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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 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, 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 10 s 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 s. 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 (CO_2), 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 $\mu g\,kg^{-1}$. If the tracheal route is used, it is likely that a dose of at least 50–100 $\mu g\,kg^{-1}$ will be needed to achieve a similar effect to 10 $\mu g\,kg^{-1}$ 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 (OHCAs) 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 Conference[40] 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.

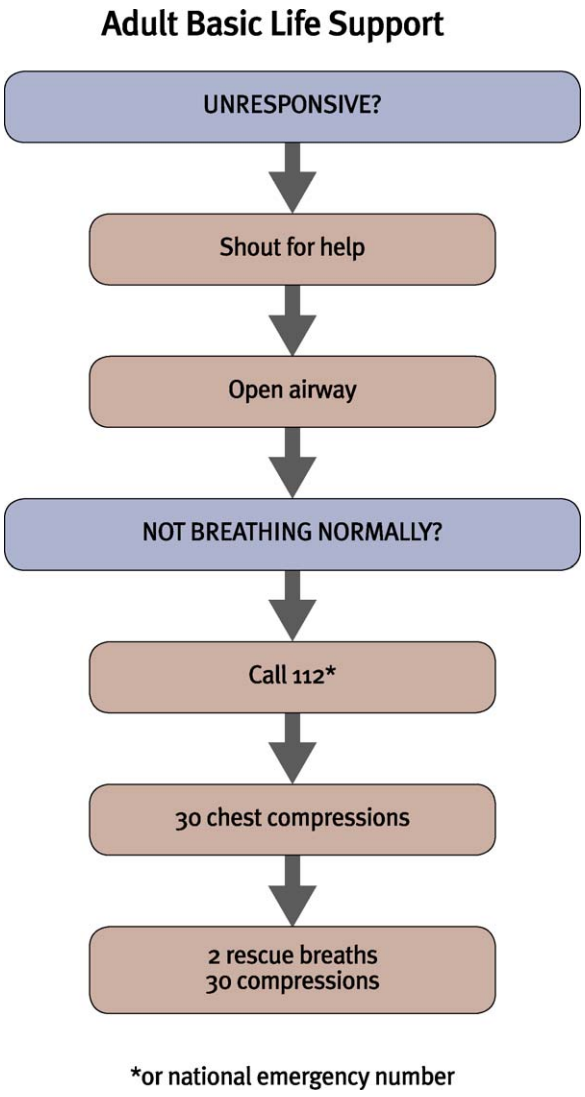

Fig. 1.2. Basic life support algorithm.

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.

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.

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 CO_2 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

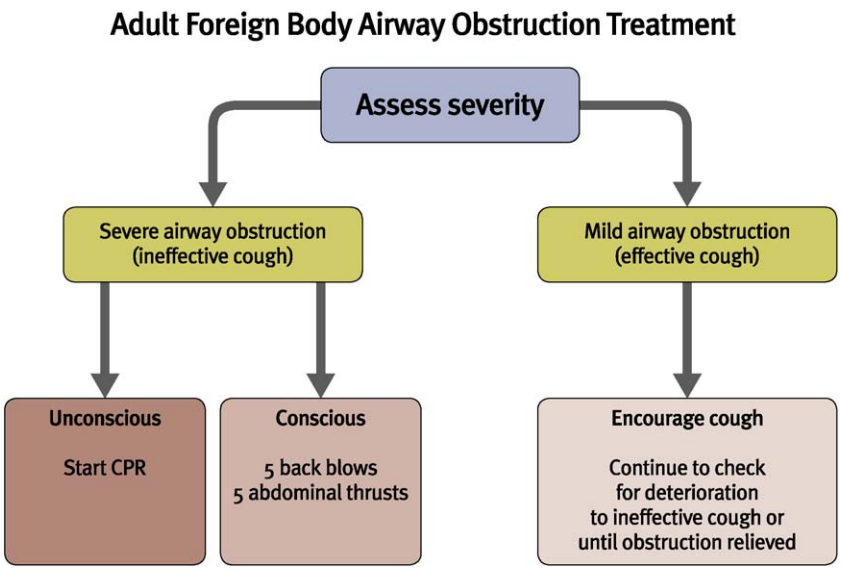

Fig. 1.3. Adult foreign-body airway obstruction (choking) sequence. © 2010 ERC.

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 casinos[55] 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%.

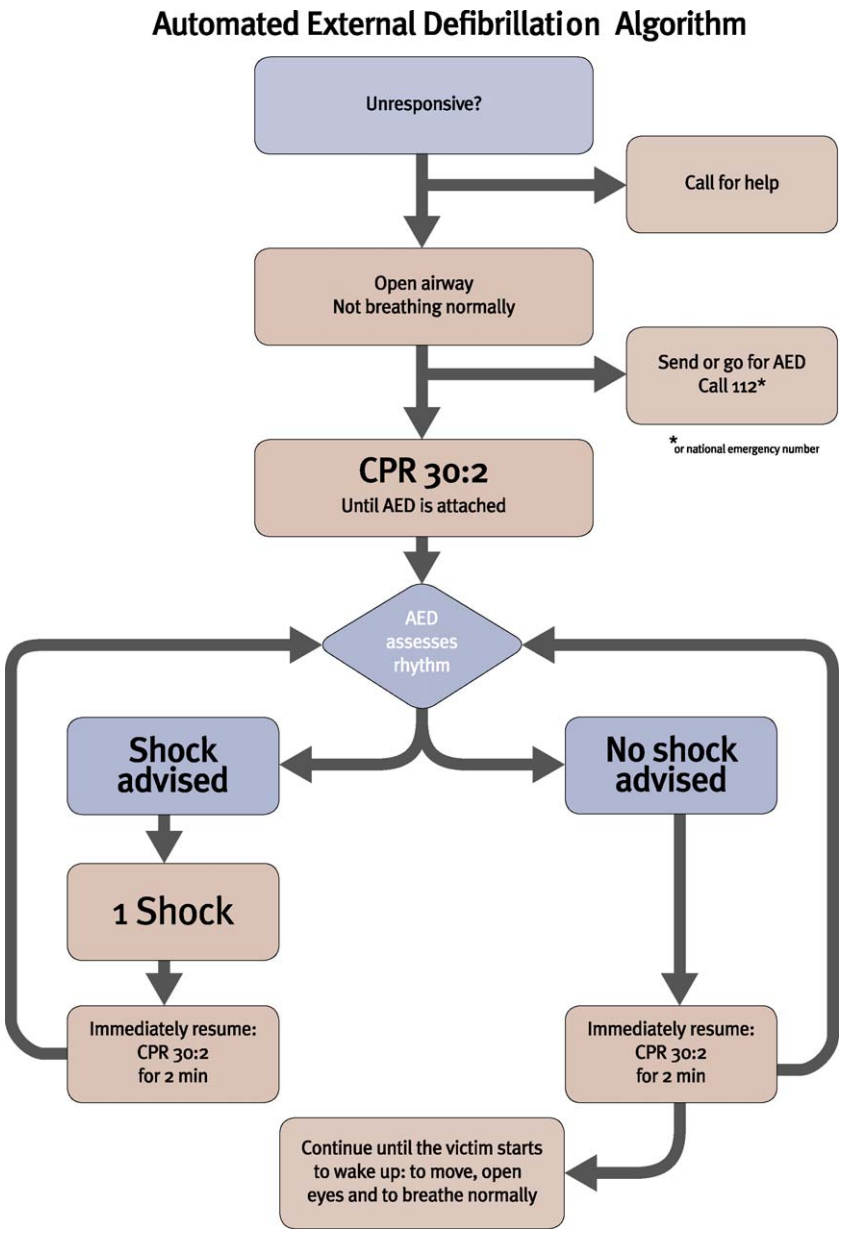

Fig. 1.4. AED algorithm. © 2010 ERC.

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-hospital[105] and pre-hospital[106] 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 survival[142] 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 protocol[148] 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 SpO_2.[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;

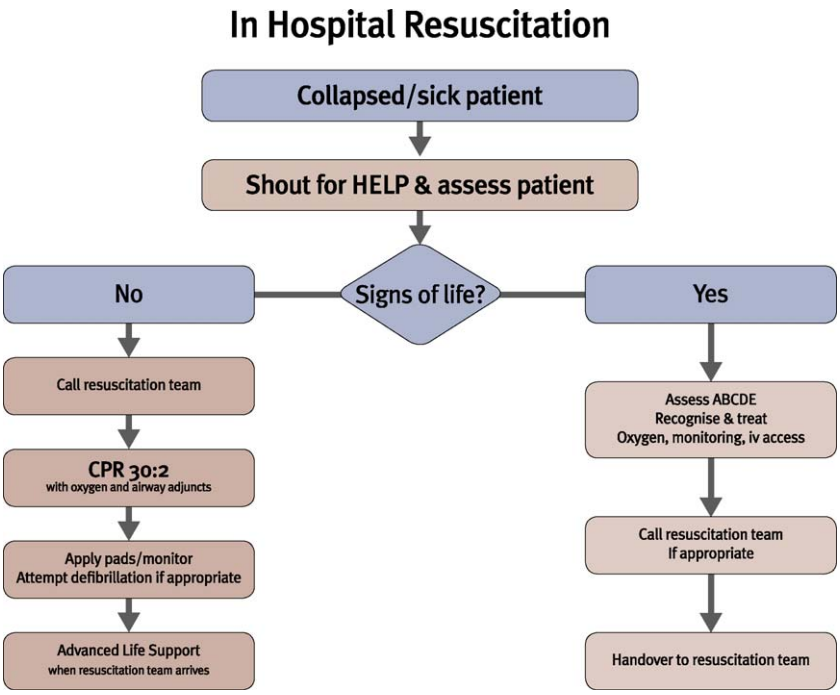

Fig. 1.5. Algorithm for the initial management of in-hospital cardiac arrest. © 2010 ERC.

- 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.

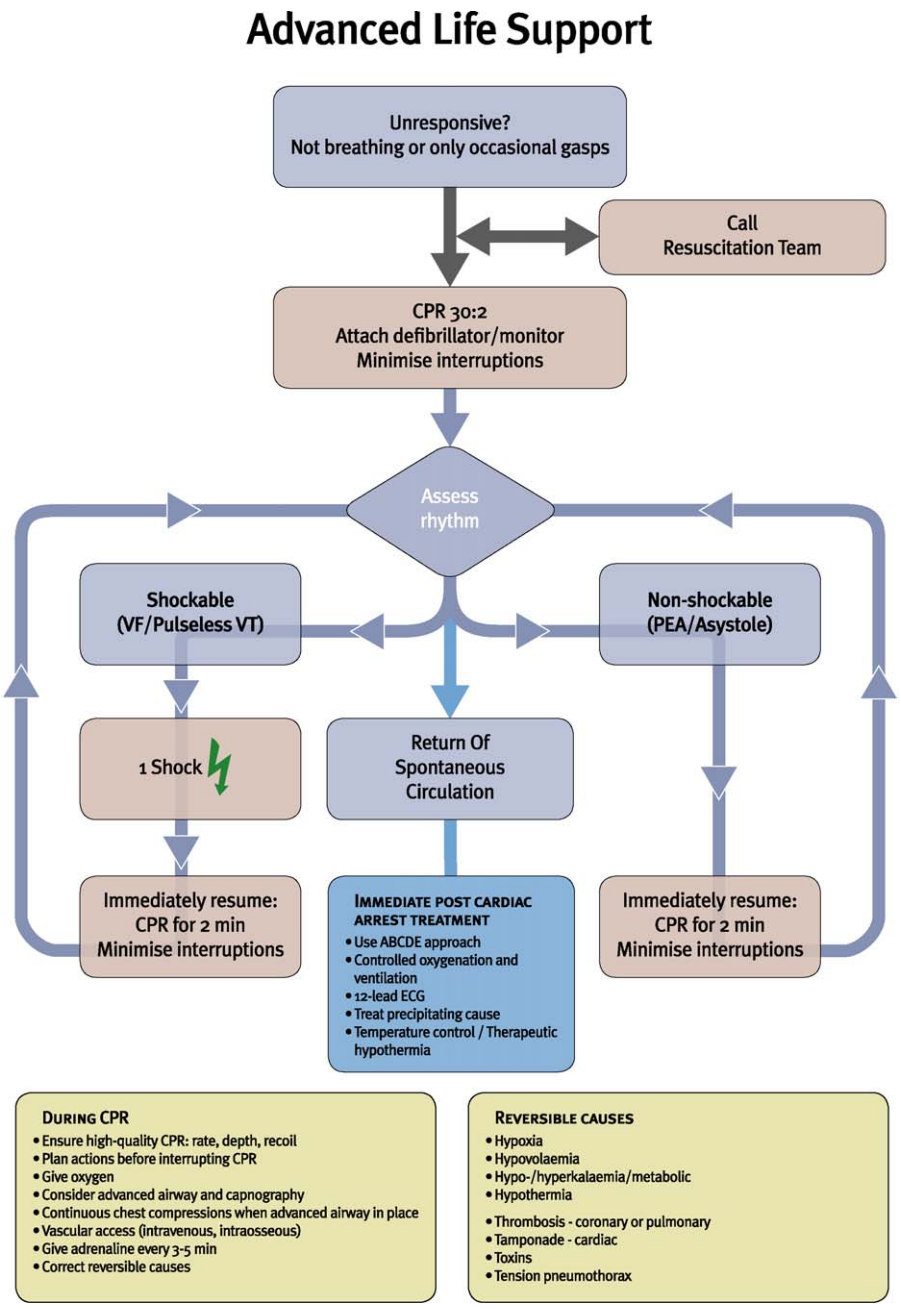

Fig. 1.6. ALS cardiac arrest algorithm. © 2010 ERC.

- 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 $ETCO_2$ monitoring can be used to indicate the quality of CPR: although an optimal target for $ETCO_2$ during CPR has not been established, a value of less than 10 mm Hg (1.4 kPa) is associated with failure to achieve ROSC and may indicate that the quality of chest compressions should be improved. If possible, the person providing chest compressions should be 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 preshock 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 established[230] 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 CO_2 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 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.

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 placebo[244] 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 (SaO_2) during CPR. There are animal data[273] and some observational clinical data indicating an association between high SaO_2 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 (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%.

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 CO_2 detectors, oesophageal detector devices and non-waveform capnometers does not exceed the accuracy of auscultation and direct visualization for confirming the tracheal position of a tube in victims of cardiac arrest. Waveform capnography is the most sensitive and specific way to confirm and continuously monitor the position of a tracheal tube in victims of cardiac arrest and should supplement clinical assessment (auscultation and visualization of tube through cords). 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 scans[300] 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, SpO_2). Whenever possible, record a 12-lead ECG; this will help determine the precise rhythm, either before treatment or retrospectively. Correct any electrolyte abnormalities (e.g., K^+, Mg^{2+}, Ca^{2+}). Consider the cause and context of arrhythmias when planning treatment.

The assessment and treatment of all arrhythmias addresses two factors: the condition of the patient (stable versus unstable), and the nature of the arrhythmia. Anti-arrhythmic drugs are slower in onset and less reliable than electrical cardioversion in converting a tachycardia to sinus rhythm; thus, drugs tend to be reserved for stable patients without adverse signs, and electrical cardioversion is usually the preferred treatment for the unstable patient displaying adverse signs.

Adverse signs

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

1. Shock – this is seen as pallor, sweating, cold and clammy extremities (increased sympathetic activity), impaired consciousness (reduced cerebral blood flow), and hypotension (e.g., systolic blood pressure <90 mm Hg).
2. Syncope – loss of consciousness, which occurs as a consequence of reduced cerebral blood flow.
3. Heart failure – arrhythmias compromise myocardial performance by reducing coronary artery blood flow. In acute situations this is manifested by pulmonary oedema (failure of the left ventricle) and/or raised jugular venous pressure, and hepatic engorgement (failure of the right ventricle).
4. Myocardial ischaemia – this occurs when myocardial oxygen consumption exceeds delivery. Myocardial ischaemia may present with chest pain (angina) or may occur without pain as an isolated finding on the 12 lead ECG (silent ischaemia). The presence of myocardial ischaemia is especially important if there is underlying coronary artery disease or structural heart disease because it may cause further life-threatening complications including cardiac arrest.

Treatment options

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

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

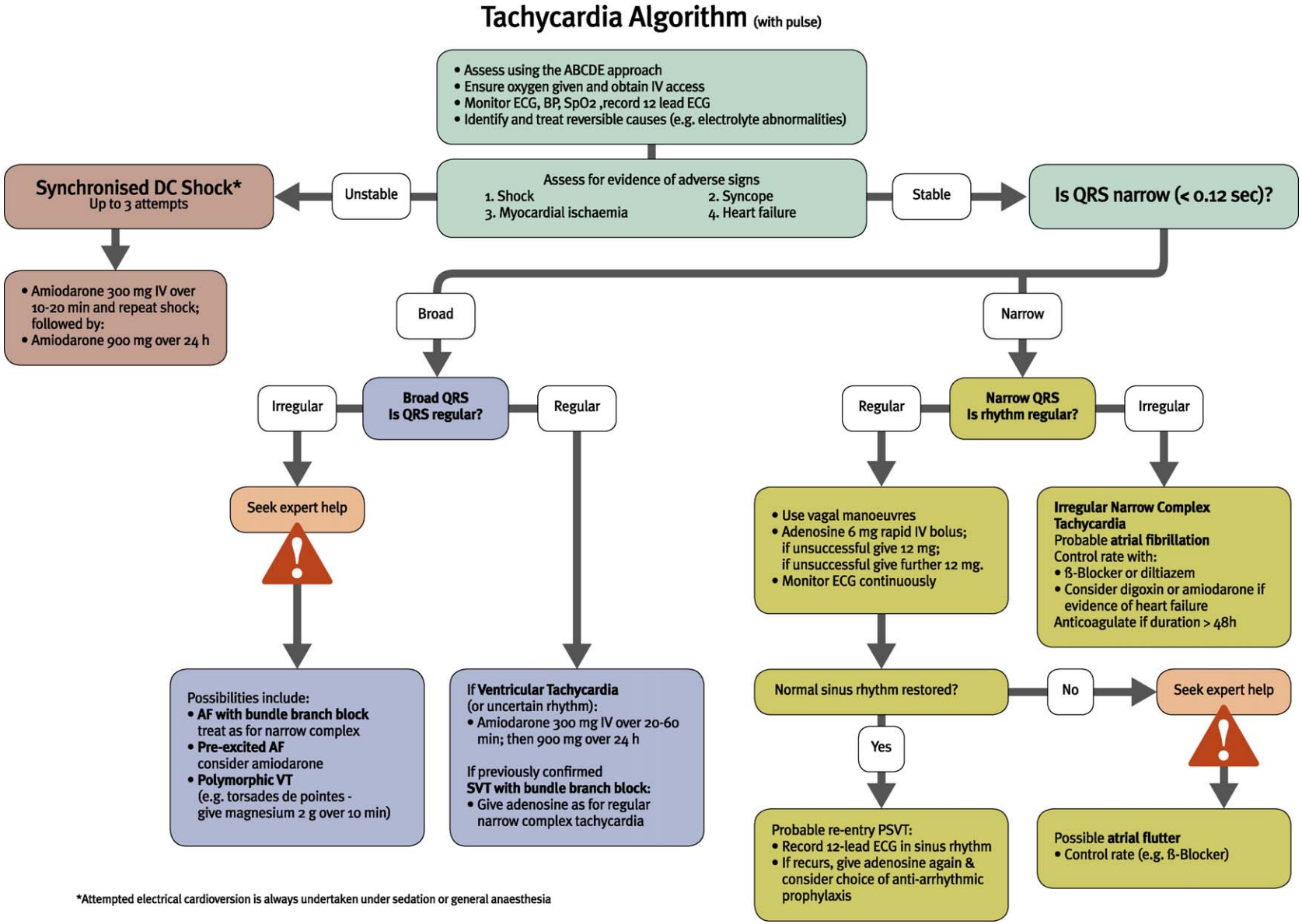

Fig. 1.7. Tachycardia algorithm. © 2010 ERC.

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

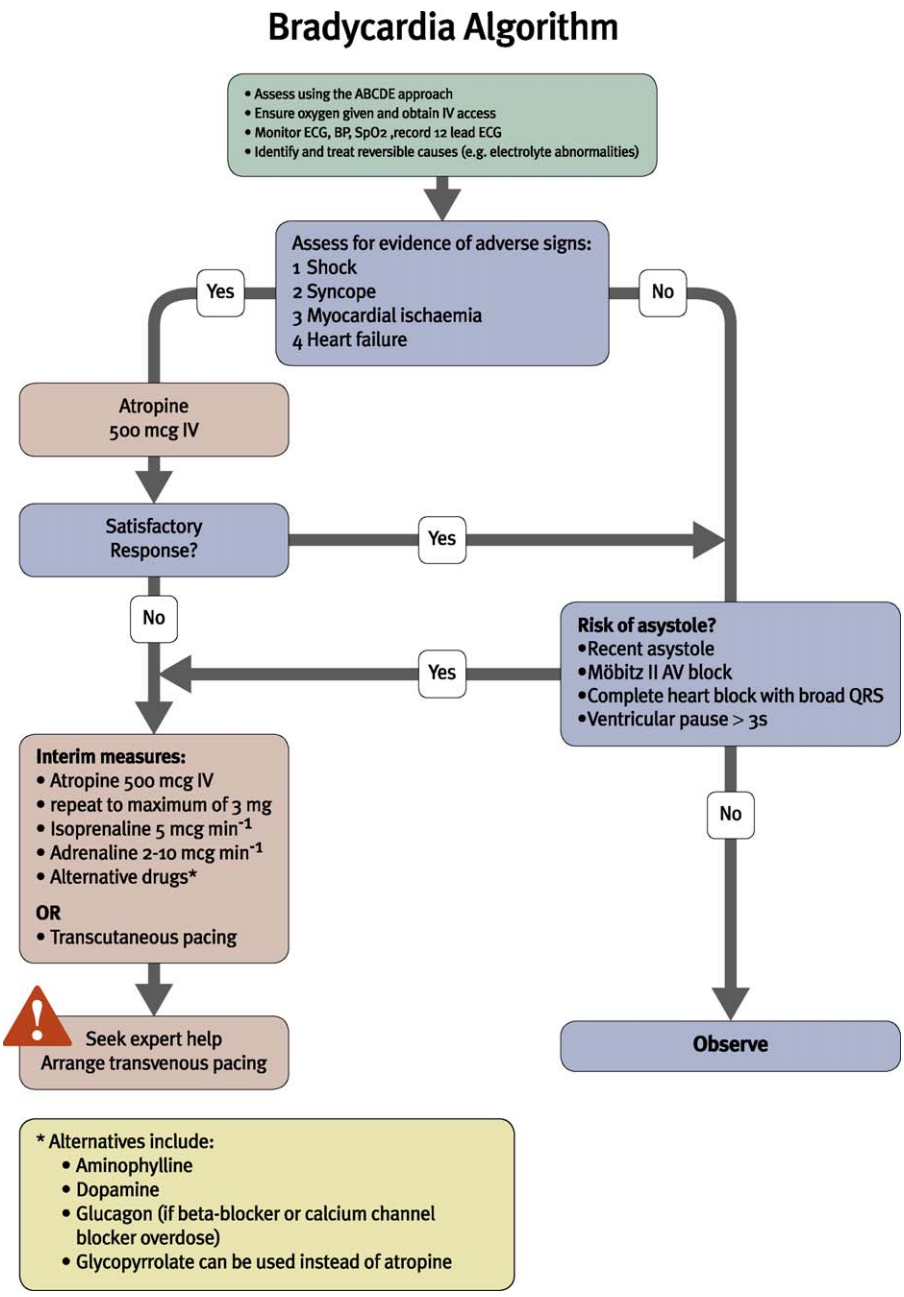

Fig. 1.8. Bradycardia algorithm. © 2010 ERC.

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 PCO_2 after resuscitation from cardiac arrest, but it is reasonable to adjust ventilation to achieve normocarbia and to monitor this using the end-tidal PCO_2 and arterial blood gas values.

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-fold[331] 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 $\leq 10\ mmol\ l^{-1}$ ($180\ mg\ dl^{-1}$).[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 $\geq 37.6\,^{\circ}C$) 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 ($CMRO_2$) by about 6% for each $1\,^{\circ}C$ reduction in temperature[354] 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 trial[355] and a pseudo-randomised trial[356] 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 with[227] and without[305] 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 countries[408]; 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.

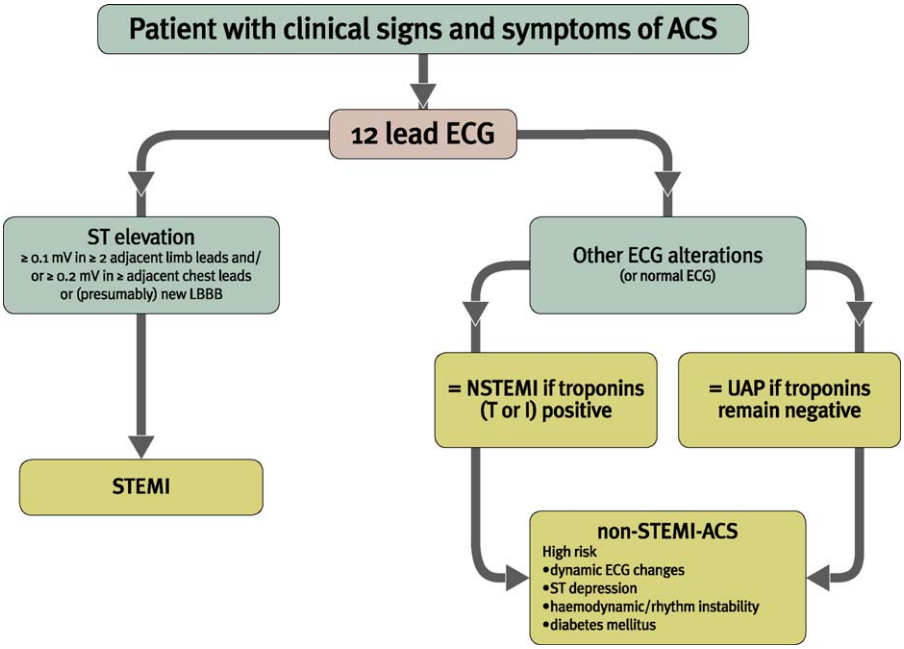

Fig. 1.9. Definitions of acute coronary syndromes (ACS); STEMI, ST elevation myocardial infarction; NSTEMI, non-ST elevation myocardial infarction; UAP, unstable angina pectoris.

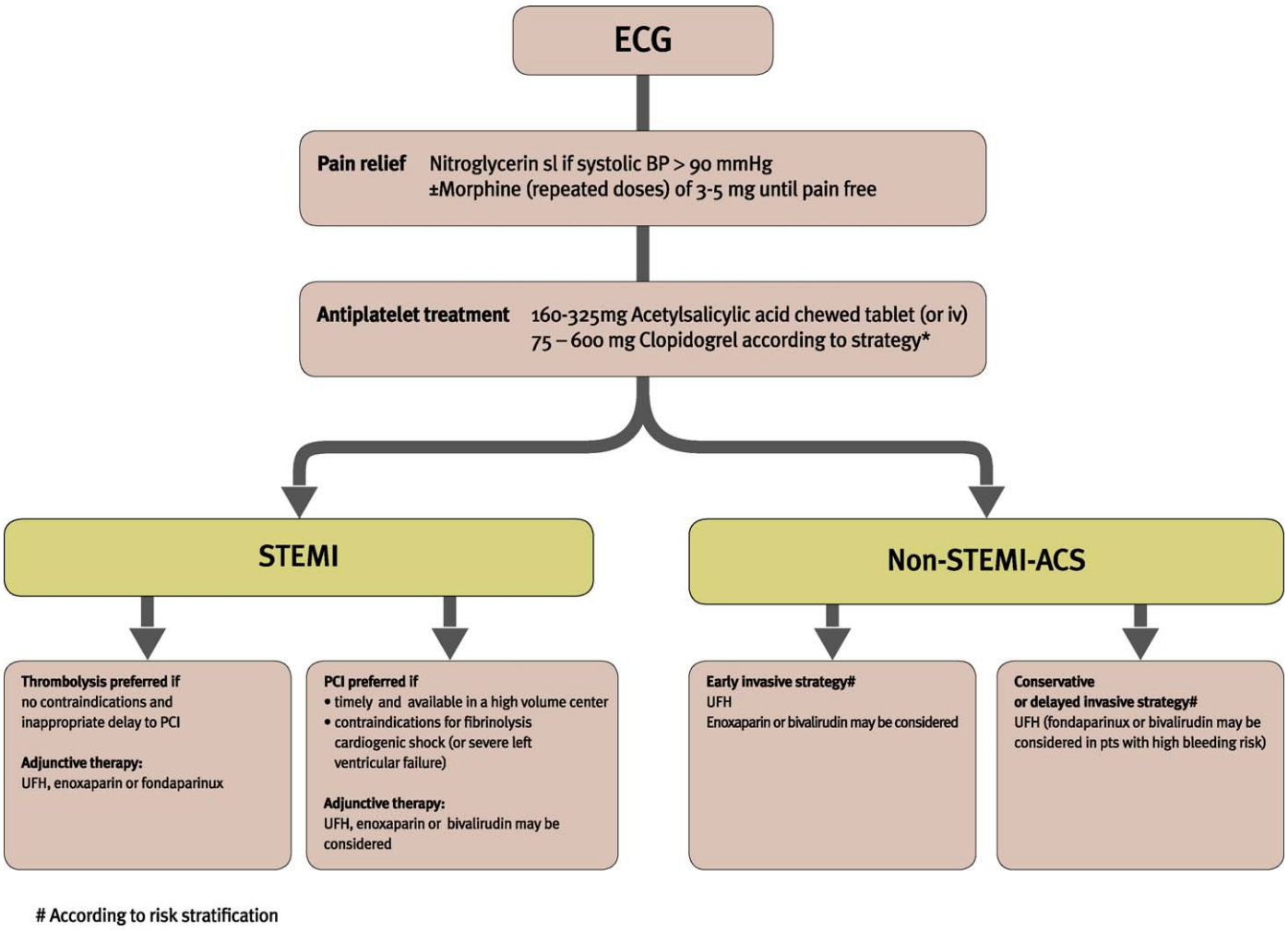

Fig. 1.10. Treatment algorithm for acute coronary syndromes; BP, blood pressure; PCI, percutaneous coronary intervention; UFH, unfractionated heparin. *Prasugrel, 60 mg loading dose, may be chosen as an alternative to clopidogrel in patients with STEMI and planned PPCI provided there is no history of 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 healthcare 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 ≤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 (SaO_2) 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-pentyltriazolo-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. Eptifibatide 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 (≥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. prehospital 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).

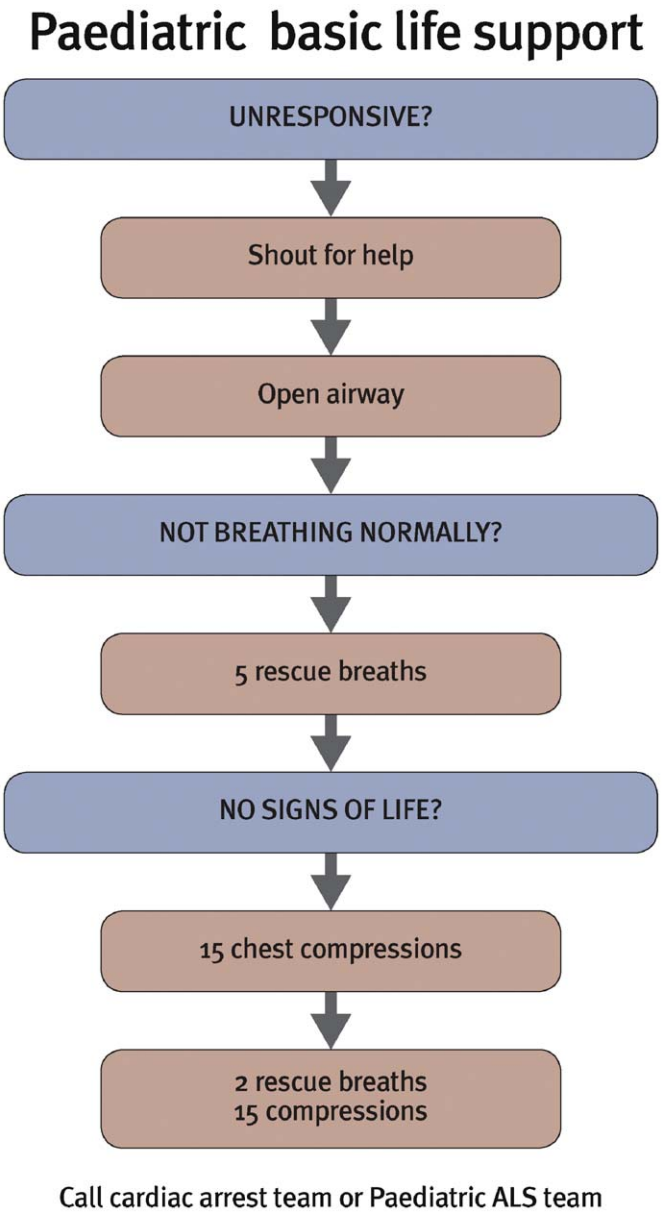

Fig. 1.11. Paediatric basic life support algorithm for those with a duty to respond.

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	

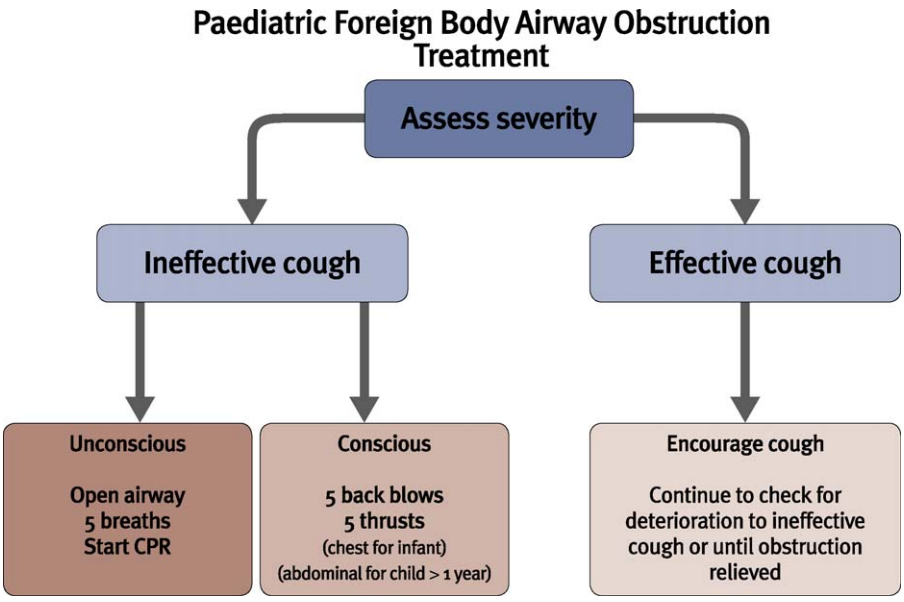

Fig. 1.12. Paediatric foreign body airway obstruction algorithm. © 2010 ERC.

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/SpO_2).
- 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 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 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.

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

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 H_2O.[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 CO_2 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 SpO_2 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 CO_2 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 (SaO_2) 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 ($PaCO_2$).

Monitoring end-tidal CO_2 ($ETCO_2$) 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 CO_2 during cardiopulmonary arrest does not guarantee tube misplacement since a low or absent end tidal CO_2 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 $ETCO_2$ remains below 15 mm Hg (2 kPa). Current evidence does not support the use of a threshold $ETCO_2$ 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 (SaO_2) is unreliable; therefore, monitor the child's peripheral oxygen saturation continuously by pulse oximetry (SpO_2).

Circulation

- Establish cardiac monitoring [first line—pulse oximetry (SpO_2), 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, 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.[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 ($ScvO_2$) 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}):

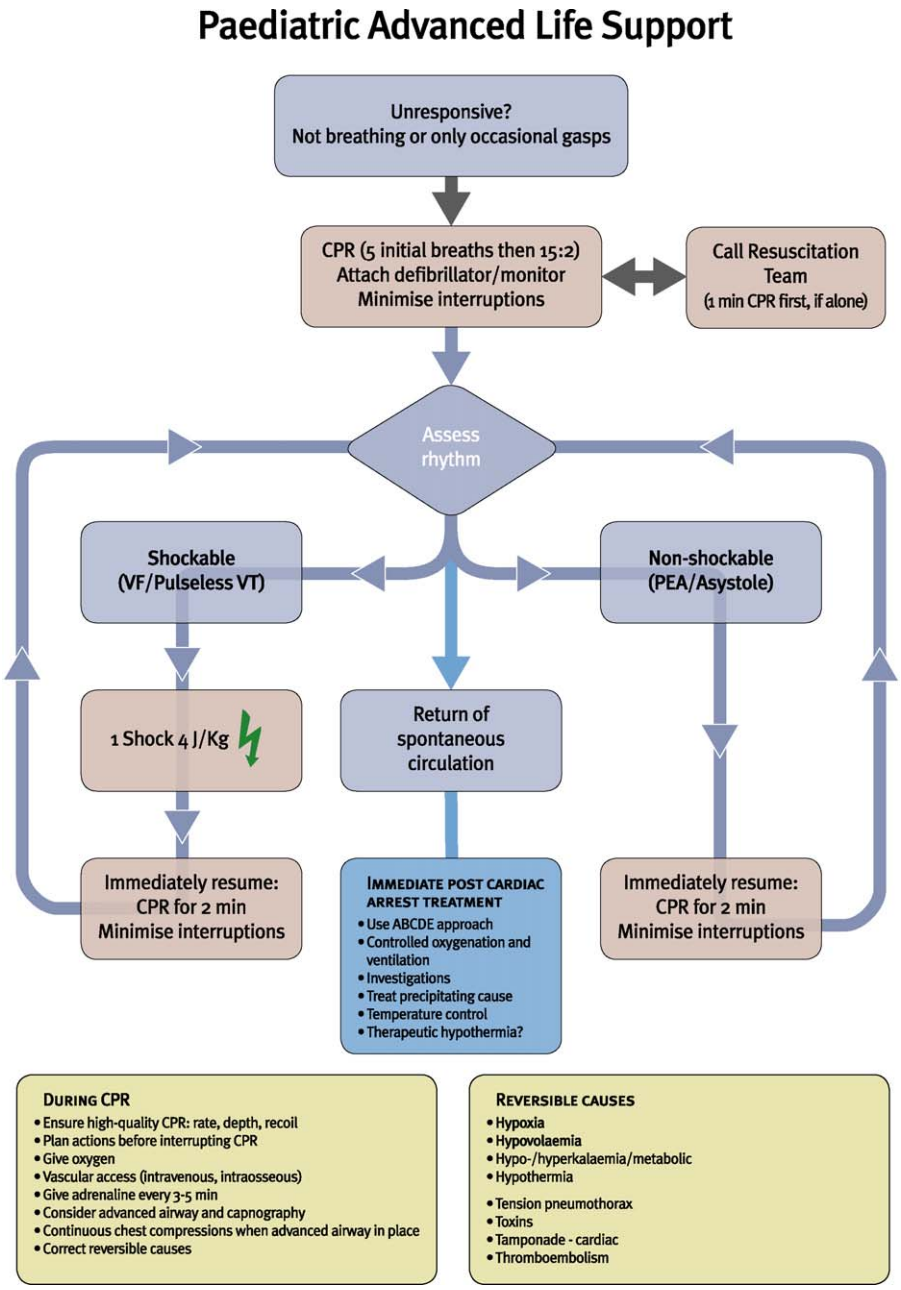

Fig. 1.13. Paediatric advanced life support algorithm. © 2010 ERC.

- 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 μg 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 visualised[527] 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 $ScvO_2$) 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 FiO_2 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 tension[573] 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,

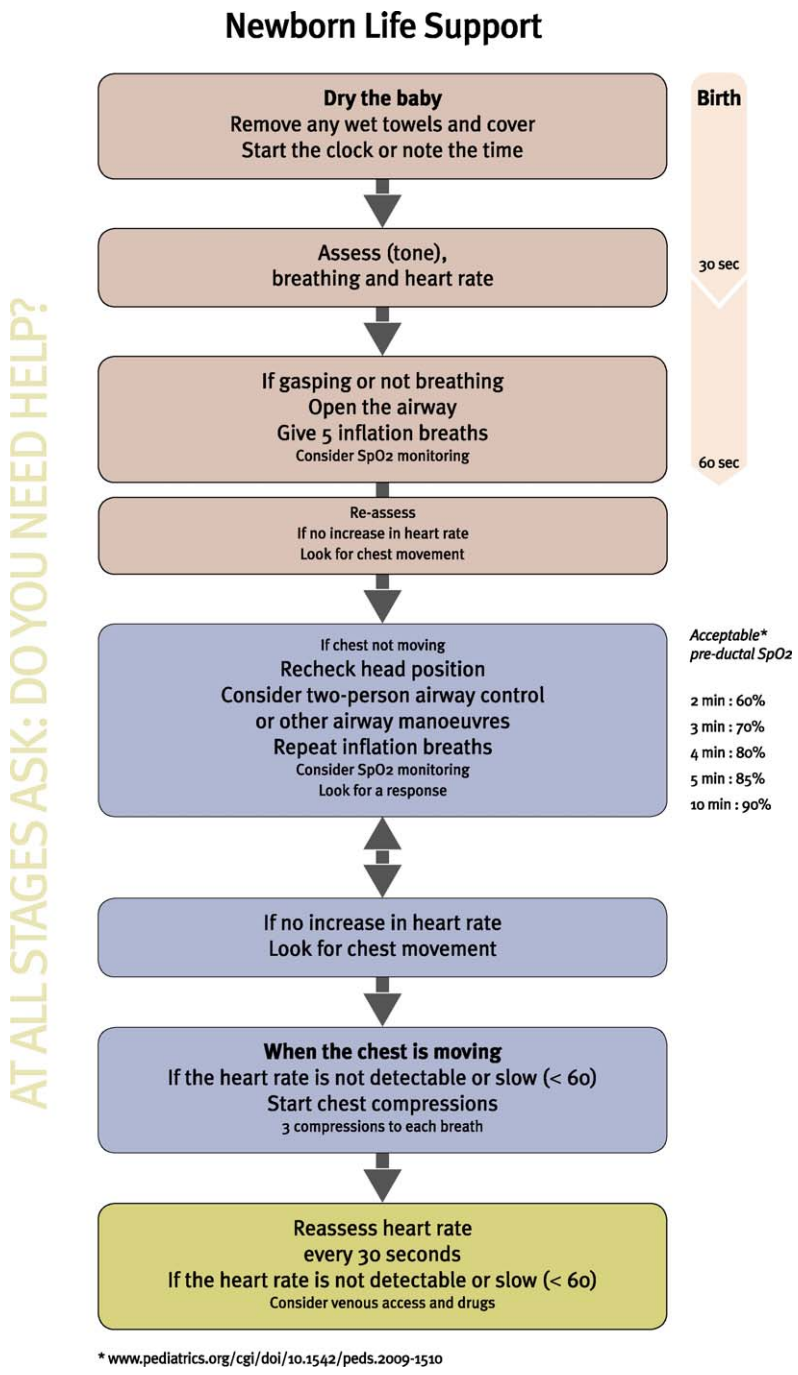

Fig. 1.14. Newborn life support algorithm. © 2010 ERC.

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] Withold 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 rewarming the patient. CPR and rewarming may have to be continued for several hours to facilitate successful defibrillation.[597]

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. Hypothermic victims with an altered consciousness should be taken to a hospital capable of active external and internal rewarming. 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}.[598]

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 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 effects the elderly during heat waves[600]; 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 $SaO_2 < 92\%$ 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 to 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 the need to perform emergency resternotomy 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 resternotomy. Further defibrillation is attempted as indicated in the universal algorithm and should be performed with internal paddles at 20 J if resternotomy 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 resternotomy.

Emergency resternotomy

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 resternotomy without delay. Emergency resternotomy 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 20 J in cardiac arrest, but 5 J 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).

Commotio cordis

Commotio 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). Commotio 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 commotio 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 pregancy.[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., 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.

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 hours[681]; 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.[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 are[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 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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