non-significant trend towards lower rates of hospital admission in patients with acute severe asthma.³¹³ Further studies are needed to confirm those findings.

Intravenous bronchodilators

Nebulised bronchodilators are the first line treatment for acute severe and life-threatening exacerbations of asthma. There is a lack of definitive evidence for or against the use of intravenous bronchodilators in this setting. Trials have primarily included spontaneously breathing patients with moderate to life-threatening exacerbations of asthma, evidence in ventilated patients with life-threatening asthma or cardiac arrest is sparse. The use of intravenous bronchodilators should generally be restricted to patients unresponsive to nebulised therapy or where nebulised/inhaled therapy is not possible (e.g., a patient receiving bag-mask ventilation).

Intravenous magnesium sulphate

Studies of intravenous magnesium sulphate in acute severe and life-threatening asthma have produced conflicting results.^{314,315} Magnesium sulphate causes bronchial smooth muscle relaxation independent of the serum magnesium level and has only minor side effects (flushing, light-headedness). Given the low risk of serious side effects from magnesium sulphate it would seem reasonable to use intravenous magnesium sulphate (1.2–2 g IV slowly) in adults with life-threatening unresponsive to nebulised therapy. The multicentre randomised controlled trial 3Mg (ISRCTN04417063) is due

to report in 2012 and should provide definitive evidence on the role of magnesium in acute severe asthma.

Aminophylline

A Cochrane review of intravenous aminophylline found no evidence of benefit and a higher incidence of adverse effects (tachycardia, vomiting) compared with standard care alone.^{316,317} Whether aminophylline has a place as an additional therapy after treatment with established medications such as inhaled beta-agonists and systemic corticosteroids remains uncertain. If after obtaining senior advice the decision is taken to administer IV aminophylline a loading dose of 5 mg kg⁻¹ is given over 20–30 min (unless on maintenance therapy), followed by an infusion of 500–700 µg kg⁻¹ h⁻¹. Serum theophylline concentrations should be maintained below 20 µg ml⁻¹ to avoid toxicity.

Beta-2 agonists

A Cochrane review on intravenous beta-2 agonists compared with nebulised beta-2 agonists found no evidence of benefit and some evidence of increased side effects compared with inhaled treatment.³¹⁸ Salbutamol may be given as either a slow IV injection (250 µg IV slowly) or continuous infusion of 3–20 µg min⁻¹.

Leukotriene receptor antagonists

There are few data on the use of intravenous leukotriene receptor antagonists.³¹⁹ Further studies are required to confirm the findings of a recent randomised controlled trial which demonstrated evidence of additional bronchodilation when intravenous LRTA montelukast was used as a rescue therapy.³²⁰

Subcutaneous or intramuscular adrenaline and terbutaline

Adrenaline and terbutaline are adrenergic agents that may be given subcutaneously to patients with acute severe asthma. The dose of subcutaneous adrenaline is 300 µg up to a total of 3 doses at 20-min intervals. Adrenaline may cause an increase in heart rate, myocardial irritability and increased oxygen demand; however, its use (even in patients over 35-years old) is well tolerated.³²¹ Terbutaline is given in a dose of 250 µg subcutaneously, which can be repeated in 30–60 min. These drugs are more commonly given to children with acute asthma and, although most studies have shown them to be equally effective,³²² one study concluded that terbutaline was superior.³²³ These alternative routes may need to be considered when IV access is impossible. Patients with asthma are at increased risk of anaphylaxis. Sometimes it may be difficult to distinguish severe life-threatening asthma from anaphylaxis. In these circumstances intramuscular adrenaline given according to the anaphylaxis guidelines may be appropriate (Section 8g).

Intravenous fluids and electrolytes

Severe or near-fatal asthma is associated with dehydration and hypovolaemia, and this will further compromise the circulation in patients with dynamic hyperinflation of the lungs. If there is evidence of hypovolaemia or dehydration, give IV fluids. Beta-2 agonists and steroids may induce hypokalaemia, which should be corrected with electrolyte supplements.

Heliox

Heliox is a mixture of helium and oxygen (usually 80:20 or 70:30). A meta-analysis of four clinical trials did not support the use of heliox in the initial treatment of patients with acute asthma.³²⁴

Referral to intensive care

Patients that fail to respond to initial treatment, or develop signs of life-threatening asthma, should be assessed by an intensive care specialist. Admission to intensive care after asthma-related cardiac arrest is associated with significantly poorer outcomes than if cardiac arrest does not occur.³²⁵

Rapid sequence induction and tracheal intubation should be considered if, despite efforts to optimize drug therapy, the patient has:

- a decreasing conscious level, coma;
- persisting or worsening hypoxaemia;
- deteriorating respiratory acidosis despite intensive therapy;
- findings of severe agitation, confusion and fighting against the oxygen mask (clinical signs of hypoxaemia);
- progressive exhaustion;
- respiratory or cardiac arrest.

Elevation of the PaCO₂ alone does not indicate the need for tracheal intubation.³²⁶ Treat the patient, not the numbers.

Non-invasive ventilation

Non-invasive ventilation decreases the intubation rate and mortality in COPD³²⁷; however, its role in patients with severe acute asthma is uncertain. There is insufficient evidence to recommend its routine use in asthma.³²⁸

Treatment of cardiac arrest

Basic life support

Give basic life support according to standard guidelines. Ventilation will be difficult because of increased airway resistance; try to avoid gastric inflation.

Advanced life support

Modifications to standard ALS guidelines include considering the need for early tracheal intubation. The peak airway pressures recorded during ventilation of patients with severe asthma (mean 67.8 ± 11.1 cm H₂O in 12 patients) are significantly higher than the normal lower oesophageal sphincter pressure (approximately 20 cm H₂O).³²⁹ There is a significant risk of gastric inflation and hypoventilation of the lungs when attempting to ventilate a severe asthmatic without a tracheal tube. During cardiac arrest this risk is even higher, because the lower oesophageal sphincter pressure is substantially less than normal.³³⁰

Respiratory rates of 8–10 breaths min⁻¹ and tidal volume required for a normal chest rise during CPR should not cause dynamic hyperinflation of the lungs (gas trapping). Tidal volume depends on inspiratory time and inspiratory flow. Lung emptying depends on expiratory time and expiratory flow. In mechanically ventilated severe asthmatics, increasing the expiratory time (achieved by reducing the respiratory rate) provides only moderate gains in terms of reduced gas trapping when a minute volume of less than 10 l min⁻¹ is used.³²⁹

There is limited evidence from case reports of unexpected ROSC in patients with suspected gas trapping when the tracheal tube is disconnected.^{331–335} If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest wall and/or a period of apnoea (disconnection of tracheal tube) may relieve gas trapping if dynamic hyperinflation occurs. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.³³⁶

Dynamic hyperinflation increases transthoracic impedance.³³⁷ Consider higher shock energies for defibrillation if initial defibrillation attempts fail.³³⁸

There is no good evidence for the use of open-chest cardiac compressions in patients with asthma-associated cardiac arrest. Working through the 4 Hs and 4 Ts will identify potentially reversible causes of asthma-related cardiac arrest. Tension pneumothorax can be difficult to diagnose in cardiac arrest; it may be indicated by unilateral expansion of the chest wall, shifting of the trachea and subcutaneous emphysema. Pleural ultrasound in skilled hands is faster and more sensitive than chest X-ray for the detection of pneumothorax.³³⁹ If pneumothorax is suspected, release air from the pleural space with needle decompression. Insert a large-gauge cannula in the second intercostal space, above the rib, in the mid-clavicular line, being careful to avoid direct puncture of the lung. If air is emitted, insert a chest tube. Always consider bilateral pneumothoraces in asthma-related cardiac arrest.

Extracorporeal life support (ECLS) can ensure both organ perfusion and gas exchange in case of otherwise untreatable respiratory and circulatory failure. Cases of successful treatment of asthma-related cardiac arrest in adults using ECLS have been reported^{340,341}; however, the role of ECLS in cardiac arrest caused by asthma has never been investigated in controlled studies. The use of ECLS requires appropriate skills and equipment which may not be available everywhere.

8g. Anaphylaxis

Definition of anaphylaxis

A precise definition of anaphylaxis is not important for its emergency treatment. There is no universally agreed definition. The European Academy of Allergology and Clinical Immunology Nomenclature Committee proposed the following broad definition³⁴²: Anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction. This is characterised by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.^{305,343}

Anaphylaxis usually involves the release of inflammatory mediators from mast cells and, or basophils triggered by an allergen interacting with cell-bound immunoglobulin E (IgE). Non-IgE-mediated or non-immune release of mediators can also occur. Histamine and other inflammatory mediator release are responsible for the vasodilatation, oedema and increased capillary permeability.

Epidemiology

The overall frequency of episodes of anaphylaxis using current data lies between 30 and 950 cases per 100,000 persons per year and a lifetime prevalence of between 50 and 2000 episodes per 100,000 persons or 0.05–2.0%.³⁴⁴ Anaphylaxis can be triggered by any of a very broad range of triggers including foods, drugs, stinging insects, and latex. Food is the commonest trigger in children and drugs the commonest in adults.³⁴⁵ Virtually any food or drug can be implicated, but certain foods (nuts) and drugs (muscle relaxants, antibiotics, NSAIDs and aspirin) cause most reactions.³⁴⁶ A significant number of cases of anaphylaxis are idiopathic.

The overall prognosis of anaphylaxis is good, with a case fatality ratio of less than 1% reported in most population-based studies. Anaphylaxis and risk of death is increased in those with pre-existing asthma, particularly if the asthma is poorly controlled, severe or in asthmatics who delay treatment with adrenaline.^{347,348} When anaphylaxis is fatal, death usually occurs very soon after

contact with the trigger. From a case-series, fatal food reactions cause respiratory arrest typically after 30–35 min; insect stings cause collapse from shock after 10–15 min; and deaths caused by intravenous medication occur most commonly within 5 min. Death never occurred more than 6 h after contact with the trigger.

Recognition of an anaphylaxis

Anaphylaxis is the likely diagnosis if a patient who is exposed to a trigger (allergen) develops a sudden illness (usually within minutes) with rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes. The reaction is usually unexpected.

Many patients with anaphylaxis are not given the correct treatment.³⁴⁹ Confusion arises because some patients have systemic allergic reactions that are less severe. For example, generalised urticaria, angioedema, and rhinitis would not be described as anaphylaxis, because the life-threatening features are not present. Anaphylaxis guidelines must therefore take into account some inevitable diagnostic errors, with an emphasis on the need for safety. Patients can have either an airway and/or breathing and/or circulation problem:

Airway problems

- Airway swelling, e.g., throat and tongue swelling (pharyngeal/laryngeal oedema).
- Hoarse voice.
- Stridor.

Breathing problems

- Shortness of breath.
- Wheeze.
- Confusion caused by hypoxia.
- Respiratory arrest.
- Life-threatening asthma with no features of anaphylaxis can be triggered by food allergy.³⁵⁰

Circulation problems

- Pale, clammy.
- Tachycardia.
- Hypotension.
- Decreased conscious level.
- Myocardial ischaemia and electrocardiograph (ECG) changes even in individuals with normal coronary arteries.³⁵¹
- Cardiac arrest.

Circulation problems (often referred to as anaphylactic shock) can be caused by direct myocardial depression, vasodilation and capillary leak, and loss of fluid from the circulation. Bradycardia is usually a late feature, often preceding cardiac arrest.³⁵²

Skin and, or mucosal changes

These should be assessed as part of the exposure when using the ABCDE approach.

- They are often the first feature and present in over 80% of anaphylaxis cases.³⁵³
- They can be subtle or dramatic.
- There may be just skin, just mucosal, or both skin and mucosal changes any where on the body.

- There may be erythema, urticaria (also called hives, nettle rash, weals or welts), or angioedema (eyelids, lips, and sometimes in the mouth and throat).

Most patients who have skin changes caused by allergy do not go on to develop anaphylaxis.

Treatment of an anaphylaxis

Use an ABCDE approach to recognise and treat anaphylaxis. Treat life-threatening problems as you find them. The basic principles of treatment are the same for all age groups. All patients who have suspected anaphylaxis should be monitored (e.g., by ambulance crew, in the emergency department etc.) as soon as possible. Minimal monitoring includes pulse oximetry, non-invasive blood pressure and 3-lead ECG.

Patient positioning

Patients with anaphylaxis can deteriorate and are at risk of cardiac arrest if made to sit up or stand up.³⁵⁴ All patients should be placed in a comfortable position. Patients with airway and breathing problems may prefer to sit up as this will make breathing easier. Lying flat with or without leg elevation is helpful for patients with a low blood pressure (circulation problem).

Remove the trigger if possible

Stop any drug suspected of causing anaphylaxis. Remove the stinger after a bee sting. Early removal is more important than the method of removal.³⁵⁵ Do not delay definitive treatment if removing the trigger is not feasible.

Cardiorespiratory arrest following an anaphylaxis

Start cardiopulmonary resuscitation (CPR) immediately and follow current guidelines. Prolonged CPR may be necessary. Rescuers should ensure that help is on its way as early advanced life support (ALS) is essential.

Airway obstruction

Anaphylaxis can cause airway swelling and obstruction. This will make airway and ventilation interventions (e.g., bag-mask ventilation, tracheal intubation, cricothyroidotomy) difficult. Call for expert help early.

Drugs and their delivery

Adrenaline (epinephrine)

Adrenaline is the most important drug for the treatment of anaphylaxis.^{356,357} Although there are no randomised controlled trials,³⁵⁸ adrenaline is a logical treatment and there is consistent anecdotal evidence supporting its use to ease breathing and circulation problems associated with anaphylaxis. As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. Its beta-receptor activity dilates the bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release. There are beta-2 adrenergic receptors on mast cells that inhibit activation, and so early adrenaline attenuates the severity of IgE-mediated allergic reactions. Adrenaline seems to work best when given early after the onset of the reaction³⁵⁹ but it is not without risk, particularly when given intravenously. Adverse effects are extremely rare with correct doses injected intramuscularly (IM).

Adrenaline should be given to all patients with life-threatening features. If these features are absent but there are other features of

a systemic allergic reaction, the patient needs careful observation and symptomatic treatment using the ABCDE approach.

Intramuscular (IM) adrenaline. The intramuscular (IM) route is the best for most individuals who have to give adrenaline to treat anaphylaxis. Monitor the patient as soon as possible (pulse, blood pressure, ECG, and pulse oximetry). This will help monitor the response to adrenaline. The IM route has several benefits:

- There is a greater margin of safety.
- It does not require intravenous access.
- The IM route is easier to learn.

The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The needle for injection needs to be long enough to ensure that the adrenaline is injected into muscle.³⁶⁰ The subcutaneous or inhaled routes for adrenaline are not recommended for the treatment of anaphylaxis because they are less effective than the IM route.^{361–363}

Adrenaline IM dose. The evidence for the recommended doses is weak. Doses are based on what is considered to be safe and practical to draw up and inject in an emergency.

(The equivalent volume of 1:1000 adrenaline is shown in brackets)

>12 years and adults:	500 µg IM (0.5 ml)
>6–12 years:	300 µg IM (0.3 ml)
>6 months–6 years:	150 µg IM (0.15 ml)
<6 months:	150 µg IM (0.15 ml)

Repeat the IM adrenaline dose if there is no improvement in the patient's condition. Further doses can be given at about 5-min intervals according to the patient's response.

Intravenous (IV) adrenaline (for specialist use only). There is a much greater risk of causing harmful side effects by inappropriate dosage or misdiagnosis of anaphylaxis when using IV adrenaline.³⁶⁴ Intravenous adrenaline should be used only by those experienced in the use and titration of vasopressors in their normal clinical practice (e.g., anaesthetists, emergency physicians, intensive care doctors). In patients with a spontaneous circulation, intravenous adrenaline can cause life-threatening hypertension, tachycardia, arrhythmias, and myocardial ischaemia. If IV access is not available or not achieved rapidly, use the IM route for adrenaline. Patients who are given IV adrenaline must be monitored – continuous ECG and pulse oximetry and frequent non-invasive blood pressure measurements as a minimum. Patients who require repeated IM doses of adrenaline may benefit from IV adrenaline. It is essential that these patients receive expert help early.

Adrenaline IV bolus dose – adult. Titrate IV adrenaline using 50 µg boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion.^{352,365}

Adrenaline IV bolus dose – children. IM adrenaline is the preferred route for children having anaphylaxis. The IV route is recommended only in specialist paediatric settings by those familiar with its use (e.g., paediatric anaesthetists, paediatric emergency physicians, paediatric intensivists) and if the patient is monitored and IV access is already available. There is no evidence on which to base a dose recommendation – the dose is titrated according to response. A child may respond to a dose as small as 1 µg kg⁻¹. This requires very careful dilution and checking to prevent dose errors.

Oxygen (give as soon as available)

Initially, give the highest concentration of oxygen possible using a mask with an oxygen reservoir.²⁰⁵ Ensure high-flow oxygen (usually greater than 10 litres min⁻¹) to prevent collapse of the reservoir during inspiration. If the patient's trachea is intubated, ventilate the lungs with high concentration oxygen using a self-inflating bag.

Fluids (give as soon as available)

Large volumes of fluid may leak from the patient's circulation during anaphylaxis. There will also be vasodilation. If there is intravenous access, infuse intravenous fluids immediately. Give a rapid IV fluid challenge (20 ml kg⁻¹) in a child or 500–1000 ml in an adult) and monitor the response; give further doses as necessary. There is no evidence to support the use of colloids over crystalloids in this setting. Consider colloid infusion as a cause in a patient receiving a colloid at the time of onset of an anaphylaxis and stop the infusion. A large volume of fluid may be needed.

If intravenous access is delayed or impossible, the intra-osseous route can be used for fluids or drugs. Do not delay the administration of IM adrenaline attempting intra-osseous access.

Antihistamines (after initial resuscitation)

Antihistamines are a second line treatment for anaphylaxis. The evidence to support their use is weak, but there are logical reasons for them.³⁶⁶ Antihistamines (H₁-antihistamine) help counter histamine-mediated vasodilation and bronchoconstriction. There is little evidence to support the routine use of an H₂-antihistamine (e.g., ranitidine, cimetidine) for the initial treatment of an anaphylaxis.

Steroids (give after initial resuscitation)

Corticosteroids may help prevent or shorten protracted reactions although the evidence is very limited.³⁶⁷ In asthma, early corticosteroid treatment is beneficial in adults and children. There is little evidence on which to base the optimum dose of hydrocortisone in anaphylaxis.

Other drugs

Bronchodilators. The presenting symptoms and signs of a severe anaphylaxis and life-threatening asthma can be the same. Consider further bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), aminophylline (IV) or magnesium (IV) (see Section 8f above). Intravenous magnesium is a vasodilator and can make hypotension worse.

Cardiac drugs. Adrenaline remains the first line vasopressor for the treatment of anaphylaxis. There are animal studies and case reports describing the use of other vasopressors and inotropes (noradrenaline, vasopressin, terlipressin, metaraminol, methoxamine, and glucagon) when initial resuscitation with adrenaline and fluids has not been successful.^{368–380} Use these drugs only in specialist settings (e.g., intensive care units) where there is experience in their use. Glucagon can be useful to treat anaphylaxis in a patient taking a beta-blocker.³⁸¹ Some case reports of cardiac arrest suggest cardiopulmonary bypass^{382,383} or mechanical support of circulation³⁸⁴ may also be helpful.

Investigations

Undertake the usual investigations appropriate for a medical emergency, e.g., 12-lead ECG, chest X-ray, urea and electrolytes, arterial blood gases etc.

Mast cell tryptase

The specific test to help confirm a diagnosis of anaphylaxis is measurement of mast cell tryptase. Tryptase is the major protein component of mast cell secretory granules. In anaphylaxis, mast cell degranulation leads to markedly increased blood tryptase concentrations. Tryptase concentrations in the blood may not increase significantly until 30 min or more after the onset of symptoms, and peak 1–2 h after onset.³⁸⁵ The half-life of tryptase is short (approximately 2 h), and concentrations may be back to normal within 6–8 h, so timing of any blood samples is very important. The time of onset of the anaphylaxis is the time when symptoms were first noticed.

- (a) Minimum: one sample at 1–2 h after the start of symptoms.
 (b) Ideally: three timed samples:

- Initial sample as soon as feasible after resuscitation has started – do not delay resuscitation to take sample.
- Second sample at 1–2 h after the start of symptoms
- Third sample either at 24 h or in convalescence (for example in a follow-up allergy clinic). This provides baseline tryptase levels – some individuals have an elevated baseline level.

Serial samples have better specificity and sensitivity than a single measurement in the confirmation of anaphylaxis.³⁸⁶

Discharge and follow-up

Patients who have had suspected anaphylaxis (i.e., an airway, breathing or circulation (ABC) problem) should be treated and then observed in a clinical area with facilities for treating life-threatening ABC problems. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation. The exact incidence of biphasic reactions is unknown. Although studies quote an incidence of 1–20%, it is not clear whether all the patients in these studies actually had an anaphylaxis and whether the initial treatment was appropriate.³⁸⁷ There is no reliable way of predicting who will have a biphasic reaction. It is therefore important that decisions about discharge are made for each patient by an experienced clinician.

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician.
- Given clear instructions to return to hospital if symptoms return.
- Considered for antihistamines and oral steroids therapy for up to 3 days. This is helpful for treatment of urticaria and may decrease the chance of further reaction.
- Considered for an adrenaline auto-injector, or given a replacement.^{388–390}
- Have a plan for follow-up, including contact with the patient's general practitioner.

An adrenaline auto-injector is an appropriate treatment for patients at increased risk of idiopathic anaphylaxis, or for anyone at continued high risk of reaction, e.g., to triggers such as venom stings and food-induced reactions (unless easy to avoid). An auto-injector is not usually necessary for patients who have suffered drug-induced anaphylaxis, unless it is difficult to avoid the drug. Ideally, all patients should be assessed by an allergy specialist and have a treatment plan based on their individual risk.

Individuals provided with an auto-injector on discharge from hospital must be given instructions and training on when and how to use it. Ensure appropriate follow-up including contact with the

patient's general practitioner. All patients presenting with anaphylaxis should be referred to an allergy clinic to identify the cause, and thereby reduce the risk of future reactions and prepare the patient to manage future episodes themselves. Patients need to know the allergen responsible and how to avoid it. Patients need to be able to recognise the early symptoms of anaphylaxis, so that they can summon help quickly and prepare to use their emergency medication. Although there are no randomised clinical trials, there is evidence that individualised action plans for self-management should decrease the risk of recurrence.³⁹¹

8h. Cardiac arrest following cardiac surgery

Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase, with a reported incidence of 0.7–2.9%.^{392–400} It is usually preceded by physiological deterioration,⁴⁰¹ although it can occur suddenly in stable patients.³⁹⁸ There are usually specific causes of cardiac arrest, such as tamponade, hypovolaemia, myocardial ischaemia, tension pneumothorax, or pacing failure. These are all potentially reversible and if treated promptly cardiac arrest after cardiac surgery has a relatively high survival rate. If cardiac arrest occurs during the first 24 h after cardiac surgery, the rate of survival to hospital discharge is 54%³⁹⁹ to 79%^{398,402} in adults and 41% in children.⁴⁰¹

Key to the successful resuscitation of cardiac arrest in these patients is the need to perform emergency re-sternotomy early, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective.

Identification of cardiac arrest

Patients in the ICU are highly monitored and an arrest is most likely to be signalled by monitoring alarms where absence of pulsation or perfusing pressure on the arterial line, loss of pulse oximeter, pulmonary artery (PA) trace, or end-tidal CO₂ trace can be sufficient to indicate cardiac arrest without the need to palpate a central pulse.

Starting CPR

Start external chest compressions immediately in all patients who collapse without an output. Consider reversible causes: hypoxia – check tube position, ventilate with 100% oxygen; tension pneumothorax – clinical examination, thoracic ultrasound; hypovolaemia, pacing failure. In asystole, secondary to a loss of cardiac pacing, external massage may be delayed momentarily as long as the surgically inserted temporary pacing wires can be connected rapidly and pacing re-established (DDD at 100 min⁻¹ at maximum amplitude). The effectiveness of compressions may be verified by looking at the arterial trace, aiming to achieve a systolic blood pressure of at least 80 mmHg at a rate of 100 min⁻¹. Inability to attain this pressure may indicate tamponade, tension pneumothorax, or exanguinating haemorrhage and should precipitate emergency re-sternotomy. Intra-aortic balloon pumps should be changed to pressure triggering during CPR. In PEA, switch off the pacemaker – a temporary pacemaker may potentially hide underlying VF.

Defibrillation

There is concern that external chest compressions can cause sternal disruption or cardiac damage.^{403–406} In the post-cardiac surgery ICU, a witnessed and monitored VF/VT cardiac arrest should be treated immediately with up to three quick successive (stacked) defibrillation attempts. Three failed shocks in the post-cardiac

surgery setting should trigger the need for emergency re-sternotomy. Further defibrillation is attempted as indicated in the universal algorithm and should be performed with internal paddles at 20 J if re-sternotomy has been performed.

Emergency drugs

Use adrenaline very cautiously and titrate to effect (intravenous doses of 100 or less micrograms in adults). In order to exclude a medication error as the cause of the arrest, stop all drug infusions and check if they are correct. If there is concern about patient awareness, restart the anaesthetic drugs. Atropine is no longer recommended for the treatment of cardiac arrest as there is little evidence to show it is effective in patients who have been given adrenaline. Individual clinicians may use atropine at their discretion in post-cardiac surgery cardiac arrest if they feel it is indicated. Treat bradycardia with atropine, according to the bradycardia algorithm (see Section 4 Advanced Life Support).^{24a}

Give amiodarone 300 mg after the 3rd failed defibrillation attempt but do not delay re-sternotomy. An irritable myocardium following cardiac surgery is caused most commonly by myocardial ischaemia and correction of this, rather than giving amiodarone, is more likely to achieve myocardial stability.

Emergency re-sternotomy

This is an integral part of resuscitation after cardiac surgery, once all other reversible causes have been excluded. Once adequate airway and ventilation has been established, and if three attempts at defibrillation have failed in VF/VT, undertake re-sternotomy without delay. Emergency re-sternotomy is also indicated in asystole or PEA, when other treatments have failed. Resuscitation teams should be well rehearsed in this technique so that it can be performed safely within 5 min of the onset of cardiac arrest. Re-sternotomy equipment should be prepared as soon as an arrest is identified. Simplification of the re-sternotomy tray and regular manikin rehearsals are key measures to ensure a prompt re-sternotomy.^{407,408} All medical members of the patient care team should be trained to perform re-sternotomy if a surgeon is not available within 5 min. Improved survival and better quality of life is well documented with rapid re-sternotomy.^{394,395,409}

Re-sternotomy should be a standard part of resuscitation within 10 days after cardiac surgery. The overall survival to discharge following internal cardiac massage is 17%³⁹⁴ to 25%³⁹⁵ although survival rates are much lower when chest opening is performed outside the specialised environment of the post-cardiac surgery ICU.³⁹⁵

Reinstitution of emergency cardiopulmonary bypass

The need for emergency cardiopulmonary bypass (CPB) occurs in approximately 0.8% patients at a mean of 7 h post-operatively³⁹⁶ and is usually indicated to correct surgical bleeding or graft occlusion and rest the myocardium. Emergency institution of CPB should be available on all units undertaking cardiac surgery. Survival to discharge rates of 32%,³⁹⁵ 42%³⁹⁶ and 56.3%⁴¹⁰ have been reported when CPB is reinstated on the ICU.

Survival rates decline rapidly when this procedure is undertaken more than 24 h after surgery and when performed on the ward rather than the ICU. Emergency CPB should probably be restricted to patients who arrest within 72 h of surgery, as surgically remediable problems are unlikely after this time.³⁹⁵ Ensuring adequate anticoagulation before starting CPB, or the use of a heparin-bonded circuit, is important. The need for a further period of cross-clamping does not preclude a favourable outcome.³⁹⁶

Patients with non-sternotomy cardiac surgery

These guidelines are appropriate for patients following non-sternotomy cardiac surgery, but surgeons performing these operations should have already given clear instructions for chest reopening. Patients undergoing port-access mitral procedures or minimally invasive coronary bypass graft surgery are likely to require an emergency sternotomy, as very poor access is obtained by opening or extending a mini-thoracotomy incision. Equipment and guidelines should be kept close to the patient.

Children

The incidence of cardiac arrest after cardiac surgery in children is 4%⁴¹¹ and survival rates are similar to those of adults. The causes are also similar, although one case-series documented primary respiratory arrest in 11%. The guidance given in this section is equally applicable to children, with appropriate modification of defibrillation energy and drug doses (see Section 6 Paediatric Life Support).^{411a} Use extreme caution and check doses carefully when giving intravenous adrenaline doses to children in cardiac arrest after cardiac surgery. Use smaller doses of adrenaline in this setting (e.g., 1 µg kg⁻¹) under the guidance of experienced clinicians.

Internal defibrillation

Internal defibrillation using paddles applied directly across the ventricles requires considerably less energy than that used for external defibrillation. Biphasic shocks are more effective than monophasic shocks for direct defibrillation.⁴¹² For biphasic shocks, starting at 5 J creates the optimum conditions for lowest threshold and cumulative energy, whereas 10–20 J offers optimum conditions for more rapid defibrillation and fewer shocks,⁴¹² thus 20 J is the most applicable energy in an arrest situation, whereas 5 J would be adequate if the patient has been placed on cardiopulmonary bypass.

Continuing cardiac compressions using the internal paddles whilst charging the defibrillator and delivering the shock during the decompression phase of compressions may improve shock success.^{413,414}

It is acceptable to perform external defibrillation after emergency re-sternotomy. Apply external pads preoperatively to all patients undergoing re-sternotomy surgery.⁴¹⁵ Use the defibrillation energy level indicated in the universal algorithm. If the sternum is widely open the impedance may be significantly increased – if external defibrillation is chosen over internal defibrillation close the sternal retractor before shock delivery.

8i. Traumatic cardiorespiratory arrest

Introduction

Cardiac arrest caused by trauma has a very high mortality, with an overall survival of just 5.6% (range 0–17%) (Table 8.4).^{416–422} For reasons that are unclear, reported survival rates in the last 5 years are better than reported previously (Table 8.4) In those who survive (and where data are available) neurological outcome is good in only 1.6% of those sustaining traumatic cardiorespiratory arrest (TCRA).

Diagnosis of traumatic cardiorespiratory arrest

The diagnosis of TCRA is made clinically: the trauma patient is unresponsive, apnoeic and pulseless. Both asystole and organised cardiac activity without cardiac output are regarded as TCRA.

Table 8.4
Survival after out of hospital traumatic cardiac arrest.

Source	Entry criteria: children or adults requiring CPR before or on hospital admission	Number of patients/survivors/ good neurological outcome	Penetrating/ survivors/good neurological outcome	Blunt/survivors/ good neurological outcome
Shimazu and Shatney ⁴¹⁷	TCRA on admission	267 7 4		
Rosemurgy et al. ⁴¹⁶	CPR before admission	138 0 0	42 0 0	96 0 0
Bouillon et al. ⁴²⁹	CPR on scene	224 4 3		
Battistella et al. ⁴¹⁸	CPR at scene, en route or in the ED	604 16 9	300 12 9	304 4 0
Fisher and Worthen ⁴³⁰	Children requiring CPR before or on admission after blunt trauma	65 1 0		65 1 0
Hazinski et al. ⁴³¹	Children requiring CPR or being severely hypotensive on admission after blunt trauma	38 1 0		38 1 0
Stratton et al. ⁴²²	Unconscious, pulseless at scene	879 9 3	497 4 3	382 5 0
Calkins et al. ⁴³²	Children requiring CPR after blunt trauma	25 2 2		25 2 2
Yanagawa et al. ⁴³³	OHCA in blunt trauma	332 6 0		332 6 0
Pickens et al. ⁴³⁴	CPR on scene	184 14 9	94 9 5	90 5 4
Di Bartolomeo et al. ⁴³⁵	CPR on scene	129 2 0		
Willis et al. ⁴³⁶	CPR on scene	89 4 4	18 2 2	71 2 2
David et al. ⁴³⁷	CPR on scene	268 5 1		
Crewdson et al. ⁴³⁸	80 children who required CPR on scene after trauma	80 7 3	7 0 0	73 7 3
Huber-Wagner et al. ⁴³⁹	CPR on scene or after arrival	757 130 28	43 ? ?	714 ? ?
Pasquale et al. ⁴²¹	CPR before or on hospital admission	106 3	21 1	85 2
Lockey et al. ⁴⁴⁰	CPR on scene	871 68	114 9	757 59
Cera et al. ⁴⁴¹	CPR on admission	161 15		
Totals		5217 293 (5.6%)	1136 37 (3.3%)	3032 94 (3.1%)

Commotio cordis

Commotio cordis is actual or near cardiac arrest caused by a blunt impact to the chest wall over the heart.^{423–427} A blow to the chest during the vulnerable phase of the cardiac cycle may cause

malignant arrhythmias (usually ventricular fibrillation). Syncope after chest wall impact may be caused by non-sustained arrhythmic events. Commotio cordis occurs mostly during sports (most commonly baseball) and recreational activities and victims are usually young males (mean age 14 years). In a series of 1866 cardiac arrests

in athletes in Minneapolis, 65 (3%) were due to commotio cordis.⁴²⁸ The registry is accruing 5–15 cases of commotio cordis each year. The overall survival rate from commotio cordis is 15%, but 25% if resuscitation is started within 3 min.⁴²⁷

Trauma secondary to medical events

A cardiorespiratory arrest due to a medical condition (e.g., cardiac arrhythmia, hypoglycaemia, seizure) can cause a secondary traumatic event (e.g., fall, road traffic accident etc). Despite the initial reported mechanism, traumatic injuries may not be the primary cause of a cardiorespiratory arrest and standard advanced life support, including chest compressions, may be appropriate.

Mechanism of injury

Blunt trauma

Of 3032 patients with cardiac arrest after blunt trauma, 94 (3.1%) survived. Only 15 out of 1476 patients (1%) were reported to have a good neurological outcome (Table 8.4).

Penetrating trauma

Of 1136 patients with cardiac arrest after penetrating injury, there were 37 (3.3%) survivors 19 of which (1.9%) had a good neurological outcome (Table 8.4).

A confounding factor in both blunt and penetrating trauma survival rates is that some studies report survival including those pronounced dead on scene and others do not.

Signs of life and initial ECG activity

There are no reliable predictors of survival for TCRA. One study reported that the presence of reactive pupils and sinus rhythm correlate significantly with survival.⁴⁴¹ In a study of penetrating trauma, pupil reactivity, respiratory activity and sinus rhythm were correlated with survival but were unreliable.⁴²² Three studies reported no survivors in patients presenting with asystole or agonal rhythms.^{418,422,442} Another reported no survivors in PEA after blunt trauma.⁴⁴³ Based on these studies, the American College of Surgeons and the National Association of EMS physicians produced pre-hospital guidelines on withholding resuscitation.⁴⁴⁴ They recommend withholding resuscitation in:

- (i) blunt trauma patients presenting with apnoea, pulselessness and without organised ECG activity;
- (ii) penetrating trauma patients found apnoeic and pulseless after rapid assessment for signs of life such as pupillary reflexes, spontaneous movement, or organised ECG activity.

Three retrospective studies question these recommendations and report survivors who would have had resuscitation withheld if the guidelines had been followed.^{434,436,440}

Treatment

Survival from TCRA is correlated with duration of CPR and pre-hospital time.^{420,445–449} Prolonged CPR is associated with a poor outcome; the maximum CPR time associated with favourable outcome is 16 min.^{420,445–447} The level of pre-hospital intervention will depend on the skills of local EMS providers, but treatment on scene should focus on good quality BLS and ALS and exclusion of reversible causes. Look for and treat any medical condition that may have precipitated the trauma event. Undertake only essential life-saving interventions on scene and, if the patient has signs of

life, transfer rapidly to the nearest appropriate hospital. Consider on scene thoracotomy for appropriate patients.^{450,451} Do not delay for unproven interventions such as spinal immobilisation.⁴⁵²

1. Treatment of reversible causes:

- Hypoxaemia (oxygenation, ventilation).
- Compressible haemorrhage (pressure, pressure dressings, tourniquets, novel haemostatic agents).
- Non-compressible haemorrhage (splints, intravenous fluid).
- Tension pneumothorax (chest decompression).
- Cardiac tamponade (immediate thoracotomy)

2. Chest compressions: although they may not be effective in hypovolaemic cardiac arrest most survivors do not have hypovolaemia and in this subgroup standard advanced life support may be life-saving.

3. Standard CPR should not delay the treatment of reversible causes (e.g., thoracotomy for cardiac tamponade).

Resuscitative thoracotomy

Pre-hospital

Resuscitative thoracotomy has been reported as futile if out of hospital time has exceeded 30 min⁴⁴⁸; others consider thoracotomy to be futile in patients with blunt trauma requiring more than 5 min of pre-hospital CPR and in patients with penetrating trauma requiring more than 15 min of CPR.⁴⁴⁹ With these time limits in mind, one UK service recommends that if surgical intervention cannot be accomplished within 10 min after loss of pulse in patients with penetrating chest injury, on scene thoracotomy should be considered.⁴⁵⁰ Based on this approach, of 71 patients who underwent thoracotomy at scene, thirteen patients survived and eleven of these made a good neurological recovery.⁴⁵³ In contrast, pre-hospital thoracotomy for 34 patients with blunt trauma in Japan has not produced any survivors.⁴⁵⁴

Hospital

A relatively simple technique for resuscitative thoracotomy has been described recently.^{451,455}

The American College of Surgeons has published practice guidelines for emergency department thoracotomy (EDT) based on a meta-analysis of 42 outcome studies including 7035 EDT's.⁴⁵⁶ The overall survival rate was 7.8% and, of 226 survivors (5%), only 34 (15%) had a neurological deficit. The investigators concluded that EDT:

1. After blunt trauma, should be limited to those with vital signs on arrival and a witnessed cardiac arrest (estimated survival rate 1.6%).
2. Is best applied to patients with penetrating cardiac injuries who arrive at the trauma centre after a short on scene and transport time with witnessed signs of life or ECG activity (estimated survival rate 31%).
3. Should be undertaken in penetrating non-cardiac thoracic injuries even though survival rates are low.
4. Should be undertaken in patients with exsanguinating abdominal vascular injury even though survival rates are low. This procedure should be used as an adjunct to definitive repair of abdominal vascular injury.

One European study reports a survival rate of 10% in blunt trauma patients undergoing EDT within twenty min after witnessed cardiac arrest. Three of the four survivors had intrabdominal haemorrhage. They conclude that in moribund patients with blunt chest or abdominal trauma EDT should be performed as early as possible.⁴⁵⁷

Airway management

Effective airway management is essential to maintain oxygenation of the severely compromised trauma patient. In one study, tracheal intubation on scene of patients with TCRA doubled the tolerated period of CPR before emergency department thoracotomy – the mean duration of CPR for survivors who were intubated in the field was 9.1 versus 4.2 min for those who were not intubated.⁴⁴⁷

Tracheal intubation in trauma patients is a difficult procedure with a high failure rate if carried out by less experienced care providers.^{458–462} Use basic airway management manoeuvres and alternative airways to maintain oxygenation if tracheal intubation cannot be accomplished immediately. If these measures fail a surgical airway is indicated.

Ventilation

In low cardiac output conditions, positive pressure ventilation causes further circulatory depression, or even cardiac arrest, by impeding venous return to the heart.⁴⁶³ Monitor ventilation with continuous waveform capnography and adjust to achieve normocapnia. This may enable slow respiratory rates and low tidal volumes and the corresponding decrease in transpulmonary pressure may increase venous return and cardiac output.

Chest decompression

Effective decompression of a tension pneumothorax can be achieved quickly by lateral or anterior thoracostomy, which, in the presence of positive pressure ventilation, is likely to be more effective than needle thoracostomy and quicker than inserting a chest tube.⁴⁶⁴

Effectiveness of chest compressions in TCRA

In hypovolaemic cardiac arrest, chest compressions are unlikely to be as effective as in cardiac arrest from other causes.⁴⁶⁵ However most survivors of TCRA have reasons other than pure hypovolaemia for their arrest and these patients may benefit from standard advanced life support interventions.^{436,438,440} Patients with cardiac tamponade are also less likely to benefit from chest compressions and should, where possible, have immediate surgical release of tamponade. Return of spontaneous circulation with advanced life support in patients with TCRA has been described and chest compressions are still the standard of care in patients with cardiac arrest irrespective of aetiology.

Haemorrhage control

Early haemorrhage control is vital. Handle the patient gently at all times to prevent clot disruption. Apply external compression, and pelvic and limb splints when appropriate. Delays in surgical haemostasis are disastrous for patients with exsanguinating trauma. Recent conflicts have seen a resurgence in the use of tourniquets to stop life-threatening limb haemorrhage.⁴⁶⁶ It is unlikely that the same benefits will be seen in civilian trauma practice.

Pericardiocentesis

In patients with suspected trauma-related cardiac tamponade, needle pericardiocentesis is probably not a useful procedure.⁴⁶⁷ There is no evidence of benefit in the literature. It may increase scene time, can cause myocardial injury and delays effective therapeutic measures such as emergency thoracotomy.

Fluids and blood transfusion on scene

Fluid resuscitation of trauma patients before haemorrhage is controlled is controversial and there is no clear consensus on when it should be started and what fluids should be given.^{468,469} Limited evidence and general consensus support a more conservative approach to intravenous fluid infusion, with permissive hypotension until surgical haemostasis is achieved.^{470,471} In the UK, the National Institute for Clinical Excellence (NICE) has published guidelines on pre-hospital fluid replacement in trauma.⁴⁷² The recommendations include giving 250 ml boluses of crystalloid solution until a radial pulse is achieved and not delaying rapid transport of trauma victims for fluid infusion in the field. Pre-hospital fluid therapy may have a role in prolonged entrapments but there is no reliable evidence for this.^{473,474}

Ultrasound

Ultrasound is a valuable tool in the evaluation of the compromised trauma patient. Haemoperitoneum, haemo- or pneumothorax and cardiac tamponade can be diagnosed reliably in minutes even in the pre-hospital phase.⁴⁷⁵ Diagnostic peritoneal lavage and needle pericardiocentesis have virtually disappeared from clinical practice since the introduction of sonography in trauma care. Pre-hospital ultrasound is now available, although its benefits are yet to be proven.⁴⁷⁶

Vasopressors

The possible role of vasopressors (e.g., vasopressin) in trauma resuscitation is unclear and is based mainly on case reports.⁴⁷⁷

8j. Cardiac arrest associated with pregnancy

Overview

Mortality related to pregnancy in developed countries is rare, occurring in an estimated 1:30,000 deliveries.⁴⁷⁸ The fetus must always be considered when an adverse cardiovascular event occurs in a pregnant woman. Fetal survival usually depends on maternal survival. Resuscitation guidelines for pregnancy are based largely on case series, extrapolation from non-pregnant arrests, manikin studies and expert opinion based on the physiology of pregnancy and changes that occur in normal labour. Studies tend to address causes in developed countries, whereas the most pregnancy-related deaths occur in developing countries. There were an estimated 342,900 maternal deaths (death during pregnancy, childbirth, or in the 42 days after delivery) worldwide in 2008.⁴⁷⁹

Significant physiological changes occur during pregnancy, e.g., cardiac output, blood volume, minute ventilation and oxygen consumption all increase. Furthermore, the gravid uterus can cause significant compression of iliac and abdominal vessels when the mother is in the supine position, resulting in reduced cardiac output and hypotension.

Causes

There are many causes of cardiac arrest in pregnant women. A review of nearly 2 million pregnancies in the UK⁴⁸⁰ showed that maternal deaths (death during pregnancy, childbirth, or in the 42 days after delivery) between 2003 and 2005 were associated with:

- cardiac disease;
- pulmonary embolism;

- psychiatric disorders;
- hypertensive disorders of pregnancy;
- sepsis;
- haemorrhage;
- amniotic-fluid embolism;
- ectopic pregnancy.

Pregnant women can also sustain cardiac arrest from the same causes as women of the same age group.

Key interventions to prevent cardiac arrest

In an emergency, use an ABCDE approach. Many cardiovascular problems associated with pregnancy are caused by aortocaval compression. Treat a distressed or compromised pregnant patient as follows:

- Place the patient in the left lateral position or manually and gently displace the uterus to the left.
- Give high-flow oxygen guided by pulse oximetry.
- Give a fluid bolus if there is hypotension or evidence of hypovolaemia.
- Immediately re-evaluate the need for any drugs being given.
- Seek expert help early. Obstetric and neonatal specialists should be involved early in the resuscitation.
- Identify and treat the underlying cause.

Modifications to BLS guidelines for cardiac arrest

After 20 weeks gestation, the pregnant woman's uterus can press down against the inferior vena cava and the aorta, impeding venous return and cardiac output. Uterine obstruction of venous return can cause pre-arrest hypotension or shock and, in the critically ill patient, may precipitate arrest.^{481,482} After cardiac arrest, the compromise in venous return and cardiac output by the gravid uterus limits the effectiveness of chest compressions.

Non-arrest studies show that left lateral tilt improves maternal blood pressure, cardiac output and stroke volume^{483–485} and improves fetal oxygenation and heart rate.^{486–488} Two studies found no improvement in maternal or fetal variables with 10–20° left lateral tilt.^{489,490} One study found more aortic compression at 15° left lateral tilt when compared with a full left lateral tilt.⁴⁸⁴ Aortic compression has been found to persist at over 30° of tilt.⁴⁹¹ Two non-arrest studies show that manual left uterine displacement with the patient supine is as good as or better than left lateral tilt in relieving aortocaval compression, as assessed by the incidence of hypotension and ephedrine use.^{492,493} Non-cardiac arrest data show that the gravid uterus can be shifted away from the cava in most cases by placing the patient in 15° of left lateral decubitus position.⁴⁹⁴ The value of relieving aortic or caval compression during CPR is, however, unknown.

Unless the pregnant victim is on a tilting operating table, left lateral tilt is not easy to perform whilst maintaining good quality chest compressions. A variety of methods to achieve a left lateral tilt have been described including placing the victim on the rescuers knees⁴⁹⁵, pillows or blankets, and the Cardiff wedge⁴⁹⁶ although their efficacy in actual cardiac arrests is unknown. Even when a tilting table is used, the angle of tilt is often overestimated.⁴⁹⁷ In a manikin study, the ability to provide effective chest compressions decreased as the angle of left lateral tilt increased and that at an angle of greater than 30° the manikin tended to roll.⁴⁹⁶

The key steps for BLS in a pregnant patient are:

- Call for expert help early (including an obstetrician and neonatologist).

- Start basic life support according to standard guidelines. Ensure good quality chest compressions with minimal interruptions.
- Manually displace the uterus to the left to remove caval compression.
- Add left lateral tilt if this is feasible – the optimal angle of tilt is unknown. Aim for between 15° and 30°. Even a small amount of tilt may be better than no tilt. The angle of tilt used needs to allow good quality chest compressions and if needed allow Caesarean delivery of the fetus.
- Start preparing for emergency Caesarean section (see below) – the fetus will need to be delivered if initial resuscitation efforts fail.

Modifications to advanced life support

There is a greater potential for gastro-oesophageal sphincter insufficiency and risk of pulmonary aspiration of gastric contents. Early tracheal intubation with correctly applied cricoid pressure decreases this risk. Tracheal intubation will make ventilation of the lungs easier in the presence of increased intra-abdominal pressure.

A tracheal tube 0.5–1 mm internal diameter (ID) smaller than that used for a non-pregnant woman of similar size may be necessary because of maternal airway narrowing from oedema and swelling.⁴⁹⁸ One study documented that the upper airways in the third trimester of pregnancy are narrower compared with their postpartum state and to non-pregnant controls.⁴⁹⁹ Tracheal intubation may be more difficult in the pregnant patient.⁵⁰⁰ Expert help, a failed intubation drill and the use of alternative airway devices may be needed (see Section 4).^{24a,501}

There is no change in transthoracic impedance during pregnancy, suggesting that standard shock energies for defibrillation attempts should be used in pregnant patients.⁵⁰² There is no evidence that shocks from a direct current defibrillator have adverse effects on the fetal heart. Left lateral tilt and large breasts will make it difficult to place an apical defibrillator paddle. Adhesive defibrillator pads are preferable to paddles in pregnancy.

Reversible causes

Rescuers should attempt to identify common and reversible causes of cardiac arrest in pregnancy during resuscitation attempts. The 4 Hs and 4 Ts approach helps identify all the common causes of cardiac arrest in pregnancy. Pregnant patients are at risk of all the other causes of cardiac arrest for their age group (e.g., anaphylaxis, drug overdose, trauma). Consider the use of abdominal ultrasound by a skilled operator to detect pregnancy and possible causes during cardiac arrest in pregnancy; however, do not delay other treatments. Specific causes of cardiac arrest in pregnancy include the following.

Haemorrhage

Life-threatening haemorrhage can occur both antenatally and postnatally. Postpartum haemorrhage is the commonest single cause of maternal death worldwide and is estimated to cause one maternal death every 7 min.⁵⁰³ Associations include ectopic pregnancy, placental abruption, placenta praevia, placenta accreta, and uterine rupture.⁴⁸⁰ A massive haemorrhage protocol must be available in all units and should be updated and rehearsed regularly in conjunction with the blood bank. Women at high risk of bleeding should be delivered in centres with facilities for blood transfusion, intensive care and other interventions, and plans should be made in advance for their management. Treatment is based on an ABCDE approach. The key step is to stop the bleeding. Consider the following:

- fluid resuscitation including use of rapid transfusion system and cell salvage⁵⁰⁴;
- oxytocin and prostaglandin analogues to correct uterine atony⁵⁰⁵;
- massaging the uterus⁵⁰⁶;
- correction of coagulopathy including use of tranexamic acid or recombinant activated factor VII^{507–509};
- uterine balloon tamponade^{510,511};
- uterine compression sutures⁵¹²;
- angiography and endovascular embolization⁵¹³;
- hysterectomy^{514,515};
- aortic cross-clamping in catastrophic haemorrhage.⁵¹⁶

Cardiovascular disease

Myocardial infarction and aneurysm or dissection of the aorta or its branches, and peripartum cardiomyopathy cause most deaths from acquired cardiac disease.^{517,518} Patients with known cardiac disease need to be managed in a specialist unit. Pregnant women may develop an acute coronary syndrome, typically in association with risk factors such as obesity, older age, higher parity, smoking, diabetes, pre-existing hypertension and a family history of ischaemic heart disease.^{480,519} Pregnant patients can have atypical features such as epigastric pain and vomiting. Percutaneous coronary intervention (PCI) is the reperfusion strategy of choice for ST-elevation myocardial infarction in pregnancy. Thrombolysis should be considered if urgent PCI is unavailable. A review of 200 cases of thrombolysis for massive pulmonary embolism in pregnancy reported a maternal death rate of 1% and concluded that thrombolytic therapy is reasonably safe in pregnancy.⁵²⁰

Increasing numbers of women with congenital heart disease are becoming pregnant.⁵²¹ Heart failure and arrhythmias are the commonest problems, especially in those with cyanotic heart disease. Pregnant women with known congenital heart disease should be managed in specialist centres.

Pre-eclampsia and eclampsia

Eclampsia is defined as the development of convulsions and/or unexplained coma during pregnancy or postpartum in patients with signs and symptoms of pre-eclampsia.^{522,523} Magnesium sulphate is effective in preventing approximately half of the cases of eclampsia developing in labour or immediately postpartum in women with pre-eclampsia.^{524–526}

Pulmonary embolism

The estimated incidence of pulmonary embolism is 1–1.5 per 10,000 pregnancies, with a case fatality of 3.5% (95% CI 1.1–8.0%).⁵²⁷ Risk factors include obesity, increased age, and immobility. Successful use of fibrinolytics for massive, life-threatening pulmonary embolism in pregnant women has been reported.^{520,528–531}

Amniotic-fluid embolism

Amniotic-fluid embolism usually presents around the time of delivery with sudden cardiovascular collapse, breathlessness, cyanosis, arrhythmias, hypotension and haemorrhage associated with disseminated intravascular coagulopathy.⁵³² Patients may have warning signs preceding collapse including breathlessness, chest pain, feeling cold, light-headedness, distress, panic, a feeling of pins and needles in the fingers, nausea, and vomiting.

The UK Obstetric Surveillance System identified 60 cases of amniotic-fluid embolism between 2005 and 2009. The reported incidence was 2.0 per 100,000 deliveries (95% CI 1.5–2.5%)⁵³³ The case fatality is 13–30% and perinatal mortality is 9–44%.⁵³²

Amniotic-fluid embolism was associated with induction of labour, multiple pregnancy, older, and ethnic-minority women. Caesarean delivery was associated with postnatal amniotic-fluid embolism.

Treatment is supportive, as there is no specific therapy based on an ABCDE approach and correction of coagulopathy. Successful use of extracorporeal life support techniques for women suffering life-threatening amniotic-fluid embolism during labour and delivery is reported.⁵³⁴

If immediate resuscitation attempts fail

Consider the need for an emergency hysterotomy or Caesarean section as soon as a pregnant woman goes into cardiac arrest. In some circumstances immediate resuscitation attempts will restore a perfusing rhythm; in early pregnancy this may enable the pregnancy to proceed to term. When initial resuscitation attempts fail, delivery of the fetus may improve the chances of successful resuscitation of the mother and fetus.^{535–537} One systematic review documented 38 cases of Caesarean section during CPR, with 34 surviving infants and 13 maternal survivors at discharge, suggesting that Caesarean section may have improved maternal and neonatal outcomes.⁵³⁸ The best survival rate for infants over 24–25 weeks gestation occurs when delivery of the infant is achieved within 5 min after the mother's cardiac arrest.^{535,539–541} This requires that the provider commence the hysterotomy at about 4 min after cardiac arrest. At older gestational ages (30–38 weeks), infant survival is possible even when delivery was after 5 min from the onset of maternal cardiac arrest.⁵³⁸ A case-series suggests increased use of Caesarean section during CPR with team training⁵⁴²; in this series no deliveries were achieved within 5 min after starting resuscitation. Eight of the twelve women had ROSC after delivery, with two maternal and five newborn survivors. Maternal case fatality rate was 83%. Neonatal case fatality rate was 58%.⁵⁴²

Delivery will relieve caval compression and may improve chances of maternal resuscitation. The Caesarean delivery also enables access to the infant so that newborn resuscitation can begin.

Decision-making for emergency hysterotomy (Caesarean section)

The gravid uterus reaches a size that will begin to compromise aortocaval blood flow at approximately 20 weeks gestation; however, fetal viability begins at approximately 24–25 weeks.⁵⁴³ Portable ultrasound is available in some emergency departments and may aid in determination of gestational age (in experienced hands) and positioning, provided its use does not delay the decision to perform emergency hysterotomy.⁵⁴⁴ Aim for delivery within 5 min of onset of cardiac arrest. This will mean that Caesarean section needs to ideally take place where the cardiac arrest has occurred to avoid delays.

- At gestational age less than 20 weeks, urgent Caesarean delivery need not be considered, because a gravid uterus of this size is unlikely to significantly compromise maternal cardiac output.
- At gestational age approximately 20–23 weeks, initiate emergency hysterotomy to enable successful resuscitation of the mother, not survival of the delivered infant, which is unlikely at this gestational age.
- At gestational age approximately \geq 24–25 weeks, initiate emergency hysterotomy to save the life of both the mother and the infant.

Post-resuscitation care

Post-resuscitation care should follow standard guidelines. Therapeutic hypothermia has been used safely and effectively in early

pregnancy with fetal heart monitoring and resulted in favourable maternal and fetal outcome after a term delivery.^{544a} Implantable cardioverter defibrillators (ICDs) have been used in patients during pregnancy.⁵⁴⁵

Preparation for cardiac arrest in pregnancy

Advanced life support in pregnancy requires coordination of maternal resuscitation, Caesarean delivery of the fetus and newborn resuscitation ideally within 5 min. To achieve this, units likely to deal with cardiac arrest in pregnancy should:

- have plans and equipment in place for resuscitation of both the pregnant woman and newborn;
- ensure early involvement of obstetric, anaesthetic and neonatal teams;
- ensure regular training in obstetric emergencies.⁵⁴⁶

8k. Electrocution

Introduction

Electrical injury is a relatively infrequent but potentially devastating multisystem injury with high morbidity and mortality, causing 0.54 deaths per 100,000 people each year. Most electrical injuries in adults occur in the workplace and are associated generally with high voltage, whereas children are at risk primarily at home, where the voltage is lower (220 V in Europe, Australia and Asia; 110 V in the USA and Canada).⁵⁴⁷ Electrocution from lightning strikes is rare, but worldwide it causes 1000 deaths each year.⁵⁴⁸

Electric shock injuries are caused by the direct effects of current on cell membranes and vascular smooth muscle. The thermal energy associated with high-voltage electrocution will also cause burns. Factors influencing the severity of electrical injury include whether the current is alternating (AC) or direct (DC), voltage, magnitude of energy delivered, resistance to current flow, pathway of current through the patient, and the area and duration of contact. Skin resistance is decreased by moisture, which increases the likelihood of injury. Electric current follows the path of least resistance; conductive neurovascular bundles within limbs are particularly prone to damage.

Contact with AC may cause tetanic contraction of skeletal muscle, which may prevent release from the source of electricity. Myocardial or respiratory failure may cause immediate death.

- Respiratory arrest may be caused by paralysis of the central respiratory control system or the respiratory muscles.
- Current may precipitate VF if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon).⁵⁴⁹ Electrical current may also cause myocardial ischaemia because of coronary artery spasm. Asystole may be primary, or secondary to asphyxia following respiratory arrest.

Current that traverses the myocardium is more likely to be fatal. A transthoracic (hand-to-hand) pathway is more likely to be fatal than a vertical (hand-to-foot) or straddle (foot-to-foot) pathway. There may be extensive tissue destruction along the current pathway.

Lightning strike

Lightning strikes deliver as much as 300 kV over a few milliseconds. Most of the current from a lightning strike passes over the surface of the body in a process called 'external flashover'. Both industrial shocks and lightning strikes cause deep burns at the point of contact. For industrial shocks the points of contact are usually on

the upper limbs, hands and wrists, whereas for lightning they are mostly on the head, neck and shoulders. Injury may also occur indirectly through ground current or current "splashing" from a tree or other object that is hit by lightning.⁵⁵⁰ Explosive force may cause blunt trauma.⁵⁵¹ The pattern and severity of injury from a lightning strike varies considerably, even among affected individuals from a single group.^{552–554} As with industrial and domestic electric shock, death is caused by cardiac^{553–557} or respiratory arrest.^{550,558} In those who survive the initial shock, extensive catecholamine release or autonomic stimulation may occur, causing hypertension, tachycardia, non-specific ECG changes (including prolongation of the QT interval and transient T-wave inversion), and myocardial necrosis. Creatine kinase may be released from myocardial and skeletal muscle. Lightning can also cause central and peripheral nerve damage; brain haemorrhage and oedema, and peripheral nerve injury are common. Mortality from lightning injuries is as high as 30%, with up to 70% of survivors sustaining significant morbidity.^{559–561}

Diagnosis

The circumstances surrounding the incident are not always known. Unconscious patients with linear or punctuate burns or feathering should be treated as a victims of lightning strike.⁵⁵⁰

Rescue

Ensure that any power source is switched off and do not approach the casualty until it is safe. High-voltage (above domestic mains) electricity can arc and conduct through the ground for up to a few metres around the casualty. It is safe to approach and handle casualties after lightning strike, although it would be wise to move to a safer environment, particularly if lightning has been seen within 30 min.⁵⁵⁰

Resuscitation

Start standard basic and advanced life support without delay.

- Airway management may be difficult if there are electrical burns around the face and neck. Early tracheal intubation is needed in these cases, as extensive soft-tissue oedema may develop causing airway obstruction. Head and spine trauma can occur after electrocution. Immobilise the spine until evaluation can be performed.
- Muscular paralysis, especially after high voltage, may persist for several hours⁵⁶⁰; ventilatory support is required during this period.
- VF is the commonest initial arrhythmia after high-voltage AC shock; treat with prompt attempted defibrillation. Asystole is more common after DC shock; use standard protocols for this and other arrhythmias.
- Remove smouldering clothing and shoes to prevent further thermal injury.
- Vigorous fluid therapy is required if there is significant tissue destruction. Maintain a good urine output to enhance the excretion of myoglobin, potassium and other products of tissue damage.⁵⁵⁷
- Consider early surgical intervention in patients with severe thermal injuries.
- Maintain spinal immobilisation if there is a likelihood of head or neck trauma.^{562,563}
- Conduct a thorough secondary survey to exclude traumatic injuries caused by tetanic muscular contraction or by the person being thrown.^{563,564}

- Electrocutation can cause severe, deep soft-tissue injury with relatively minor skin wounds, because current tends to follow neurovascular bundles; look carefully for features of compartment syndrome, which will necessitate fasciotomy.

Patients struck by lightning are most likely to die if they sustain immediate cardiac or respiratory arrest and are not treated rapidly. When multiple victims are struck simultaneously by lightning, rescuers should give highest priority to patients in respiratory or cardiac arrest. Victims with respiratory arrest may require only ventilation to avoid secondary hypoxic cardiac arrest. Resuscitative attempts may have higher success rates in lightning victims than in patients with cardiac arrest from other causes, and efforts may be effective even when the interval before the resuscitative attempt is prolonged.⁵⁵⁸ Dilated or non-reactive pupils should never be used as a prognostic sign, particularly in patients suffering a lightning strike.⁵⁵⁹

There are conflicting reports on the vulnerability of the fetus to electric shock. The clinical spectrum of electrical injury ranges from a transient unpleasant sensation for the mother with no effect on her fetus, to fetal death either immediately or a few days later. Several factors, such as the magnitude of the current and the duration of contact, are thought to affect outcome.⁵⁶⁵

Further treatment and prognosis

Immediate resuscitation of young victims in cardiac arrest from electrocution can result in long-term survival. Successful resuscitation has been reported after prolonged life support. All those who survive electrical injury should be monitored in hospital if they have a history of cardiorespiratory problems or have had:

- loss of consciousness;
- cardiac arrest;
- electrocardiographic abnormalities;
- soft-tissue damage and burns.

Severe burns (thermal or electrical), myocardial necrosis, the extent of central nervous system injury, and secondary multisystem organ failure determine the morbidity and long-term prognosis. There is no specific therapy for electrical injury, and the management is symptomatic. Prevention remains the best way to minimize the prevalence and severity of electrical injury.

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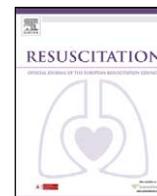
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European Resuscitation Council Guidelines for Resuscitation 2010 Section 9. Principles of education in resuscitation

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Introduction

Survival from cardiac arrest is determined by the quality of the scientific evidence behind the guidelines, the effectiveness of education and the resources for implementation of the guidelines.¹ An additional factor is how readily guidelines can be applied in clinical practice and the effect of human factors on putting the theory into practice.² Implementation of Guidelines 2010 is likely to be more successful with a carefully planned, comprehensive implementation strategy that includes education. Delays in providing training materials and freeing staff for training were cited as reasons for delays in the implementation of the 2005 guidelines.^{3,4}

This chapter includes the key educational issues identified by the International Liaison Committee on Resuscitation (ILCOR) evidence evaluation,⁵ discusses the scientific basis of basic and advanced level resuscitation training and provides an update on the European Resuscitation Council (ERC) life support courses.⁶

Key educational recommendations

The key issues identified by the Education, Implementation and Teams (EIT) task force of ILCOR during the Guidelines 2010 evidence evaluation process⁵ that are relevant to this chapter are:

- Educational interventions should be evaluated to ensure that they reliably achieve the learning objectives. The aim is to ensure that learners acquire and retain the skills and knowledge that will enable them to act correctly in actual cardiac arrests and improve patient outcomes.
- Short video/computer self-instruction courses, with minimal or no instructor coaching, combined with hands-on practice can be considered as an effective alternative to instructor-led basic life support (cardiopulmonary resuscitation [CPR] and automated external defibrillator [AED]) courses.
- Ideally all citizens should be trained in standard CPR that includes compressions and ventilations. There are circumstances however where training in compression-only CPR is appropriate (e.g., opportunistic training with very limited time). Those trained in compression-only CPR should be encouraged to learn standard CPR.
- Basic and advanced life support knowledge and skills deteriorate in as little as three to six months. The use of frequent assessments will identify those individuals who require refresher training to help maintain their knowledge and skills.
- CPR prompt or feedback devices improve CPR skill acquisition and retention and should be considered during CPR training for laypeople and healthcare professionals.
- An increased emphasis on non-technical skills (NTS) such as leadership, teamwork, task management and structured communication will help improve the performance of CPR and patient care.
- Team briefings to plan for resuscitation attempts, and debriefings based on performance during simulated or actual resuscitation

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attempts should be used to help improve resuscitation team and individual performance.

- Research about the impact of resuscitation training on actual patient outcomes is limited. Although manikin studies are useful, researchers should be encouraged to study and report the impact of educational interventions on actual patient outcomes.

Who and how to train

Ideally all citizens should have some knowledge of CPR. There is insufficient evidence for or against the use of training interventions that focus on high risk populations. However, training can reduce family member and, or patient anxiety, improve emotional adjustment and empowers individuals to feel that they would be able to start CPR.⁵

People that require resuscitation training range from laypeople, those without formal healthcare training but with a role that places a duty of care upon them (e.g., lifeguards, first aiders), and healthcare professionals working in a variety of settings including the community, emergency medical systems (EMS), general hospital wards and critical care areas.

Training should be tailored to the needs of different types of learners and learning styles to ensure acquisition and retention of resuscitation knowledge and skills. Those who are expected to perform CPR regularly need to have knowledge of current guidelines and be able to use them effectively as part of a multi-professional team. These individuals require more complex training including both technical and non-technical skills (e.g., teamwork, leadership, structured communication skills).^{7,8} In the next section we have arbitrarily divided these into basic level and advanced level training interventions whereas in truth this is a continuum. Most research in this area is based on training rescuers in adult resuscitation skills. Much of this research also applies to training in resuscitation of children and of the newborn.

Basic level and AED training

Bystander CPR and early defibrillation saves lives. Many factors decrease the willingness of bystanders to start CPR, including panic, fear of disease, harming the victim or performing CPR incorrectly.^{9–24} Providing CPR training to laypeople increases willingness to perform CPR.^{12,18–20,25–30}

CPR training and doing CPR during an actual cardiac arrest is safe in most circumstances. Individuals undertaking CPR training should be advised of the nature and extent of the physical activity required during the training program. Learners who develop significant symptoms (e.g., chest pain, severe shortness of breath) during CPR training should be advised to stop. Rescuers who develop significant symptoms during actual CPR should consider stopping CPR (see basic life support guidelines for further information about risks to the rescuer).³¹

Basic life support and AED curriculum

The curriculum for basic life support and AED training should be tailored to the target audience and kept as simple as possible. The following should be considered as core elements of the basic life support and AED curriculum^{5,32}:

- Personal and environmental risks before starting CPR.
- Recognition of cardiac arrest by assessment of responsiveness, opening of the airway and assessment of breathing.^{31,32}
- Recognition of gasping or abnormal breathing as a sign of cardiac arrest in unconscious unresponsive individuals.^{33,34}

- Good quality chest compressions (including adherence to rate, depth, full recoil and minimizing hands-off time) and rescue breathing.
- Feedback/prompts (including from devices) during CPR training should be considered to improve skill acquisition and retention during basic life support training.³⁵
- All basic life support and AED training should aim to teach standard CPR including rescue breathing/ventilations. Chest compression-only CPR training has potential advantages over chest compression and ventilation in certain specific situations.^{10,15,18,23,24,27,36,37} An approach to teaching CPR is suggested below.

Standard CPR versus chest compression-only CPR teaching

There is controversy about which CPR skills different types of rescuers should be taught. Compression-only CPR is easier and quicker to teach especially when trying to teach a large number of individuals who would not otherwise access CPR training. In many situations however, standard CPR (which includes ventilation/rescuer breathing) is better, for example in children,³⁸ asphyxial arrests, and when bystander CPR is required for more than a few minutes.³² A simplified, education-based approach is therefore suggested:

- Ideally, full CPR skills (compressions and ventilation using a 30:2 ratio) should be taught to all citizens.
- When training is time-limited or opportunistic (e.g., EMS telephone instructions to a bystander, mass events, publicity campaigns, YouTube 'viral' videos, or the individual does not wish to train), training should focus on chest compression-only CPR.
- For those trained in compression-only CPR, subsequent training should include training in ventilation as well as chest compressions. Ideally these individuals should be trained in compression-only CPR and then offered training in chest compressions with ventilation at the same training session.
- Those laypersons with a duty of care, such as first aid workers, lifeguards, and child minders, should be taught how to do chest compressions and ventilations.
- For children, rescuers should be encouraged to use whichever adult sequence they have been taught, as outcome is worse if they do nothing. Non-specialists who wish to learn paediatric resuscitation because they have responsibility for children (e.g., parents, teachers, school nurses, lifeguards etc), should be taught that it is preferable to modify adult basic life support and give five initial breaths followed by approximately 1 min of CPR before they go for help, if there is no-one to go for them. Chest compression depth for children is at least one-third of the A-P diameter of the chest.³⁹
- Citizen-CPR training should be promoted for all. However being untrained should not be a barrier to performing chest compression-only CPR, preferably with dispatcher telephone advice.

Basic life support and AED training methods

There are numerous methods to deliver basic life support and AED training. Traditional, instructor-led training courses remain the most frequently used method for basic life support and AED training.⁴⁰ When compared with traditional instructor-led training, well designed self-instruction programmes (e.g., video, DVD, computer driven) with minimal or no instructor coaching can be effective alternatives to instructor-led courses for laypeople and healthcare providers learning basic life support and AED skills.^{41–55} It is essential that courses include hands-on practice as part of the programme.

The use of AEDs by individuals without prior formal training can be beneficial and may be life saving.^{45,56–60} Performance in the use of an AED (e.g., speed of use, correct pad placement) can be further improved with brief training of laypeople and healthcare professionals.^{45,50,61,62}

Duration and frequency of instructor-led basic life support and AED training courses

The optimal duration of instructor-led basic life support and AED training courses has not been determined and is likely to vary according to the characteristics of the participants (e.g., lay or healthcare; previous training; age), the curriculum, the ratio of instructors to participants, the amount of hands-on training and the use of end of course assessments.

Most studies show that CPR skills such as calling for help, chest compressions and ventilations decay within three to six months after initial training.^{43,46,63–68} AED skills are retained for longer than basic life support skills alone.^{59,64,69}

CPR performance can be retained or improved with re-evaluation and, if required, a brief refresher, or retraining after as little as three to six months.^{64,70–73}

Use of CPR prompt/feedback devices

The use of CPR prompt/feedback devices may be considered during CPR training for laypeople and healthcare professionals.³⁵ Devices can be prompting (i.e., signal to perform an action e.g., metronome for compression rate or voice feedback), give feedback (i.e., after event information based on effect of an action such as visual display of compression depth), or a combination of prompts and feedback. Training using a prompt/feedback device can improve CPR skill performance, acquisition and retention. In these studies acquisition and retention was measured by testing on a manikin without using the device.^{63,74–78} Instructors and rescuers should be made aware that a compressible support surface (e.g., mattress) can cause a prompt/feedback device to overestimate depth of compression.^{79,80}

Advanced level training

Advanced level training curriculum

Advanced level training is usually for healthcare providers. Curricula should be tailored to match individual learning needs, patient case mix and the individual's role within the healthcare systems response to cardiac arrest. There is limited evidence about specific interventions that enhance learning and retention from advanced level life support courses. The ERC Advanced Life Support (ALS) course following Guidelines 2005 has been shown to reduce "no-flow" fraction but not other elements of quality of CPR performance in cardiac arrest simulations.⁸¹ Increased clinical experience of learners seems to improve long-term retention of knowledge and skills.^{82,83}

Studies of advanced life support in actual or simulated in-hospital arrests,^{84–94} show improved resuscitation team performance when specific team and, or leadership training is added to advanced level courses. Team training and rhythm recognition skills will be essential to minimize hands-off time when using the 2010 manual defibrillation strategy that includes charging during chest compressions.^{95,96}

Core elements for advanced life support curricula should include:

- Cardiac arrest prevention.^{97,98}

- Good quality chest compressions including adherence to rate, depth, full recoil and minimizing hands-off time, and ventilation using basic skills (e.g., pocket mask, bag mask).
- Defibrillation including charging during compressions for manual defibrillation.
- Advanced life support algorithms.
- Non-technical skills (e.g., leadership and team training, communication).

Extended training may cover advanced airway management, management of peri-arrest arrhythmias; resuscitation in special circumstances, vascular access, cardiac arrest drugs, post-resuscitation care and ethics.

Advanced level training methods

Pre-course training

A variety of methods (such as reading manuals, pretests and e-learning can be used to prepare candidates before attending a life support course.^{99–107} A recent large randomized controlled study of use of a commercially available e-learning simulation programme before attending an advanced life support course compared with standard preparation with a course manual showed no improvement in cognitive or psychomotor skills during cardiac arrest simulation testing.^{107,108}

There are numerous studies of alternative teaching methods that claim equivalence or benefit for computer or video-based training and decrease the time instructors spend with learners.^{100,101,106,109–123} Any method of pre-course preparation that is aimed at improving knowledge and skills or reducing instructor to learner face-to-face time should be formally assessed to ensure equivalent or improved learning outcomes compared with standard instructor-led courses. A large multicentre randomised controlled trial to test if a 1-day face-to-face ALS course supplemented by e-learning material is equivalent to the 2-day face-to-face standard ALS course with respect to the course learning outcomes is ongoing [ISRCTN86380392].

Simulation and realistic training techniques

Simulation training is an essential part of resuscitation training. There is large variation in how simulation can be and is used for resuscitation training.¹²⁴ The lack of consistent definitions (e.g., high vs. low fidelity simulation) makes comparisons of studies of different types of simulation training difficult.

Simulation training has fairly consistently,^{33,125–136} although not universally^{137–143} been shown to improve knowledge and skill performance on manikins. Evidence of change in real life performance is more limited. A small number of before and after studies examining the effects of resuscitation training (including simulation) on real life performance have documented improvement in actual patient outcomes.^{144–148} These studies are limited by their inability to separate the effect of simulation training from other educational and temporal factors. One randomised controlled trial and a prospective case control study which allocated participants to simulator or standard resuscitation training showed improved real life performance of those skills.^{127,149}

There are conflicting data on the effect of increasing realism (e.g., use of actual resuscitation settings, high fidelity manikins) on learning, and few data on patient outcomes.^{125,128,133,135,137,138,140,141,150–154} One study reported a significant increase in knowledge when using manikins or live patient models for trauma teaching compared with no manikins or live models.¹⁵³ In this study there was no difference in knowl-

edge acquisition between using manikins or live patient models, although learners preferred using the manikins.

There is insufficient evidence for or against the use of more realistic techniques (e.g., high-fidelity manikins, in situ training) to improve outcomes (e.g., skills performance on manikins, skills performance in real arrests, willingness to perform) when compared with standard training (e.g., low fidelity manikins, education centre) in basic and advanced life support. The incremental cost effectiveness of higher fidelity simulators should be determined.¹⁴¹

Future studies should focus on measuring the effect of training interventions (including simulation) on patient and real life process focused outcomes. Chart note review,¹⁵⁵ quality assurance studies¹⁴⁹ and quality of CPR monitoring technology^{89,156} have confirmed the feasibility of this approach.

Advanced life support training intervals

Knowledge and skill retention declines rapidly after initial resuscitation training. Refresher training is invariably required to maintain knowledge and skills; however, the optimal frequency for refresher training is unclear. Most studies show that ALS skills and knowledge decayed when tested at three to six months after training,^{65,157–164} two studies suggested seven to twelve months,^{165,166} and one study eighteen months.¹⁶⁷

Assessment on advanced level courses

The best method of assessment during courses is unknown. Written tests in ALS courses do not reliably predictor practical skill performance and should not be used as a substitute for demonstration of clinical skill performance.^{168–171} Assessment at the end of training does seem to have a beneficial effect on performance and retention and should be considered.^{172,173}

Alternative strategies that may improve advanced life support performance

Use of checklists and cognitive aids

Cognitive aids such as checklists may be used to improve adherence to guidelines as long as they do not cause delays in starting CPR and the correct checklist is used.^{174–186} Checklists should be tested in simulated resuscitations before implementation.^{84–94}

Mock codes

Mock cardiac arrest codes and drills provide the opportunity to test the individual and system responses to cardiac arrest. Mock codes can improve advanced life support provider knowledge,¹⁸⁷ skill performance,¹⁸⁸ confidence,¹⁸⁹ familiarity with the environment¹⁹⁰ and identify common system and user errors.^{191,192}

Team briefings and debriefings

Briefings and debriefings should be used during both learning and actual clinical activities.

Successful teams such as sports teams meet before and after events. Surveys in the UK^{193,194} and Canada⁹⁰ show that resuscitation teams rarely have formal briefings and debriefings. Debriefings and feedback are two separate but related entities in that various forms of feedback are components of debriefing. Debriefing tends to be face-to-face and involves both parties engaging in discussion. Feedback tends to provide information about prior events and can use several methods (video recordings, defibrillator downloads or trained observer feedback). Debriefing appears to be an effective method for improving resuscitation performance and, potentially, patient outcomes as long as objective data forms the basis for the

discussion.^{87,89,127,129,149,187,195–205} The ideal format for debriefing remains to be determined.

European Resuscitation Council resuscitation courses

The ERC has a portfolio of training courses that aim to equip learners with the ability to undertake resuscitation in a real clinical situation at the level that they would be expected to perform – be they laypeople, first responders in the community or the hospital, or a healthcare professional working for an EMS, on a general ward, in an acute area, or as a member of a resuscitation team.

ERC courses focus on teaching in small groups using interactive discussion and hands-on practice for skills and clinical simulations using resuscitation manikins.^{6,206} Courses have a high ratio of instructors to candidates (e.g. 1:3–1:6 depending on the type of course). Full up to date information about ERC courses and terminology is available on the ERC website www.erc.edu.

Ethos

ERC courses are taught by instructors who have been trained in teaching and assessment. The ethos of ERC courses is to create a positive environment that promotes learning. First names are encouraged among both faculty and candidates to reduce apprehension. Interactions between faculty and candidates are designed to be positive and teaching is conducted by encouragement with constructive feedback and debriefing on performance. A mentor/mentee system is used to enhance feedback and support for the candidate. Some stress is inevitable,²⁰⁷ particularly during assessment, but the aim of the instructors is to enable the candidates to do their best.

Course management

Courses are overseen by specialist committees within each National Resuscitation Council and by the ERC international course committee. The ERC has developed a web-based course management system (<http://courses.erc.edu>). The system can be used to register all ERC courses and enables course organizers to register a course from any country, assign instructors, record candidate attendance and outcomes, and file the course director's report directly with the ERC. Candidates may sign up online to a course, or may contact the organizer to register their interest in the course. At the end of the course the system will generate course certificates for the candidates and faculty. These certificates are assigned a unique number and can be accessed at any time by course organizers and directors. Participants that successfully complete courses are referred to as providers. For example someone that successfully completes an ALS course is known as an ALS provider. National Resuscitation Councils have access to information about courses organised in their country.

Language

Initially, the ERC courses were taught in English by an international faculty.²⁰⁶ As local instructors have been trained, and manuals and course materials have been translated into different languages, courses are now mainly taught in the native language. Early translation of guidelines and course materials is essential as delays in translation into the local language can cause significant delays to implementation of guidelines.³

Instructors

A tried and tested method has evolved for identifying and training instructors.

Identification of instructor potentials (IP)

These will be individuals who, in the opinion of the faculty, have passed and demonstrated a high level of performance during a provider course and, importantly, have shown qualities of leadership, team working and clinical credibility, with skills that include being articulate, supportive, and motivated. These individuals will be invited to take part in an instructor course and are called instructor potentials. Instructor potentials wishing to teach on Advanced Life Support (ALS), European Paediatric Life Support (EPLS), Newborn Life Support (NLS), Immediate Life Support (ILS), and European Paediatric Immediate Life Support (EPILS) courses should attend the Generic Instructor Course (GIC); for those wishing to teach only on the ERC Basic Life Support (BLS)/Automated External Defibrillation (AED) Course there is a specific BLS/AED Instructor Course.

Instructor courses

These are conducted by experienced instructors and, in the case of the Generic Instructor Course (see below), include an educator who has undertaken specific training in medical educational practice and the principles of adult learning. Assessment is formative by the faculty and feedback is given as appropriate.

Instructor candidate (IC) stage

Following successful completion of an instructor course (see below) the individual is designated instructor candidate (IC) status and normally will teach on two separate courses, under supervision, receiving constructive feedback on his or her performance. Following successful completion of these two courses the IC normally progresses to full instructor status. Occasionally the faculty will decide that a further course is required or, rarely, that the candidate is not suitable to progress to be an instructor. An appeal can be lodged with the relevant ERC International Course Committee who will make the final decision.

Course Director (CD) status

Each ERC course is led by an approved Course Director. Individuals are selected for approval as Course Directors through nomination by their peers and approved by their National Resuscitation Council (NRC) or the ERC International Course Committee. Course Directors are relatively senior individuals who are clinically credible, have demonstrated their qualities as a teacher and assessor and possess the leadership skills to lead a faculty of instructors. They will have embraced the educational principles inherent in the instructor course. A key component of ERC courses are the faculty meetings. These usually take place at the start and end of each day of the course. They are led by the course director. The aim of these meetings is to brief the teaching faculty and to facilitate evaluation of each candidate's performance. At the end of each course a final faculty meeting is held. During this meeting the faculty will review the performance of each candidate and decide whether they have successfully completed the course. As described above, candidates that have shown exceptional ability are selected for invitation to train as instructors. Where there are instructor candidates on the courses, their performance is also evaluated and feedback provided by their mentor or the course director. This faculty meeting also

gives the instructors an opportunity to debrief at the end of the course.

The Basic Life Support (BLS) and Automated External Defibrillator (AED) Courses

BLS/AED courses are appropriate for a wide range of providers. These may include clinical and non-clinical healthcare professionals (particularly those who are less likely to be faced with having to manage a cardiac arrest), general practitioners, dentists, medical students, first-aid workers, lifeguards, those with a duty of care for others (such as school teachers and care workers), and members of first responder schemes, as well as members of the general public. Separate BLS and AED provider courses are available, but the ERC encourages candidates to combine BLS skills with the use of an AED.

Provider course format

The aim of this provider course is to enable each candidate to gain competency in BLS and the use of an AED. Each BLS/AED provider course lasts approximately half a day and consists of skill demonstrations and hands-on practice, with a minimum number of lectures. The recommended ratio of instructor to candidates is 1:6, with at least one manikin and one AED for each group of 6 candidates. Formal assessment is not usually undertaken, but each candidate receives individual feedback on their performance. Those who need a certificate of competency for professional or personal use may be assessed continuously during the course or definitively at the end.

BLS/AED Instructor Course

Many of the candidates attending a BLS/AED provider course are laypeople, and some want subsequently to become instructors themselves. For this reason, the ERC has developed a one-day BLS/AED instructor course. Candidates for this course must be healthcare professionals, or laypeople who hold the ERC BLS/AED provider certificate and are designated as instructor potentials. The aim is to be as inclusive as possible regarding course attendance, the overriding criterion being that all candidates should have the potential and knowledge to teach the subject. The BLS/AED instructor course follows the principles of the Generic Instructor Course (GIC), with an emphasis on teaching people. Following successful completion of the course, each candidate becomes an instructor candidate (IC) and teaches on two BLS/AED courses before becoming a full instructor.

The Immediate Life Support (ILS) Course

The Immediate Life Support (ILS) course is for the majority of healthcare professionals who attend cardiac arrests rarely but have the potential to be first responders or resuscitation team members.²⁰⁸ The course teaches healthcare professionals the skills that are most likely to result in successful resuscitation whilst awaiting the arrival of the resuscitation team.²⁰⁹ Importantly, ILS also includes a section on the initial care of the sick adult and preventing cardiac arrest and complements other short courses that focus on the initial treatment of sick patients.²¹⁰ A recent cohort study found that the number of cardiac arrest calls decreased while pre-arrest calls increased after implementing a programme that included ILS teaching 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.²¹¹

Potential ILS candidates include nurses, nursing students, doctors, medical students, dentists, physiotherapists, radiographers, and cardiac technicians.

Course format

The ILS course is delivered over one day and comprises lectures, hands-on skills teaching and cardiac arrest simulation teaching (CASTeach) using manikins. The programme includes several options that enable instructors to tailor the course to their candidate group. The ILS course is designed to be straightforward to run. Most courses are conducted in hospitals with small groups of candidates (average 12 candidates). Course centres should try as far as possible to train candidates to use the equipment (e.g., defibrillator type) that is available locally.

Course content

The course covers those skills that are most likely to result in successful resuscitation: causes and prevention of cardiac arrest including use of the ABCDE approach, starting CPR, basic airway skills and defibrillation (AED or manual). The course includes an optional session on issues relevant to the candidate group (e.g., anaphylaxis, equipment checks). Once all the skills have been covered there is a cardiac arrest demonstration by the instructors that outlines the first responder role to the candidates. This is followed by the CASTeach station where candidates practice. ILS candidates are not usually expected to undertake the role of team leader. Candidates should be able to start a resuscitation attempt and continue until more experienced help arrives. When appropriate, the instructor takes over as resuscitation team leader. This is not always necessary because in some simulations resuscitation may be successful before more experienced help arrives. Standardised simulations are used that can be adapted to the workplace and clinical role of the candidate.

Assessment

Candidates are assessed continuously and must show their competence throughout the ILS course. There are no formal testing stations at the end of the course. Candidates are sent assessment forms with the pre-course materials. The forms indicate clearly how their performance will be measured against pre-determined criteria. Assessment on the ILS course enables the candidate to see what is expected, and frame their learning around achievement of these outcomes. The following practical skills are assessed on the ILS course: airway management, CPR and defibrillation. With a supportive approach, most candidates achieve the course learning outcomes.

The Advanced Life Support (ALS) Course

The target candidates for this course are doctors and senior nurses working in acute areas of the hospital and those who may be resuscitation team leaders and members.^{212,213} The course is also suitable for senior paramedics and some hospital technicians. The ILS course is more suitable for first responder nurses, doctors who rarely encounter cardiac arrest in their practice, and emergency medical technicians.

Each instructor acts as a mentor for a small group of candidates. The course normally lasts for 2 or 2.5 days.

Course format

The course format has very few formal lectures and teaching concentrates on hands-on skills, clinically-based simulations in small groups with emphasis on the team leader approach, and interactive group discussions. A formal mentor/mentee session is included to enable candidates to give and receive feedback.

Course content

The course content is based on the current ERC Guidelines for Resuscitation. Candidates are expected to have studied the ALS course materials carefully before the course.

The course aims to train candidates to highlight the causes of cardiac arrest and identify sick patients in danger of deterioration and to manage cardiac arrest and the immediate peri-arrest problems encountered in and around the first hour or so of the event. It is not a course in advanced intensive care or cardiology. Competence in basic life support is expected before the candidate enrolls for the course.

Emphasis is placed on the techniques of safe defibrillation and ECG interpretation, the management of the airway and ventilation, the management of peri-arrest rhythms, simple acid-base balance, and of special circumstances relating to cardiac arrest. Post-resuscitation care, ethical aspects related to resuscitation and care of the bereaved are included in the course.

Assessment and testing

Each candidate is assessed continuously during the course and reviewed at the end of each day at a faculty meeting. Feedback is given as required. Candidates are expected to be able to use the ABCDE approach to assess and treat the sick patient, recognise cardiac arrest, provide good quality CPR and safe defibrillation throughout the course. There is a cardiac arrest simulation testing (CASTest) towards the end of the course. This tests the participant's applied knowledge and skills during a simulated cardiac arrest. The reliability and measurement properties of the CASTest have been reported.^{169,214,215} A multiple choice question (MCQ) paper taken at the end of the course tests core knowledge. Candidates are required to achieve 75% to pass this test. The measurement properties of the multiple choice papers have been assessed from over 8000 candidates and found to have high internal consistency and discrimination properties (Data from Resuscitation Council (UK) and Dr Carl Gwinnutt).

The European Paediatric Life Support (EPLS) Course

The EPLS course is designed for healthcare workers who are involved in the resuscitation of a newborn, an infant or a child whether in or out-of-hospital. The course aims at providing caregivers with knowledge and skills for the management of the critically ill child during the first hour of illness and to prevent progression of diseases to cardiac arrest.

EPLS is not a course in neonatal or paediatric intensive care aimed for advanced providers.

Competence in paediatric basic life support is a prerequisite although refresher teaching in basic life support and relief of foreign body airway obstruction is included. The EPLS course is suitable for doctors, nurses, emergency medical technicians, paramedics etc who have a duty to respond to sick newborns, infants and children in their practice.^{216,217}

Experience in paediatrics is necessary to keep simulations realistic and answer to candidates' questions so a minimum of 50% of the faculty must have regular experience in neonatal or paediatric practice. The course lasts for 2–2.5 days.

Course format

The course format has few formal lectures. Teaching of knowledge and skills is delivered in small groups using clinically based simulations (e.g., cardiac arrest, cardiac and respiratory failure, delivery room simulations). The emphasis is on assessment

and treatment of the sick child, team working and leadership.

Course content

The course content follows the current ERC guidelines for neonatal and paediatric resuscitation. The course candidates are expected to have studied the manual before attending the course. A pre-course MCQ is sent with the manual to candidates 4–6 weeks before the course to encourage candidates to read the course materials.

The EPLS course is aimed at training candidates to understand the causes and mechanisms of cardiorespiratory arrest in neonates and children, to recognise and treat the critically ill neonate, infant or child and to manage cardiac arrest. Skills taught include airway management, bag-mask ventilation, log roll and cervical collar placement, oxygen delivery, an introduction to intubation and vascular access, safe defibrillation, cardioversion and AED use.

Each candidate is assessed individually and reviewed by the faculty. Feedback is given as required. A BLS assessment follows the BLS refresher course and a simulation-based test at the end of the course emphasises the assessment of the sick child and other core skills. An end of course MCQ with a pass mark of 74% tests core knowledge.

The European Paediatric Immediate Life Support (EPILS) Course

Course format

EPILS is a one-day course comprising one lecture, hands-on skills and simulation teaching. The programme includes options to enable teaching to be tailored for candidate groups.

Course content

The course is aimed at training nurses, EMS personnel, and doctors to recognize and treat critically ill infants and children, prevent cardiorespiratory arrest and to treat children in cardiorespiratory arrest during the first few minutes whilst awaiting the arrival of a resuscitation team. This interactive course is based on short practical simulations adapted to the workplace and to the actual clinical role of candidates.

Basic life support, bag-mask ventilation, chest compressions, choking, and intraosseous access are included; drugs during cardiac arrest and laryngeal mask insertion are optional. The EPILS course is designed to be simple to run. Most courses are conducted in hospitals with small groups of candidates (average 5–6 candidates with one instructor). There needs to be at least one baby and one child manikin for every 6 candidates. Course centres should try as far as possible to train candidates to use the equipment (e.g., defibrillator type) that is available in their clinical setting.

Assessment

Candidates are sent a pre-course MCQ paper with pre-course materials to help them prepare for the course. The MCQ paper helps to ensure that candidates read the course materials before attending the course and does not count towards the final assessment. There are no formal testing stations during the course. Candidate's performances are assessed continuously. Assessment forms are given to the candidates at the beginning of the course and instructors provide feedback throughout the course. The following practical skills are assessed on the EPILS course: basic life support,

bag-mask ventilation and AED use. With a supportive approach, most candidates achieve the course learning outcomes.

The Newborn Life Support (NLS) Course

This one-day course is designed for healthcare workers likely to be present at the birth of a baby in the course of their job. It aims to give those who may be called upon to start resuscitation at birth the background knowledge and skills to approach the management of the newborn infant during the first 10–20 min. The course is suitable for midwives, nurses, EMS personnel, and doctors and, like most such courses, works best with candidates from a mixture of specialties.

Course format

The NLS manual is sent to each of the candidates four weeks before the course. Each candidate receives a MCQ together with the manual and is asked to complete this and bring it with them to the course. There is an introduction followed by two short lectures. The candidates are then divided into four groups and undertake three workstations before lunch. The afternoon is then taken up by a demonstration simulation followed by two hours of simulation teaching in small groups and, finally, a theoretical and practical assessment by an MCQ and an individual practical airway test. The course places appropriate emphasis on airway management but also covers chest compression, umbilical venous access and drugs.

Both basic infant and four infant advanced manikins should be available as well as other airway adjuncts. Resuscitaires, ideally complete with sufficient gas cylinders for the whole day, should also be available.

The Generic Instructor Course (GIC)

This course is for candidates who have been recommended as instructor potential (IP) emanating from ERC provider courses (ALS, EPLS, NLS, ILS, EPILS). Candidates with IP status from certain other provider courses can also attend (e.g., European Trauma Course, Pre Hospital Trauma Care, Italy). There should be a maximum of 24 candidates with a ratio of at least one instructor to three candidates. Instructors must be full and experienced ERC instructors who have been through a formal process of training to become a GIC instructor. Groups should not exceed six candidates. The emphasis of the course is on developing teaching and assessment skills, as well as promoting team leadership and providing constructive feedback. Core knowledge of the original provider course is assumed. The course lasts for 2 days or 2.5 days.

Course format

The course format is largely interactive. An ERC medical educator plays a key role leading the educational process, the discussions and feedback. This lecture is interspersed with group activities. The remainder of the course is conducted in small group discussions and skill and simulation based hands on sessions. Mentor/mentee sessions are included and there is a faculty meeting at the beginning of the course and at the end of each day.

Course content

Candidates are given pre-course reading material and are expected to have read this before attending. The theoretical background of adult learning and effective teaching and assessment is covered by the educator at the beginning of the course. Each teaching and assessment skill is demonstrated by the faculty. The

candidates then get the opportunity to practice: equipment familiarisation, lecturing, teaching skills by means of the four stage approach, intermediate fidelity simulation sessions using simulations, small group teaching sessions (open and closed discussions), and assessment.

For each teaching tool, a “mini-topic” is extracted from the original provider course material. Throughout the course, emphasis is placed on the role of the instructor and each candidate has the opportunity to adopt the instructor role. The concept of constructive feedback is a key element and is also emphasised. Finally, the roles and qualities of an ERC Instructor are discussed.

Assessment

Each candidate is assessed formatively by the faculty throughout the course. Candidates' performances and attitudes are discussed at the daily faculty meetings and feedback given as required. Successful candidates may proceed to the status of instructor candidate (IC). Candidates who successfully complete the course but who are considered by the faculty to need specific support in their development may be recommended to undertake their IC placements at nominated centres.

The Educator Master Class

Medical Educators are an essential component of the GIC Faculty. This two-day course is designed for those aspiring to become medical educators for the ERC and is run when there is a need for expansion of Educator numbers. Suitable candidates are selected by the ERC Educational Advisory Group (EAG) following a written application and generally must have a background and qualification in medical education or have demonstrated a special commitment to educational practice over a number of years. They should have experience of a provider course and a GIC and should have studied the background reading for the course.

The instructors for the course are experienced educators.

Course format

The course consists mainly of closed discussion groups for the whole course, led by one or two of the instructors, together with breakout small group discussions and problem solving.

Course content

The course covers the theoretical framework for medical educators, assessment and quality control, teaching methodologies, critical appraisal, the role of the mentor, multi-professional education strategies and continued development of the medical educator.

Assessment

Each candidate is assessed formatively by the faculty throughout the course. Successful candidates may proceed to the status of educator candidate where they will be supervised and assessed by an experienced educator and course director until it is decided whether or not they will be suitable educators to work on their own.

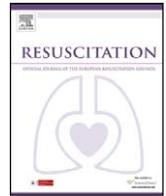
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European Resuscitation Council Guidelines for Resuscitation 2010 Section 10. The ethics of resuscitation and end-of-life decisions

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Introduction

Sudden unexpected cardiac arrest is an event with often devastating consequences to the individual victim, family and friends. While some resuscitation attempts are successful with good long-term outcome, the majority are not, despite significant efforts and some improvements during the last decade.

Healthcare professionals are obliged to do what is necessary to protect and save lives. Society as a whole and especially emergency medical services (EMS), hospitals and other healthcare institutions need to plan for, organise and provide an appropriate response in case of sudden cardiac arrest. This often implies the use of many resources and high costs, especially in the more affluent countries. New technology and medical evidence and increasing expectations of the public have rendered ethical considerations an important part of any end-of-life intervention or decision. This includes achieving the best results for the individual patient, relatives and for society as whole by appropriate allocation of available resources.

Several considerations are required to ensure that decisions to attempt or withhold resuscitation attempts are appropriate, and that patients are treated with dignity. These decisions are complex and may be influenced by individual, international and local cultural, legal, traditional, religious, social and economic factors.^{1–11}

Sometimes the decisions can be made in advance, but often these difficult decisions have to be made in a matter of seconds or minutes at the time of the emergency and especially in the out-of-hospital setting, based upon limited information. Therefore it is important that healthcare providers understand the principles involved before they are faced with a situation where a decision to resuscitate or not must be made. For healthcare professionals end-of-life decisions and ethical considerations should be made in advance and in the context of the society. Although there is little science to guide end-of-life decision-making, the subject is impor-

tant, which is why information for healthcare providers is included in these resuscitation guidelines.

This section of the guidelines deals with some recurring ethical aspects and end-of-life decisions.

- Key principles of ethics
- Sudden death in a global perspective
- Outcome and prognostication
- When to start and when to stop resuscitation attempts
- Advance directives and do-not-attempt resuscitation orders
- Organ procurement
- Family presence during resuscitation
- Research in resuscitation and informed consent
- Research and training on the recently dead

Principles of ethics

The key principles of ethics are referred to as autonomy, beneficence, non-maleficence, justice and further more dignity and honesty.¹²

Autonomy is the right of the patient to accept or refuse any treatment. Autonomy relates to patients being able to make informed decisions on their own behalf, rather than being subjected to paternalistic decisions being made for them by healthcare professionals. This principle has been introduced during the past 40 years, arising from legislature, primarily the Helsinki Declaration of Human Rights and its subsequent modifications and amendments.¹³ Autonomy requires that the patient is adequately informed, competent, free from undue pressure and that there is consistency in the patient's preferences. The principle is considered universal in medical practice; however, it may often be difficult to apply in an emergency, such as sudden cardiac arrest.

Non-maleficence means doing no harm or, even more appropriate, no further harm. Resuscitation should not be attempted in obviously futile cases.

Beneficence implies that healthcare providers must provide benefits in the best interest of the individual patient while balancing benefit and risks. Commonly, this will involve attempting resusci-

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tation, but on occasion it will mean withholding cardiopulmonary resuscitation (CPR).

Justice implies the concern and duty to distribute limited health resources equally within a society, and the decision of who gets what treatment (fairness and equality). If resuscitation is provided, it should be made available to all who will benefit from it within the frame of available resources.

Dignity and honesty are frequently added as essential elements of ethics. Patients always have the right to be treated with dignity and information should be honest without suppressing important facts. Transparency and disclosure of conflict of interests (COI) is another important part of the ethics of medical professionalism. The importance of this is emphasized by the COI policy operated by the International Liaison Committee on Resuscitation (ILCOR).¹⁴

Sudden death in a global perspective

In Europe, with 46 countries and with a population on the European continent of 730 million, the incidence of sudden cardiac arrest is estimated at between 0.4 and 1 per 1000 inhabitants per year, thus involving between 350,000 and 700,000 people.¹⁵ Approximately, 275,000 persons have a cardiac arrest treated by the EMS in Europe.¹⁶ Out-of-hospital cardiac arrest is the third leading cause of death in the USA.¹⁷ In Europe and USA ischaemic heart disease is considered the main cause of sudden cardiac arrest.

Health challenges look different in a worldwide perspective. In the World Health Organization (WHO) 2002 Annual Report, two extreme findings are found almost side by side: 170 million children in poor countries were underweight, causing over three million deaths yearly, and on the other extreme at least 300 million adults worldwide were overweight or clinically obese with high risk of sudden cardiac arrest.¹⁸ In parallel, the cause of sudden death differs widely across the world. Outside Europe and North America, cardiac arrest of non-cardiac aetiology, such as trauma, drowning or newborn asphyxia, is more important than cardiac aetiology. More than 1.3 million people die yearly in road traffic accidents.¹⁹ In 2008, there were 8.8 million deaths among children less than 5 years old, with considerable inequities between countries. Diarrhoea and pneumonia still kill almost 3 million children less than 5 years old annually, especially in low-income countries. And about one third of deaths among children less than five years of age occur in the first month of life. More than 500,000 women die of complications during pregnancy or childbirth, 99% of them in developing countries.^{20,21} Worldwide, it is estimated that approximately 150,000 people die from drowning each year, and the majority are children.²²

In summary, sudden death is a worldwide challenge. Aetiology differs and treatment and prevention have to be tailored to the local problems and resources. The obligation and challenges to protect and save lives are evident both from the local and the global perspective.

Outcome from sudden cardiac arrest

Resuscitation efforts often focus on sudden and unexpected cardiac arrest that should have been prevented. Included in the decision on whether to commence resuscitation is the likelihood of success and, if initially successful, the quality of life that can be expected following hospital discharge. Reliable and valid data are therefore essential to guide healthcare providers. Resuscitation attempts are unsuccessful in 70–98% of cases and death ultimately is inevitable.

Several studies have demonstrated that successful resuscitation after cardiac arrest produces a good quality of life in most survivors. There is little evidence to suggest that resuscitation leads to a large

pool of survivors with an unacceptable quality of life. Cardiac arrest survivors may experience post-arrest problems including anxiety, depression, post-traumatic stress, and difficulties with cognitive function. Clinicians should be aware of these potential problems, screen for them and, if found, treat them.^{23–38} Future interventional resuscitation studies should include long-term follow up evaluation.

Prognostication in cardiac arrest

In well-developed pre-hospital systems, about one third to one half of patients may achieve Return of Spontaneous Circulation (ROSC) with CPR, with a smaller proportion surviving to the hospital critical care unit, and an even smaller proportion surviving to hospital discharge with good neurological outcome. Prognostication is of the essence to guide clinicians, and it would be important to be able to predict poor outcome with high specificity to reduce unnecessary burden on the patient, family members and health care providers, and reduce inappropriate use of resources. Unfortunately, there are currently no valid tools for prognostication of poor outcome in the emergency setting, including the first few hours after ROSC. In fact, prediction of final neurological outcome in patients remaining comatose after ROSC is difficult during the first 3 days.³⁹ The inclusion of therapeutic hypothermia has further challenged the previously established prognostic criteria.⁴⁰

Certain circumstances, for example hypothermia at the time of cardiac arrest, will enhance the chances of recovery without neurological damage, and the normal prognostic criteria (such as asystole persisting for more than 20 min) are not applicable.⁴¹

When to start and when to stop resuscitation attempts?

In all cases of sudden cardiac arrest the healthcare provider is being challenged with two main questions: when to start and when to stop resuscitation attempts? In the individual case, the decision to start, continue or to terminate resuscitation attempts, is based on the difficult balance between the benefits, risks and cost these interventions will place on patient, family members and healthcare providers. In a broader perspective, cost to the society and health care system is part of this. The standard of care remains the prompt initiation of CPR. However, ethical principles such as beneficence, non-maleficence, autonomy, and justice have to be applied in the unique setting of emergency medicine. Physicians have to consider the therapeutic efficacy of CPR, the potential risks, and the patient's preferences.^{42,43}

Resuscitation is inappropriate and should not be provided when there is clear evidence that it will be futile or is against the expressed wishes of the patient. Systems should be established to communicate these prospective decisions and simple algorithms should be developed to assist rescuers in limiting the burden of futile and unnecessary costly treatments. One prospective study demonstrated that a basic life support termination of resuscitation rule (no shockable rhythm, unwitnessed by EMS and no return of spontaneous circulation) was predictive of death when applied by defibrillation-only emergency medical technicians.⁴⁴ Subsequent studies showed external generalisability of this rule, but it has also been challenged.^{45–47} Prospectively validated termination of resuscitation rules are recommended to guide termination of pre-hospital CPR in adults. Other rules for various provider levels, including in-hospital providers, may be helpful to reduce variability in decision-making; but all rules should be validated prospectively before implementation. The implementation of a termination rule will carry a self-fulfilling prophecy, and should be challenged periodically as new treatments evolve.

Who should decide not to attempt resuscitation?

Resuscitation protocols or standard operating procedures should define who has the obligation and responsibility to make the difficult decision not to attempt resuscitation or to abandon further attempts. This goes for the pre-hospital and in-hospital setting and might vary according to legislation, culture or local tradition.

In hospital, the decision is usually made, after appropriate consultations, by the senior physician in charge of the patient or the leader of the resuscitation team when called. Medical emergency teams (METs), acting in response to concern about a patient's condition from ward staff, can initiate DNAR decisions.^{48–50} In the pre-hospital setting, in the absence of doctors, the decision can be made according to standard protocols or after consultation with a physician.

Legislation on who can make decisions about death varies within countries. Many out-of-hospital cardiac arrest cases are attended by emergency medical technicians (EMTs) or paramedics, who face similar dilemmas about when to determine if resuscitation is futile and when it should be abandoned. In general, resuscitation is started in out-of-hospital cardiac arrest unless there is a valid advanced directive to the contrary or it is clear that resuscitation would be futile in cases of a mortal injury, such as decapitation, rigor mortis, dependent lividity and fetal maceration. In such cases, the non-physician is making a diagnosis of death but is not certifying death, which, in most countries, can be done only by a physician.

What constitutes futility?

Futility exists if resuscitation will be of no benefit in terms of prolonging life of acceptable quality. It is problematic that, although predictors for non-survival after attempted resuscitation have been published, none have been tested on an independent patient sample with sufficient predictive value, apart from end-stage multi-organ failure with no reversible cause.^{51–56} Furthermore, studies on resuscitation are particularly dependent on system factors such as time to start of CPR, time to defibrillation, etc. These intervals may be prolonged in any study cohort but are often not applicable to an individual case. Inevitably, judgements will have to be made, and there will be grey areas where subjective opinions are required in patients with heart failure and severe respiratory compromise, asphyxia, major trauma, head injury and neurological disease. The age of the patient may influence the decision but age itself is only a relatively weak independent predictor of outcome.^{56–58} However, high age is frequently associated with comorbidity, which does have an influence on prognosis. At the other end of the scale, most physicians will err on the side of intervention in children for emotional reasons, even though the overall prognosis in children is often worse than in adults. It is therefore important that clinicians understand the factors that influence resuscitation success.

When to abandon further resuscitation attempts

The vast majority of resuscitation attempts do not succeed and therefore have to be abandoned. Several factors will influence the decision to stop the resuscitative effort. These will include the medical history and anticipated prognosis from factors such as the period between cardiac arrest and start of CPR by bystanders and by healthcare professionals, the initial ECG rhythm, the interval to defibrillation and the period of advanced life support (ALS) with continuing asystole, no reversible causes and no ROSC.⁵⁹

In many cases, particularly in out-of-hospital cardiac arrest, the underlying cause of arrest may be unknown or merely surmised, and the decision is made to start resuscitation while further infor-

mation is gathered. If it becomes clear that the underlying cause renders the situation to be futile, then resuscitation should be abandoned if the patient remains in asystole with all ALS measures in place. Additional information such as an advance directive may become available and may render discontinuation of the resuscitation attempt ethically correct.

In general, resuscitation should be continued as long as VF persists. It is generally accepted that ongoing asystole for more than 20 min in the absence of a reversible cause, and with ongoing ALS, constitutes grounds for abandoning further resuscitation attempts.⁶⁰ There are, of course, reports of exceptional cases that do not support the general rule, and each case must be assessed individually. Ultimately, the decision is based on the clinical judgement that the patient's arrest is unresponsive to ALS. In out-of-hospital cardiac arrest of cardiac origin, if recovery is going to occur, ROSC usually takes place on site. Patients with primary cardiac arrest, who require ongoing CPR without any return of a pulse during transport to hospital, rarely survive neurologically intact.^{61,62}

Many will persist with the resuscitation attempt for longer if the patient is a child. This decision is not generally justified on scientific grounds, though new data are encouraging.⁶³ Nevertheless, the decision to persist in the distressing circumstances of the death of a child is understandable, and the potential enhanced recruitment of cerebral cells in children after an ischaemic insult is an as yet unknown factor. If faced with a newly born baby with no detectable heart rate, which remains undetectable for 10 min, it is appropriate to then consider stopping resuscitation.⁶⁴

Advance directives

Advance directives have been introduced in many countries, emphasizing the importance of patient autonomy. Advance directives are a method of communicating the patient's wishes concerning future care, particularly towards the end-of-life, and must be expressed while the patient is mentally competent and not under duress. Advance directives are likely to specify limitations concerning terminal care, including the withholding of CPR. This may help healthcare attendants in assessing the patient's wishes should the patient later become mentally incompetent. However, challenges can arise. The relative may misinterpret the wishes of the patient, or may have a vested interest in the death (or continued existence) of the patient. On the other hand, healthcare providers tend to underestimate sick patients' desire to live.

Written directions by the patient, legally administered living wills or powers of attorney may eliminate some of these problems but are not without limitations. The patient should describe as precisely as possible the situation envisaged when life support should be withheld or discontinued. This may be aided by a medical adviser. For instance, most people would prefer not to undergo CPR in the presence of end-stage multi-organ failure with no obvious reversible cause, but the same persons would welcome the attempt at resuscitation should ventricular fibrillation (VF) occur in association with a remediable primary cardiac cause. Patients often change their minds with changes in circumstances, and therefore the advanced directive should be as recent as possible and take into account any change of circumstances.

In sudden out-of-hospital cardiac arrest, the attendants usually do not know the patient's situation and wishes, and an advance directive is often not readily available. In these circumstances, resuscitation should begin immediately and questions addressed later. There is no ethical difference in stopping the resuscitation attempt that has started if the healthcare providers are later presented with an advance directive limiting care. There is considerable international variation in the medical attitude towards

written advance directives.¹ In some countries, the written advance directive is considered to be legally binding; in others not.

DNAR orders

A do-not-attempt resuscitation (DNAR) order (also described more recently as a DNACPR decision) is a binding legal document that states that resuscitation should not be attempted in the event of cardiac or respiratory arrest; meaning that CPR should not be performed. Other treatment should be continued, particularly pain relief and sedation, as required and indicated, if they are considered to be contributing to the quality of life. If not, orders not to continue or initiate any such treatments should be specified independently of DNAR orders. For many years, DNAR orders in many countries were written by single doctors, often without consulting the patient, relatives or other health personnel, but there are now clear procedural requirements in many countries.⁶⁵

Although the ultimate responsibility and decision for DNAR rests with the senior doctor in charge of the patient, it is wise for this individual to consult others before making the decision. Following the principle of patient autonomy it is wise, if possible, to ascertain the patient's wishes about a resuscitation attempt. This must be done in advance, when the patient is able to make an informed choice. Opinions vary as to whether such discussions should occur routinely for every hospital admission or only if the diagnosis of a potentially life-threatening condition is made. In presenting the facts to the patient, the doctor must be as certain as possible of the diagnosis and prognosis and may seek a second medical opinion in this matter. It is vital that the doctor should not allow personal life values to distort the discussion—in matters of acceptability of a certain quality of life, the patient's opinion should prevail. It is considered essential for the doctor to have discussions with close relatives if at all possible. Whereas they may influence the doctor's decision, it should be made clear to them that the ultimate responsibility and decision will be that of the doctor. It is neither fair nor reasonable to place the burden of decision on the relative.

According to the principle of autonomy, patients have the right to refuse treatment; however, they do not have an automatic right to demand a specific treatment—they cannot insist that resuscitation must be attempted in any circumstance. A doctor is required only to provide treatment that is likely to benefit the patient, and is not required to provide treatment that would be futile. However, it would be wise to seek a second opinion in making this decision, for fear that the doctor's own personal values, or the question of available resources, might influence his or her opinion.⁶⁶

In adult cardiac arrest various studies have addressed the impact of advance directives and DNAR orders in directing appropriate resuscitation efforts. Most of these studies are old and often contradictory.^{67–76} Standardised orders for limiting life-sustaining treatments decrease the incidence of futile resuscitation attempts and should ensure that adult patient wishes are honoured. Instructions should be specific, detailed, and transferable across health care settings, and easily understood. Processes, protocols, and systems should be developed that fit within local cultural norms and legal limitations to allow providers to honour patient wishes regarding resuscitation efforts.

Organ procurement

The issue of initiating life-prolonging treatment or continuing otherwise futile resuscitation attempts with the sole purpose of harvesting organs is debatable.^{77,78} There is variation between countries and cultures about the ethics of this process; at present no consensus exists. If considering prolonging CPR and other resuscitative

measures to enable organ donation to take place mechanical chest compressions may be valuable in these circumstances.^{79,80}

Family presence during resuscitation

The concept of a family member being present during the resuscitation process was introduced in the 1980s and has become accepted practice in many countries.^{81–86} Many relatives would like to be present during resuscitation attempts and, of those who have had this experience, over 90% would wish to do so again. Most parents would wish to be with their child at this time.⁸²

Relatives have considered several benefits from being permitted to be present during a resuscitation attempt, including coming to terms with the reality of death. However, this is a choice entirely to be made by the relatives. Several measures are required to ensure that the experience of the relative is the best under the circumstances. This includes allocating personnel to take care of the relatives.^{87,88}

In the event of an out-of-hospital arrest, the relatives may already be present, and possibly performing basic life support (BLS). They should be offered the same choices and appreciation of their effort as bystander offering BLS. With increasing experience of family presence during resuscitation attempts, it is clear that problems rarely arise. Fifteen years ago, most staff would not have countenanced the presence of relatives during resuscitation, but there is an increasingly open attitude and appreciation of the autonomy of both patient and relatives.¹ Cultural and social variations still exist, and must be understood and appreciated with sensitivity.

Research in resuscitation and informed consent

There is an essential need to improve the quality of resuscitation and thereby the long-term outcome. To achieve this, research and randomised clinical trials are crucial, not only to introduce new and better interventions, but also to abandon the use of inefficient and costly procedures and medications, whether old or new. As the ILCOR 2010 consensus on CPR and ECC Science clearly reveals many current practises are based upon tradition and not on science.^{89,90}

There are important ethical issues relating to undertaking randomised clinical trials for patients in cardiac arrest who cannot give informed consent to participate in research studies. Progress in improving the dismal rates of successful resuscitation will only come through the advancement of science through clinical studies. The utilitarian concept in ethics looks to the greatest good for the greatest number of people. This must be balanced with respect for patient autonomy, according to which patients should not be enrolled in research studies without their informed consent. Over the past decade, legal directives have been introduced into the USA and the European Union^{91,92} that place significant barriers to research on patients during resuscitation without informed consent from the patient or immediate relative.⁹³ There are data showing that such regulations deter research progress in resuscitation.⁹⁴ It can be argued that these directives may in themselves conflict with the fundamental human right to good medical treatment as set down in the Helsinki Declaration.¹³ The US authorities have, to a very limited extent, sought to introduce methods of exemption,⁹⁵ but these are still associated with problems and almost insurmountable difficulties.^{94,96,97}

Research and training on the recently dead

Research on the recently dead encounters similar restrictions unless previous permission is granted as part of an advance direc-

tive by the patient, or permission can be given immediately by the relative. The management of resuscitation can be taught using scenarios with manikins and simulators or animal models, but training in certain skills required during resuscitation is difficult. Therefore the question arises as to whether it is ethically and morally appropriate to undertake training and practice on the living or the dead. There is a wide diversity of opinion on this matter.^{98,99} Many, particularly those in the Islamic nations, find the concept of any skills training and practice on the recently dead completely unacceptable because of an innate respect for the deceased. Others will accept the practice of non-invasive procedures that do not leave a mark; and some accept that any procedure may be learned on the dead body with the justification that the learning of skills is paramount for the well being of future patients. One option is to request informed consent for the procedure from the relative of the deceased. It is advised that healthcare professionals learn local and hospital policies regarding this issue and follow the established policy.

Summary

Sudden unexpected cardiac arrest is a global challenge. Some deaths are preventable and some arrests can be treated successfully and result in a very good long-term outcome. However, most resuscitation attempts are futile and death is inevitable. End-of-life decision is an important part of resuscitation.

Scientific evidence does not provide much guidance for end-of-life-decisions. Nevertheless, because of the importance of the subject, the ERC has produced this guidance on this important and difficult topic for healthcare providers. End-of-life decisions are complex and may be influenced by individual, international and local cultural, legal, traditional, religious, social and economic factors. Solutions should be tailored accordingly. Sometimes the decisions can be made in advance, but often these difficult decisions have to be made in an emergency and based upon limited information. Therefore it is important that healthcare providers understand the principles involved, the challenges and the need for research in resuscitation. End-of-life decisions and ethical considerations should be reflected in advance through education, discussions and debriefings for health care professionals to further strengthen individual ethical competence.

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**EUROPEAN
RESUSCITATION
COUNCIL**



Summary of the main changes in the Resuscitation Guidelines

ERC GUIDELINES 2010





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Version 1.2

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Summary of main changes since 2005 Guidelines

Basic life support

Changes in basic life support (BLS) since the 2005 guidelines include:

- ◆ Dispatchers should be trained to interrogate callers with strict protocols to elicit information. This information should focus on the recognition of unresponsiveness and the quality of breathing. In combination with unresponsiveness, absence of breathing or any abnormality of breathing should start a dispatch protocol for suspected cardiac arrest. The importance of gasping as sign of cardiac arrest is emphasised.
- ◆ All rescuers, trained or not, should provide chest compressions to victims of cardiac arrest. A strong emphasis on delivering high quality chest compressions remains essential. The aim should be to push to a depth of at least 5 cm at a rate of at least 100 compressions min^{-1} , to allow full chest recoil, and to minimise interruptions in chest compressions. Trained rescuers should also provide ventilations with a compression–ventilation (CV) ratio of 30:2. Telephone-guided chest compression-only CPR is encouraged for untrained rescuers.
- ◆ The use of prompt/feedback devices during CPR will enable immediate feedback to rescuers and is encouraged. The data stored in rescue equipment can be used to monitor and

improve the quality of CPR performance and provide feedback to professional rescuers during debriefing sessions.

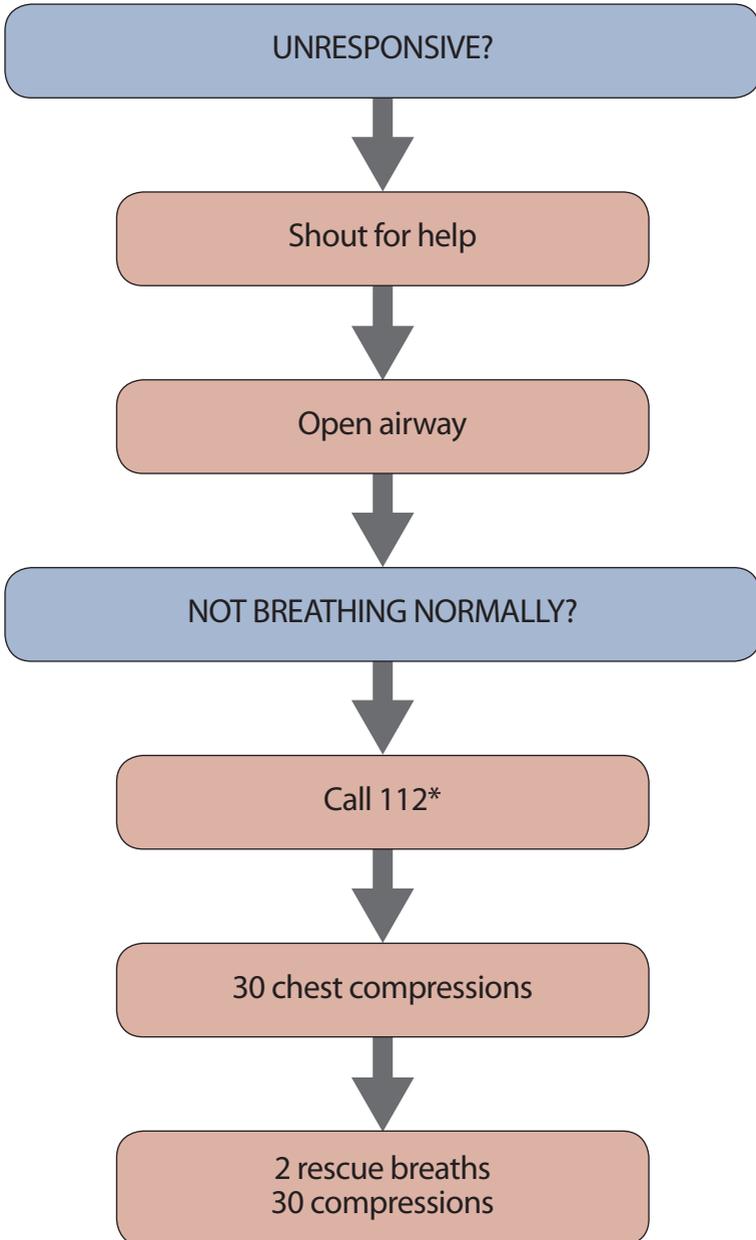
Electrical therapies: automated external defibrillators, defibrillation, cardioversion and pacing

The most important changes in the 2010 ERC Guidelines for electrical therapies include:

- ◆ The importance of early, uninterrupted chest compressions is emphasised throughout these guidelines.
- ◆ Much greater emphasis on minimising the duration of the pre-shock and post-shock pauses; the continuation of compressions during charging of the defibrillator is recommended.
- ◆ Immediate resumption of chest compressions following defibrillation is also emphasised; in combination with continuation of compressions during defibrillator charging, the delivery of defibrillation should be achievable with an interruption in chest compressions of no more than 5 seconds.
- ◆ Safety of the rescuer remains paramount, but there is recognition in

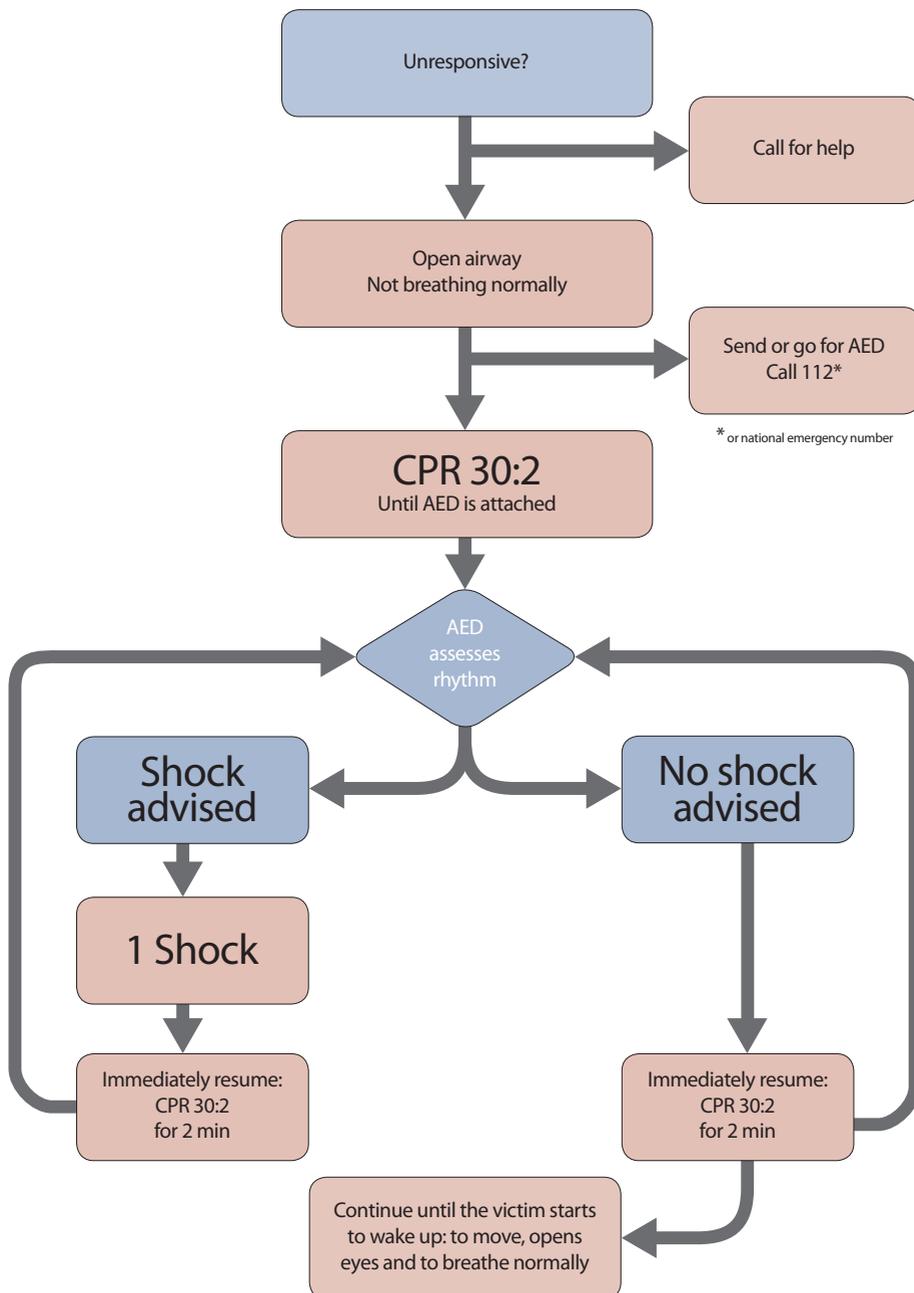


Adult Basic Life Support

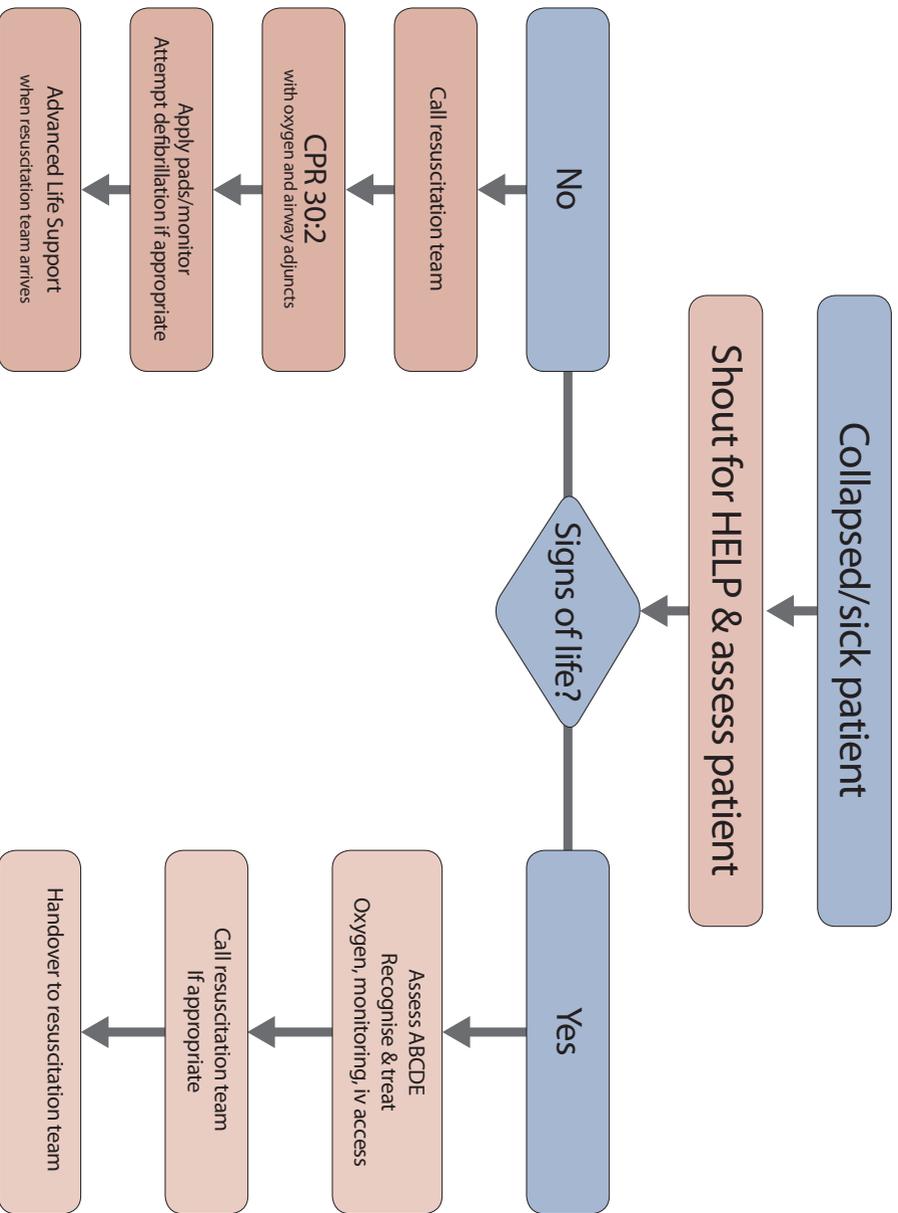


*or national emergency number

Automated External Defibrillation



In Hospital Resuscitation



these guidelines that the risk of harm to a rescuer from a defibrillator is very small, particularly if the rescuer is wearing gloves. The focus is now on a rapid safety check to minimise the pre-shock pause.

◆ When treating out-of-hospital cardiac arrest, emergency medical services (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., two or three minutes) before rhythm analysis and a shock is delivered is no longer recommended. For some EMS that 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.

◆ The use of up to three-stacked shocks may be considered if VF/VT occurs during cardiac catheterisation or in the early post-operative period following cardiac surgery. This three-shock strategy may also be considered for an initial, witnessed VF/VT cardiac arrest when the patient is already connected to a manual defibrillator.

◆ Further development of AED programmes is encouraged – there is a need for further deployment of AEDs in both public and residential areas.

Adult advanced life support

The most important changes in the 2010 ERC 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.

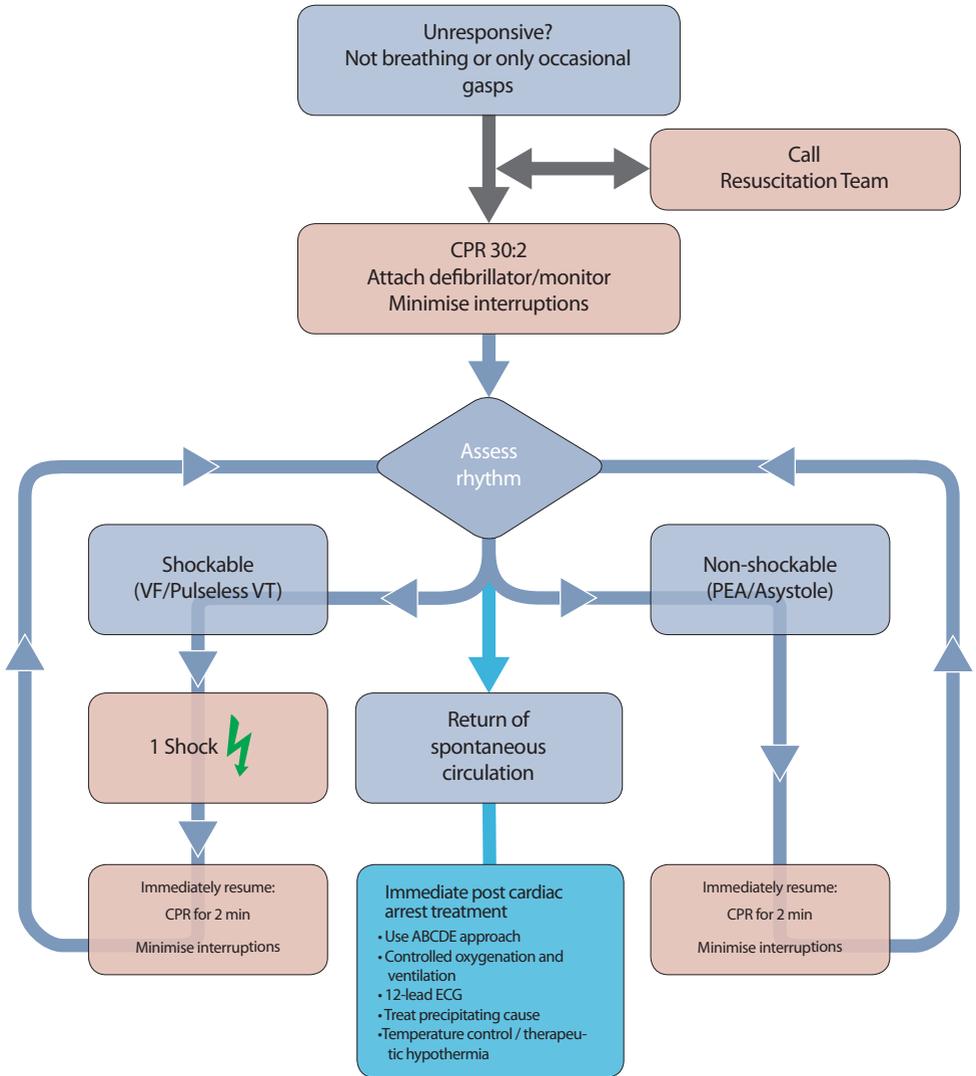
◆ Removal of the recommendation for a pre-specified period of cardiopulmonary resuscitation (CPR) before out-of-hospital defibrillation following cardiac arrest unwitnessed by the EMS.

◆ Continuation of chest compressions while a defibrillator is charged - this will minimise the pre-shock pause.

◆ The role of the precordial thump is de-emphasised.



Advanced Life Support



During CPR

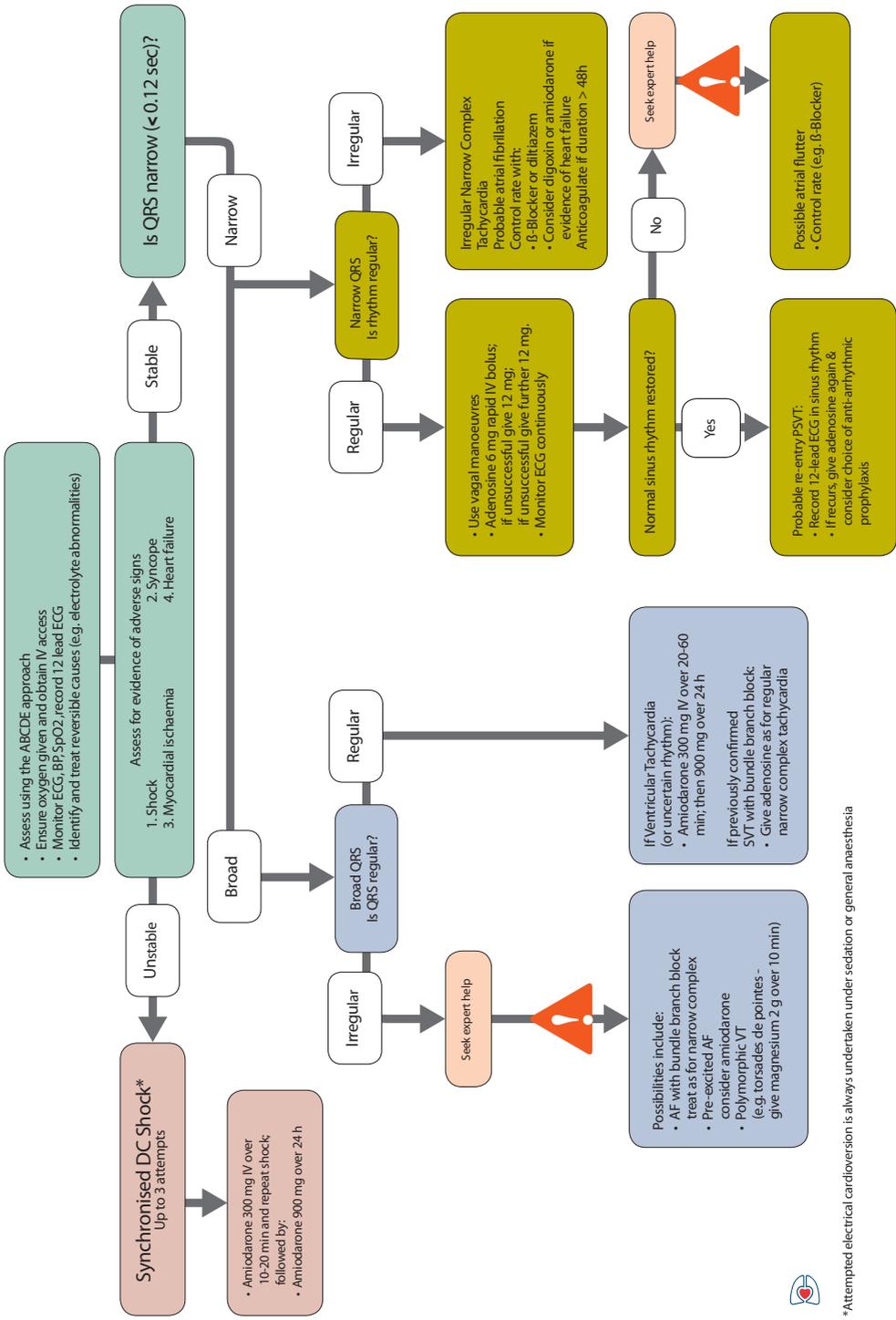
- Ensure high-quality CPR: rate, depth, recoil
- Plan actions before interrupting CPR
- Give oxygen
- Consider advanced airway and capnography
- Continuous chest compressions when advanced airway in place
- Vascular access (intravenous, intraosseous)
- Give adrenaline every 3-5 min
- Correct reversible causes

Reversible causes

- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypothermia
- Thrombosis
- Tamponade - cardiac
- Toxins
- Tension pneumothorax



Tachycardia (with pulse)



*Attempted electrical cardioversion is always undertaken under sedation or general anaesthesia



Bradycardia

• Assess using the ABCDE approach
• Ensure oxygen given and obtain IV access
• Monitor ECG, BP, SpO₂, record 12 lead ECG
• Identify and treat reversible causes (e.g. electrolyte abnormalities)

Assess for evidence of adverse signs:
1 Shock
2 Syncope
3 Myocardial ischaemia
4 Heart failure

Yes

No

Atropine
500 mcg IV

Satisfactory
Response?

Yes

No

Risk of asystole?
• Recent asystole
• Möbitz II AV block
• Complete heart block with broad QRS
• Ventricular pause > 3s

Yes

No

Interim measures:
• Atropine 500 mcg IV repeat to maximum of 3 mg
• Isoprenaline 5 mcg min⁻¹
• Adrenaline 2-10 mcg min⁻¹
• Alternative drugs*
OR
• Transcutaneous pacing

 Seek expert help
Arrange transvenous pacing

Observe

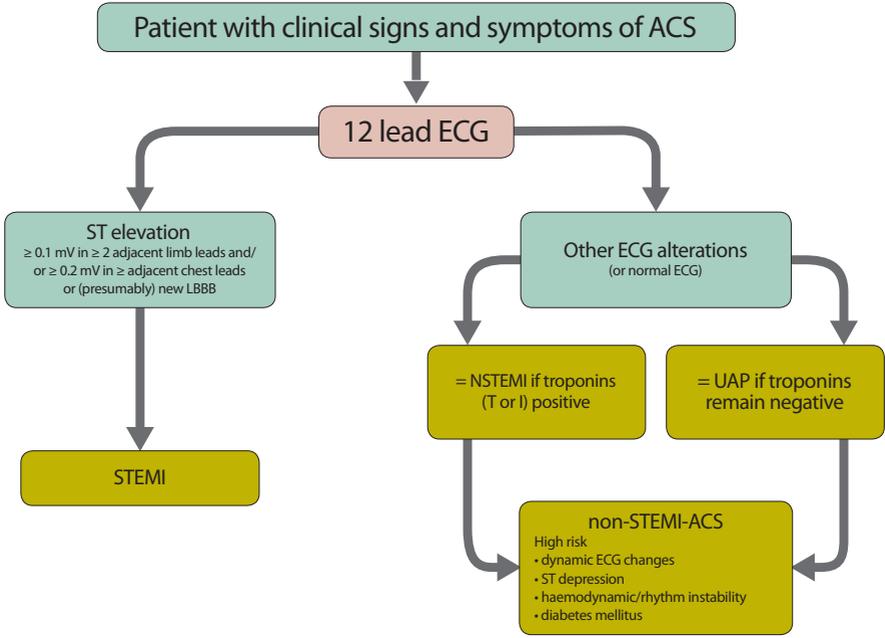
* Alternatives include:
• Aminophylline
• Dopamine
• Glucagon (if beta-blocker or calcium channel blocker overdose)
• Glycopyrrolate can be used instead of atropine



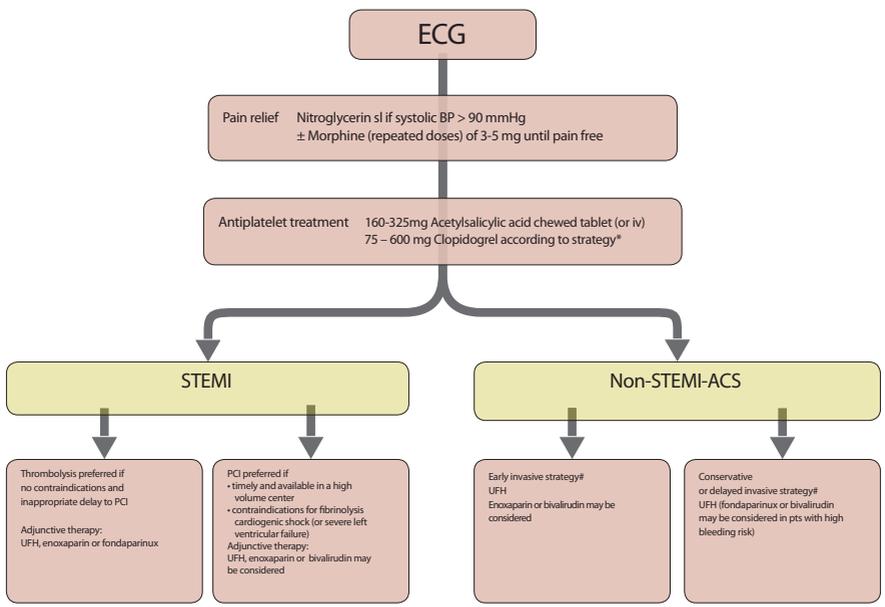
- ◆ The use of up to three quick successive (stacked) shocks for ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) occurring in the cardiac catheterisation laboratory or in the immediate post-operative period following cardiac surgery.
- ◆ Delivery of drugs via a tracheal tube is no longer recommended – if intravenous access cannot be achieved, drugs should be given by the intraosseous (IO) 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 minutes (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 (PEA).
- ◆ 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 (including 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 $>10 \text{ mmol l}^{-1}$ ($>180 \text{ mg dl}^{-1}$) should be treated but hypoglycaemia must be avoided.



ACS



ECG



- ◆ 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, especially if the patient has been treated with therapeutic hypothermia.
- ◆ The role of chest pain observation units (CPUs) is to identify, by using repeated clinical examinations, ECG and biomarker testing, those patients who require admission for invasive procedures. This may include provocative testing and, in selected patients, imaging procedures such as cardiac computed tomography, magnetic resonance imaging etc.
- ◆ Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided.

Initial management of acute coronary syndromes

Changes in the management of acute coronary syndrome since the 2005 guidelines include:

- ◆ The term non-ST-elevation myocardial infarction-acute coronary syndrome (non-STEMI-ACS) has been introduced for both NSTEMI and unstable angina pectoris because the differential diagnosis is dependent on biomarkers that may be detectable only after several hours, whereas decisions on treatment are dependent on the clinical signs at presentation.
- ◆ History, clinical examinations, biomarkers, ECG criteria and risk scores are unreliable for the identification of patients who may be safely discharged early.
- ◆ Nitrates should not be used for diagnostic purposes.
- ◆ Supplementary oxygen is to be given only to those patients with hypoxaemia, breathlessness or pulmonary congestion. Hyperoxaemia may be harmful in uncomplicated infarction.
- ◆ Guidelines for treatment with acetyl salicylic acid (ASA) have been made more liberal: ASA may now be given by bystanders with or without EMS dispatcher assistance.
- ◆ Revised guidance for new anti-platelet and anti-thrombin treatment for patients with STEMI and non-STEMI-ACS based on therapeutic strategy.



◆ Gp IIb/IIIa inhibitors before angiography/percutaneous coronary intervention (PCI) are discouraged.

◆ The reperfusion strategy in ST-elevation myocardial infarction has been updated:

- Primary PCI (PPCI) is the preferred reperfusion strategy provided it is performed in a timely manner by an experienced team.

- A nearby hospital may be bypassed by emergency medical services (EMS) provided PPCI can be achieved without too much delay.

- The acceptable delay between start of fibrinolysis and first balloon inflation varies widely between about 45 and 180 minutes depending on infarct localisation, age of the patient, and duration of symptoms.

- 'Rescue PCI' should be undertaken if fibrinolysis fails.

- The strategy of routine PCI immediately after fibrinolysis ('facilitated PCI') is discouraged.

- Patients with successful fibrinolysis but not in a PCI-capable hospital should be transferred for angiography and eventual PCI, performed optimally 6 – 24 hours after fibrinolysis (the 'pharmaco-invasive' approach).

- Angiography and, if necessary, PCI may be reasonable in patients with return of spontaneous circulation (ROSC) after cardiac arrest and may be part of a standardised post-cardiac arrest protocol.

- To achieve these goals, the creation of networks including EMS, non PCI capable hospitals and PCI hospitals is useful.

◆ Recommendations for the use of beta-blockers are more restricted: there is no evidence for routine intravenous beta-blockers except in specific circumstances such as for the treatment of tachyarrhythmias. Otherwise, beta-blockers should be started in low doses only after the patient is stabilised.

◆ Guidelines on the use of prophylactic anti-arrhythmics, angiotensin converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs) and statins are unchanged.

Paediatric life support

Major changes in these new guidelines for paediatric life support include:

◆ Recognition of cardiac arrest - Healthcare providers cannot reliably determine the presence or absence of a pulse in less than 10 seconds in



infants or children. Healthcare providers should look for signs of life and if they are confident in the technique, they may add pulse palpation for diagnosing cardiac arrest and decide whether they should begin chest compressions or not. The decision to begin CPR must be taken in less than 10 seconds. According to the child's age, carotid (children), brachial (infants) or femoral pulse (children and infants) checks may be used.

◆ The compression ventilation (CV) ratio used for children should be based on whether one, or more than one rescuer is present. Lay rescuers, who usually learn only single-rescuer techniques, should be taught to use a ratio of 30 compressions to 2 ventilations, which is the same as the adult guidelines and enables anyone trained in BLS to resuscitate children with minimal additional information. Rescuers with a duty to respond should learn and use a 15:2 CV ratio; however, they can use the 30:2 ratio if they are alone, particularly if they are not achieving an adequate number of compressions. Ventilation remains a very important component of CPR in asphyxial arrests. Rescuers who are unable or unwilling to provide mouth-to-mouth ventilation should be encouraged to perform at least compression-only CPR.

◆ The emphasis is on achieving quality compressions of an adequate depth with minimal interruptions to

minimise no-flow time. Compress the chest to at least 1/3 of the anterior-posterior chest diameter in all children (i.e., approximately 4 cm in infants and approximately 5 cm in children). Subsequent complete release is emphasised. For both infants and children, the compression rate should be at least 100 but not greater than 120 min⁻¹. The compression technique for infants includes two-finger compression for single rescuers and the two-thumb encircling technique for two or more rescuers. For older children, a one- or two-hand technique can be used, according to rescuer preference.

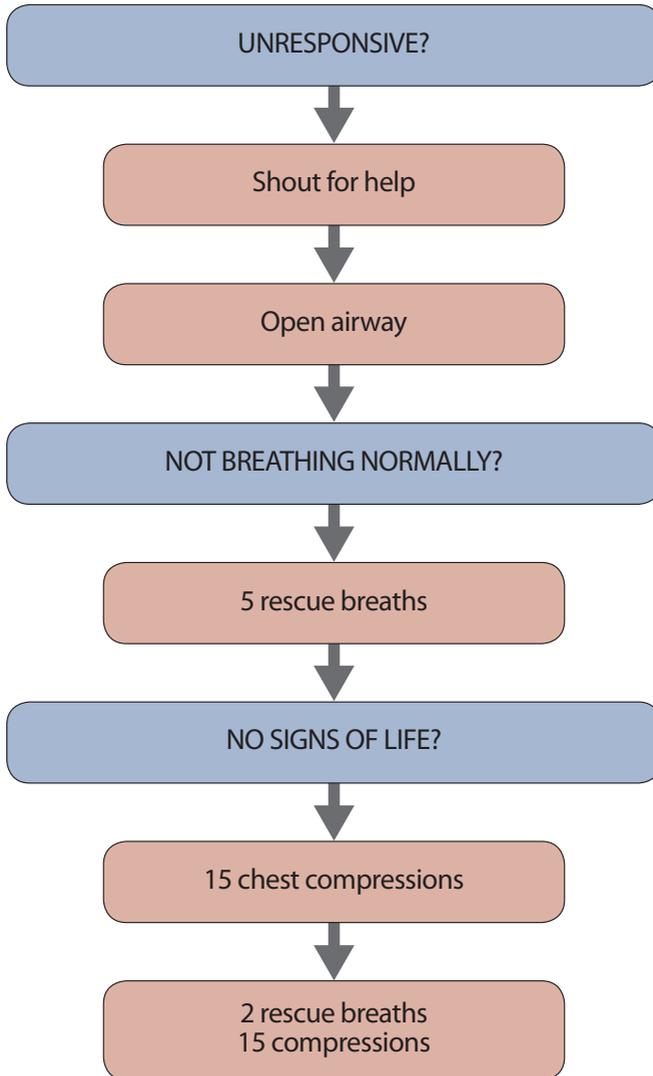
◆ Automated external defibrillators (AEDs) are safe and successful when used in children older than one year of age. Purpose-made paediatric pads or software attenuate the output of the machine to 50–75 J and these are recommended for children aged 1–8 years. If an attenuated shock or a manually adjustable machine is not available, an unmodified adult AED may be used in children older than 1 year. There are case reports of successful use of AEDs in children aged less than 1 year; in the rare case of a shockable rhythm occurring in a child less than 1 year, it is reasonable to use an AED (preferably with dose attenuator).

◆ To reduce the no flow time, when using a manual defibrillator, chest compressions are continued while applying and charging the paddles or



Paediatric Basic Life Support

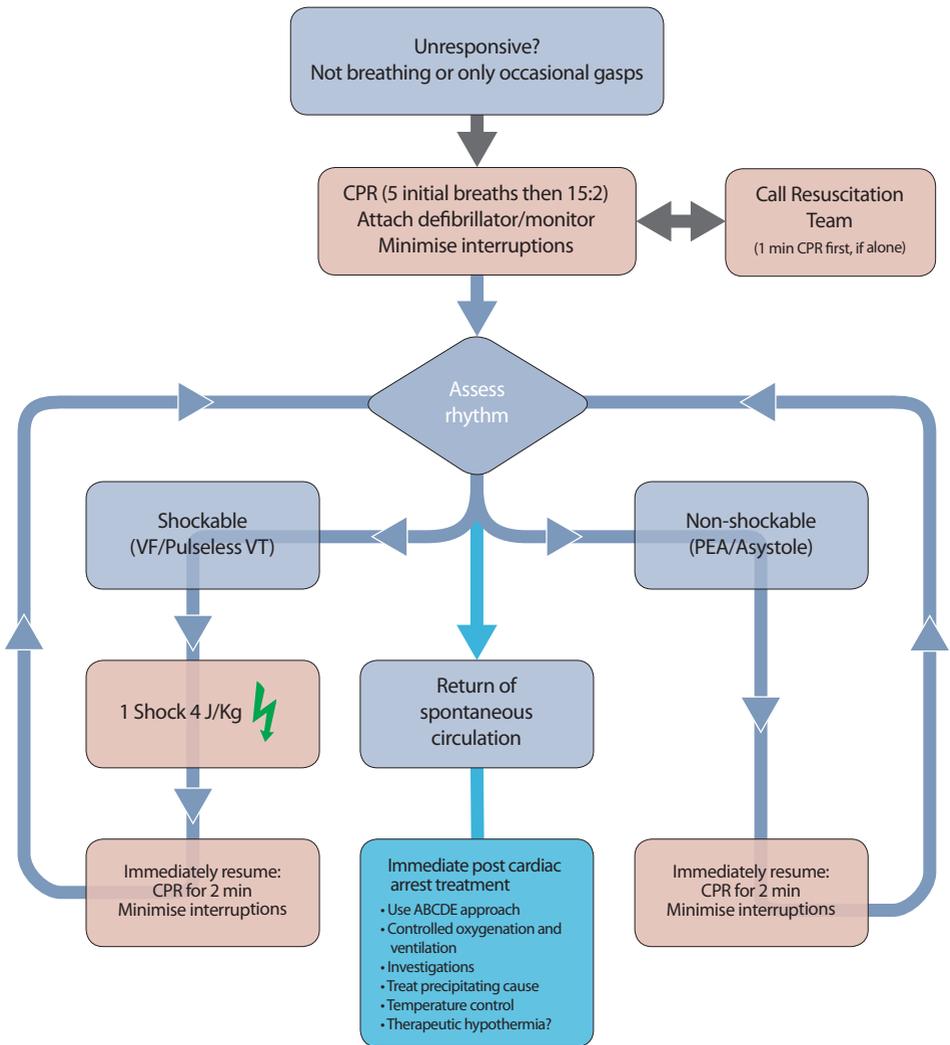
Health professionals with a duty to respond



Call cardiac arrest team or Paediatric ALS team



Paediatric Advanced Life Support



During CPR

- Ensure high-quality CPR: rate, depth, recoil
- Plan actions before interrupting CPR
- Give oxygen
- Vascular access (intravenous, intraosseous)
- Give adrenaline every 3-5 min
- Consider advanced airway and capnography
- Continuous chest compressions when advanced airway in place
- Correct reversible causes

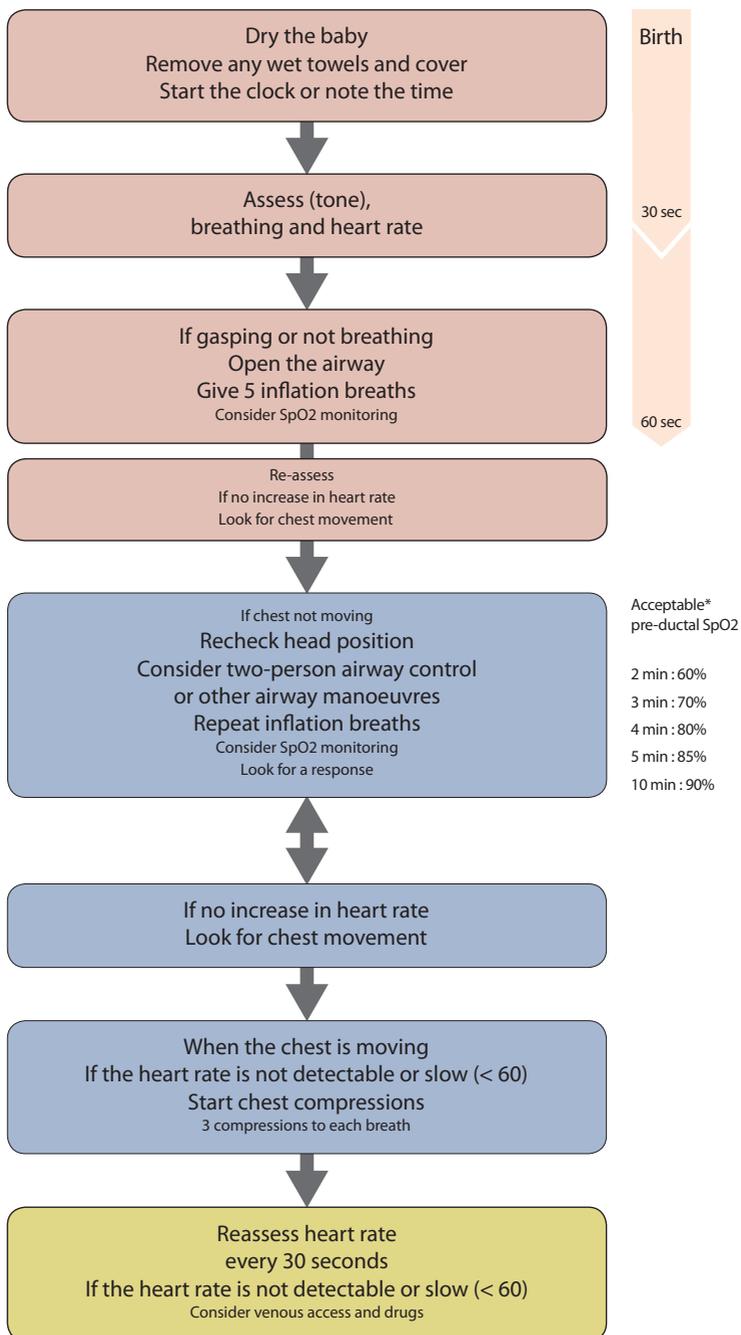
Reversible causes

- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypothermia
- Tension pneumothorax
- Toxins
- Tamponade - cardiac
- Thromboembolism



Newborn Life Support

AT ALL STAGES ASK: DO YOU NEED HELP?



self-adhesive pads (if the size of the child's chest allows this). Chest compressions are paused briefly once the defibrillator is charged to deliver the shock. For simplicity and consistency with adult BLS and ALS guidance, a single-shock strategy using a non-escalating dose of 4 J kg^{-1} (preferably biphasic, but monophasic is acceptable) is recommended for defibrillation in children.

- ◆ Cuffed tracheal tubes can be used safely in infants and young children. The size should be selected by applying a validated formula.

- ◆ The safety and value of using cricoid pressure during tracheal intubation is not clear. Therefore, the application of cricoid pressure should be modified or discontinued if it impedes ventilation or the speed or ease of intubation.

- ◆ Monitoring exhaled carbon dioxide (CO_2), ideally by capnography, is helpful to confirm correct tracheal tube position and recommended during CPR to help assess and optimise its quality.

- ◆ Once spontaneous circulation is restored, inspired oxygen should be titrated to limit the risk of hyperoxaemia.

- ◆ Implementation of a rapid response system in a paediatric inpatient setting may reduce rates of cardiac and respiratory arrest and in-hospital mortality.

- ◆ New topics in the 2010 guidelines include channelopathies and several new special circumstances: trauma, single ventricle pre and post 1st stage repair, post Fontan circulation, and pulmonary hypertension.

Resuscitation of babies at birth

The following are the main changes that have been made to the guidelines for resuscitation at birth in 2010:

- ◆ For uncompromised babies, a delay in cord clamping of at least one minute from the complete delivery of the infant, is now recommended. As yet there is insufficient evidence to recommend an appropriate time for clamping the cord in babies who are severely compromised at birth.

- ◆ For term infants, air should be used for resuscitation at birth. If, despite effective ventilation, oxygenation (ideally guided by oximetry) remains unacceptable, use of a higher concentration of oxygen should be considered.



- ◆ Preterm babies less than 32 weeks gestation may not reach the same transcutaneous oxygen saturations in air as those achieved by term babies. Therefore blended oxygen and air should be given judiciously and its use guided by pulse oximetry. If a blend of oxygen and air is not available use what is available.
- ◆ Preterm babies of less than 28 weeks gestation should be completely covered in a food-grade plastic wrap or bag up to their necks, without drying, immediately after birth. They should then be nursed under a radiant heater and stabilised. They should remain wrapped until their temperature has been checked after admission. For these infants delivery room temperatures should be at least 26°C.
- ◆ The recommended compression:ventilation ratio for CPR remains at 3:1 for newborn resuscitation.
- ◆ Attempts to aspirate meconium from the nose and mouth of the unborn baby, while the head is still on the perineum, are not recommended. If presented with a floppy, apnoeic baby born through meconium it is reasonable to rapidly inspect the oropharynx to remove potential obstructions. If appropriate expertise is available, tracheal intubation and suction may be useful. However, if attempted intubation is prolonged or unsuccessful, start mask ventilation, particularly if there is persistent bradycardia.
- ◆ If adrenaline is given then the intravenous route is recommended using a dose of 10-30 microgram kg^{-1} . If the tracheal route is used, it is likely that a dose of at least 50-100 microgram kg^{-1} will be needed to achieve a similar effect to 10 microgram kg^{-1} intravenously.
- ◆ Detection of exhaled carbon dioxide in addition to clinical assessment is recommended as the most reliable method to confirm placement of a tracheal tube in neonates with spontaneous circulation.
- ◆ Newly born infants born at term or near-term with evolving moderate to severe hypoxic – ischaemic encephalopathy should, where possible, be treated with therapeutic hypothermia. This does not affect immediate resuscitation but is important for post-resuscitation care.



Principles of education in resuscitation

The key issues identified by the Education, Implementation and Teams (EIT) task force of the International Liaison Committee on Resuscitation (ILCOR) during the Guidelines 2010 evidence evaluation process are:

- ◆ Educational interventions should be evaluated to ensure that they reliably achieve the learning objectives. The aim is to ensure that learners acquire and retain the skills and knowledge that will enable them to act correctly in actual cardiac arrests and improve patient outcomes.
- ◆ Short video/computer self-instruction courses, with minimal or no instructor coaching, combined with hands-on practice can be considered as an effective alternative to instructor-led basic life support (CPR and AED) courses.
- ◆ Ideally all citizens should be trained in standard CPR that includes compressions and ventilations. There are circumstances however where training in compression-only CPR is appropriate (e.g., opportunistic training with very limited time). Those trained in compression-only CPR should be encouraged to learn standard CPR.
- ◆ Basic and advanced life support knowledge and skills deteriorate in as little as three to six months. The use of frequent assessments will identify those individuals who require refresher training to help maintain their knowledge and skills.
- ◆ CPR prompt or feedback devices improve CPR skill acquisition and retention and should be considered during CPR training for laypeople and healthcare professionals.
- ◆ An increased emphasis on non-technical skills (NTS) such as leadership, teamwork, task management and structured communication will help improve the performance of CPR and patient care.
- ◆ Team briefings to plan for resuscitation attempts, and debriefings based on performance during simulated or actual resuscitation attempts should be used to help improve resuscitation team and individual performance.
- ◆ Research about the impact of resuscitation training on actual patient outcomes is limited. Although manikin studies are useful, researchers should be encouraged to study and report the impact of educational interventions on actual patient outcomes.



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