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European Resuscitation Council Guidelines for Resuscitation 2015 Section 1. Executive summary

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Introduction

This executive summary provides the essential treatment algorithms for the resuscitation of children and adults and highlights the main guideline changes since 2010. Detailed guidance is provided in each of the ten sections, which are published as individual papers within this issue of Resuscitation. The sections of the ERC Guidelines 2015 are:

1. Executive summary
2. Adult basic life support and automated external defibrillation[1]
3. Adult advanced life support[2]
4. Cardiac arrest in special circumstances[3]
5. Post-resuscitation care[4]
6. Paediatric life support[5]
7. Resuscitation and support of transition of babies at birth[6]
8. Initial management of acute coronary syndromes[7]
9. First aid[8]
10. Principles of education in resuscitation[9]
11. The ethics of resuscitation and end-of-life decisions[10]

The ERC Guidelines 2015 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 the changes since the 2010 Guidelines

Adult basic life support and automated external defibrillation

- The ERC Guidelines 2015 highlight the critical importance of the interactions between the emergency medical dispatcher, the bystander who provides CPR and the timely deployment of an AED. An effective, co-ordinated community response that draws these elements together is key to improving survival from out-of-hospital cardiac arrest (Fig. 1.1).
- The emergency medical dispatcher plays an important role in the early diagnosis of cardiac arrest, the provision of dispatcher-assisted CPR (also known as telephone CPR), and the location and dispatch of an AED.
- The bystander who is trained and able should assess the collapsed victim rapidly to determine if the victim is unresponsive and not breathing normally and then immediately alert the emergency services.

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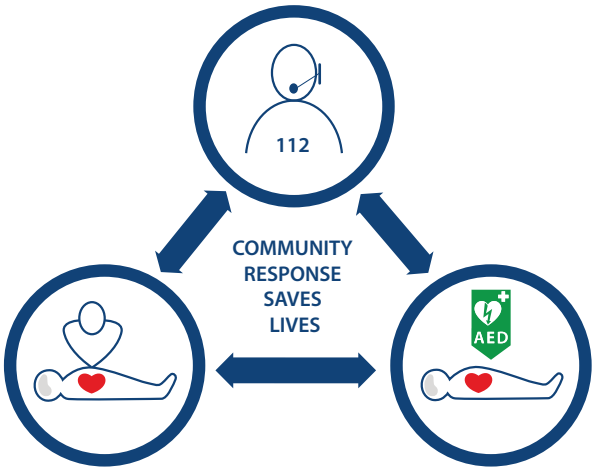


Fig. 1.1. The interactions between the emergency medical dispatcher, the bystander who provides CPR and the timely use of an automated external defibrillator are the key ingredients for improving survival from out of hospital cardiac arrest.

- The victim who is unresponsive and not breathing normally is in cardiac arrest and requires CPR. Bystanders and emergency medical dispatchers should be suspicious of cardiac arrest in any patient presenting with seizures and should carefully assess whether the victim is breathing normally.
- CPR providers should perform chest compressions for all victims in cardiac arrest. CPR providers trained and able to perform rescue breaths should combine chest compressions and rescue breaths. Our confidence in the equivalence between chest compression-only and standard CPR is not sufficient to change current practice.
- High-quality CPR remains essential to improving outcomes. The guidelines on compression depth and rate have not changed. CPR providers should ensure chest compressions of adequate depth (at least 5 cm but no more than 6 cm) with a rate of 100–120 compressions min^{-1}. After each compression allow the chest to recoil completely and minimise interruptions in compressions. When providing rescue breaths/ventilations spend approximately 1 s inflating the chest with sufficient volume to ensure the chest rises visibly. The ratio of chest compressions to ventilations remains 30:2. Do not interrupt chest compressions for more than 10 s to provide ventilations.
- Defibrillation within 3–5 min of collapse can produce survival rates as high as 50–70%. Early defibrillation can be achieved through CPR providers using public access and on-site AEDs. Public access AED programmes should be actively implemented in public places that have a high density of citizens.
- The adult CPR sequence can be used safely in children who are unresponsive and not breathing normally. Chest compression depths in children should be at least one third of the depth of the chest (for infants that is 4 cm, for children 5 cm).
- A foreign body causing severe airway obstruction is a medical emergency and requires prompt treatment with back blows and, if that fails to relieve the obstruction, abdominal thrusts. If the victim becomes unresponsive CPR should be started immediately whilst help is summoned.

Adult advanced life support

The ERC 2015 ALS Guidelines emphasise improved care and implementation of the guidelines in order to improve patient focused outcomes.[11] The key changes since 2010 are:

- Continued emphasis on the use of rapid response systems for care of the deteriorating patient and prevention of in-hospital cardiac arrest.
- Continued emphasis on minimally interrupted high-quality chest compressions throughout any ALS intervention: chest compressions are paused briefly only to enable specific interventions. This includes minimising interruptions in chest compressions for less than 5 s to attempt defibrillation.
- Keeping the focus on the use of self-adhesive pads for defibrillation and a defibrillation strategy to minimise the preshock pause, although we recognise that defibrillator paddles are used in some settings.
- There is a new section on monitoring during ALS with an increased emphasis on the use of waveform capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of return of spontaneous circulation (ROSC).
- There are a variety of approaches to airway management during CPR and a stepwise approach based on patient factors and the skills of the rescuer is recommended.
- The recommendations for drug therapy during CPR have not changed, but there is greater equipoise concerning the role of drugs in improving outcomes from cardiac arrest.
- The routine use of mechanical chest compression devices is not recommended, but they are a reasonable alternative in situations where sustained high-quality manual chest compressions are impractical or compromise provider safety.
- Peri-arrest ultrasound may have a role in identifying reversible causes of cardiac arrest.
- Extracorporeal life support techniques may have a role as a rescue therapy in selected patients where standard ALS measures are not successful.

Cardiac arrest in special circumstances

Special causes

This section has been structured to cover the potentially reversible causes of cardiac arrest that must be identified or excluded during any resuscitation. They are divided into two groups of four – 4Hs and 4Ts: hypoxia; hypo-/hyperkalaemia and other electrolyte disorders; hypo-/hyperthermia; hypovolaemia; tension pneumothorax; tamponade (cardiac); thrombosis (coronary and pulmonary); toxins (poisoning).

- Survival after an asphyxia-induced cardiac arrest is rare and survivors usually have severe neurological impairment. During CPR, early effective ventilation of the lungs with supplementary oxygen is essential.
- A high degree of clinical suspicion and aggressive treatment can prevent cardiac arrest from electrolyte abnormalities. The new algorithm provides clinical guidance to emergency treatment of life-threatening hyperkalaemia.
- Hypothermic patients without signs of cardiac instability can be rewarmed externally using minimally invasive techniques. Patients with signs of cardiac instability should be transferred directly to a centre capable of extracorporeal life support (ECLS).
- Early recognition and immediate treatment with intramuscular adrenaline remains the mainstay of emergency treatment for anaphylaxis.
- A new treatment algorithm for traumatic cardiac arrest was developed to prioritise the sequence of life-saving measures.
- Transport with continuing CPR may be beneficial in selected patients where there is immediate hospital access to the catheterisation laboratory and experience in percutaneous coronary intervention (PCI) with ongoing CPR.
- Recommendations for administration of fibrinolytics when pulmonary embolism is the suspected cause of cardiac arrest remain unchanged.

Special environments

The special environments section includes recommendations for the treatment of cardiac arrest occurring in specific locations. These locations are specialised healthcare facilities (e.g. operating theatre, cardiac surgery, catheterisation laboratory, dialysis unit, dental surgery), commercial airplanes or air ambulances, field of play, outside environment (e.g. drowning, difficult terrain, high altitude, avalanche burial, lightning strike and electrical injuries) or the scene of a mass casualty incident.

- A new section covers the common causes and relevant modification to resuscitative procedures in patients undergoing surgery.
- In patients following major cardiac surgery, key to successful resuscitation is recognising the need to perform immediate emergency resternotomy, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective.
- Cardiac arrest from shockable rhythms (ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT)) during cardiac catheterisation should immediately be treated with up to three stacked shocks before starting chest compressions. Use of mechanical chest compression devices during angiography is recommended to ensure high-quality chest compressions and to reduce the radiation burden to personnel during angiography with ongoing CPR.
- AEDs and appropriate CPR equipment should be mandatory on board of all commercial aircraft in Europe, including regional and low-cost carriers. Consider an over-the-head technique of CPR if restricted access precludes a conventional method.
- Sudden and unexpected collapse of an athlete on the field of play is likely to be cardiac in origin and requires rapid recognition and early defibrillation.
- Submersion exceeding 10 min is associated with poor outcome. Bystanders play a critical role in early rescue and resuscitation. Resuscitation strategies for those in respiratory or cardiac arrest continue to prioritise oxygenation and ventilation.
- The chances of good outcome from cardiac arrest in difficult terrain or mountains may be reduced because of delayed access and prolonged transport. There is a recognised role of air rescue and availability of AEDs in remote but often-visited locations.
- The cut-off criteria for prolonged CPR and extracorporeal rewarming of avalanche victims in cardiac arrest have become more stringent to reduce the number of futile cases treated with extracorporeal life support (ECLS).
- Safety measures are emphasised when providing CPR to the victim of an electrical injury.
- During mass casualty incidents (MCIs), if the number of casualties overwhelms healthcare resources, withhold CPR for those without signs of life.

Special patients

The section on special patients gives guidance for CPR in patients with severe comorbidities (asthma, heart failure with ventricular assist devices, neurological disease, obesity) and those with specific physiological conditions (pregnancy, elderly people).

- In patients with ventricular assist devices (VADs), confirmation of cardiac arrest may be difficult. If during the first 10 days after surgery, cardiac arrest does not respond to defibrillation, perform resternotomy immediately.
- Patients with subarachnoid haemorrhage may have ECG changes that suggest an acute coronary syndrome (ACS). Whether a computed tomography (CT) brain scan is done before or after coronary angiography will depend on clinical judgement.
- No changes to the sequence of actions are recommended in resuscitation of obese patients, but delivery of effective CPR may be challenging. Consider changing rescuers more frequently than the standard 2-min interval. Early tracheal intubation is recommended.
- For the pregnant woman in cardiac arrest, high-quality CPR with manual uterine displacement, early ALS and delivery of the foetus if early return of spontaneous circulation (ROSC) is not achieved remain key interventions.

Post-resuscitation care

This section is new to the European Resuscitation Council Guidelines; in 2010 the topic was incorporated into the section on ALS.[12] The ERC has collaborated with the European Society of Intensive Care Medicine to produce these post-resuscitation care guidelines, which recognise the importance of high-quality post-resuscitation care as a vital link in the Chain of Survival.[13]

The most important changes in post-resuscitation care since 2010 include:

- There is a greater emphasis on the need for urgent coronary catheterisation and percutaneous coronary intervention (PCI) following out-of-hospital cardiac arrest of likely cardiac cause.
- Targeted temperature management remains important but there is now an option to target a temperature of 36 °C instead of the previously recommended 32–34 °C. The prevention of fever remains very important.
- Prognostication is now undertaken using a multimodal strategy and there is emphasis on allowing sufficient time for neurological recovery and to enable sedatives to be cleared.
- A novel section has been added which addresses rehabilitation after survival from a cardiac arrest. Recommendations include the systematic organisation of follow-up care, which should include screening for potential cognitive and emotional impairments and provision of information.

Paediatric life support

Guideline changes have been made in response to convincing new scientific evidence and, by using clinical, organisational and educational findings, they have been adapted to promote their use and ease for teaching.

Basic life support

- The duration of delivering a breath is about 1 s, to coincide with adult practice.
- For chest compressions, the lower sternum should be depressed by at least one third the anterior-posterior diameter of the chest (4 cm for the infant and 5 cm for the child).

Managing the seriously ill child

- If there are no signs of septic shock, then children with a febrile illness should receive fluid with caution and reassessment following its administration. In some forms of septic shock, restricting fluids with isotonic crystalloid may be of benefit as compared to liberal use of fluids.
- For cardioversion of a supraventricular tachycardia (SVT), the initial dose has been revised to 1 J kg^{-1}.

Paediatric cardiac arrest algorithm

- Many of the features are common with adult practice.

Post-resuscitation care

- Prevent fever in children who have return of spontaneous circulation (ROSC) from an out-of-hospital setting.

- Targeted temperature management of children post-ROSC should be either normothermia or mild hypothermia.
- There is no single predictor for when to stop resuscitation.

Resuscitation and support of transition of babies at birth

The following are the main changes that have been made to the ERC guidelines for resuscitation at birth in 2015:

- **Support of transition**: Recognising the unique situation of the baby at birth, who rarely requires resuscitation but sometimes needs medical help during the process of postnatal transition. The term support of transition has been introduced to better distinguish between interventions that are needed to restore vital organ functions (resuscitation) or to support transition.
- **Cord clamping**: For uncompromised babies, a delay in cord clamping of at least 1 min from the complete delivery of the infant, is now recommended for term and preterm babies. As yet there is insufficient evidence to recommend an appropriate time for clamping the cord in babies who require resuscitation at birth.
- **Temperature**: The temperature of newly born non-asphyxiated infants should be maintained between 36.5 °C and 37.5 °C after birth. The importance of achieving this has been highlighted and reinforced because of the strong association with mortality and morbidity. The admission temperature should be recorded as a predictor of outcome as well as a quality indicator.
- **Maintenance of temperature**: At <32 weeks gestation, a combination of interventions may be required in addition to maintain the temperature between 36.5 °C and 37.5 °C after delivery through admission and stabilisation. These may include warmed humidified respiratory gases, increased room temperature plus plastic wrapping of body and head, plus thermal mattress or a thermal mattress alone, all of which have been effective in reducing hypothermia.
- **Optimal assessment of heart rate**: It is suggested in babies requiring resuscitation that the ECG can be used to provide a rapid and accurate estimation of heart rate.
- **Meconium**: Tracheal intubation should not be routine in the presence of meconium and should only be performed for suspected tracheal obstruction. The emphasis should be on initiating ventilation within the first minute of life in non-breathing or ineffectively breathing infants and this should not be delayed.
- **Air/oxygen**: Ventilatory support of term infants should start with air. For preterm infants, either air or a low concentration of oxygen (up to 30%) should be used initially. If, despite effective ventilation, oxygenation (ideally guided by oximetry) remains unacceptable, use of a higher concentration of oxygen should be considered.
- **CPAP**: Initial respiratory support of spontaneously breathing preterm infants with respiratory distress may be provided by CPAP rather than intubation.

Acute coronary syndromes

The following is a summary of the most important new views and changes in recommendations for the diagnosis and treatment of acute coronary syndromes (ACS).

Diagnostic Interventions in ACS

- Pre-hospital recording of a 12-lead electrocardiogram (ECG) is recommended in patients with suspected ST segment elevation acute myocardial infarction (STEMI). For those with STEMI this expedites pre-hospital and in-hospital reperfusion and reduces mortality.
- Non-physician ECG STEMI interpretation with or without the aid of computer ECG STEMI interpretation is suggested if adequate diagnostic performance can be maintained through carefully monitored quality assurance programmes.
- Pre-hospital STEMI activation of the catheterisation laboratory may not only reduce treatment delays but may also reduce patient mortality.
- The use of negative high-sensitivity cardiac troponins (hs-cTn) during initial patient evaluation cannot be used as a standalone measure to exclude an ACS, but in patients with very low risk scores may justify early discharge.

Therapeutic Interventions in ACS

- Adenosine diphosphate (ADP) receptor antagonists (clopidogrel, ticagrelor, or prasugrel-with specific restriction), may be given either pre-hospital or in the ED for STEMI patients planned for primary PCI.
- Unfractionated heparin (UFH) can be administered either in the pre-hospital or in-hospital setting in patients with STEMI and a planned primary PCI approach.
- Pre-hospital enoxaparin may be used as an alternative to pre-hospital UFH for STEMI.
- Patients with acute chest pain with presumed ACS do not need supplemental oxygen unless they present with signs of hypoxia, dyspnoea, or heart failure.

Reperfusion decisions in STEMI

Reperfusion decisions have been reviewed in a variety of possible local situations.

- When fibrinolysis is the planned treatment strategy, we recommend using pre-hospital fibrinolysis in comparison to in-hospital fibrinolysis for STEMI where transport times are >30 min and pre-hospital personnel are well trained.
- In geographic regions where PCI facilities exist and are available, direct triage and transport for PCI is preferred to pre-hospital fibrinolysis for STEMI.
- Patients presenting with STEMI in the emergency department (ED) of a non-PCI capable hospital should be transported immediately to a PCI centre provided that treatment delays for PPCI are less than 120 min (60–90 min for early presenters and those with extended infarctions), otherwise patients should receive fibrinolysis and be transported to a PCI centre.
- Patients who receive fibrinolytic therapy in the emergency department of a non-PCI centre should be transported if possible for early routine angiography (within 3–24 h from fibrinolytic therapy) rather than be transported only if indicated by the presence of ischaemia.
- PCI in less than 3 h following administration of fibrinolytics is not recommended and can be performed only in case of failed fibrinolysis.

Hospital reperfusion decisions after return of spontaneous circulation

- We recommend emergency cardiac catheterisation lab evaluation (and immediate PCI if required), in a manner similar to patients with STEMI without cardiac arrest, in selected adult patients with ROSC after out-of-hospital cardiac arrest (OHCA) of suspected cardiac origin with ST-elevation on ECG.
- In patients who are comatose and with ROSC after OHCA of suspected cardiac origin without ST-elevation on ECG It is reasonable

to consider an emergency cardiac catheterisation lab evaluation in patients with the highest risk of coronary cause cardiac arrest.

First aid

A section on first aid is included for the first time in the 2015 ERC Guidelines.

Principles of education in resuscitation

The following is a summary of the most important new views or changes in recommendations for education in resuscitation since the last ERC guidelines in 2010.

Training

- In centres that have the resources to purchase and maintain high fidelity manikins, we recommend their use. The use of lower fidelity manikins however is appropriate for all levels of training on ERC courses.
- Directive CPR feedback devices are useful for improving compression rate, depth, release, and hand position. Tonal devices improve compression rates only and may have a detrimental effect on compression depth while rescuers focus on the rate.
- The intervals for retraining will differ according to the characteristics of the participants (e.g. lay or healthcare). It is known that CPR skills deteriorate within months of training and therefore annual retraining strategies may not be frequent enough. Whilst optimal intervals are not known, frequent 'low dose' retraining may be beneficial.
- Training in non-technical skills (e.g. communication skills, team leadership and team member roles) is an essential adjunct to the training of technical skills. This type of training should be incorporated into life support courses.
- Ambulance service dispatchers have an influential role to play in guiding lay rescuers how to deliver CPR. This role needs specific training in order to deliver clear and effective instructions in a stressful situation.

Implementation

- Data-driven performance-focused debriefing has been shown to improve performance of resuscitation teams. We highly recommend its use for teams managing patients in cardiac arrest.
- Regional systems including cardiac arrest centres are to be encouraged, as there is an association with increased survival and improved neurological outcome in victims of out-of-hospital cardiac arrest.
- Novel systems are being developed to alert bystanders to the location of the nearest AED. Any technology that improves the delivery of swift bystander CPR with rapid access to an AED is to be encouraged.
- "It takes a system to save a life" [http://www.resuscitationacademy.com/]. Healthcare systems with a responsibility for the management of patients in cardiac arrest (e.g. EMS organisations, cardiac arrest centres) should evaluate their processes to ensure that they are able to deliver care that ensures the best achievable survival rates.

The ethics of resuscitation and end-of-life decisions

The 2015 ERC Guidelines include a detailed discussion of the ethical principles underpinning cardiopulmonary resuscitation.

The international consensus on cardiopulmonary resuscitation science

The International Liaison Committee on Resuscitation (ILCOR, www.ilcor.org) 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), the 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 most recent International Consensus Conference was held in Dallas in February 2015 and the published conclusions and recommendations from this process form the basis of these ERC Guidelines 2015.[14]

In addition to the six ILCOR task forces from 2010 (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)) a First Aid task force was created. The task forces identified topics requiring evidence evaluation and invited international experts to review them. As in 2010, a comprehensive conflict of interest (COI) policy was applied.[14]

For each topic, two expert reviewers were invited to undertake independent evaluations. Their work was supported by a new and unique online system called SEERS (Scientific Evidence Evaluation and Review System), developed by ILCOR. To assess the quality of the evidence and the strength of the recommendations, ILCOR adopted the GRADE (Grading of Recommendations Assessment, Development and Evaluation) methodology.[15] The ILCOR 2015 Consensus Conference was attended by 232 participants representing 39 countries; 64% of the attendees came from outside the United States. This participation ensured that this final publication represents a truly international consensus process. During the three years leading up to this conference, 250 evidence reviewers from 39 countries reviewed thousands of relevant, peer-reviewed publications to address 169 specific resuscitation questions, each in the standard PICO (Population, Intervention, Comparison, Outcome) format. Each science statement summarised the experts' interpretation of all relevant data on the specific topic and the relevant ILCOR task force added consensus draft treatment recommendations. Final wording of science statements and treatment recommendations was completed after further review by ILCOR member organisations and by the editorial board, and published in Resuscitation and Circulation as the 2015 Consensus on Science and Treatment Recommendations (CoSTR).[16,17] The member organisations forming ILCOR will publish resuscitation guidelines that are consistent with this CoSTR document, but will also consider geographic, economic and system differences in practice, and the availability of medical devices and drugs.

From science to guidelines

These ERC Guidelines 2015 are based on the 2015 CoSTR document and represent consensus among the members of the ERC General Assembly. New to the ERC Guidelines 2015 are the First Aid Guidelines, created in parallel with the First Aid Task Force of ILCOR, and guidelines on post-resuscitation care. For each section of the ERC Guidelines 2015, a writing group was assigned that drafted and agreed on the manuscript prior to approval by the General Assembly and the ERC Board. In areas where ILCOR had not conducted a systematic review, the ERC writing group undertook focused literature reviews. The ERC considers these new guidelines 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. Some of the recommendations made in the ERC Guidelines 2010 remain unchanged in 2015, either because no new studies have been published or because new evidence since 2010 has merely strengthened the evidence that was already available.

Adult basic life support and automated external defibrillation

The basic life support (BLS) and automated external defibrillation (AED) chapter contains guidance on the techniques used during the initial resuscitation of an adult cardiac arrest victim. This includes BLS (airway, breathing and circulation support without the use of equipment other than a protective device) and the use of an AED. In addition, simple techniques used in the management of choking (foreign body airway obstruction) are included. Guidelines for the use of manual defibrillators and starting in-hospital resuscitation are found in section 3.[2] A summary of the recovery position is included, with further information provided in the First Aid Chapter.

The guidelines are based on the ILCOR 2015 Consensus on Science and Treatment Recommendations (CoSTR) for BLS/AED.[18] The ILCOR review focused on 23 key topics leading to 32 Treatment Recommendations in the domains of early access and cardiac arrest prevention, early, high-quality CPR, and early defibrillation.

Cardiac arrest

Sudden cardiac arrest (SCA) is one of the leading causes of death in Europe. On initial heart-rhythm analysis, about 25–50% of SCA victims have ventricular fibrillation (VF)[19–21] but when the rhythm is recorded soon after collapse, in particular by an on-site AED, the proportion of victims in VF can be as high as 76%.[22,23] The recommended treatment for VF cardiac arrest is immediate bystander CPR and early electrical defibrillation. Most cardiac arrests of non-cardiac origin have respiratory causes, such as drowning (among them many children) and asphyxia. Rescue breaths as well as chest compressions are critical for successful resuscitation of these victims.

The chain of survival

The Chain of Survival summarises the vital links needed for successful resuscitation (Fig. 1.2). Most of these links apply to victims of both primary cardiac and asphyxial arrest.[13]

1: Early recognition and call for help

Recognising the cardiac origin of chest pain, and calling the emergency services before a victim collapses, enables the emergency medical service to arrive sooner, hopefully before cardiac arrest has occurred, thus leading to better survival.[24–26]

Once cardiac arrest has occurred, early recognition is critical to enable rapid activation of the EMS and prompt initiation of bystander CPR. The key observations are unresponsiveness and not breathing normally.

2: Early bystander CPR

The immediate initiation of CPR can double or quadruple survival after cardiac arrest.[27–29] If able, bystanders with CPR training should give chest compressions together with ventilations. When a bystander has not been trained in CPR, the emergency medical dispatcher should instruct him or her to give chest-compression-only CPR while awaiting the arrival of professional help.[30–32]

3: Early defibrillation

Defibrillation within 3–5 min of collapse can produce survival rates as high as 50–70%. This can be achieved by public access and onsite AEDs.[21,23,33]

4: Early advanced life support and standardised post-resuscitation care

Advanced life support with airway management, drugs and correcting causal factors may be needed if initial attempts at resuscitation are un-successful.

The critical need for bystanders to act

In most communities, the median time from emergency call to emergency medical service arrival (response interval) is 5–8 min,[22,34–36] or 8–11 min to a first shock.[21,28] During this time the victim's survival depends on bystanders who initiate CPR and use an automated external defibrillator (AED).[22,37]

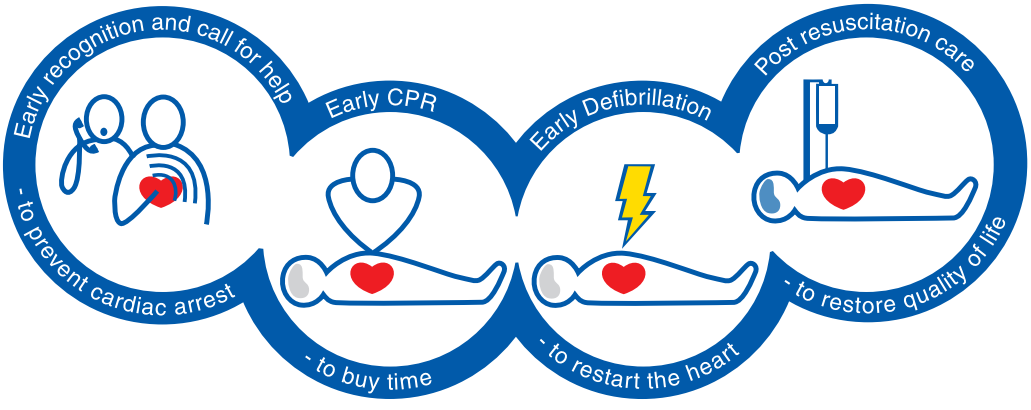


Fig. 1.2. The Chain of Survival.

Recognition of cardiac arrest

Recognising cardiac arrest can be challenging. Both bystanders and emergency call handlers (emergency medical dispatchers) have to diagnose cardiac arrest promptly in order to activate the chain of survival. Checking the carotid pulse (or any other pulse) has proved to be an inaccurate method for confirming the presence or absence of circulation.[38–42] Agonal breathing may be present in up to 40% of victims in the first minutes after cardiac arrest, and if responded to as a sign of cardiac arrest, is associated with higher survival rates.[43] The significance of agonal breathing should be emphasised during basic life support training.[44,45] Bystanders should suspect cardiac arrest and start CPR if the victim is unresponsive and not breathing normally. Bystanders should be suspicious of cardiac arrest in any patient presenting with seizures.[46,47]

Role of the emergency medical dispatcher

Dispatcher recognition of cardiac arrest

Patients who are unresponsive and not breathing normally should be presumed to be in cardiac arrest. Agonal breathing is often present, and callers may mistakenly believe the victim is still breathing normally.[48–57] Offering dispatchers additional education, specifically addressing the identification and significance of agonal breathing, can improve cardiac arrest recognition, increase the provision of telephone-CPR,[55,57] and reduce the number of missed cardiac arrest cases.[52]

If the initial emergency call is for a person suffering seizures, the call taker should be highly suspicious of cardiac arrest, even if the caller reports that the victim has a prior history of epilepsy.[49,58]

Dispatcher-assisted CPR

Bystander CPR rates are low in many communities. Dispatcher-assisted CPR (telephone-CPR) instructions improve bystander CPR rates,[56,59–62] reduce the time to first CPR,[57,59,62–64] increase the number of chest compressions delivered[60] and improve patient outcomes following out-of-hospital cardiac arrest (OHCA) in all patient groups.[30–32,56,61,63,65] Dispatchers should provide telephone-CPR instructions in all cases of suspected cardiac arrest unless a trained provider is already delivering CPR. Where instructions are required for an adult victim, dispatchers should provide chest-compression-only CPR instructions. If the victim is a child, dispatchers should instruct callers to provide both ventilations and chest compressions.

Adult BLS sequence

Fig. 1.3 presents the step-by-step sequence for the trained provider. It continues to highlight the importance of ensuring rescuer, victim and bystander safety. Calling for additional help (if required) is incorporated in the alerting emergency services step below. For clarity the algorithm is presented as a linear sequence of steps. It is recognised that the early steps of checking response, opening the airway, checking for breathing and calling the emergency medical dispatcher may be accomplished simultaneously or in rapid succession.

Those who are not trained to recognise cardiac arrest and start CPR would not be aware of these guidelines and therefore require dispatcher assistance whenever they make the decision to call 112 (Fig. 1.4).

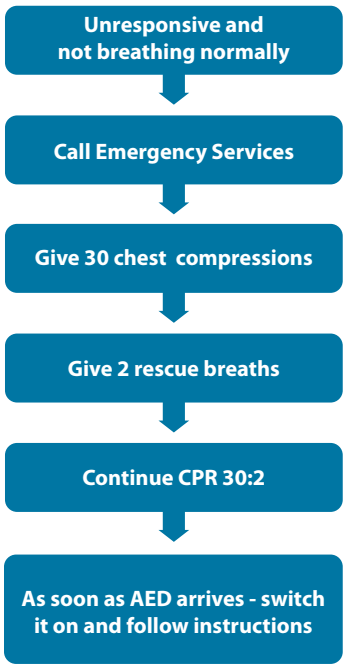


Fig. 1.3. The basic life support/automated external defibrillation (BLS/AED) algorithm.

Opening the airway and checking for breathing

The trained provider should assess the collapsed victim rapidly to determine if they are responsive and breathing normally. Open the airway using the head tilt and chin lift technique whilst assessing whether the person is breathing normally.

Alerting emergency services

112 is the European emergency phone number, available everywhere in the EU, free of charge. It is possible to call 112 from fixed and mobile phones to contact any emergency service: an ambulance, the fire brigade or the police. Early contact with the emergency services will facilitate dispatcher assistance in the recognition of cardiac arrest, telephone instruction on how to perform CPR, emergency medical service/first responder dispatch, and on locating and dispatching of an AED.[66–69]

Starting chest compressions

In adults needing CPR, there is a high probability of a primary cardiac cause. When blood flow stops after cardiac arrest, the blood in the lungs and arterial system remains oxygenated for some minutes. To emphasise the priority of chest compressions, it is recommended that CPR should start with chest compressions rather than initial ventilations.

When providing manual chest compressions:

1. Deliver compressions 'in the centre of the chest'
2. Compress to a depth of at least 5 cm but not more than 6 cm
3. Compress the chest at a rate of 100–120 min^{-1} with as few interruptions as possible
4. Allow the chest to recoil completely after each compression; do not lean on the chest

Hand position

Experimental studies show better haemodynamic responses when chest compressions are performed on the lower half of the

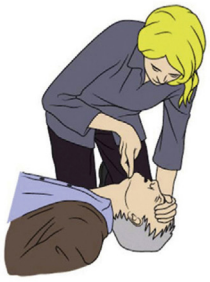
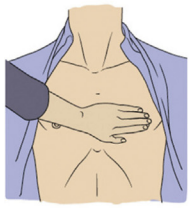
SEQUENCE / Action		Technical description
SAFETY **Make sure you, the victim and any bystanders are safe**		
RESPONSE **Check the victim for a response**		Gently shake his shoulders and ask loudly: “Are you all right?" If he responds leave him in the position in which you find him, provided there is no further danger; try to find out what is wrong with him and get help if needed; reassess him regularly
AIRWAY **Open the airway**		Turn the victim onto his back if necessary 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
BREATHING **Look, listen and feel for normal breathing**		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 seconds to determine whether the victim is breathing normally. If you have any doubt whether breathing is normal, act as if it is they are not breathing normally and prepare to start CPR
UNRESPONSIVE AND NOT BREATHING NORMALLY **Alert emergency services**		Ask a helper to call the emergency services (112) if possible otherwise call them yourself Stay with the victim when making the call if possible Activate speaker function on phone to aid communication with dispatcher
SEND FOR AED **Send someone to get AED**		Send someone to find and bring an AED if available. If you are on your own, do not leave the victim, start CPR

Fig. 1.4. Step by step sequence of actions for use by the BLS/AED trained provider to treat the adult cardiac arrest victim.

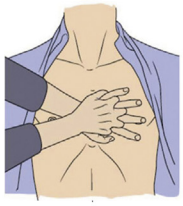
CIRCULATION

Start chest compressions



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 bony sternum (breastbone)

Position yourself vertically above the victim's chest and press down on the sternum at least 5 cm but not more than 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 100-120 min^{-1}

IF TRAINED AND ABLE

Combine chest compressions 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 second 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. Do not interrupt compressions by more than 10 seconds to deliver two breaths. Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions

Fig. 1.4. (*Continued*)

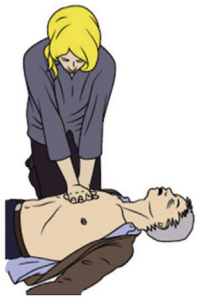
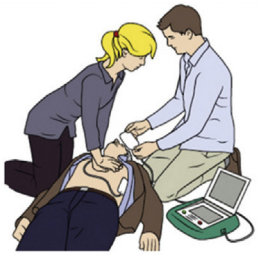
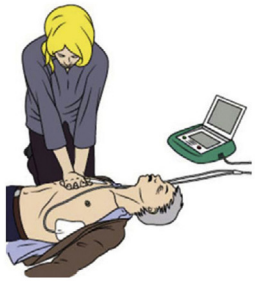
		Continue with chest compressions and rescue breaths in a ratio of 30:2
IF UNTRAINED OR UNABLE TO DO RESCUE BREATHS **Continue compression only CPR**		Give chest compressions only CPR (continuous compressions at a rate of 100-120 min^{-1})
WHEN AED ARRIVES **Switch on the AED and attach the electrode pads**		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		Ensure that nobody is touching the victim while the AED is analysing the rhythm
If a shock is indicated, **deliver shock**		Ensure that nobody is touching the victim Push shock button as directed (fully automatic AEDs will deliver the shock automatically) Immediately restart CPR 30:2 Continue as directed by the voice / visual prompts
If no shock is indicated, continue CPR		Immediately resume CPR. Continue as directed by the voice/visual prompts

Fig. 1.4. (*Continued*)

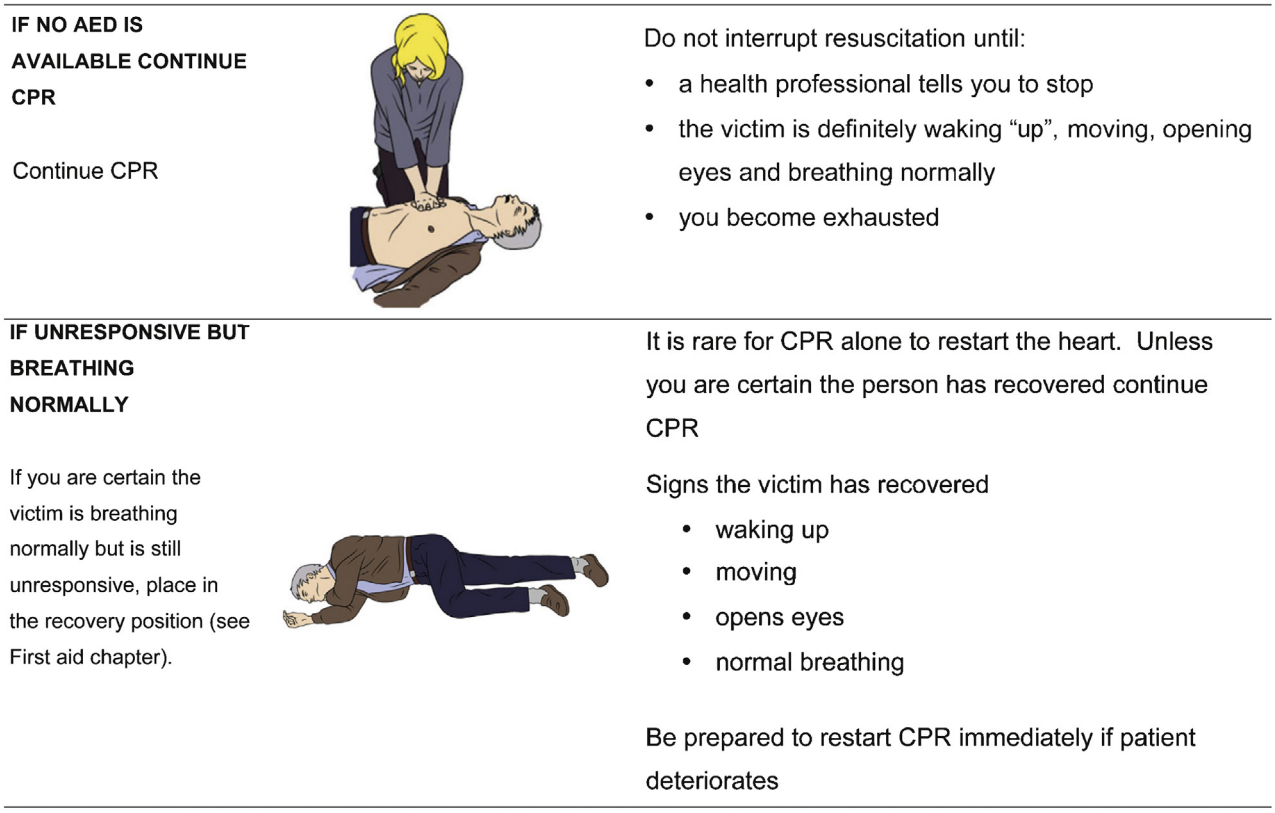


Fig. 1.4. (*Continued*)

sternum.[70–72] It is recommended that this location be taught in a simplified way, such as, "place the heel of your hand in the centre of the chest with the other hand on top". This instruction should be accompanied by a demonstration of placing the hands on the lower half of the sternum.[73,74]

Chest compressions are most easily delivered by a single CPR provider kneeling by the side of the victim, as this facilitates movement between compressions and ventilations with minimal interruptions. Over-the-head CPR for single CPR providers and straddle-CPR for two CPR providers may be considered when it is not possible to perform compressions from the side, for example when the victim is in a confined space.[75,76]

Compression depth

Data from four recent observational studies suggest that a compression depth range of 4.5–5.5 cm in adults leads to better outcomes than all other compression depths during manual CPR.[77–80] One of these studies found that a compression depth of 46 mm was associated with the highest survival rate.[79] The ERC, therefore, endorses the ILCOR recommendation that it is reasonable to aim for a chest compression depth of approximately 5 cm but not more than 6 cm in the average sized adult.[81] In line with the ILCOR recommendation, the ERC decided to retain the 2010 guidance to compress the chest at least 5 cm but not more than 6 cm.

Compression rate

Two studies found higher survival among patients who received chest compressions at a rate of 100–120 min^{-1}. Very high chest compression rates were associated with declining chest compression depths.[82,83] The ERC recommends, therefore, that chest compressions should be performed at a rate of 100–120 min^{-1}.

Minimising pauses in chest compressions

Pre- and post-shock pauses of less than 10 s, and chest compression fractions >60% are associated with improved outcomes.[84–88] Pauses in chest compressions should be minimised.

Firm surface

CPR should be performed on a firm surface whenever possible. Air-filled mattresses should be routinely deflated during CPR.[89] The evidence for the use of backboards is equivocal.[90–94] If a backboard is used, take care to avoid interrupting CPR and dislodging intravenous lines or other tubes during board placement.

Chest wall recoil

Allowing complete recoil of the chest after each compression results in better venous return to the chest and may improve the effectiveness of CPR.[95–98] CPR providers should, therefore, take care to avoid leaning after each chest compression.

Duty cycle

There is very little evidence to recommend any specific duty cycle and, therefore, insufficient new evidence to prompt a change from the currently recommended ratio of 50%.

Feedback on compression technique

None of the studies on feedback or prompt devices has demonstrated improved survival to discharge with feedback.[99] The use of CPR feedback or prompt devices during CPR should only be considered as part of a broader system of care that should include comprehensive CPR quality improvement initiatives,[99,100] rather than as an isolated intervention.

Rescue breaths

We suggest that during adult CPR tidal volumes of approximately 500–600 ml (6–7 ml kg^{-1}) are delivered. Practically, this is the volume required to cause the chest to rise visibly.[101] CPR providers should aim for an inflation duration of about 1 s, with enough volume to make the victim's chest rise, but avoid rapid or forceful breaths. The maximum interruption in chest compression to give two breaths should not exceed 10 s.[102]

Compression–ventilation ratio

A ratio of 30:2 was recommended in ERC Guidelines 2010 for the single CPR provider attempting resuscitation of an adult. Several observational studies have reported slightly improved outcomes after implementation of the guideline changes, which included switching from a compression ventilation ratio of 15:2 to 30:2.[103–106] The ERC continues, therefore, to recommend a compression to ventilation ratio of 30:2.

Compression-only CPR

Observational studies, classified mostly as very low-quality evidence, have suggested equivalence of chest-compression-only CPR and chest compressions combined with rescue breaths in adults with a suspected cardiac cause for their cardiac arrest.[27,107–118] Our confidence in the equivalence between chest-compression-only and standard CPR is not sufficient to change current practice. The ERC, therefore, endorses the ILCOR recommendations that all CPR providers should perform chest compressions for all patients in cardiac arrest. CPR providers trained and able to perform rescue breaths should perform chest compressions and rescue breaths as this may provide additional benefit for children and those who sustain an asphyxial cardiac arrest[111,119,120] or where the EMS response interval is prolonged.[115]

Use of an automated external defibrillator

AEDs are safe and effective when used by laypeople with minimal or no training.[121] AEDs make it possible to defibrillate many minutes before professional help arrives. CPR providers should continue CPR with minimal interruption of chest compressions while attaching an AED and during its use. CPR providers should concentrate on following the voice prompts immediately when they are spoken, in particular resuming CPR as soon as instructed, and minimising interruptions in chest compression. Standard AEDs are suitable for use in children older than 8 years.[122–124] For children between 1 and 8 years use paediatric pads, together with an attenuator or a paediatric mode if available.

CPR before defibrillation

Continue CPR while a defibrillator or AED is being brought onsite and applied, but defibrillation should not be delayed any longer.

Interval between rhythm checks

Pause chest compressions every 2 min to assess the cardiac rhythm.

Voice prompts

It is critically important that CPR providers pay attention to AED voice prompts and follow them without any delay. Voice prompts are usually programmable, and it is recommended that they be set in accordance with the sequence of shocks and timings for CPR given above. Devices measuring CPR quality may in addition provide real-time CPR feedback and supplemental voice/visual prompts.

In practice, AEDs are used mostly by trained rescuers, where the default setting of AED prompts should be for a compression to ventilation ratio of 30:2. If (in an exception) AEDs are placed in a setting where such trained rescuers are unlikely to be available or present, the owner or distributor may choose to change the settings to compression only.

Public access defibrillation (PAD) programmes

Placement of AEDs in areas where one cardiac arrest per 5 years can be expected is considered cost-effective and comparable to other medical interventions.[125–127] Registration of AEDs for public access, so that dispatchers can direct CPR providers to a nearby AED, may also help to optimise response.[128] The effectiveness of AED use for victims at home is limited.[129] The proportion of patients found in VF is lower at home than in public places, however the absolute number of potentially treatable patients is higher at home.[129] Public access defibrillation (PAD) rarely reaches victims at home.[130] Dispatched lay CPR providers, local to the victim and directed to a nearby AED, may improve bystander CPR rates[33] and help reduce the time to defibrillation.[37]

Universal AED signage

ILCOR has designed a simple and clear AED sign that may be recognised worldwide and this is recommended to indicate the location of an AED.[131]

In-hospital use of AEDs

There are no published randomised trials comparing in-hospital use of AEDs with manual defibrillators. Three observational studies showed no improvements in survival to hospital discharge for in-hospital adult cardiac arrest when using an AED compared with manual defibrillation.[132–134] Another large observational study showed that in-hospital AED use was associated with a lower survival-to-discharge rate compared with no AED use.[135] This suggests that AEDs may cause harmful delays in starting CPR, or interruptions in chest compressions in patients with non-shockable rhythms.[136] We recommend the use of AEDs in those areas of the hospital where there is a risk of delayed defibrillation,[137] because it will take several minutes for a resuscitation team to arrive, and first responders do not have skills in manual defibrillation. The goal is to attempt defibrillation within 3 min of collapse. In hospital areas where there is rapid access to manual defibrillation, either from trained staff or a resuscitation team, manual defibrillation should be used in preference to an AED. Hospitals should monitor collapse-to-first shock intervals and audit resuscitation outcomes.

Risks to the CPR provider and recipients of CPR

In victims who are eventually found not to be in cardiac arrest, bystander CPR extremely rarely leads to serious harm. CPR providers should not, therefore, be reluctant to initiate CPR because of concern of causing harm.

Foreign body airway obstruction (choking)

Foreign body airway obstruction (FBAO) is an uncommon but potentially treatable cause of accidental death.[138] As victims initially are conscious and responsive, there are often opportunities for early interventions which can be life saving.

Recognition

FBAO usually occurs while the victim is eating or drinking. Fig. 1.5 presents the treatment algorithm for the adult with FBAO. Foreign bodies may cause either mild or severe obstruction. It is important to ask the conscious victim "Are you choking?". The victim that is able to speak, cough and breathe has mild obstruction. The victim that is unable to speak, has a weakening cough, is struggling or unable to breathe, has severe airway obstruction.

Treatment for mild airway obstruction

Encourage the victim to cough as coughing generates high and sustained airway pressures and may expel the foreign body.

Action		Technical description
SUSPECT CHOKING **Be alert to choking particularly if victim is eating**		
ENCOURAGE TO COUGH **Instruct victim to cough**		
GIVE BACK BLOWS **If cough becomes ineffective give up to 5 back blows**		If the victim shows signs of severe airway obstruction and is conscious apply five back blows Stand to the side and slightly behind the victim Support the chest with one hand and lean the victim well forwards so that when the obstructing object is dislodged it comes out of the mouth rather than goes further down the airway; Give five sharp blows between the shoulder blades with the heel of your other hand.
GIVE ABDOMINAL THRUSTS **If back blows are ineffective give up to 5 abdominal thrutsts**		If five back blows fail to relieve the airway obstruction, give up to five abdominal thrusts as follows: Stand behind the victim and put both arms round the upper part of the abdomen; Lean the victim forwards; Clench your fist and place it between the umbilicus (navel) and the ribcage; Grasp this hand with your other hand and pull sharply inwards and upwards; Repeat up to five times. If the obstruction is still not relieved, continue alternating five back blows with five abdominal thrusts.

Fig. 1.5. Step by step sequence of actions for the treatment of the adult victim with foreign body airway obstruction.

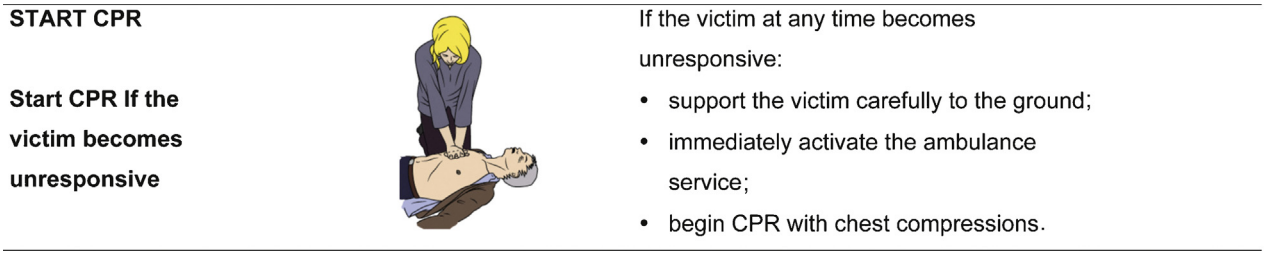


Fig. 1.5. (*Continued*)

Treatment for severe airway obstruction

For conscious adults and children over one year of age with complete FBAO, case reports have demonstrated the effectiveness of back blows or 'slaps', abdominal thrusts and chest thrusts.[139] The likelihood of success is increased when combinations of back blows or slaps, and abdominal and chest thrusts are used.[139]

Treatment of foreign body airway obstruction in an unresponsive victim

A randomised trial in cadavers[140] and two prospective studies in anaesthetised volunteers[141,142] showed that higher airway pressures can be generated using chest thrusts compared with abdominal thrusts. Chest compressions should, therefore, be started promptly if the victim becomes unresponsive or unconscious. After 30 compressions attempt 2 rescue breaths, and continue CPR until the victim recovers and starts to breathe normally.

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

Resuscitation of children (see also section 6) and victims of drowning (see also section 4)

Many children do not receive resuscitation because potential CPR providers fear causing harm if they are not specifically trained in resuscitation for children. This fear is unfounded: it is far better to use the adult BLS sequence for resuscitation of a child than to do nothing. For ease of teaching and retention, laypeople should be taught that the adult sequence may also be used for children who are not responsive and not breathing normally. The following minor modifications to the adult sequence will make it even more suitable for use in children:

- Give 5 initial rescue breaths before starting chest compressions
- Give CPR for 1 min before going for help in the unlikely event the CPR provider is alone
- Compress the chest by at least one third of its depth; use 2 fingers for an infant under one year; use 1 or 2 hands for a child over 1 year as needed to achieve an adequate depth of compression

The same modifications of 5 initial breaths and 1 min of CPR by the lone CPR provider before getting help, may improve outcome for victims of drowning. This modification should be taught only to those who have a specific duty of care to potential drowning victims (e.g. lifeguards).

Adult advanced life support

Guidelines for 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.[13] Once cardiac arrest occurs, only about 20% of patients who have an in-hospital cardiac arrest will survive to go home.[143,144] Hospitals should provide a system of care that includes: (a) educating staff about the signs of patient deterioration and the rationale for rapid response to illness, (b) appropriate, and frequent monitoring of patients' vital signs, (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 help.[145]

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

Most SCD victims have a history of cardiac disease and warning signs, most commonly chest pain, in the hour before cardiac arrest.[146] 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.[147–151] Screening programmes for athletes vary between countries.[152,153] Identification of individuals with inherited conditions and screening of family members can help prevent deaths in young people with inherited heart disorders.[154–156]

Prehospital resuscitation

CPR versus defibrillation first for out-of-hospital cardiac arrest

EMS personnel should provide high-quality CPR while a defibrillator is retrieved, applied and charged. Defibrillation should not be delayed longer than needed to establish the need for defibrillation and charging.

Termination of resuscitation rules

The 'basic life support termination of resuscitation rule' is predictive of death when applied by defibrillation-only emergency medical technicians.[157] The rule recommends termination when there is no ROSC, no shocks are administered and EMS personnel did not witness the arrest. Several studies have shown external generalisability of this rule.[158–164] More recent studies show that EMS systems providing ALS interventions can also use this BLS rule and therefore termed it the 'universal' termination of resuscitation rule.[159,165,166]

In-hospital resuscitation

After in-hospital cardiac arrest, the division between BLS and ALS is arbitrary; in practice, the resuscitation process is a

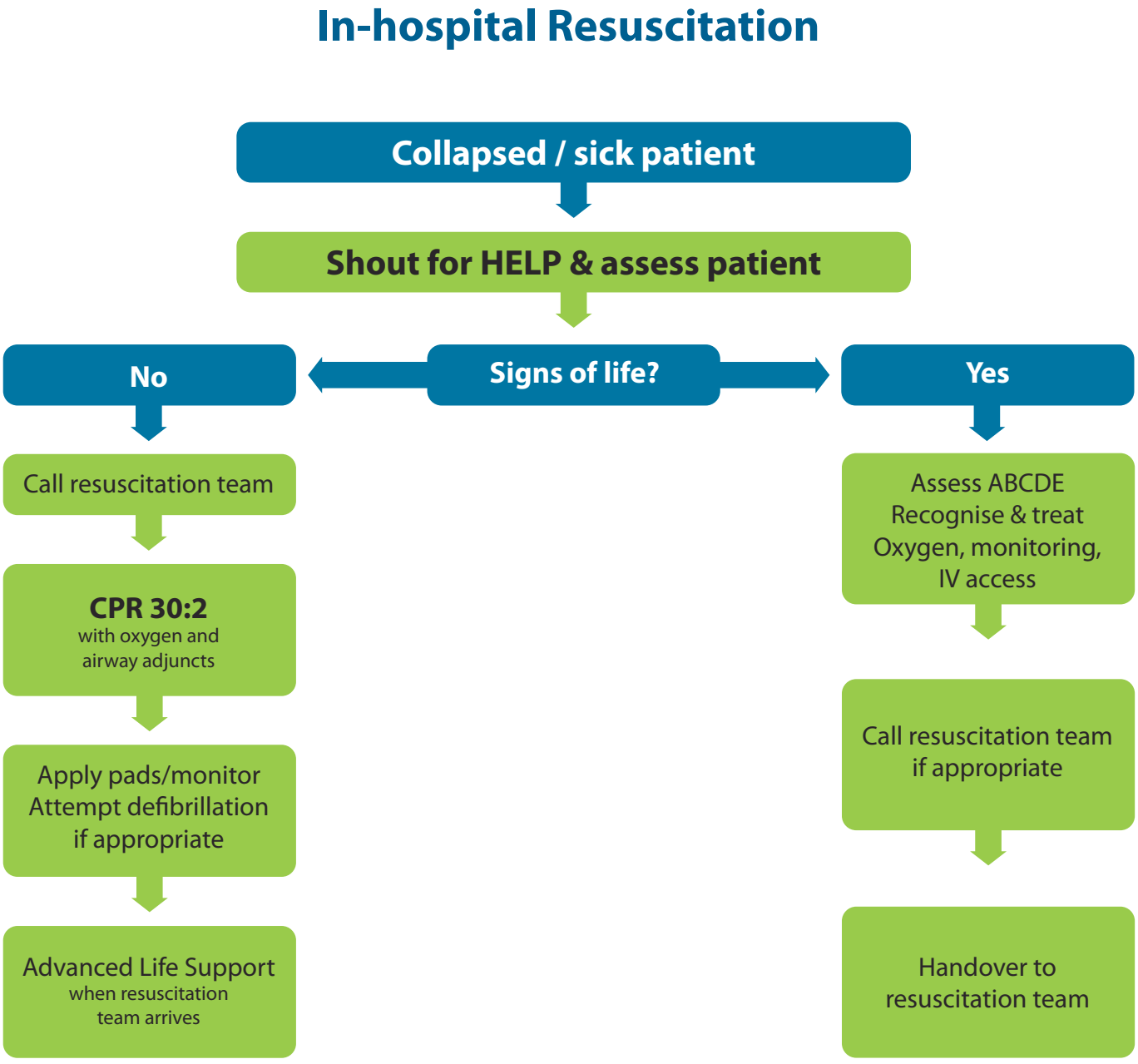


Fig. 1.6. In-hospital resuscitation algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure IV – intravenous; CPR – cardiopulmonary resuscitation.

continuum and is based on common sense. An algorithm for the initial management of in-hospital cardiac arrest is shown in Fig. 1.6.

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

The responsive patient

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

The unresponsive patient

The exact sequence will depend on the training of staff and experience in assessment of breathing and circulation. Trained healthcare staff cannot assess the breathing and pulse sufficiently reliably to confirm cardiac arrest.[39,40,42,44,167–172]

Agonal breathing (occasional gasps, slow, laboured or noisy breathing) is common in the early stages of cardiac arrest and is a sign of cardiac arrest and should not be confused as a sign of life.[43,53,54,56] Agonal breathing can also occur during chest compressions as cerebral perfusion improves, but is not indicative of ROSC. Cardiac arrest can cause an initial short seizure-like episode that can be confused with epilepsy[46,47] Finally changes in skin colour, notably pallor and bluish changes associated with cyanosis are not diagnostic of cardiac arrest.[46]

- Shout for help (if not already)
  Turn the victim on to his back and then open the airway:
- Open airway and check breathing:
  - Open the airway using a head tilt chin lift
  - Keeping the airway open, look, listen and feel for normal breathing (an occasional gasp, slow, laboured or noisy breathing is not normal):
    - Look for chest movement
    - Listen at the victim's mouth for breath sounds
    - Feel for air on your cheek
- Look, listen and feel for no more than 10 seconds to determine if the victim is breathing normally.
- Check for signs of a circulation:
  - It may be difficult to be certain that there is no pulse. If the patient has no signs of life (consciousness, purposeful movement, normal breathing, or coughing), or if there is doubt, start CPR immediately until more experienced help arrives or the patient shows signs of life.
  - Delivering chest compressions to a patient with a beating heart is unlikely to cause harm.[173] However, delays in diagnosing cardiac arrest and starting CPR will adversely effect survival and must be avoided.
  - Only those experienced in ALS should try to assess the carotid pulse whilst simultaneously looking for signs of life. This rapid assessment should take no more than 10 s. Start CPR if there is any doubt about the presence or absence of a pulse.
- If there are signs of life, urgent medical assessment is required. Depending on the local protocols, this may take the form of a resuscitation team. While awaiting this team, give the patient oxygen, attach monitoring and insert an intravenous cannula. When a reliable measurement of oxygen saturation of arterial blood (e.g. pulse oximetry (SpO2)) can be achieved, titrate the inspired oxygen concentration to achieve a SpO2 of 94–98%.
- If there is no breathing, but there is a pulse (respiratory arrest), ventilate the patient's lungs and check for a circulation every 10 breaths. Start CPR if there is any doubt about the presence or absence of a pulse.

Starting in-hospital CPR

The key steps are listed here. Supporting evidence can be found in the sections on specific interventions that follow.

- 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.
- Compress to a depth of at least 5 cm but no more than 6 cm.
- Chest compressions should be performed at a rate of 100–120 min^{-1}.
- Allow the chest to recoil completely after each compression; do not lean on the chest.
- Minimise interruptions and ensure high-quality compressions.
- Undertaking high-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. Pocket mask ventilation or two-rescuer bag-mask ventilation, which can be supplemented with an oral airway, should be started. Alternatively, use a supraglottic airway device (SGA) and self-inflating bag. Tracheal intubation should be attempted only by those who are trained, competent and experienced in this skill.
- Waveform capnography must be used for confirming tracheal tube placement and monitoring ventilation rate. Waveform capnography can also be used with a bag-mask device and SGA. The further use of waveform capnography to monitor CPR quality and potentially identify ROSC during CPR is discussed later in this section.[174]
- Use an inspiratory time of 1 s and give enough volume to produce a normal chest rise. Add supplemental oxygen to give the highest feasible inspired oxygen as soon as possible.[175]
- Once the patient's trachea has been intubated or a SGA has been inserted, continue uninterrupted chest compressions (except for defibrillation or pulse checks when indicated) at a rate of 100–120 min^{-1} and ventilate the lungs at approximately 10 breaths min^{-1}. Avoid hyperventilation (both excessive rate and tidal volume).
- 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 unable to do this, do chest compressions until help or airway equipment arrives.
- When the defibrillator arrives, apply self-adhesive defibrillation pads to the patient whilst chest compressions continue and then briefly analyse the rhythm. If self-adhesive defibrillation pads are not available, use paddles. Pause briefly to assess the heart rhythm. With a manual defibrillator, if the rhythm is VF/pVT charge the defibrillator while another rescuer continues chest compressions. Once the defibrillator is charged, pause the chest compressions and then give one shock, and immediately resume chest compressions. Ensure no one is touching the patient during shock delivery. Plan and ensure safe defibrillation before the planned pause in chest compressions.
- If using an automated external defibrillator (AED) follow the AED's audio-visual prompts, and similarly aim to minimise pauses in chest compressions by rapidly following prompts.
- In some settings where self-adhesive defibrillation pads are not available, alternative defibrillation strategies using paddles are used to minimise the preshock pause.
- In some countries a defibrillation strategy that involves charging the defibrillator towards the end of every 2 min cycle of CPR in preparation for the pulse check is used.[176,177] If the rhythm is VF/pVT a shock is given and CPR resumed. Whether this leads to any benefit is unknown, but it does lead to defibrillator charging for non-shockable rhythms.
- Restart chest compressions immediately after the defibrillation attempt. Minimise interruptions to chest compressions. When using a manual defibrillator it is possible to reduce the pause between stopping and restarting of chest compressions to less than five seconds.
- Continue resuscitation until the resuscitation team arrives or the patient shows signs of life. Follow the voice prompts if using an AED.
- 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).[178,179] Locate the patient's records.
- The quality of chest compressions during in-hospital CPR is frequently sub-optimal.[180,181] The importance of uninterrupted chest compressions cannot be over emphasised. Even short interruptions to chest compressions are disastrous for outcome and every effort must be made to ensure that continuous, effective chest compression is maintained throughout the resuscitation attempt. Chest compressions should commence at the beginning of a resuscitation attempt and continue uninterrupted unless they are paused briefly for a specific intervention (e.g. rhythm

check). Most interventions can be performed without interruptions to chest compressions. The team leader should monitor the quality of CPR and alternate CPR providers if the quality of CPR is poor.
- Continuous E_TCO_2 monitoring during CPR can be used to indicate the quality of CPR, and a rise in E_TCO_2 can be an indicator of ROSC during chest compressions.[174,182–184]
- If possible, the person providing chest compressions should be changed every 2 min, but without pauses in chest compressions.

ALS treatment algorithm

Although the ALS cardiac arrest algorithm (Fig. 1.7) is applicable to all cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances (see Section 4).[3]

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander basic life support (BLS), uninterrupted, high-quality chest compressions and early defibrillation for VF/pVT. The use of adrenaline has been shown to increase ROSC but not survival to discharge. Furthermore there is a possibility that it causes worse long-term neurological survival. Similarly, the evidence to support the use of advanced airway interventions during ALS remains limited.[175,185–192] 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 injected every 3–5 min until ROSC is achieved – the timing of the initial dose of adrenaline is described below. In VF/pVT, a single dose of amiodarone 300 mg is indicated after a total of three shocks and a further dose of 150 mg can be considered after five shocks. The optimal CPR cycle time is not known and algorithms for longer cycles (3 min) exist which include different timings for adrenaline doses.[193]

Shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia)

Having confirmed cardiac arrest, summon help (including the request for a defibrillator) and start CPR, beginning with chest compressions, with a compression: ventilation (CV) ratio of 30:2. When the defibrillator arrives, continue chest compressions while applying the defibrillation electrodes. Identify the rhythm and treat according to the ALS algorithm.

- If VF/pVT 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.
- Defibrillation shock energy levels are unchanged from the 2010 guidelines.[194] For biphasic waveforms, use an initial shock energy of at least 150 J. With manual defibrillators it is appropriate to consider escalating the shock energy if feasible, after a failed shock and for patients where refibrillation occurs.[195,196]
- Minimise the delay between stopping chest compressions and delivery of the shock (the preshock pause); even a 5–10 s delay will reduce the chances of the shock being successful.[84,85,197,198]
- Without pausing to reassess the rhythm or feel for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions to limit the post-shock pause and the total peri-shock pause.[84,85]
- Continue CPR for 2 min, then pause briefly to assess the rhythm; if still VF/pVT, give a second shock (150–360 J biphasic). Without pausing to reassess the rhythm or feel 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/pVT, give a third shock (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, during the next 2 min of CPR give adrenaline 1 mg and amiodarone 300 mg.[199]
- The use of waveform capnography may enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. Several human studies have shown that there is a significant increase in E_TCO_2 when ROSC occurs.[174,182–184,200,201] If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- If ROSC has not been achieved with this 3rd shock, the adrenaline may improve myocardial blood flow and increase the chance of successful defibrillation with the next shock.
- Timing of adrenaline dosing can cause confusion amongst ALS providers and this aspect needs to be emphasised during training.[202] Training should emphasise that giving drugs must not lead to interruptions in CPR and delay interventions such as defibrillation. Human data suggests drugs can be given without affecting the quality of CPR.[186]
- 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 feel a pulse. Ensure that rhythm checks are brief, and pulse checks are 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, immediately resume CPR. If ROSC has been achieved, begin post-resuscitation care.

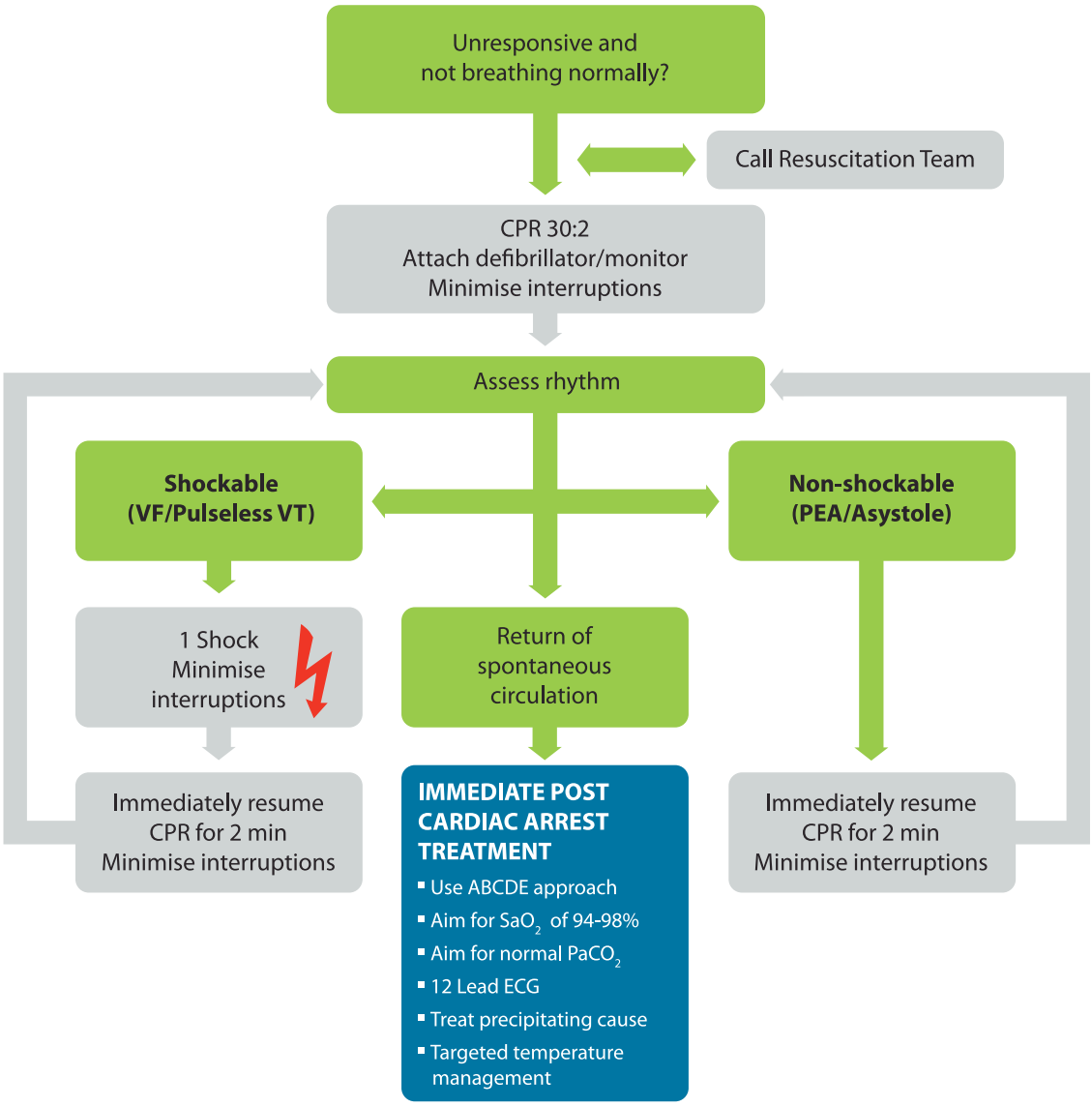
During treatment of VF/pVT, healthcare providers must practice efficient coordination between CPR and shock delivery whether using a manual defibrillator or an AED. Reduction in the peri-shock pause (the interval between stopping compressions to resuming compressions after shock delivery) by even a few seconds can increase the probability of shock success.[84,85,197,198] High-quality CPR may improve the amplitude and frequency of the VF and improve the chance of successful defibrillation to a perfusing rhythm.[203–205]

Regardless of the arrest rhythm, after the initial adrenaline dose has been given, give further doses of adrenaline 1 mg every 3–5 min until ROSC is achieved; in practice, this will be about once every two cycles of the algorithm. If signs of life return during CPR (purposeful movement, normal breathing or coughing), or there is an increase in E_TCO_2, check the monitor; if an organised rhythm is present, check for a pulse. If a pulse is palpable, start post-resuscitation care. If no pulse is present, continue CPR.

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

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and, if appropriate, ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

Advanced Life Support



DURING CPR

- Ensure high quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Continuous compressions when advanced airway in place
- Vascular access (intravenous or intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks

TREAT REVERSIBLE CAUSES

Hypoxia	Thrombosis – coronary or pulmonary
Hypovolaemia	Tension pneumothorax
Hypo-/hyperkalaemia/metabolic	Tamponade – cardiac
Hypothermia/hyperthermia	Toxins

CONSIDER

- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR

Fig. 1.7. Advanced Life Support algorithm. CPR – cardiopulmonary resuscitation; VF/Pulseless VT – ventricular fibrillation/pulseless ventricular tachycardia; PEA – pulseless electrical activity; ABCDE – Airway, Breathing Circulation, Disability, Exposure; SaO_2 – oxygen saturation; $PaCO_2$ – partial pressure carbon dioxide in arterial blood; ECG – electrocardiogram.

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

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. 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. Tracheal intubation must not delay defibrillation attempts. 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 be less than 5 s. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until ROSC. No RCTs 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 100–120 min^{-1} without pausing during ventilation.

In the absence of personnel skilled in tracheal intubation, a supraglottic airway (SGA) (e.g. laryngeal mask airway, laryngeal tube or i-gel) is an acceptable alternative. Once a SGA has been inserted, attempt to deliver continuous chest compressions, uninterrupted by ventilation.[206] If excessive gas leakage causes inadequate ventilation of the patient's lungs, chest compressions will have to be interrupted to enable ventilation (using a CV ratio of 30:2).

Intravenous access and drugs. 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 and elevation of the extremity for 10–20 s to facilitate drug delivery to the central circulation. If intravenous access is difficult or impossible, consider the IO route. This is now established as an effective route in adults.[207–210] Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a vein.[211,212]

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity (other than ventricular tachyarrhythmia) that would normally be associated with a palpable pulse.[213] 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. If asystole is displayed, without stopping CPR, check 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 as soon as venous or intraosseous access is achieved, and repeat every alternate CPR cycle (i.e. about every 3–5 min). If a pulse is present, begin post-resuscitation care. If signs of life return during CPR, check the rhythm and check for a pulse. If ROSC is suspected during CPR withhold adrenaline and continue CPR. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves, because this may respond to cardiac pacing. There is no benefit in attempting to pace true asystole. In addition, if there is doubt about whether the rhythm is asystole or extremely fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Continuing high-quality CPR however may improve the amplitude and frequency of the VF and improve the chance of successful defibrillation to a perfusing rhythm.[203–205]

The optimal CPR time between rhythm checks may vary according to the cardiac arrest rhythm and whether it is the first or subsequent loop.[214] Based on expert consensus, for the treatment of asystole or PEA, following a 2-min cycle of CPR, if the rhythm has changed to VF, follow the algorithm for shockable rhythms. Otherwise, continue CPR and give adrenaline every 3–5 min following the failure to detect a palpable pulse with the pulse check. If VF is identified on the monitor midway through a 2-min cycle of CPR, complete the cycle of CPR before formal rhythm and shock delivery if appropriate – this strategy will minimise interruptions in chest compressions.

Potentially reversible causes

Potential causes or aggravating factors for which specific treatment exists must be considered during any cardiac arrest. For ease of memory, these are divided into two groups of four, based upon their initial letter: either H or T. More details on many of these conditions are covered in Section 4 (Special Circumstances).[3]

Use of ultrasound imaging during advanced life support. Several studies have examined the use of ultrasound during cardiac arrest to detect potentially reversible causes.[215–217] 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. The integration of ultrasound into advanced life support requires considerable training if interruptions to chest compressions are to be minimised.

Monitoring during advanced life support

There are several methods and emerging technologies to monitor the patient during CPR and potentially help guide ALS interventions. These include:

- Clinical signs such as breathing efforts, movements and eye opening can occur during CPR. These can indicate ROSC and require verification by a rhythm and pulse check, but can also occur because CPR can generate a sufficient circulation to restore signs of life including consciousness.[218]
- The use of CPR feedback or prompt devices during CPR is addressed in Section 2 Basic Life Support.[1] The use of CPR feedback or prompt devices during CPR should only be considered as part of a broader system of care that should include comprehensive CPR quality improvement initiatives. [99,219]
- Pulse checks when there is an ECG rhythm compatible with an output can be used to identify ROSC, but may not detect pulses in those with low cardiac output states and a low blood pressure.[220] The value of attempting to feel arterial pulses during chest compressions to assess the effectiveness of chest compressions is unclear. There are no valves in the inferior vena cava and retrograde blood flow into the venous system can produce femoral vein pulsations.[221] Carotid pulsation during CPR

does not necessarily indicate adequate myocardial or cerebral perfusion.
- Monitoring the heart rhythm through pads, paddles or ECG electrodes is a standard part of ALS. Motion artefacts prevent reliable heart rhythm assessment during chest compressions forcing rescuers to stop chest compressions to assess the rhythm, and preventing early recognition of recurrent VF/pVT. Some modern defibrillators have filters that remove artefacts from compressions but there are no human studies showing improvements in patient outcomes from their use. We suggest against the routine use of artefact-filtering algorithms for analysis of ECG rhythm during CPR unless as part of a research programme.[18]
- The use of waveform capnography during CPR has a greater emphasis in Guidelines 2015 and is addressed in more detail below.
- Blood sampling and analysis during CPR can be used to identify potentially reversible causes of cardiac arrest. Avoid finger prick samples in critical illness because they may not be reliable; instead, use samples from veins or arteries.
- Blood gas values are difficult to interpret during CPR. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid–base state.[222] Analysis of central venous blood may provide a better estimation of tissue pH. Central venous oxygen saturation monitoring during ALS is feasible but its role in guiding CPR is not clear.
- Invasive arterial pressure monitoring will enable the detection of low blood pressure values when ROSC is achieved. Consider aiming for an aortic diastolic pressure of greater than 25 mmHg during CPR by optimising chest compressions.[223] In practice this would mean measuring an arterial diastolic pressure. Although haemodynamic-directed CPR showed some benefit in experimental studies [224–227] there is currently no evidence of improvement in survival with this approach in humans.[175]
- Ultrasound assessment is addressed above to identify and treat reversible causes of cardiac arrest, and identify low cardiac output states ('pseudo-PEA'). Its use has been discussed above.
- Cerebral oximetry using near-infrared spectroscopy measures regional cerebral oxygen saturation (rSO2) non-invasively.[228–230] This remains an emerging technology that is feasible during CPR. Its role in guiding CPR interventions including prognostication during and after CPR is yet to be established.[231]

Waveform capnography during advanced life support. Waveform capnography enables continuous real-time E_TCO_2 to be monitored during CPR. During CPR, E_TCO_2 values are low, reflecting the low cardiac output generated by chest compression. There is currently no evidence that use of waveform capnography during CPR improves patient outcomes, although the prevention of unrecognised oesophageal intubation is clearly beneficial. The role of waveform capnography during CPR includes:

- Ensuring tracheal tube placement in the trachea (see below for further details).
- Monitoring ventilation rate during CPR and avoiding hyperventilation.
- Monitoring the quality of chest compressions during CPR. E_TCO_2 values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value.[232] Whether this can be used to guide care and improve outcome requires further study.[174]
- Identifying ROSC during CPR. An increase in E_TCO_2 during CPR may indicate ROSC and prevent unnecessary and potentially harmful dosing of adrenaline in a patient with ROSC.[174,182,200,201] If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- Prognostication during CPR. Lower E_TCO_2 values may indicate a poor prognosis and less chance of ROSC;[175] however, we recommend that a specific E_TCO_2 value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO_2 values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

Extracorporeal Cardiopulmonary Resuscitation (eCPR)

Extracorporeal CPR (eCPR) should be considered as a rescue therapy for those patients in whom initial ALS measures are unsuccessful and, or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI) or pulmonary thrombectomy for massive pulmonary embolism).[233,234] There is an urgent need for randomised studies of eCPR and large eCPR registries to identify the circumstances in which it works best, establish guidelines for its use and identify the benefits, costs and risks of eCPR.[235,236]

Defibrillation

The defibrillation strategy for the ERC Guidelines 2015 has changed little from the former guidelines:

- The importance of early, uninterrupted chest compressions remains emphasised throughout these guidelines, together with minimising the duration of pre-shock and post-shock pauses.
- Continue chest compressions during defibrillator charging, deliver defibrillation with an interruption in chest compressions of no more than 5 s and immediately resume chest compressions following defibrillation.
- Self-adhesive defibrillation pads have a number of advantages over manual paddles and should always be used in preference when they are available.
- CPR should be continued while a defibrillator or automated external defibrillator (AED) is retrieved and applied, but defibrillation should not be delayed longer than needed to establish the need for defibrillation and charging.
- The use of up to three-stacked shocks may be considered if initial VF/pVT occurs during a witnessed, monitored arrest with a defibrillator immediately available, e.g. cardiac catheterisation.
- Defibrillation shock energy levels are unchanged from the 2010 guidelines.[194] For biphasic waveforms deliver the first shock with an energy of at least 150 J, the second and subsequent shocks at 150–360 J. The shock energy for a particular defibrillator should be based on the manufacturer's guidance. It is appropriate to consider escalating the shock energy if feasible, after a failed shock and for patients where refibrillation occurs.[195,196]

Strategies for 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.[84,85,87,197,198,237] The pre-shock pause can 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.[176,238] The safety check to avoid rescuer contact with the patient at the moment of defibrillation should be undertaken rapidly but efficiently. The post-shock pause is minimised by resuming chest compressions immediately after shock delivery (see below). The entire process of manual defibrillation

should be achievable with less than a 5 second interruption to chest compressions.

Airway management and ventilation

The optimal strategy for managing the airway has yet to be determined. Several observational studies have challenged the premise that advanced airway interventions (tracheal intubation or supraglottic airways) improve outcomes.[239] The ILCOR ALS Task Force has suggested using either an advanced airway (tracheal intubation or supraglottic airway (SGA)) or a bag-mask for airway management during CPR.[175] This very broad recommendation is made because of the total absence of high quality data to indicate which airway strategy is best. In practice a combination of airway techniques will be used stepwise during a resuscitation attempt.[240] The best airway, or combination of airway techniques will vary according to patient factors, the phase of the resuscitation attempt (during CPR, after ROSC), and the skills of rescuers.[192]

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. The ILCOR ALS Task Force recommends using waveform capnography to confirm and continuously monitor the position of a tracheal tube during CPR in addition to clinical assessment (strong recommendation, low quality evidence). Waveform capnography is given a strong recommendation as it may have other potential uses during CPR (e.g. monitoring ventilation rate, assessing quality of CPR). The ILCOR ALS Task Force recommends that if waveform capnography is not available, a non-waveform carbon dioxide detector, oesophageal detector device or ultrasound in addition to clinical assessment is an alternative.

Drugs and fluids for cardiac arrest

Vasopressors

Despite the continued widespread use of adrenaline and the use of vasopressin during resuscitation in some countries, there is no placebo-controlled study that shows that the routine use of any vasopressor during human cardiac arrest increases survival to hospital discharge, although improved short-term survival has been documented.[186,187,189]

Our current recommendation is to continue the use of adrenaline during CPR as for Guidelines 2010. We have considered the benefit in short-term outcomes (ROSC and admission to hospital) and our uncertainty about the benefit or harm on survival to discharge and neurological outcome given the limitations of the observational studies.[175,241,242] We have decided not to change current practice until there is high-quality data on long-term outcomes.

A series of randomised controlled trials[243–247] demonstrated no difference in outcomes (ROSC, survival to discharge, or neurological outcome) with vasopressin versus adrenaline as a first line vasopressor in cardiac arrest. Other studies comparing adrenaline alone or in combination with vasopressin also demonstrated no difference in ROSC, survival to discharge or neurological outcome.[248–250] We suggest vasopressin should not be used in cardiac arrest instead of adrenaline. Those healthcare professionals working in systems that already use vasopressin may continue to do so because there is no evidence of harm from using vasopressin when compared to adrenaline.[175]

Anti-arrhythmics

As with vasopressors, the evidence that anti-arrhythmic drugs are of benefit in cardiac arrest is limited. No anti-arrhythmic drug given during human cardiac arrest has been shown to increase survival to hospital discharge, although amiodarone has been shown to increase survival to hospital admission.[251,252] Despite the lack of human long-term outcome data, the balance of evidence is in favour of the use anti-arrhythmic drugs for the management of arrhythmias in cardiac arrest. Following three initial shocks, amiodarone in shock-refractory VF improves the short-term outcome of survival to hospital admission compared with placebo[251] or lidocaine.[252] Amiodarone also appears to improve the response to defibrillation when given to humans or animals with VF or haemodynamically unstable ventricular tachycardia.[253–257] There is no evidence to indicate the optimal time at which amiodarone should be given when using a single-shock strategy. In the clinical studies to date, the amiodarone was given if VF/pVT persisted after at least three shocks. For this reason, and in the absence of any other data, amiodarone 300 mg is recommended if VF/pVT persists after three shocks.

Lidocaine is recommended for use during ALS when amiodarone is unavailable.[252] Do not use magnesium routinely for the treatment of cardiac arrest.

Other drug therapy

Do not give sodium bicarbonate routinely during cardiac arrest and CPR or after ROSC. Consider sodium bicarbonate for life-threatening hyperkalaemia, for cardiac arrest associated with hyperkalaemia and for tricyclic overdose.

Fibrinolytic therapy should not be used routinely in cardiac arrest. Consider fibrinolytic therapy when cardiac arrest is caused by proven or suspected acute pulmonary embolism. 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.[258–260] 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 balanced crystalloid solutions such as Hartmann's solution or 0.9% sodium chloride. Avoid dextrose, which is redistributed away from the intravascular space rapidly and causes hyperglycaemia, and may worsen neurological outcome after cardiac arrest.[261]

CPR techniques and devices

Although manual chest compressions are often performed very poorly,[262–264] no adjunct has consistently been shown to be superior to conventional manual CPR.

Mechanical chest compression devices

Since Guidelines 2010 there have been three large RCTs enrolling 7582 patients that have shown no clear advantage from the routine use of automated mechanical chest compression devices for OHCA.[36,265,266] We suggest that automated mechanical chest compression devices are not used routinely to replace manual chest compressions. We suggest that automated mechanical chest compression devices are a reasonable alternative to high-quality manual chest compressions in situations where sustained high-quality manual chest compressions are impractical or compromise provider safety, such as CPR in a moving ambulance, prolonged CPR

(e.g. hypothermic arrest), and CPR during certain procedures (e.g. coronary angiography or preparation for extracorporeal CPR.[175] Interruptions to CPR during device deployment should be avoided. Healthcare personnel who use mechanical CPR should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills.

Impedance threshold device (ITD)

An RCT of the ITD with standard CPR compared to standard CPR alone with 8718 OHCA patients failed to show any benefit with ITD use in terms of survival and neurological outcome.[267] We therefore recommend that the ITD is not used routinely with standard CPR. Two RCTs did not show a benefit in terms of survival to hospital discharge of the ITD with active compression decompression CPR when compared with active compression decompression CPR alone.[268,269] Results of a large trial of a combination of ITD with active compression decompression CPR (ACD CPR) compared to standard CPR were reported in two publications.[270,271] There was no difference for survival to discharge and neurologically favourable survival at 12 months, and after consideration of the number needed to treat a decision was made not to recommend the routine use of the ITD and ACD.[175]

Peri-arrest arrhythmias

The correct identification and treatment of arrhythmias in the critically ill patient may prevent cardiac arrest from occurring or reoccurring after successful initial resuscitation. The initial assessment and treatment of a patient with an arrhythmia should follow the ABCDE approach. 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. Algorithms for the treatment of tachycardia and bradycardia are unchanged from 2010 and are show in Figs. 1.8 and 1.9.

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 mmHg).
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.

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

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

Cardiac arrest in special circumstances

Special causes

Hypoxia

Cardiac arrest caused by hypoxaemia is usually a consequence of asphyxia, which accounts for most of the non-cardiac causes of cardiac arrest. Survival after cardiac arrest from asphyxia is rare and most survivors sustain severe neurological injury. Those who are unconscious but have not progressed to a cardiac arrest are much more likely to make a good neurological recovery.[272,273]

Hypo-/hyperkalaemia and other electrolyte disorders

Electrolyte abnormalities can cause cardiac arrhythmias or cardiac arrest. Life-threatening arrhythmias are associated most commonly with potassium disorders, particularly hyperkalaemia.

Hypothermia (accidental)

Accidental hypothermia is defined as an involuntary drop of the body core temperature <35 °C. Cooling of the human body decreases cellular oxygen consumption by about 6% per 1 °C decrease in core temperature.[274] At 18 °C the brain can tolerate cardiac arrest for up to 10 times longer than at 37 °C. This results in hypothermia exerting a protective effect on the brain and heart,[275] and intact neurological recovery may be possible even after prolonged cardiac arrest if deep hypothermia develops before asphyxia. If an ECLS centre is not available, rewarming may be attempted in hospital using a combination of external and internal rewarming techniques (e.g. forced warm air, warm infusions, forced peritoneal lavage).[276]

Hyperthermia

Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds that normally maintained by homeostatic mechanisms.

Hyperthermia is a continuum of heat-related conditions, starting with heat stress, progressing to heat exhaustion, heat stroke and finally multiple organ dysfunction and cardiac arrest.[277] The mainstay of treatment is supportive therapy and rapidly cooling the patient.[278–280] Start cooling in the prehospital setting if possible. Aim to rapidly reduce the core temperature to approximately 39 °C. If cardiac arrest occurs, follow standard guidelines and continue cooling the patient. Use the same cooling techniques as for targeted temperature management after cardiac arrest

Hypovolaemia

Hypovolaemia is a potentially treatable cause of cardiac arrest that usually results from a reduced intravascular volume (i.e. haemorrhage), but relative hypovolaemia may also occur in patients with severe vasodilation (e.g. anaphylaxis, sepsis).

Depending on the suspected cause, initiate volume therapy with warmed blood products and/or crystalloids, in order to rapidly restore intravascular volume. At the same time, initiate immediate intervention to control haemorrhage, e.g. surgery, endoscopy, endovascular techniques,[281] or treat the primary cause (e.g. anaphylactic shock).

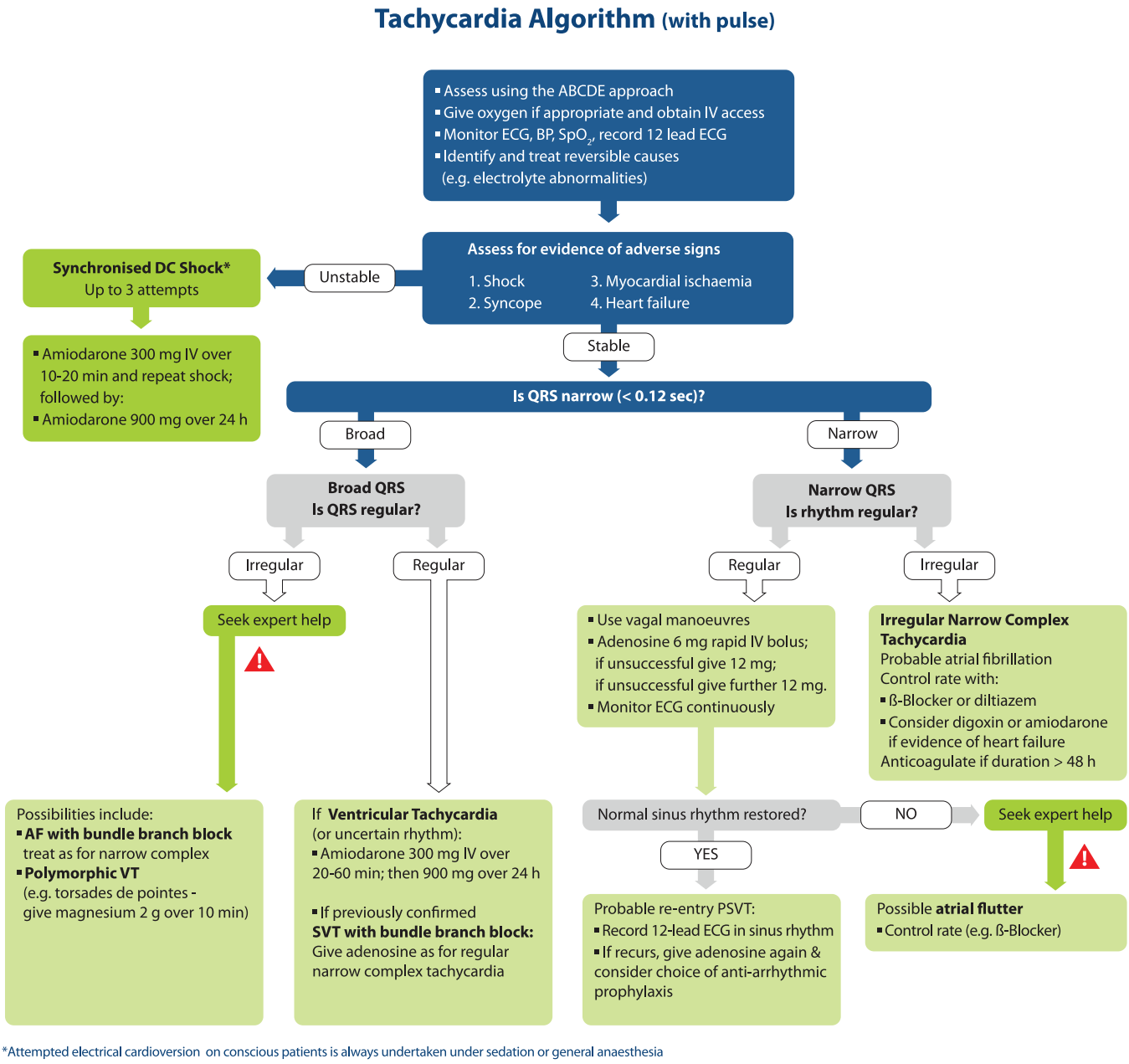


Fig. 1.8. Tachycardia algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure; IV – intravenous; SpO_2 – oxygen saturation measured by pulse oximetry; BP – blood pressure; ECG – electrocardiogram; DC – direct current; AF – atrial fibrillation; VT – ventricular tachycardia; SVT – supraventricular tachycardia; PSVT – paroxysmal supraventricular tachycardia.

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.[282–285] Adrenaline is the most important drug for the treatment of anaphylaxis.[286,287] The treatment algorithm for anaphylaxis, including the correct doses for adrenaline, is shown in Fig. 1.10. Adrenaline is most effective when given early after the onset of the reaction,[288] and adverse effects are extremely rare with correct IM doses. Repeat the IM adrenaline dose if there is no improvement in the patient's condition within 5 min. IV adrenaline should only be used by those experienced in the use and titration of vasopressors in their normal clinical practice.

Traumatic cardiac arrest. Traumatic cardiac arrest (TCA) carries a very high mortality, but in those where ROSC can be achieved, neurological outcome in survivors appears to be much better than in other causes of cardiac arrest.[289,290] It is vital that a medical cardiac arrest is not misdiagnosed as a TCA as it must be treated with the universal ALS algorithm. In cardiac arrest caused by hypovolaemia, cardiac tamponade or tension pneumothorax, chest compressions are unlikely to be as effective as in normovolaemic cardiac arrest.[291,292] For this reason, chest compressions take a lower priority than the immediate treatment of reversible causes, e.g. thoracotomy, controlling haemorrhage etc. (Fig. 1.11)

Tension pneumothorax

The incidence of tension pneumothorax is approximately 5% in major trauma patients treated in the prehospital setting (13% of

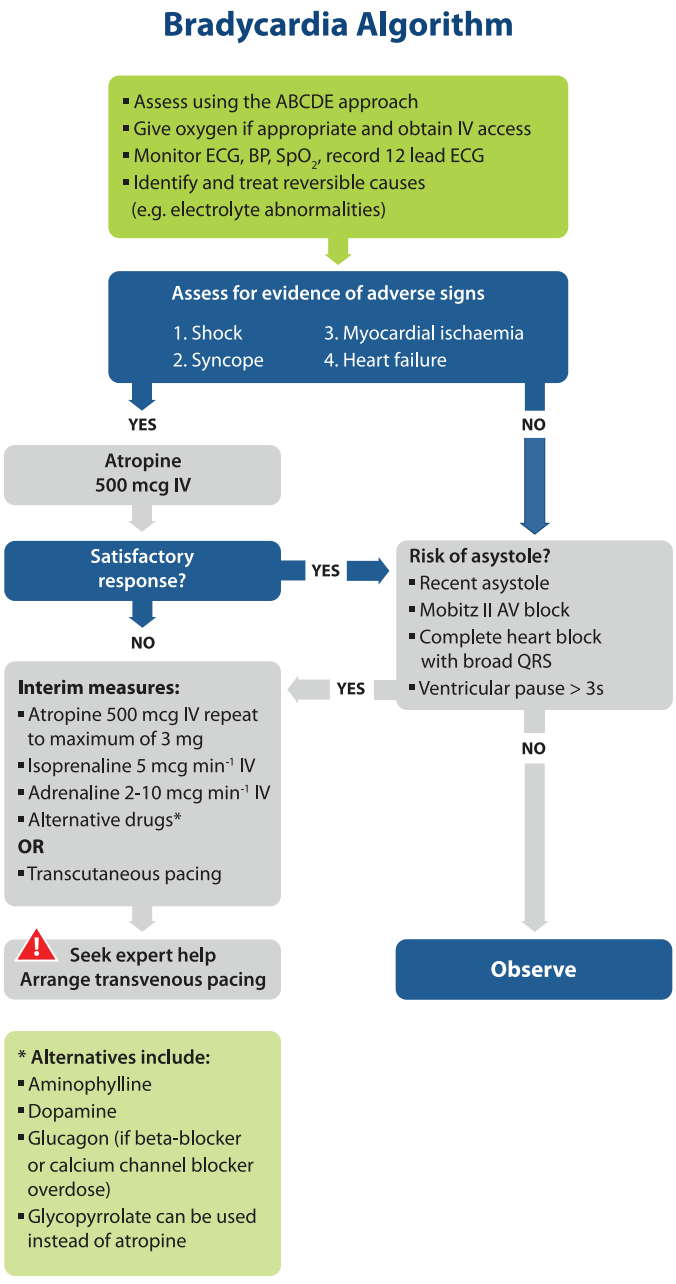


Fig. 1.9. Bradycardia algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure; IV – intravenous; SpO_2 – oxygen saturation measured by pulse oximetry; BP – blood pressure; ECG – electrocardiogram; AV – atrioventricular.

those developing TCA).[293–295] Needle chest decompression is rapid and within the skill set of most ambulance personnel but is of limited value.[296,297] Simple thoracostomy is easy to perform and used routinely by several prehospital physician services.[298,299] This consists of the first stage of standard chest tube insertion – a simple incision and rapid dissection into the pleural space in the positive pressure ventilated patient

Tamponade (cardiac)

The mortality after cardiac tamponade is high and immediate decompression of the pericardium is required to give any chance of survival. If thoracotomy is not possible, consider ultrasound-guided pericardiocentesis to treat cardiac arrest associated with suspected traumatic or non-traumatic cardiac tamponade. Non-image guided pericardiocentesis is an alternative, only if ultrasound is not available.

Thrombosis

Pulmonary embolism. Cardiac arrest from acute pulmonary embolism is the most serious clinical presentation of venous thromboembolism.[300] The reported incidence of cardiac arrest caused by pulmonary embolism is 2–9% of all OHCAs,[183,301–303] and 5–6% of all in-hospital cardiac arrests.[304,305] Diagnosis of acute pulmonary embolism during cardiac arrest is difficult. Clinical history and assessment, capnography and echocardiography (if available) can all assist in the diagnosis of acute pulmonary embolism during CPR with varying degrees of specificity and sensitivity. Consider administration of fibrinolytic therapy when acute pulmonary embolism is a known or suspected cause of cardiac arrest. Ongoing CPR is not a contraindication to fibrinolysis. The potential benefit of fibrinolysis in terms of improved survival outweighs potential risks in a location where no alternative exists, e.g. in the prehospital setting.[258] Once a fibrinolytic drug is administered, continue CPR for at least 60–90 min before terminating resuscitation attempts.[258,259]

Coronary thrombosis. Although proper diagnosis of the cause of cardiac arrest may be difficult in a patient already in cardiac arrest, if the initial rhythm is VF it is most likely that the cause is coronary artery disease with an occluded large coronary vessel. In these cases, transport with ongoing CPR and immediate access to the catheterisation laboratory may be considered if a prehospital and in-hospital infrastructure is available with teams experienced in mechanical haemodynamic support and primary percutaneous coronary intervention (PPCI) with ongoing CPR. A decision to transport with ongoing CPR should take into consideration a realistic chance of survival (e.g. witnessed cardiac arrest with initial shockable rhythm (VF/pVT) and bystander CPR). Intermittent ROSC also strongly favours a decision to transport.[306]

Toxins

Overall, poisoning rarely causes cardiac arrest or death.[307] There are few specific therapeutic measures for poisoning that improve outcomes: decontamination, enhancing elimination, and the use of specific antidotes.[308–310] The preferred method of gastrointestinal decontamination in patients with an intact or protected airway is activated charcoal. It is most effective if given within 1 h of ingestion.[311]

Special environments

Perioperative cardiac arrest

The commonest cause of anaesthesia-related cardiac arrest involves airway management.[312,313] Cardiac arrest caused by bleeding had the highest mortality in non-cardiac surgery, with only 10.3% of these patients surviving to hospital discharge.[314] Patients in the operating room are normally fully monitored and, as such, there should be little or no delay in diagnosing cardiac arrest.

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 0.7–8%.[315,316] Emergency resternotomy 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/pVT, undertake resternotomy without delay. Emergency resternotomy is also indicated in asystole or PEA, when other

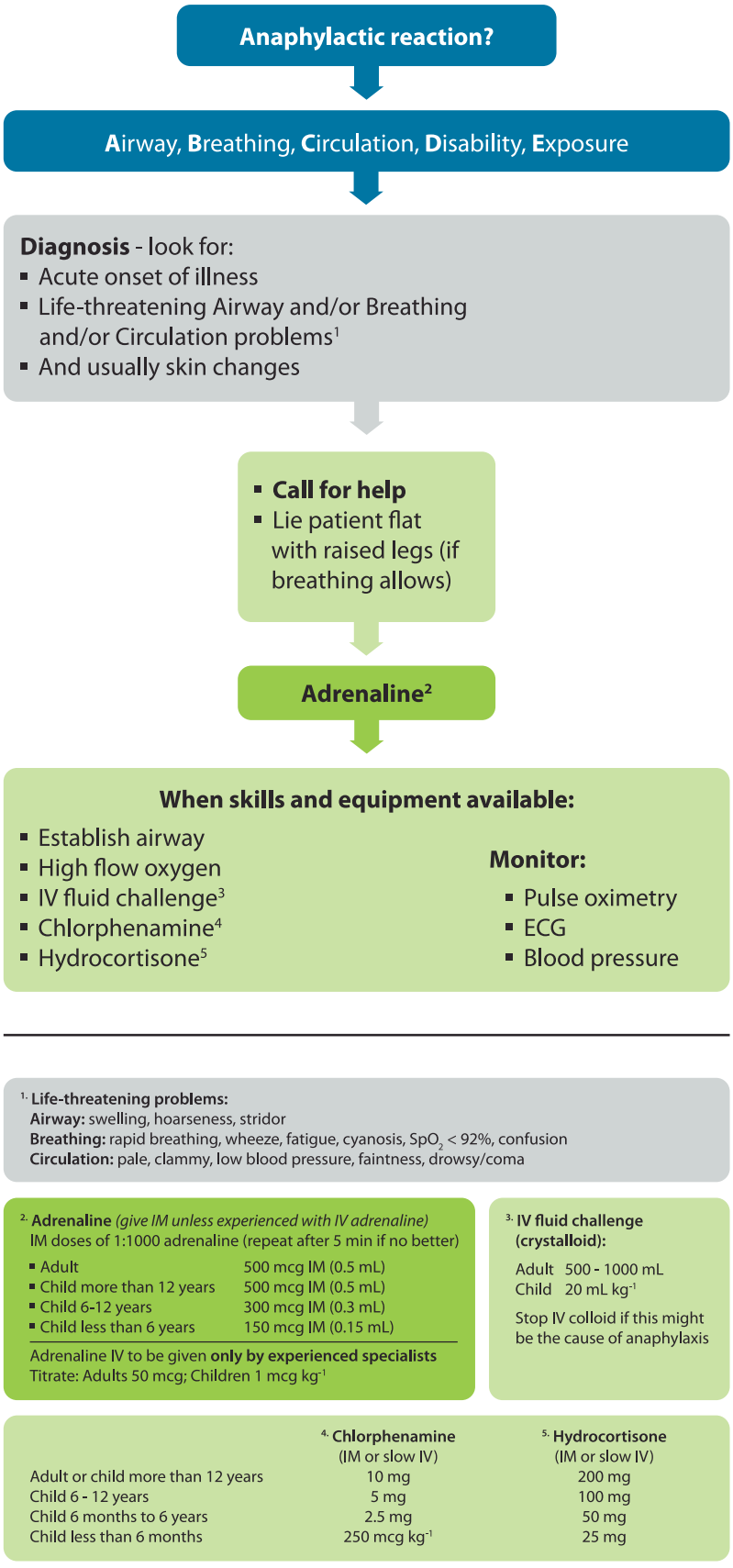


Fig. 1.10. Anaphylaxis treatment algorithm.[282]

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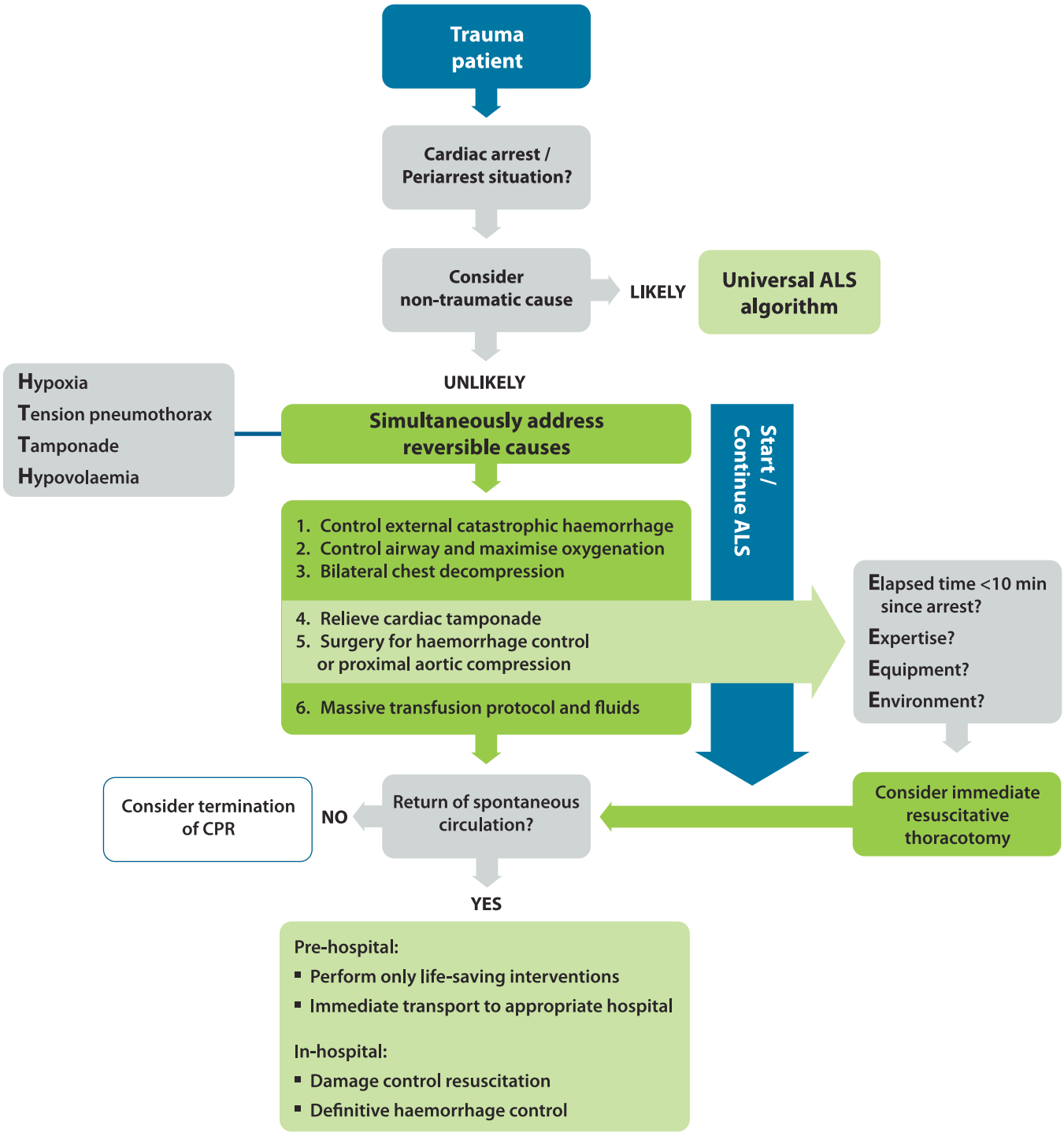

Fig. 1.11. Traumatic cardiac arrest algorithm.

treatments have failed, and should be performed within 5 min of the cardiac arrest by anyone with appropriate training.

Cardiac arrest in a cardiac catheterisation laboratory

Cardiac arrest (commonly VF) may occur during percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI) or non-STEMI, but it may also be a complication of angiography. In this special setting with immediate response to monitored VF, defibrillation without preceding chest compressions is recommended. If needed for failed defibrillation or immediately recurring VF, immediate defibrillation may be repeated up to two times. If VF persists after the initial three shocks or ROSC not immediately established with certainty, chest compressions and ventilations must be initiated without further delay and a cause for the unresolved problem sought with further coronary angiography. On an angiography table with the image intensifier above the patient, delivering chest compressions with adequate depth and rate is almost impossible and exposes the rescuers to dangerous radiation. Therefore, early transition to the use of a mechanical chest compression device is strongly recommended.[317,318] If the problem is not rapidly resolved, very low quality evidence suggests that the use of extracorporeal life support (ECLS) can be considered as

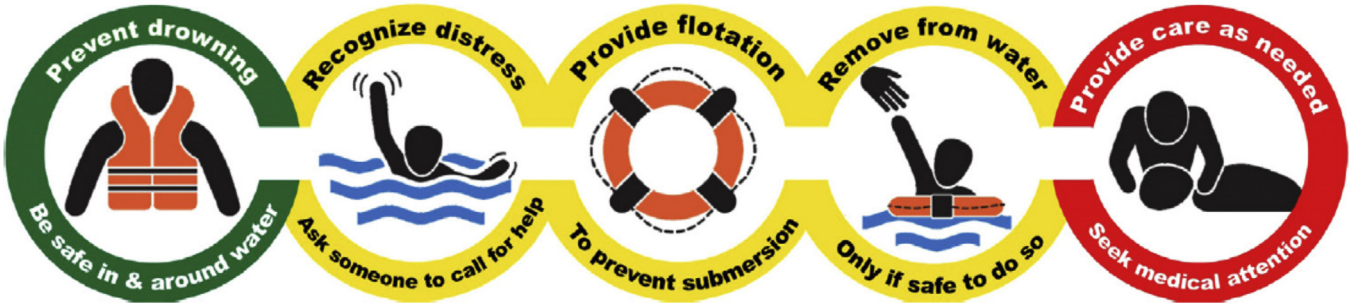

Fig. 1.12. Drowning chain of survival.[337]

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a rescue strategy if the infrastructure is available, and probably to be preferred over intra-aortic balloon pump (IABP).[319]

Cardiac arrest in a dialysis unit

Sudden cardiac death is the most common cause of death in haemodialysis patients and is usually preceded by ventricular arrhythmias.[320] Hyperkalaemia contributes to 2–5% of deaths amongst haemodialysis patients [321] A shockable rhythm (VF/pVT) is more common in patients undergoing haemodialysis. [320,322,323] Most haemodialysis machine manufacturers recommend disconnection from the dialysis equipment prior to defibrillation.[324]

Cardiac arrest in transportation vehicles

In-flight emergencies aboard airplanes. Cardiac arrest on board has an incidence of 1 per 5–10 million passenger flights. An initial shockable rhythm is present in 25–31% patients,[325–328] and the in-flight use of an AED can result in 33–50% survival to hospital discharge.[325,328,329]

Cardiac arrest in HEMS and air ambulances. Air ambulance services operate either a helicopter emergency medical service (HEMS) or fixed-wing air ambulances that routinely transport critically ill patients. Cardiac arrest may occur in flight, both in patients being transported from an accident site and also critically ill patients being transported between hospital.[330,331]

If a shockable rhythm (VF/pVT) is recognised in a monitored patient and defibrillation can be accomplished rapidly, immediately give up to three-stacked shocks before starting chest compressions. Mechanical chest compression devices enable delivery of high quality chest compressions in the confined space of an air ambulance and their use should be considered.[332,333] If a cardiac arrest during flight is thought to be a possibility, consider fitting the patient within a mechanical chest compression device during packaging before flight.[334,335]

Cardiac arrest during sports activities

The sudden and unexpected collapse, not associated with contact or trauma, of an athlete on the field of play is probably cardiac in origin and requires rapid recognition and effective treatment if the victim is to survive. If there is no immediate response to treatment and there is an organised medical team, consider moving the patient to an area shielded from media and spectators. If the patient is in VF/pVT, delay moving them until after the first three defibrillation attempts (defibrillation is most likely to be successful in the first three shocks).

Water rescue and drowning

Drowning is a common cause of accidental death.[336] The Drowning Chain of Survival [337] describes five critical links for improving survival from drowning (Fig. 1.12).

Bystanders play a critical role in initial attempts at rescue and resuscitation.[338–340] ILCOR reviewed specific prognostic indicators and noted that submersion durations of less than 10 min were associated with a very high chance of favourable outcome.[18] Age, emergency medical services (EMS) response time, fresh or salt water, water temperature, and witness status were not useful for predicting survival. Submersion in ice-cold water may prolong the window of survival and justify extended search and rescue activities.[341–343] The BLS sequence in drowning (Fig. 1.13) reflects the critical importance of rapid alleviation of hypoxia.

Wilderness and environmental emergencies

Difficult terrain and remote areas. Compared to urban areas some terrains will be more difficult to access and are remote from organised medical care. The chances of a good outcome from cardiac arrest may be reduced due to delayed access and prolonged transport.

Whenever possible, transport the patient with air rescue.[344,345] The organisation of the helicopter emergency medical service (HEMS) affects the outcome.[346–348]

High altitude illness. Given the increasing popularity of travel at altitude, an increasing number of tourists at altitude have

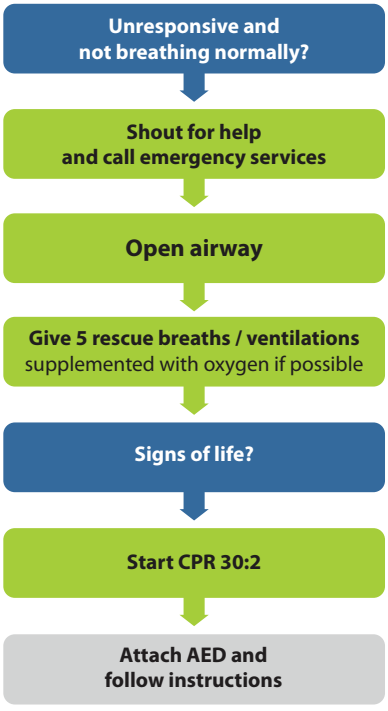


Fig. 1.13. Drowning treatment algorithm for rescuers with a duty to respond.

cardiovascular and metabolic risk factors for cardiac arrest. Resuscitation at high altitude does not differ from standard CPR. With the lower pO_2, CPR is more exhausting for the rescuer than at sea level, and the average number of effective chest compressions may decrease within the first minute.[349–351] Use mechanical chest compression devices whenever possible. In situations where transport is not possible, and correction of reversible causes is not possible, further resuscitation is futile and CPR should be terminated.

Avalanche burial. In Europe and North America together, there are about 150 snow avalanche deaths each year. Fatalities are mainly due to asphyxia, sometimes associated with trauma and hypothermia. Prognostic factors are severity of injury, duration of complete burial, airway patency, core temperature and serum potassium.[352] The cut-off criteria for prolonged CPR and extracorporeal rewarming of avalanche victims in cardiac arrest have become more stringent to reduce the number of futile cases treated with extracorporeal life support (ECLS). An algorithm for the management of the buried avalanche victim is shown in Fig. 1.14.

Lightning strike and electrical injuries. 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. Ensure that any power source is switched off and do not approach the casualty until it is safe. Electrocution from lightning strikes is rare, but worldwide it causes 1000 deaths each year.[353] Unconscious patients with linear or punctuate burns (feathering) should be treated as victims of lightning strike.[354] Severe burns (thermal or electrical), myocardial necrosis, the extent of central nervous system injury, and secondary multisystem organ failure determine the morbidity and long-term prognosis.

Mass casualty incidents

Use a triage system to prioritise treatment. The decision to use a mass casualty incident (MCI) triage sieve, and withhold CPR to those with imminent death, (including victims without signs of life), is the responsibility of a medical commander who is usually the most experienced EMS clinician on scene. Training allows fast and correct recognition of those needing life-saving procedures, and reduces the risk of inappropriate care given to futile cases.

Special patients

Cardiac arrest associated with concomitant diseases

Asthma. The majority of asthma-related deaths occur before admission to hospital.[355]

Cardiac arrest in a person with asthma is often a terminal event after a period of hypoxaemia. Modifications to standard ALS guidelines include considering the need for early tracheal intubation. If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest while disconnecting tracheal tube may relieve air trapping.

Patients with ventricular assist devices. Confirming cardiac arrest in these patients may be difficult. A patient with invasive monitoring should be considered to have arrested if the arterial line reads the same as the central venous pressure (CVP) line. In patients without invasive monitoring, if the patient has no signs of life and is not breathing, then they should be considered to have suffered a cardiac arrest. Patients with an implantable left ventricular assist device (LVAD) should have the same algorithm followed as the algorithm for arrest after cardiac surgery. In pulseless electrical activity (PEA), turn the pacing off and verify there is no underlying VF, which must be treated by defibrillation. External chest compressions should be performed if immediate resuscitative efforts fail. Importantly, the airway and breathing checks should always be performed. It is possible for a patient to have asystole or VF, but still have adequate cerebral blood flow due to adequate and continued pump flow. If the patient is conscious and responding then you will have more time in which to resolve this arrhythmia and external chest compressions will not be needed. Resternotomy should be performed in an established cardiac arrest within 10 days of surgery.

Cardiac arrest associated with neurological disease. Cardiac arrest associated with acute neurological disease is relatively uncommon and can occur with subarachnoid haemorrhage, intracerebral haemorrhage, epileptic seizures, and ischaemic stroke.[356] Cardiac or respiratory arrest occurs in between 3 and 11% of patents with subarachnoid haemorrhage,[357] and the initial rhythm is usually non-shockable. However, patients with subarachnoid haemorrhage may have ECG changes that suggest an acute coronary syndrome.[358] Individuals with neurological prodromal symptoms who achieve ROSC may be considered for CT brain scan. Whether this is done before or after coronary angiography will depend on clinical judgement with consideration of the likelihood of a subarachnoid haemorrhage versus an acute coronary syndrome.[4]

Obesity. In 2014, more than 1.9 billion (39%) adults were overweight, and of these over 600 million (13%) were obese. Traditional cardiovascular risk factors (hypertension, diabetes, lipid profile, prevalent coronary heart disease, heart failure, and left ventricular hypertrophy) are common in obese patients. Obesity is associated with increased risk of sudden cardiac death.[359] No changes to the sequence of actions are recommended in resuscitation of obese patients, but delivery of effective CPR may be challenging.

Cardiac arrest associated with pregnancy

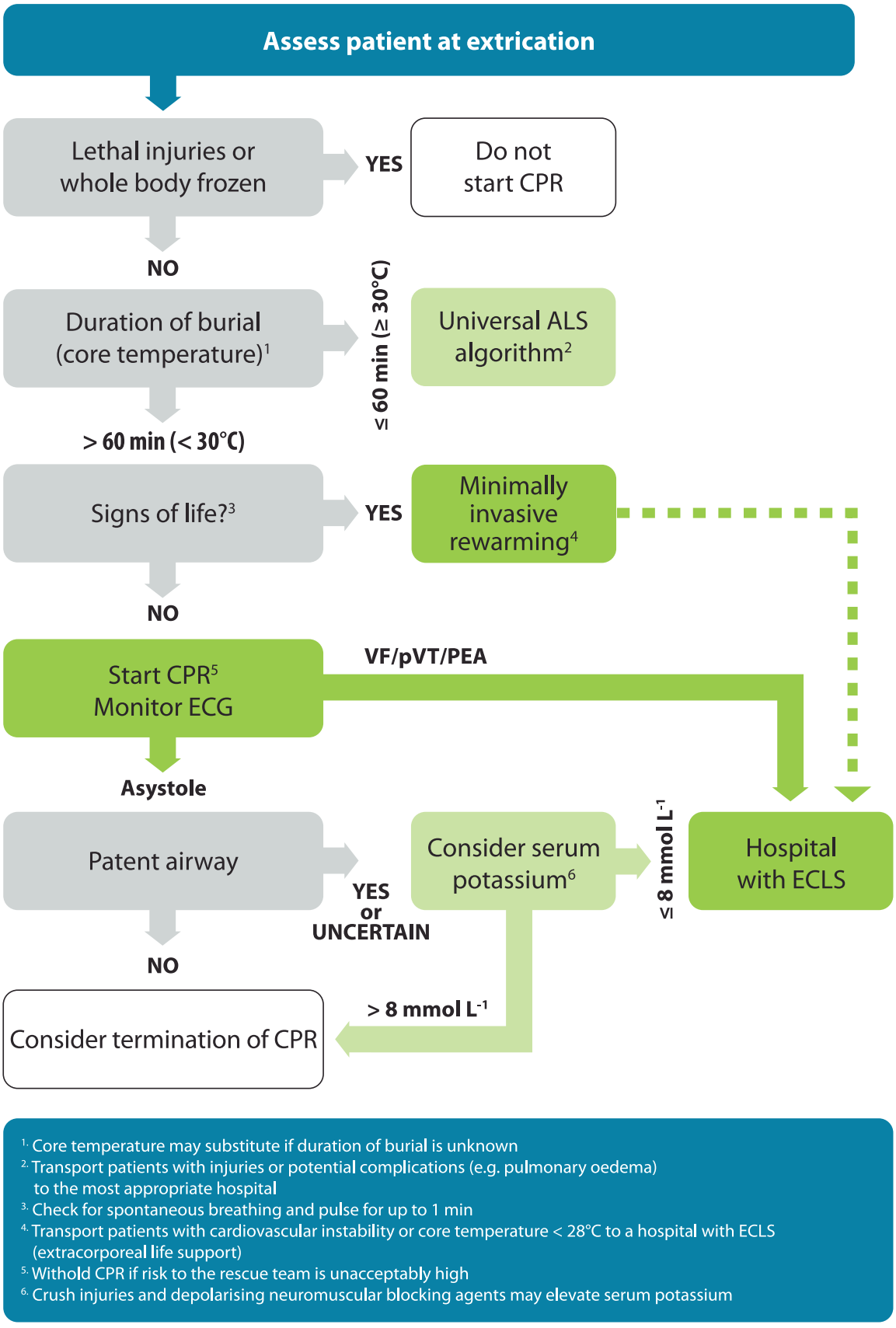
From 20 weeks' gestation, the uterus can compress both the inferior vena cava (IVC) and aorta, impeding venous return and cardiac output. The hand position for chest compressions may need to be slightly higher on the sternum for patients with advanced pregnancy e.g. third trimester.[360] Manually displace the uterus to the left to reduce IVC compression. Add left lateral tilt if this is feasible and ensure the chest remains supported on a firm surface (e.g. in the operating room). Consider the need for an emergency hysterotomy or Caesarean section as soon as a pregnant woman goes into cardiac arrest. 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.[361]

Elderly people

More than 50% of people resuscitated from OHCA are aged 65 years or older.[362] No modifications of standard resuscitation protocols are needed when managing aged patients in cardiac arrest. Rescuers, however, should be aware that the risk of both sternal and rib fractures is higher in elderly.[363–365] The incidence of CPR-related injuries increases with duration of CPR.[365]

Post-resuscitation care

Successful return of spontaneous circulation (ROSC) is the first step towards the goal of complete recovery from cardiac arrest. The complex pathophysiological processes that occur following whole body ischaemia during cardiac arrest and the subsequent reperfusion response during CPR and following successful resuscitation have been termed the post-cardiac arrest syndrome.[366]



1. Core temperature may substitute if duration of burial is unknown
2. Transport patients with injuries or potential complications (e.g. pulmonary oedema) to the most appropriate hospital
3. Check for spontaneous breathing and pulse for up to 1 min
4. Transport patients with cardiovascular instability or core temperature < 28°C to a hospital with ECLS (extracorporeal life support)
5. Withold CPR if risk to the rescue team is unacceptably high
6. Crush injuries and depolarising neuromuscular blocking agents may elevate serum potassium

Fig. 1.14. Avalanche accident algorithm.

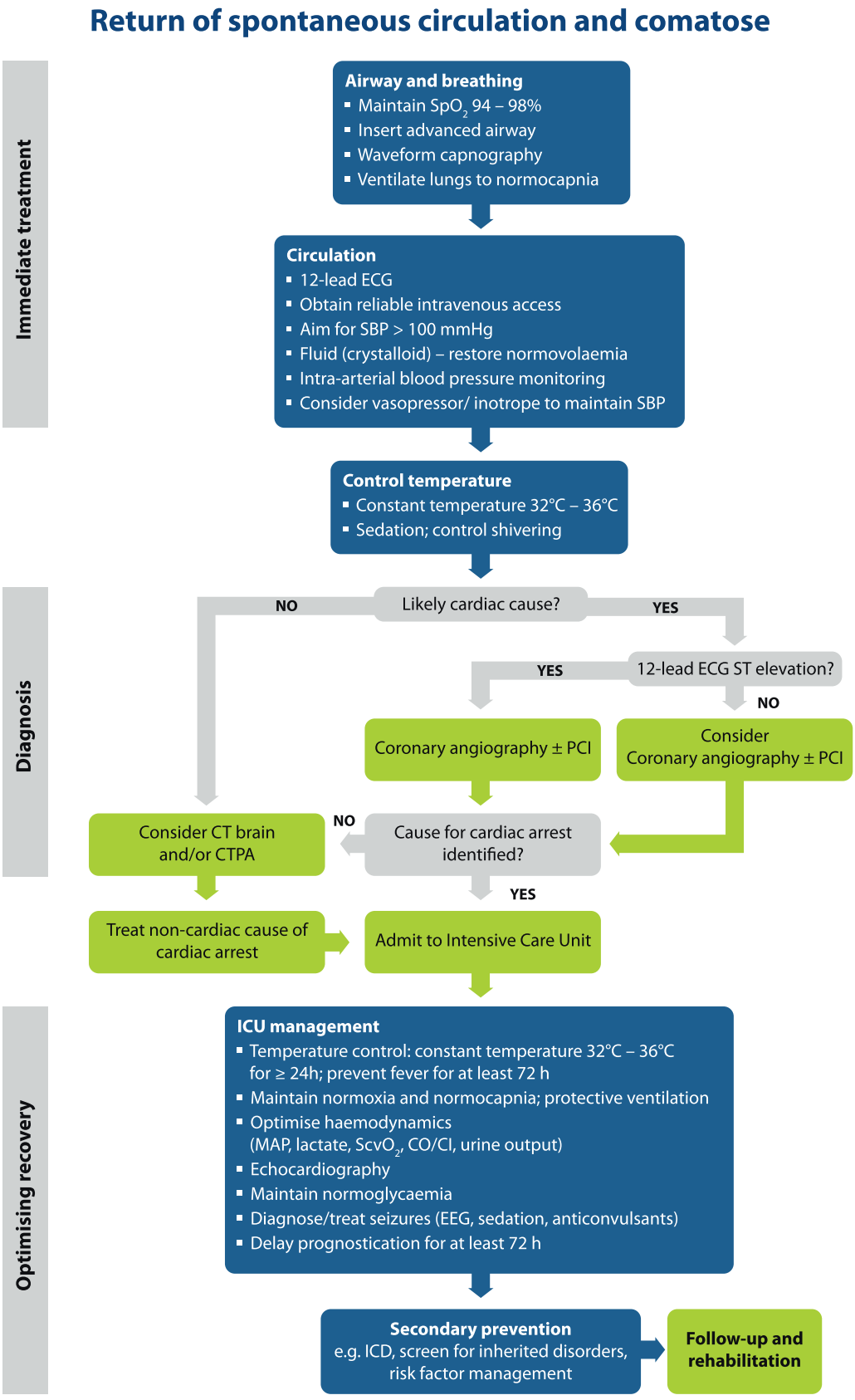


Fig. 1.15. Post resuscitation care algorithm. SBP – systolic blood pressure; PCI – percutaneous coronary intervention; CTPA – computed tomography pulmonary angiogram; ICU – intensive care unit; MAP – mean arterial pressure; $ScvO_2$ – central venous oxygenation; CO/CI – cardiac output/cardiac index; EEG – electroencephalography; ICD – implanted cardioverter defibrillator.

Depending on the cause of the arrest, and the severity of the post-cardiac arrest syndrome, many patients will require multiple organ support and the treatment they receive during this post-resuscitation period influences significantly the overall outcome and particularly the quality of neurological recovery.[367–373] The post-resuscitation care algorithm (Fig. 1.15) outlines some of the key interventions required to optimise outcome for these patients.

Post-cardiac arrest syndrome

The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and the persistent precipitating pathology.[366,374,375] 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. Cardiovascular failure accounts for most deaths in the first three days, while brain injury accounts for most of the later deaths.[376–378] Withdrawal of life sustaining therapy (WLST) is the most frequent cause of death (approximately 50%) in patients with a prognosticated bad outcome,[378,379] emphasising the importance of the prognostication plan (see below). Post-cardiac arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypotension, hypercarbia, hypoxaemia, hyperoxaemia, pyrexia, hypoglycaemia, hyperglycaemia and seizures. Significant myocardial dysfunction is common after cardiac arrest but typically starts to recover by 2–3 days, although full recovery may take significantly longer.[380–382] The whole body ischaemia/reperfusion of cardiac arrest activates immune and coagulation pathways contributing to multiple organ failure and increasing the risk of infection.[383] Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion, vasodilation, endothelial injury and abnormalties of the microcirculation.[384–390]

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 early after ROSC causes oxidative stress and harms post-ischaemic neurones.[391] Virtually all human data is derived from intensive care unit registries and they have produced conflicting results on the potential impact of hyperoxaemia after resuscitation from cardiac arrest.[392] A recent study of air versus supplemental oxygen in ST-elevation myocardial infarction showed that supplemental oxygen therapy increased myocardial injury, recurrent myocardial infarction and major cardiac arrhythmia and was associated with larger infarct size at 6 months.[393] Given the evidence of harm after myocardial infarction and the possibility of increased neurological injury after cardiac arrest, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%. Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration.

Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. After cardiac arrest, hypocapnia induced by hyperventilation causes cerebral ischaemia.[394–396] Observational studies using cardiac arrest registries document an association between hypocapnia and poor neurological outcome.[397,398] Until prospective data are available, it is reasonable to adjust ventilation to achieve normocarbia and to monitor this using the end-tidal CO_2 and arterial blood gas values.

Circulation

Acute coronary syndrome (ACS) is a frequent cause of out-of-hospital cardiac arrest (OHCA): in a recent meta-analysis, the prevalence of an acute coronary artery lesion ranged from 59% to 71% in OHCA patients without an obvious non-cardiac aetiology.[399] Many observational studies have shown that emergent cardiac catheterisation laboratory evaluation, including early percutaneous coronary intervention (PCI), is feasible in patients with ROSC after cardiac arrest.[400,401] The invasive management (i.e. early coronary angiography followed by immediate PCI if necessary) of these patients, particularly those having prolonged resuscitation and nonspecific ECG changes, has been controversial because of the lack of specific evidence and significant implications on use of resources (including transfer of patients to PCI centres).

Percutaneous coronary intervention following ROSC with ST-Elevation

Based on available data, emergent cardiac catheterisation laboratory evaluation (and immediate PCI if required) should be performed in adult patients with ROSC after OHCA of suspected cardiac origin with STE on the ECG. This recommendation is based on low quality of evidence from selected populations. Observational studies also indicate that optimal outcomes after OHCA are achieved with a combination of TTM and PCI, which can be included in a standardised post–cardiac arrest protocol as part of an overall strategy to improve neurologically intact survival.[401–403]

Percutaneous coronary intervention following ROSC without ST-Elevation

In contrast to the usual presentation of ACS in non-cardiac arrest patients, the standard tools to assess coronary ischaemia in cardiac arrest patients are less accurate. The sensitivity and specificity of the usual clinical data, ECG and biomarkers to predict an acute coronary artery occlusion as the cause of OHCA are unclear.[404–407] Several large observational series showed that absence of STE may also be associated with ACS in patients with ROSC following OHCA.[408–411] In these non-STE patients, there are conflicting data from observational studies on the potential benefit of emergent cardiac catheterization laboratory evaluation.[410,412,413] It is reasonable to discuss and consider emergent cardiac catheterisation laboratory evaluation after ROSC in patients with the highest risk of a coronary cause for their cardiac arrest. Factors such as patient age, duration of CPR, haemodynamic instability, presenting cardiac rhythm, neurological status upon hospital arrival, and perceived likelihood of cardiac aetiology can influence the decision to undertake the intervention in the acute phase or to delay it until later on in the hospital stay.

Indications and timing of computed tomography (CT) scanning

Cardiac causes of OHCA have been extensively studied in the last few decades; conversely, little is known about non-cardiac causes. Early identification of a respiratory or neurological cause would enable transfer of the patient to a specialised ICU for optimal care. Improved knowledge of prognosis also enables discussion about the appropriateness of specific therapies, including TTM. Early identification of a respiratory or neurological cause can be achieved by performing a brain and chest CT-scan at hospital admission, before or after coronary angiography. In the absence of signs or symptoms suggesting a neurological or respiratory cause (e.g. headache, seizures or neurological deficits for neurological causes, shortness of breath or documented hypoxia in patients suffering from

a known and worsening respiratpry disease) or if there is clinical or ECG evidence of myocardial ischaemia, coronary angiography is undertaken first, followed by CT scan in the absence of causative lesions. Several case series showed that this strategy enables diagnosis of non-cardiac causes of arrest in a substantial proportion of patients.[358,414]

Haemodynamic management

Post-resuscitation myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias.[380,415] Perform early echocardiography in all patients in order to detect and quantify the degree of myocardial dysfunction.[381,416] Post-resuscitation myocardial dysfunction often requires inotropic support, at least transiently.

Treatment may be guided by blood pressure, heart rate, urine output, rate of plasma lactate clearance, and central venous oxygen saturation. Serial echocardiography may also be used, especially in haemodynamically unstable patients. In the ICU an arterial line for continuous blood pressure monitoring is essential.

Similarly to the early goal-directed therapy that is recommended in the treatment of sepsis, [417] although challenged by several recent studies,[418–420] a bundle of therapies, including a specific blood pressure target, has been proposed as a treatment strategy after cardiac arrest.[370] 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.[366] These targets may vary depending on individual physiology and co-morbid status. Importantly, hypothermia may increase urine output[421] and impair lactate clearance.[415]

Implantable cardioverter defibrillators

Consider insertion of an implantable cardioverter defibrillator (ICD) in ischaemic patients with significant left ventricular dysfunction, who have been resuscitated from a ventricular arrhythmia that occurred later than 24–48 h after a primary coronary event.[422–424]

Disability (optimising neurological recovery)

Cerebral perfusion

Animal studies show that immediately after ROSC there is a short period of multifocal cerebral no-reflow followed by transient global cerebral hyperaemia lasting 15–30 min.[425–427] This is followed by up to 24 h of cerebral hypoperfusion while the cerebral metabolic rate of oxygen gradually recovers. After asphyxial cardiac arrest, brain oedema may occur transiently after ROSC but it is rarely associated with clinically relevant increases in intracranial pressure.[428,429] In many patients, autoregulation of cerebral blood flow is impaired (absent or right-shifted) for some time after cardiac arrest, which means that cerebral perfusion varies with cerebral perfusion pressure instead of being linked to neuronal activity.[430,431] Thus, after ROSC, maintain mean arterial pressure near the patient's normal level.[12]

Sedation

Although it has been common practice to sedate and ventilate patients for at least 24 h after ROSC, there are no high-level data to support a defined period of ventilation, sedation and neuromuscular blockade after cardiac arrest.

Control of seizures

Seizures are common after cardiac arrest and occur in approximately one-third of patients who remain comatose after ROSC. Myoclonus is most common and occurs in 18–25%, the remainder having focal or generalised tonic–clonic seizures or a combination of seizure types.[376,432–434] Clinical seizures, including myoclonus may or may not be of epileptic origin. Other motor manifestations could be mistaken for seizures and there are several types of myoclonus, the majority being non-epileptic.[435,436] Use intermittent electroencephalography (EEG) to detect epileptic activity in patients with clinical seizure manifestations. Consider continuous EEG to monitor patients with a diagnosed status epilepticus and effects of treatment. Seizures may increase the cerebral metabolic rate[437] and have the potential to exacerbate brain injury caused by cardiac arrest: treat with sodium valproate, levetiracetam, phenytoin, benzodiazepines, propofol, or a barbiturate. Myoclonus can be particularly difficult to treat; phenytoin is often ineffective. Propofol is effective to suppress post-anoxic myoclonus.[438] Clonazepam, sodium valproate and levetiracetam are antimyoclonic drugs that may be effective in post-anoxic myoclonus.[436]

Glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome.[261,439,440] Based on the available data, following ROSC maintain the blood glucose at $\leq 10\,mmol\,l^{-1}$ ($180\,mg\,dl^{-1}$) and avoid hypoglycaemia.[441] Do not implement strict glucose control in adult patients with ROSC after cardiac arrest because it increases the risk of hypoglycaemia.

Temperature control

A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest.[261,442–445] Several studies document an association between post-cardiac arrest pyrexia and poor outcomes.[261,442,444–447] Although the effect of elevated temperature on outcome is not proven, it seems reasonable to treat hyperthermia occurring after cardiac arrest with antipyretics and to consider active cooling in unconscious patients.

Animal and human data indicate that mild induced hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia.[448,449] All studies of post-cardiac arrest mild induced hypothermia have included only patients in coma. One randomised trial and a pseudo-randomised trial demonstrated improved neurological outcome at hospital discharge or at 6 months in comatose patients after out-of-hospital VF cardiac arrest.[450,451] Cooling was initiated within minutes to hours after ROSC and a temperature range of 32–34 °C was maintained for 12–24 h.

In the Targeted Temperature Management (TTM) trial, 950 all-rhythm OHCA patients were randomised to 36 h of temperature control (comprising 28 h at the target temperature followed by slow rewarm) at either 33 °C or 36 °C.[376] Strict protocols were followed for assessing prognosis and for withdrawal of life-sustaining treatment (WLST). There was no difference in the primary outcome – all cause mortality, and neurological outcome at 6 months was also similar (hazard ratio (HR) for mortality at end of trial 1.06, 95% CI 0.89–1.28; relative risk (RR) for death or poor neurological outcome at 6 months 1.02, 95% CI 0.88–1.16). Detailed neurological outcome at 6 months was also similar.[452,453] Importantly, patients in both arms of this trial had their temperature well controlled so that fever was prevented in both groups.

The term targeted temperature management or temperature control is now preferred over the previous term therapeutic hypothermia. The ALS Task Force of the International Liaison Committee on Resuscitation made several treatment recommendations on targeted temperature management[175] and these are reflected in these ERC guidelines:

- Maintain a constant, target temperature between 32 °C and 36 °C for those patients in whom temperature control is used (strong recommendation, moderate-quality evidence).

- Whether certain subpopulations of cardiac arrest patients may benefit from lower (32–34 °C) or higher (36 °C) temperatures remains unknown, and further research may help elucidate this.
- TTM is recommended for adults after OHCA with an initial shockable rhythm who remain unresponsive after ROSC (strong recommendation, low-quality evidence).
- TTM is suggested for adults after OHCA with an initial non-shockable rhythm who remain unresponsive after ROSC (weak recommendation, very low-quality evidence).
- TTM is suggested for adults after IHCA with any initial rhythm who remain unresponsive after ROSC (weak recommendation, very low-quality evidence).
- If targeted temperature management is used, it is suggested that the duration is at least 24 h (as undertaken in the two largest previous RCTs[376,450]) (weak recommendation, very low-quality evidence).

When to control temperature?. Whichever target temperature is selected, active temperature control is required to achieve and maintain the temperature in this range. Prior recommendations suggest that cooling should be initiated as soon as possible after ROSC, but this recommendation was based only on preclinical data and rational conjecture.[454] Animal data indicate that earlier cooling after ROSC produces better outcomes.[455,456] Observational studies are confounded by the fact that there is an association between patients who cool faster spontaneously and worse neurological outcome.[457–459] It is hypothesised that those with the most severe neurological injury are more prone to losing their ability to control body temperature.

A randomised trial of prehospital cooling using a rapid infusion of large volumes of cold intravenous fluid immediately after ROSC versus cooling delayed until hospital admission showed increased rates of re-arrest during transport and pulmonary oedema.[460] Although uncontrolled prehospital infusion of cold fluid is not recommended, it may still be reasonable to infuse cold intravenous fluid where patients are well monitored and a lower target temperature (e.g. 33 °C) is the goal. Early cooling strategies, other than rapid infusion of large volumes of cold intravenous fluid, and cooling during cardiopulmonary resuscitation in the prehospital setting have not been studied adequately.

How to control temperature?. As yet, there are no data indicating that any specific cooling technique increases survival when compared with any other cooling technique; however, internal devices enable more precise temperature control compared with external techniques.[461,462] Rebound hyperthermia is associated with worse neurological outcome.[463,464] Thus, rewarming should be achieved slowly: the optimal rate is not known, but the consensus is currently about 0.25–0.5 °C of rewarming per hour.[465]

Prognostication

This section on prognostication has been adapted from the Advisory Statement on Neurological Prognostication in comatose survivors of cardiac arrest,[466] written by members of the ERC ALS Working Group and of the Trauma and Emergency Medicine (TEM) Section of the European Society of Intensive Care Medicine (ESICM), in anticipation of the 2015 Guidelines.

Hypoxic-ischaemic brain injury is common after resuscitation from cardiac arrest.[467] Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury; this has been shown both before[468] and after[376–378] the implementation of target temperature management (TTM) for post-resuscitation care. Most of these deaths are due to active withdrawal of life sustaining treatment (WLST) based on prognostication of a poor neurological outcome.[377,378] For this reason, when dealing with patients who are comatose after resuscitation from cardiac arrest minimising the risk of a falsely pessimistic prediction is essential. Ideally, when predicting a poor outcome the false positive rate (FPR) should be zero with the narrowest possible confidence interval (CI). However, most prognostication studies include so few patients that even if the FPR is 0%, the upper limit of the 95% CI is often high.[469,470] Moreover, many studies are confounded by self-fulfilling prophecy, which is a bias occurring when the treating physicians are not blinded to the results of the outcome predictor and use it to make a decision on WLST.[469,471] Finally, both TTM itself and sedatives or neuromuscular blocking drugs used to maintain it may potentially interfere with prognostication indices, especially those based on clinical examination.[472] A multimodal approach to prognostication is essential and includes: clinical examination, electrophysiology, biomarkers and imaging.

A careful clinical neurological examination remains the foundation for prognostication of the comatose patient after cardiac arrest.[473] Perform a thorough clinical examination daily to detect signs of neurological recovery such as purposeful movements or to identify a clinical picture suggesting that brain death has occurred.

The process of brain recovery following global post-anoxic injury is completed within 72 h from arrest in most patients.[474,475] However, in patients who have received sedatives ≤12 h before the 72 h post-ROSC neurological assessment, the reliability of clinical examination may be reduced.[472] Before decisive assessment is performed, major confounders must be excluded;[476,477] apart from sedation and neuromuscular blockade, these include hypothermia, severe hypotension, hypoglycaemia, and metabolic and respiratory derangements. Suspend sedatives and neuromuscular blocking drugs for long enough to avoid interference with clinical examination. Short-acting drugs are preferred whenever possible. When residual sedation/paralysis is suspected, consider using antidotes to reverse the effects of these drugs.

The prognostication strategy algorithm (Fig. 1.16) is applicable to all patients who remain comatose with an absent or extensor motor response to pain at ≥72 h from ROSC. Results of earlier prognostic tests are also considered at this time point.

Evaluate the most robust predictors first. These predictors have the highest specificity and precision (FPR <5% with 95% CIs <5% in patients treated with controlled temperature) and have been documented in several studies from at least three different groups of investigators. They include bilaterally absent pupillary reflexes at ≥72 h from ROSC and bilaterally absent somatosensory evoked potential (SSEP) N20 wave after rewarming (this last sign can be evaluated at ≥24 h from ROSC in patients who have not been treated with controlled temperature). Based on expert opinion, we suggest combining the absence of pupillary reflexes with those of corneal reflexes for predicting poor outcome at this time point. Ocular reflexes and SSEPs maintain their predictive value irrespective of target temperature.[478,479]

If none of the signs above is present to predict a poor outcome, a group of less accurate predictors can be evaluated, but the degree of confidence in their prediction will be lower. These have FPR < 5% but wider 95% CIs than the previous predictors, and/or their definition/threshold is inconsistent in prognostication studies. These predictors include the presence of early status myoclonus (within 48 h from ROSC), high values of serum neuron specific enolase (NSE) at 48–72 h after ROSC, an unreactive malignant EEG pattern (burst-suppression, status epilepticus) after rewarming, the presence of a marked reduction of the grey matter to white matter (GM/WM) ratio or sulcal effacement on brain CT within 24 h after ROSC or the presence of diffuse ischaemic changes on brain magnetic resonance imaging (MRI) at 2–5 days after ROSC. Based on expert opinion, we suggest waiting at least 24 h after the first prognostication assessment and confirming unconsciousness with a Glasgow motor score

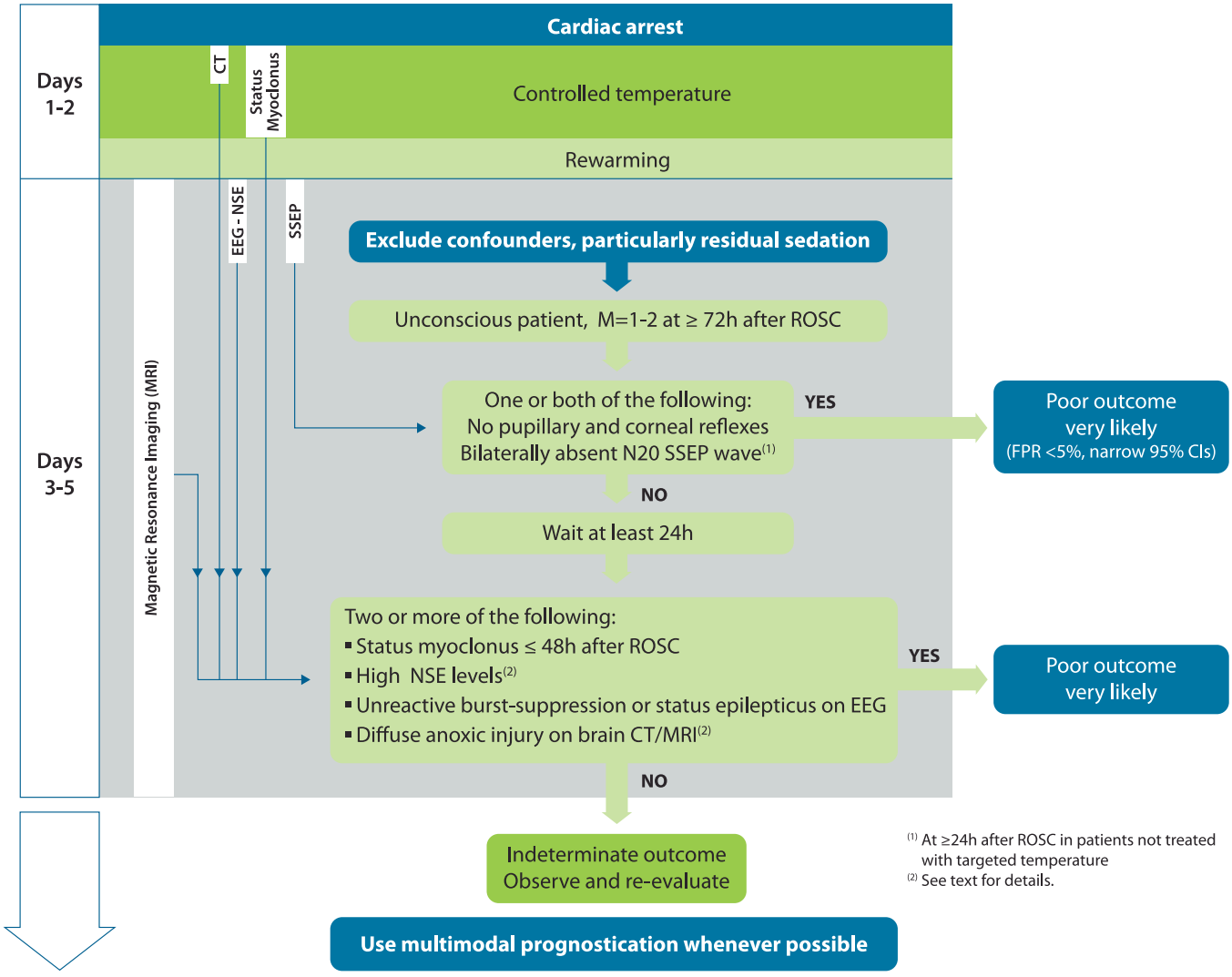


Fig. 1.16. Prognostication strategy algorithm. EEG – electroencephalography; NSE – neuron specific enolase; SSEP – somatosensory evoked potentials; ROSC – return of spontaneous circulation; M – Motor score of Glasgow Coma Scale.

of 1–2 before using this second set of predictors. We also suggest combining at least *two* of these predictors for prognostication.

No specific NSE threshold for prediction of poor outcome with 0% FPR can be recommended at present. Ideally, every hospital laboratory assessing NSE should create its own normal values and cut-off levels based on the test kit used. Sampling at multiple timepoints is recommended to detect trends in NSE levels and to reduce the risk of false positive results.[480]

Although the most robust predictors showed no false positives in most studies, none of them singularly predicts poor outcome with absolute certainty. Moreover, those predictors have often been used for WLST decisions, with the risk of a self-fulfilling prophecy. For this reason, we recommend that prognostication should be multimodal whenever possible, even in presence of one of these predictors. Apart from increasing safety, limited evidence also suggests that multimodal prognostication increases sensitivity.[481–484]

When dealing with an uncertain outcome, clinicians should consider prolonged observation. Absence of clinical improvement over time suggests a worse outcome. Although awakening has been described as late as 25 days after arrest,[485–487] most survivors will recover consciousness within one week.[376,488–491] In a recent observational study,[490] 94% of patients awoke within 4.5 days from rewarming and the remaining 6% awoke within ten days. Even those awakening late can still have a good neurological outcome.[490]

Rehabilitation

Although neurological outcome is considered to be good for the majority of cardiac arrest survivors, cognitive and emotional problems and fatigue are common.[452,492–494] Long-term cognitive impairments, mostly mild, are present in half of survivors.[453,495,496] Mild cognitive problems are often not recognised by health care professionals and cannot be detected with standard outcome scales such as the Cerebral Performance Categories (CPC) or the Mini-Mental State Examination (MMSE).[452,497] Both cognitive and emotional problems have significant impact and can affect a patient's daily functioning, return to work and quality of life.[494,498,499] after hospital discharge should be organised systematically and can be provided by a physician or specialised nurse. It should at least include screening for cognitive impairments and for emotional problems, and the provision of information.

Organ donation

Organ donation should be considered in those who have achieved ROSC and who fulfil criteria for death using neurological criteria.[500] In those comatose patients in whom a decision is made to withdraw life-sustaining therapy, organ donation should be considered after circulatory death occurs. Organ donation can also be

considered in individuals where CPR is not successful in achieving ROSC. All decisions concerning organ donation must follow local legal and ethical requirements, as these vary in different settings.

Screening for inherited disorders

Many sudden death victims have silent structural heart disease, most often coronary artery disease, but also primary arrhythmia syndromes, cardiomyopathies, familial hypercholesterolaemia and premature ischaemic heart disease. Screening for inherited disorders is crucial for primary prevention in relatives as it may enable preventive antiarrhythmic treatment and medical follow-up.[154,155,501]

Cardiac arrest centres

There is wide variability in survival among hospitals caring for patients after resuscitation from cardiac arrest.[261,371,502–506] Many studies have reported an association between survival to hospital discharge and transport to a cardiac arrest centre but there is inconsistency in the hospital factors that are most related to patient outcome.[368,371,504,507,508] There is also inconsistency in the services that together define a cardiac arrest centre. Most experts agree that such a centre must have a cardiac catheterisation laboratory that is immediately accessible 24/7 and the facility to provide targeted temperature management.

Paediatric life support

This section of the ERC GL 2015 on Paediatric Life Support includes:

- Basic life support
- Management of foreign bodies in the airway
- Prevention of cardiac arrest
- Advanced life support during cardiac arrest
- Post-resuscitation care

Paediatric basic life support

From the ILCOR CoSTR statement on the sequence for manoeuvres in BLS, there was found to be equipoise between the CAB sequence (compression for circulation, airway and breathing) and the ABC sequence (airway, breathing and compression for circulation).[509–511] Given that the ABC sequence has become an established and well recognised method for the delivery of CPR to children in Europe, the ERC PLS Writing Group determined that the use of this sequence should continue, particularly as the previous guidelines have led to its instruction to many hundreds of thousands of healthcare providers and lay people.

Sequence of actions in basic life support

Rescuers who have been taught adult BLS or the chest compression-only sequence and have no specific knowledge of paediatric resuscitation may use this, as the outcome is worse if they do nothing. However, it is better to provide rescue breaths as part of the resuscitation sequence when applied to children as the asphyxial nature of most paediatric cardiac arrests necessitates ventilation as part of effective CPR.[119,120] 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 1 min of CPR before they go for help (see adult BLS guidelines).

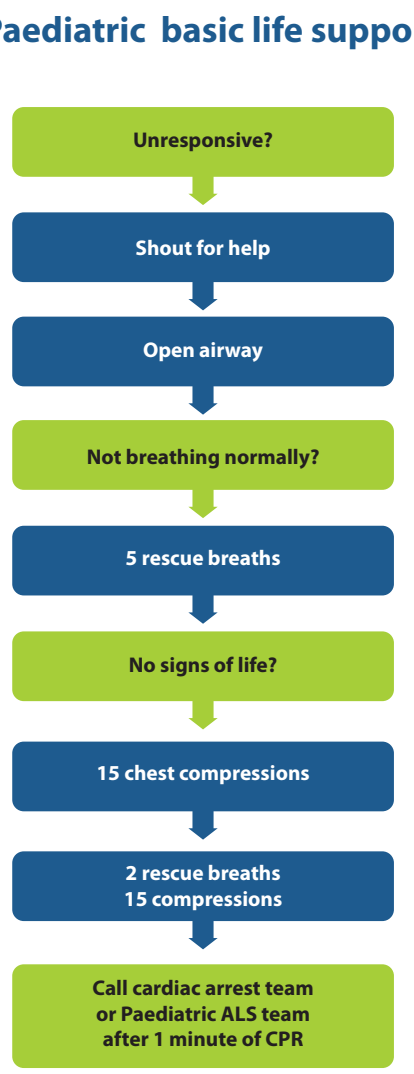


Fig. 1.17. Paediatric basic life support algorithm.

Basic life support 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 professionals) (Fig. 1.17). Although the following sequence describes expired air ventilation, health professionals with a responsibility for treating children will usually have access to, and training in the use of bag mask ventilation (BMV), and these should be used to provide rescue breaths.

1. Ensure the safety of rescuer and child

2. Check the child's responsiveness

- Stimulate the child and ask loudly: Are you all right?

3A. If the child responds by answering, crying or moving:

- Leave the child in the position in which you find him (provided he is not in further danger).
- Check his condition and call for help.
- Reassess him regularly.

3B. If the child does not respond:

- Shout for help.
- Turn the child carefully 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

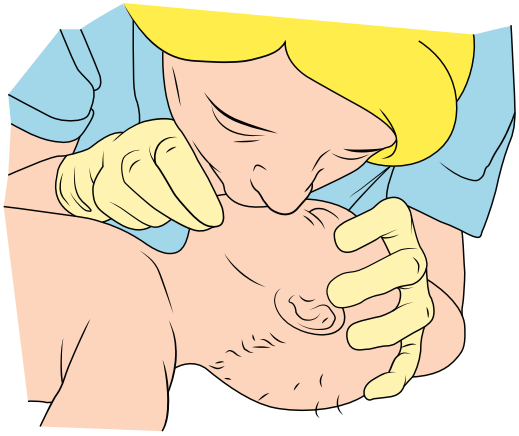


Fig. 1.18. Mouth-to-mouth-and-nose ventilation – infant.

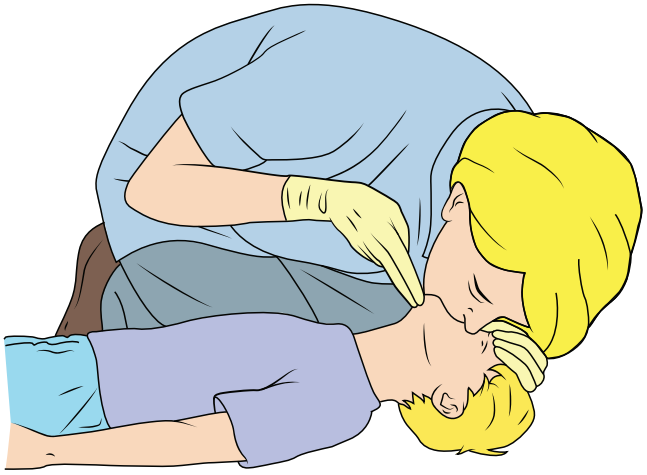


Fig. 1.19. Mouth-to-mouth ventilation – child.

the chin as this may obstruct the airway. This is especially important in infants.

- If you still have difficulty in opening the airway, try a jaw thrust: place the first two fingers of each hand behind each side of the child's mandible and push the jaw forward.

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

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

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

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). If there is a history of trauma, cervical spine injury should be considered.
- Send or go for help – call the emergency services.
- Check for continued breathing.

5B. If breathing is not normal or absent:

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

Rescue breaths for an infant

- Ensure a neutral position of the head as an infant's head is usually flexed when supine, this may require some extension (a rolled towel/blanket under the upper part of the body may help to maintain the position) 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) (Fig. 1.18).
- Blow steadily into the infant's mouth and nose for about 1 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.

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 (Fig. 1.19).
- Blow steadily into the mouth for about 1 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.

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.
- Reposition the head. 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 (gasps or infrequent, irregular breaths are abnormal). If you check the pulse, ensure that you take no more than 10 s. Pulse check is unreliable and therefore the complete picture of how the patient appears must guide whether BLS is required, i.e. if there are no signs of life, start BLS.[40,41]

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 his side (into the recovery position, with caution if there is a history of trauma) if he remains unconscious.
- Re-assess the child frequently.

7B. If there are no signs of life

- Start chest compressions.

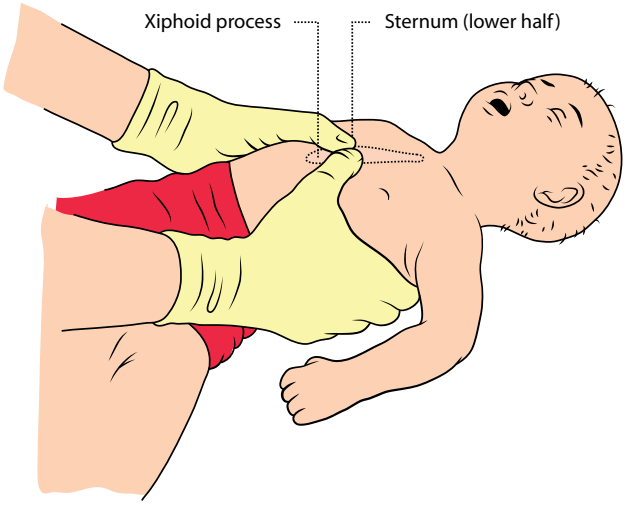


Fig. 1.20. Chest compression – infant.

- Combine rescue breathing and chest compressions at a ratio of 15 compressions to 2 ventilations.

Chest compressions. For all children, compress the lower half of the sternum. The compression should be sufficient to depress the sternum by at least one third of the anterior-posterior diameter of the chest. Release the pressure completely and repeat at a rate 100–120 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.

Chest compression in infants. The lone rescuer compresses the sternum with the tips of two fingers (Fig. 1.20). 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 both hands with the fingers together to encircle the lower part of the infant's rib cage. The fingers should support the infant's back. For both methods, depress the lower sternum by at least one third the anterior-posterior dimension of the infant's chest or by 4 cm.[512]

Chest compression in children over 1 year of age. To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Place the heel of one hand on the sternum one finger's breadth above this. Lift the fingers to ensure that pressure is not applied onto the child's ribs. Position yourself above the victim's chest and, with your arm straight, compress the sternum to at least one third of the anterior-posterior dimension of the chest or by 5 cm (Fig. 1.21).[512,513] In larger children or for small rescuers, this is achieved most easily by using both hands, with the rescuer's fingers interlocked (Fig. 1.22).

8. Do not interrupt resuscitation until:

- The child shows signs of life (starts to wake up, to move, opens eyes and to breathe normally).
- More healthcare workers arrive and can either assist or take 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.

Fig. 1.21. Chest compression with one hand – child.

- If only one rescuer is present, undertake resuscitation for about 1 min or 5 cycles of CPR before going for assistance. To minimise interruption in CPR, it may be possible to carry an infant or small child whilst summoning help.
- If you are on your own, witness a child suddenly collapse and you suspect a primary cardiac arrest, call for help first and then start CPR as the child will likely need urgent defibrillation. This is an uncommon situation.

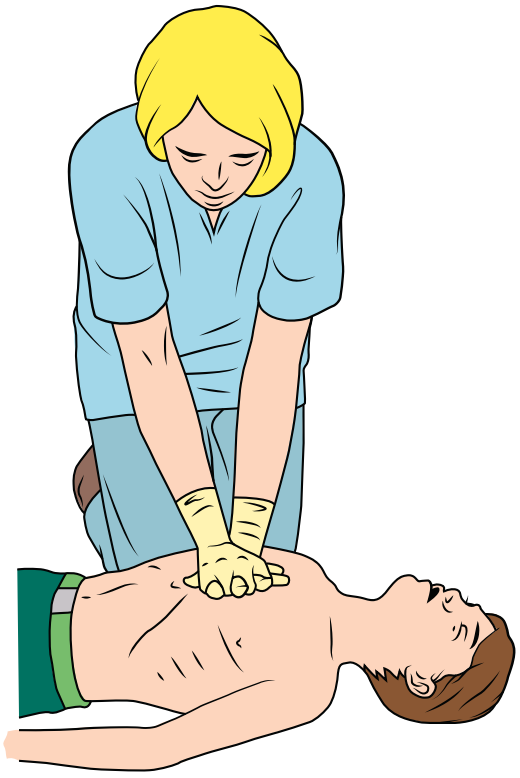


Fig. 1.22. Chest compression with two hands – child.

Table 1.1
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 cough	**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	

Automated external defibrillation and basic life support

Continue with CPR until the AED arrives. Attach the AED and follow the instructions. For 1–8 year old, use attenuated pads if available, as explained in the section on Adult Basic Life Support and Automated External Defibrillation.[1]

Recovery position

An unconscious child whose airway is clear, and who is breathing normally, should be turned on his side into the recovery position. There are several recovery positions; they all aim to prevent airway obstruction and reduce the likelihood of fluids such as saliva, secretions or vomit from entering into the upper airway.

Foreign body airway obstruction (FBAO)

Suspect FBAO if the onset was very sudden and there are no other signs of illness; there may be clues to alert the rescuer, e.g. a history of eating or playing with small items immediately before the onset of symptoms (Table 1.1)

Back blows, chest thrusts and abdominal thrusts all increase intra-thoracic pressure and can expel foreign bodies from the airway. If one is unsuccessful, try the others in rotation until the object is cleared (Fig. 1.23).

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. For this reason, the guidelines for the treatment of FBAO are different between infants and children.

Recognition of foreign body airway obstruction

Active interventions to relieve FBAO are required only when coughing becomes ineffective, but they then need to be commenced rapidly and confidently

Relief of FBAO

1. Safety and summoning assistance. The principle of do no harm should be applied, i.e. if the child is able to breathe and cough, even with difficulty, encourage these spontaneous efforts. Do not intervene at this point as this may move the foreign body and worsen the problem, e.g. by causing full airway obstruction.

- If the child is coughing effectively, no manoeuvre is necessary. Encourage the child to cough and continue monitoring the child's condition.
- If the child's coughing is (or is becoming) ineffective, shout for help immediately and determine the child's conscious level.

2. Conscious child with FBAO.

- If the child is still conscious but has absent or ineffective coughing, give back blows.
- If back blows do not relieve the FBAO, give chest thrusts to infants or abdominal thrusts to children. These manoeuvres create an artificial cough, increasing intrathoracic pressure and dislodging the foreign body.

If back blows fail to dislodge the object, and the child is still conscious, use chest thrusts for infants or abdominal thrusts for children. Do not use abdominal thrusts (Heimlich manoeuvre) in infants.

Following the chest or abdominal thrusts, reassess the child. If the object has not been expelled and the victim is still conscious, continue the sequence of back blows and chest (for

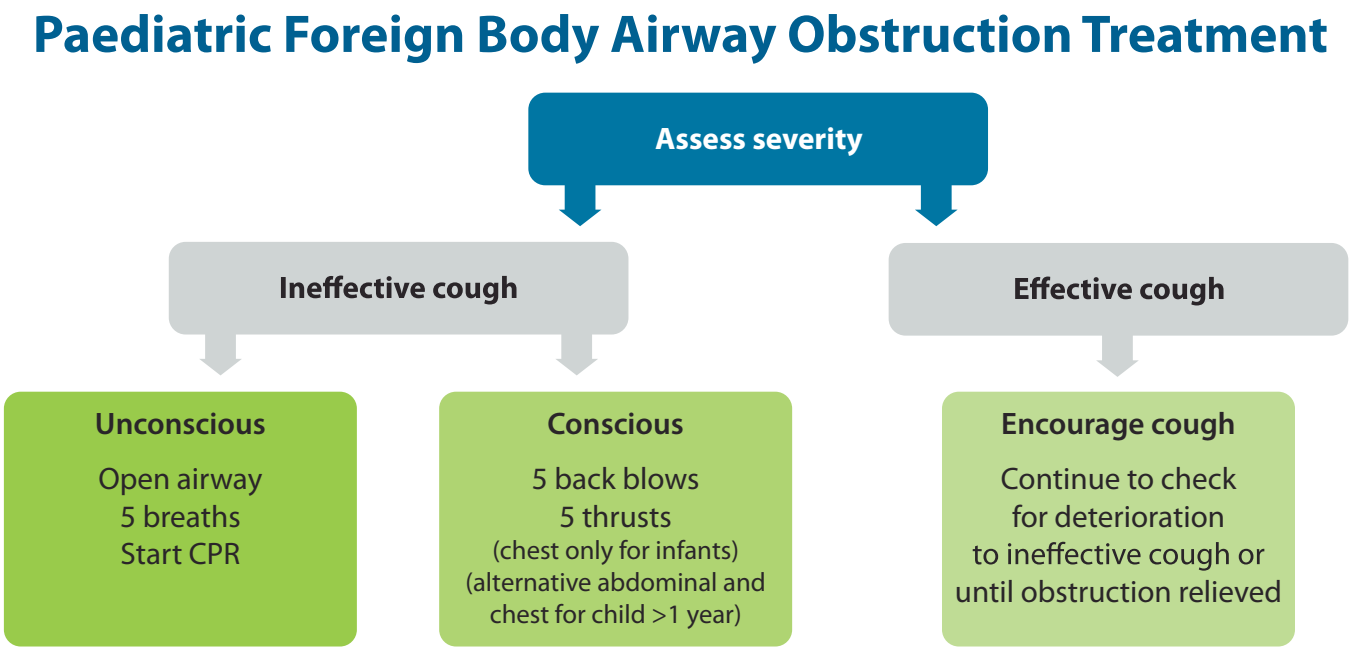


Fig. 1.23. Paediatric foreign body airway obstruction algorithm.

infant) or abdominal (for children) thrusts. Call out, or send, for help if it is still not available. Do not leave the child at this stage.

If the object is expelled successfully, assess the child's clinical condition. It is possible that part of the object may remain in the respiratory tract and cause complications. If there is any doubt, seek medical assistance. Abdominal thrusts may cause internal injuries and all victims treated with abdominal thrusts should be examined by a doctor.[514]

3. Unconscious child with FBAO. If the child with FBAO is, or becomes, unconscious, place him on a firm, flat surface. Call out, or send, for help if it is still not available. Do not leave the child at this stage; proceed as follows.

Airway opening. Open the mouth and look for any obvious object. If one is seen, make an attempt to remove it with a single finger sweep. Do not attempt blind or repeated finger sweeps – these could push the object deeper into the pharynx and cause injury.

Rescue breaths. Open the airway using a head tilt/chin lift and attempt five rescue breaths. Assess the effectiveness of each breath: if a breath does not make the chest rise, reposition the head before making the next attempt.

Chest compressions and CPR.

- Attempt five rescue breaths and if there is no response (moving, coughing, spontaneous breaths) proceed to chest compressions without further assessment of the circulation.
- Follow the sequence for single rescuer CPR for approximately a minute or 5 cycles of 15 compressions to 2 ventilations before summoning the EMS (if this has not already been done by someone else).
- When the airway is opened for attempted delivery of rescue breath, check if the foreign body can be seen in the mouth.
- If an object is seen and can be reached, attempt to remove it with a single finger sweep.
- If it appears the obstruction has been relieved, open and check the airway as above; deliver rescue breaths if the child is not breathing.
- If the child regains consciousness and exhibits spontaneous effective breathing, place him in a safe position on his side (recovery position) and monitor breathing and the level of consciousness whilst awaiting the arrival of the EMS.

Paediatric advanced life support

Assessment of the seriously ill or injured child – the 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.[147,515–524] So-called asphyxial arrests or respiratory arrests are also more common in young adulthood (e.g. trauma, drowning and poisoning).[119,525]

As the outcome from cardiopulmonary arrest in children is poor, identifying the preceding stages of circulatory or respiratory failure is a priority as effective early intervention in these stages may be lifesaving.

The order of assessment and intervention for any seriously ill child follows the ABCDE principles.

- A indicates airway.
- B indicates breathing.
- C indicates circulation.
- D indicates disability.
- E indicates exposure.

The topics of D and E are beyond the remit of these guidelines but are taught in paediatric life support courses.

Summoning a paediatric rapid response team or medical emergency team may reduce the risk of respiratory and/or cardiac arrest in hospitalised children outside the intensive care setting but the evidence is limited on this point as the literature tends not to separate out the team response alone from the other systems in place to identify early deterioration.[526–529] Processes to detect the early deterioration are key in reducing the morbidity and mortality of seriously ill and injured children. Specific scores can be used (e.g. the paediatric early warning score, PEWS),[530] but there is no evidence that these improve the decision making process, or the clinical outcome.[512,531]

Diagnosing respiratory failure: assessment of A and B. The assessment of a potentially critically ill child starts with the assessment of airway (A) and breathing (B). The signs of respiratory failure may include:

- **Respiratory rate** outside the normal range for the child's age – either too fast or too slow.[532]
- Initially increased **work of breathing**, which may progress to inadequate/decreased work of breathing as the child tires or compensatory mechanisms fail.
- **Additional noises** such as stridor, wheeze, crackles, grunting, or the loss of breath sounds.
- Decreased **tidal volume** marked by shallow breathing, decreased chest expansion or decreased air entry at auscultation.
- **Hypoxaemia** (without/with supplemental oxygen) generally identified by cyanosis but it is often detectable prior to this by pulse oximetry.

There may be associated signs in other organ systems. Even though the primary problem is respiratory, other organ systems will be involved to try to ameliorate the overall physiological disturbance.

These are detectable in step C of the assessment and include:

- Increasing tachycardia (compensatory mechanism to increase tissue oxygen delivery).
- Pallor.
- Bradycardia (an ominous indicator of the loss of compensatory mechanisms).
- Alteration in the level of consciousness (a sign that compensatory mechanisms are failing) owing to poor perfusion of the brain.

Diagnosing circulatory failure: assessment of C. Circulatory failure is characterised by a mismatch between the metabolic demand by the tissues, and the delivery of oxygen and nutrients by the circulation.[532,533] Signs of circulatory failure might include:

- Increased **heart rate** (bradycardia is an ominous sign of physiological decompensation).[532]
- Decreased systemic **blood pressure**.
- Decreased **peripheral perfusion** (prolonged capillary refill time, decreased skin temperature, pale or mottled skin) – signs of increased vascular resistance.
- Bounding pulses, vasodilation with widespread erythema may be seen in conditions with decreased vascular resistance.
- Weak or absent **peripheral pulses**.
- Decreased **intravascular volume**.
- Decreased urine output.

The transition from a compensatory state to decompensation may occur in an unpredictable way. Therefore, the child should be

monitored, to detect and correct any deterioration in their physiological parameters promptly.

Diagnosing cardiopulmonary arrest

Signs of cardiopulmonary arrest include:

- Unresponsiveness to pain (coma)
- Apnoea or gasping respiratory pattern
- Absent circulation
- Pallor or deep cyanosis

Palpation of a pulse is not reliable as the sole determinant of the need for chest compressions.[40,169,534,535] In the absence of signs of life, rescuers (lay and professional) should begin CPR unless they are certain that they can feel a central pulse within 10 seconds (infants – brachial or femoral artery; children – carotid or femoral artery). If there is any doubt, start CPR.[42,169,170,536] If personnel skilled in echocardiography are available, this investigation may help to detect cardiac activity and potentially treatable causes for the arrest.[534]

Management of respiratory and circulatory failure

Airway and breathing.

- Open the airway.
- Optimise ventilation.
- Ensure adequate oxygenation, start with 100% oxygen.
- Establish respiratory monitoring (first line – pulse oximetry/peripheral oxygen saturation – SpO_2).
- Achieving adequate ventilation and oxygenation – this may require the use of airway adjuncts +/− bag-mask ventilation (BMV), the use of a laryngeal mask airway or other supraglottic airway, securing a definitive airway by tracheal intubation and positive pressure ventilation.
- For intubated children, it is standard practice that their end tidal carbon dioxide levels are monitored. End tidal carbon dioxide monitoring can also be used in non-intubated critically ill patients.
- Very rarely, a surgical airway may be required.

Circulation.

- Establish cardiac monitoring (first line – pulse oximetry/SpO_2, electrocardiography (ECG) and non-invasive blood pressure (NIBP)).
- Secure intravascular access. This may be achieved by peripheral intravenous (IV) or by intraosseous (IO) route. 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) to treat circulatory failure due to hypovolaemia, e.g. from fluid loss or maldistribution, as seen in septic shock and anaphylaxis.
- Consider carefully the use of fluid bolus in primary cardiac functioning disorders, e.g. myocarditis, cardiomyopathy.
- Do not give a fluid bolus in severe febrile illness when circulatory failure is absent.[512,537–539]
- Isotonic crystalloids are recommended as initial resuscitation fluid in infants and children with any type of shock, including septic shock.[512,540–545]
- Assess and re-assess the child repeatedly, beginning each time with the airway before proceeding to breathing and then the circulation. Blood gas and lactate measurement may be helpful.
- 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 treatment of respiratory and/or circulatory failure.[225,226] Whilst the evidence for the use of these techniques is of low quality, the general principles of monitoring and assessing the impact of any interventions and those responses are key in managing seriously ill children.

Table 1.2
Paediatric tracheal tube size in internal diameters (ID) based on age. 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 indicated by resuscitation tapes.

	Uncuffed	Cuffed
Premature neonates	Gestational age in weeks/10	Not used
Full term neonates	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

Airway. Open the airway by using basic life support techniques. Oropharyngeal and nasopharyngeal airway adjuncts can help maintain the airway.

Supraglottic airways devices (SADs) (including LMA). Although bag-mask ventilation (BMV) remains the recommended first line method for achieving airway control and ventilation in children, the SADs represent a range of acceptable airway devices that may assist providers trained in their use.[546,547]

Tracheal intubation. Tracheal intubation is the most secure and effective way to establish and maintain the airway. The oral route for tracheal intubation is preferable during resuscitation. In the conscious child, the judicious use of anaesthetics, sedatives and neuromuscular blocking drugs is essential to avoid multiple intubation attempts or intubation failure.[548,549] Only skilled and experienced practitioners should perform intubation.

Clinical examination and capnography should be used to ensure that the tracheal tube remains secured and vital signs should be monitored.[550]

Intubation during cardiopulmonary arrest. The child who is in cardiopulmonary arrest does not require sedation or analgesia to be intubated. Appropriate tracheal tube sizes are shown in Table 1.2.

A correctly sized cuffed tracheal tube is as safe as an uncuffed tube for infants and children (not for neonates) provided attention is paid to its placement, size and cuff inflation pressure.[551–553] 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.[553]

Confirmation of correct tracheal tube placement. Displaced, misplaced or obstructed tubes occur frequently in the intubated child and are associated with an increased risk of death.[554,555] No single technique is 100% reliable for distinguishing oesophageal from tracheal intubation. If the child is in cardiopulmonary arrest and exhaled CO_2 is not detected despite adequate chest compressions, or if there is any doubt as to the tube position, confirm the placement of the tracheal tube by direct laryngoscopy. After correct placement and confirmation, secure the tracheal tube and reassess its position. Maintain the child's head in the neutral position as flexion of the head will drive the tube further into the trachea whereas extension may pull it out of the airway.[556]

Breathing.

Oxygenation. Give oxygen at the highest concentration (i.e. 100%) during initial resuscitation.

Once the child is stabilised and/or there is ROSC, titrate the fraction of inspired oxygen (FiO_2) to achieve normoxaemia, or at least (if arterial blood gas is not available), maintain SpO_2 in the range of 94–98%.[557,558]

Ventilation. Healthcare providers commonly provide excessive ventilation during CPR and this may be harmful. A simple guide to deliver an appropriate tidal volume is to achieve normal chest wall rise. Use a ratio of 15 chest compressions to 2 ventilations and a compression rate of 100–120 min^{-1}. Once the airway is protected by tracheal intubation, continue positive pressure ventilation at 10 breaths min^{-1} without interrupting the chest compressions. Take care to ensure that lung inflation is adequate during chest compressions. Once ROSC has been achieved, provide normal ventilation (rate/volume) based on the child's age, and by monitoring end-tidal CO_2 and blood gas values, to achieve a normal arterial carbon dioxide tension ($PaCO_2$) and arterial oxygen levels. Both hypocarbia and hypercarbia are associated with poor outcomes following cardiac arrest.[559] This means that the child with ROSC should usually be ventilated at 12–24 breaths min^{-1}, according to their age normal values.

Bag mask ventilation. Bag mask ventilation (BMV) is effective and safe for a child requiring assisted ventilation for a short period.[560,561] Assess the effectiveness of BMV by observing adequate chest rise, monitoring heart rate and auscultating for breath sounds, and measuring SpO_2. Any healthcare provider with a responsibility for treating children must be able to deliver BMV effectively.

Monitoring of breathing and ventilation.

End-tidal CO_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 a child.[562–565] 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. The absence of exhaled CO_2 during cardiopulmonary arrest does not guarantee tube misplacement since a low or absent $EtCO_2$ may reflect low or absent pulmonary blood flow.[200,566–568] Although an $EtCO_2$ higher than 2 kPa (15 mmHg) may be an indicator of adequate resuscitation, current evidence does not support the use of a threshold $EtCO_2$ value as an indicator for the quality of CPR or for the discontinuation of resuscitation.[512]

Peripheral pulse oximetry. Clinical evaluation to determine the degree of oxygenation in a child is unreliable; therefore, monitor the child's peripheral oxygen saturation continuously by pulse oximetry. Pulse oximetry can be unreliable under certain conditions, e.g. if the child is in circulatory failure, in cardiopulmonary arrest or has poor peripheral perfusion.

Circulation.

Vascular access. Vascular access is essential to enable drugs and fluids to be given, and blood samples obtained. Venous access can be difficult to establish during resuscitation of an infant or child. In critically ill children, if attempts at establishing intravenous (IV) access are unsuccessful after one minute, insert an intra-osseous (IO) needle.[208,569]

Intraosseous access. Intraosseous (IO) access is a rapid, safe, and effective route to give drugs, fluids and blood products.[570,571] The onset of action and time to achieve adequate plasma drug concentrations are similar to that achieved via the central venous route.[212,572–574] Bone marrow samples can be used to cross match for blood type or group for chemical analysis[575–577] and for blood gas measurement (the values may be comparable to central venous blood gases if no drug has been injected in the cavity).[212] Inject large boluses of fluid using manual pressure or a pressure bag.[578] Maintain IO access until definitive IV access has been established.

Intravenous access and other routes. Central venous lines provide more secure long-term access but, compared with IO or peripheral IV access, offer no advantages during resuscitation.[209] The tracheal route for the administration of drugs is no longer recommended.[579]

Fluids and drugs. Isotonic crystalloids are recommended as the initial resuscitation fluid for infants and children with any type of circulatory failure.[580,581] If there are signs that the systemic perfusion is inadequate, give a bolus of 20 ml kg^{-1} of an isotonic crystalloid even if the systemic blood pressure is normal. Following each bolus, re-assess the child's clinical state, using the ABCDE system of assessment, to decide whether a further bolus or other treatment is required. In some children, early inotropic or vasopressor support may be needed.[582,583] There is growing evidence to prefer the use of balanced crystalloids as these induce less hyperchloraemic acidosis.[584–587]

In life-threatening hypovolaemic shock, as may be seen in rapid blood loss in trauma, limiting the use of crystalloids in favour of a regime of massive blood transfusion may be required. There are varying regimes of combining plasma, platelets and other blood products in delivering massive blood transfusion,[588,589] so the regime used should be according to local protocols.

Adrenaline. Adrenaline (epinephrine) plays a central role in the cardiac arrest treatment algorithms for non-shockable and shockable rhythms. For cardiopulmonary resuscitation, 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. The use of single higher doses of adrenaline (above 10 μg kg^{-1}) is not recommended because it does not improve survival or neurological outcome after cardiopulmonary arrest.[590–594]

Amiodarone for shock-resistant paediatric VF/pulseless VT. Amiodarone can be used to treat paediatric shock-resistant VF/pulseless VT (pVT). It is given after the third shock as a 5 mg kg^{-1} bolus (and can be repeated following the fifth shock). When treating other cardiac rhythm disturbances, amiodarone must be injected slowly (over 10–20 min) with systemic blood pressure and ECG monitoring to avoid causing hypotension.[595] This side effect is less common with the aqueous solution.[257]

Atropine. Atropine is recommended only for bradycardia caused by increased vagal tone or cholinergic drug toxicity.[596–598] The commonly used dose is 20 μg kg^{-1}. In bradycardia with poor perfusion unresponsive to ventilation and oxygenation, the first line drug is adrenaline, not atropine.

Calcium. Calcium is essential for myocardial function,[599] but the routine use of calcium does not improve the outcome from cardiopulmonary arrest.[600,601] Calcium is indicated in the presence of hypocalcaemia, calcium channel blocker overdose, hypermagnesaemia and hyperkalaemia.[602]

Glucose. Data from neonates, children and adults indicate that both hyper- and hypo-glycaemia are associated with poor outcome after cardiopulmonary arrest,[603] but it is uncertain if this is causative or merely an association. Check blood or plasma glucose concentration and monitor closely in any ill or injured child, including after cardiac arrest. Do not give glucose-containing fluids during CPR unless hypoglycaemia is present.[604] Avoid hyper- and hypoglycaemia following ROSC.[605]

Magnesium. There is no evidence for giving magnesium routinely during cardiopulmonary arrest.[606,607] Magnesium treatment is indicated in the child with documented hypomagnesaemia or with torsade de pointes VT (50 mg kg^{-1}), regardless of the cause.[608]

Sodium bicarbonate. There is no evidence for giving sodium bicarbonate routinely during cardiopulmonary arrest.[609–611] Sodium bicarbonate may be considered for the child with prolonged cardiopulmonary arrest and/or severe metabolic acidosis. Sodium bicarbonate may also be considered in case of

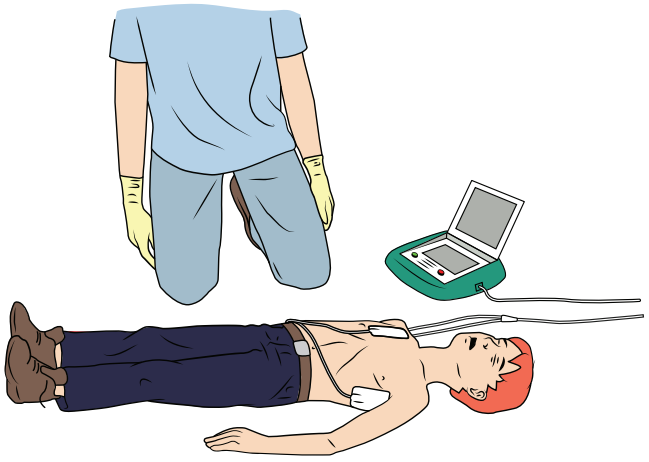


Fig. 1.24. Paddle positions for defibrillation – child.

haemodynamic instability and co-existing hyperkalaemia, or in the management of tricyclic antidepressant drug overdose.

Vasopressin–terlipressin. There is currently insufficient evidence to support or refute the use of vasopressin or terlipressin as an alternative to, or in combination with, adrenaline in any cardiac arrest rhythm in adults or children.[246,248,249,612–616]

Defibrillators

Manual defibrillators capable of delivering the full energy requirements from neonates upwards must be available within hospitals and in other healthcare facilities caring for children at risk of cardiopulmonary arrest. Automated external defibrillators (AEDs) are pre-set for all variables including the energy dose.

Pad/paddle size for defibrillation

Select the largest possible available paddles to provide good contact with the chest wall. The ideal size is unknown but there should be good separation between the pads.[617,618] Recommended sizes are 4.5 cm diameter for infants and children weighing <10 kg, and 8–12 cm diameter for children weighing >10 kg (older than one year). Self-adhesive pads facilitate continuous good quality CPR.

Position of the paddles

Apply the paddles firmly to the bare chest in the antero-lateral position, one paddle placed below the right clavicle and the other in the left axilla (Fig. 1.24). If the paddles are too large and there is a danger of charge arcing across the paddles, one should be placed on the upper back, below the left scapula and the other on the front, to the left of the sternum.

Energy dose in children. In Europe we continue to recommend a dose of 4 J kg^{-1} for initial and subsequent defibrillation. Doses higher than 4 J kg^{-1} (as much as 9 J kg^{-1}) have defibrillated children effectively with negligible side effects.[619,620]

If no manual defibrillator is available, use an AED that can recognise paediatric shockable rhythms.[621–623] The AED should be equipped with a dose attenuator that decreases the delivered energy to a value more suitable for children aged 1–8 years (50–75 J).[624,625] If such an AED in not available, use a standard adult AED and the pre-set adult energy levels. For children older than 8 years, use a standard AED with standard paddles. Experience with the use of AEDs (preferably with dose attenuator) in children younger than 1 year is limited; their use is acceptable if no other option is available.

Advanced management of cardiopulmonary arrest

The paediatric advanced life support algorithm is shown in Fig. 1.25. More detailed algorithms for the treatment of non-shockable (Fig. 1.26) and shockable rhythms (Fig. 1.27) also shown.

Cardiac monitoring. Position the cardiac monitor leads or self-adhesive pads as soon as possible to enable differentiation between a shockable and a non-shockable cardiac rhythm. Non-shockable rhythms are pulseless electrical activity (PEA), bradycardia (<60 min^{-1} with no signs of circulation) and asystole. PEA and bradycardia often have wide QRS complexes. Shockable rhythms are pVT and VF. These rhythms are more likely after sudden collapse in children with heart disease or in adolescents.

Non-shockable rhythms. Most cardiopulmonary arrests in children and adolescents are of respiratory origin.[626] A period of immediate CPR is therefore mandatory in this age group before searching for an AED or manual defibrillator, as its immediate availability will not improve the outcome of a respiratory arrest. The most common ECG patterns in infants, children and adolescents with cardiopulmonary arrest are asystole and PEA. PEA is characterised by electrical activity on the ECG, and absent pulses. It commonly follows a period of hypoxia or myocardial ischaemia, but occasionally can have a reversible cause (i.e., one of the 4 Hs and 4 Ts) that led to a sudden impairment of cardiac output.

Shockable rhythms. Primary VF occurs in 3.8–19% of cardiopulmonary arrests in children, the incidence of pVT/VF increases as the age increases.[123,340,627–634] The primary determinant of survival from VT/pVT cardiopulmonary arrest is the time to defibrillation. Pre-hospital defibrillation within the first 3 min of witnessed adult VF arrest results in >50% survival. However, the success of defibrillation decreases dramatically the longer the time until defibrillation: for every minute delay in defibrillation (without any CPR), survival decreases by 7–10%. Secondary VF is present at some point in up to 27% of in-hospital resuscitation events. It has a much poorer prognosis than primary VF.[635]

Extracorporeal life support. Extracorporeal life support should be considered for children with cardiac arrest refractory to conventional CPR with a potentially reversible cause, if the arrest occurs where expertise, resources and system are available to rapidly initiate extracorporeal life support (ECLS).

Arrhythmias

Unstable arrhythmias

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

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

Paediatric Advanced Life Support

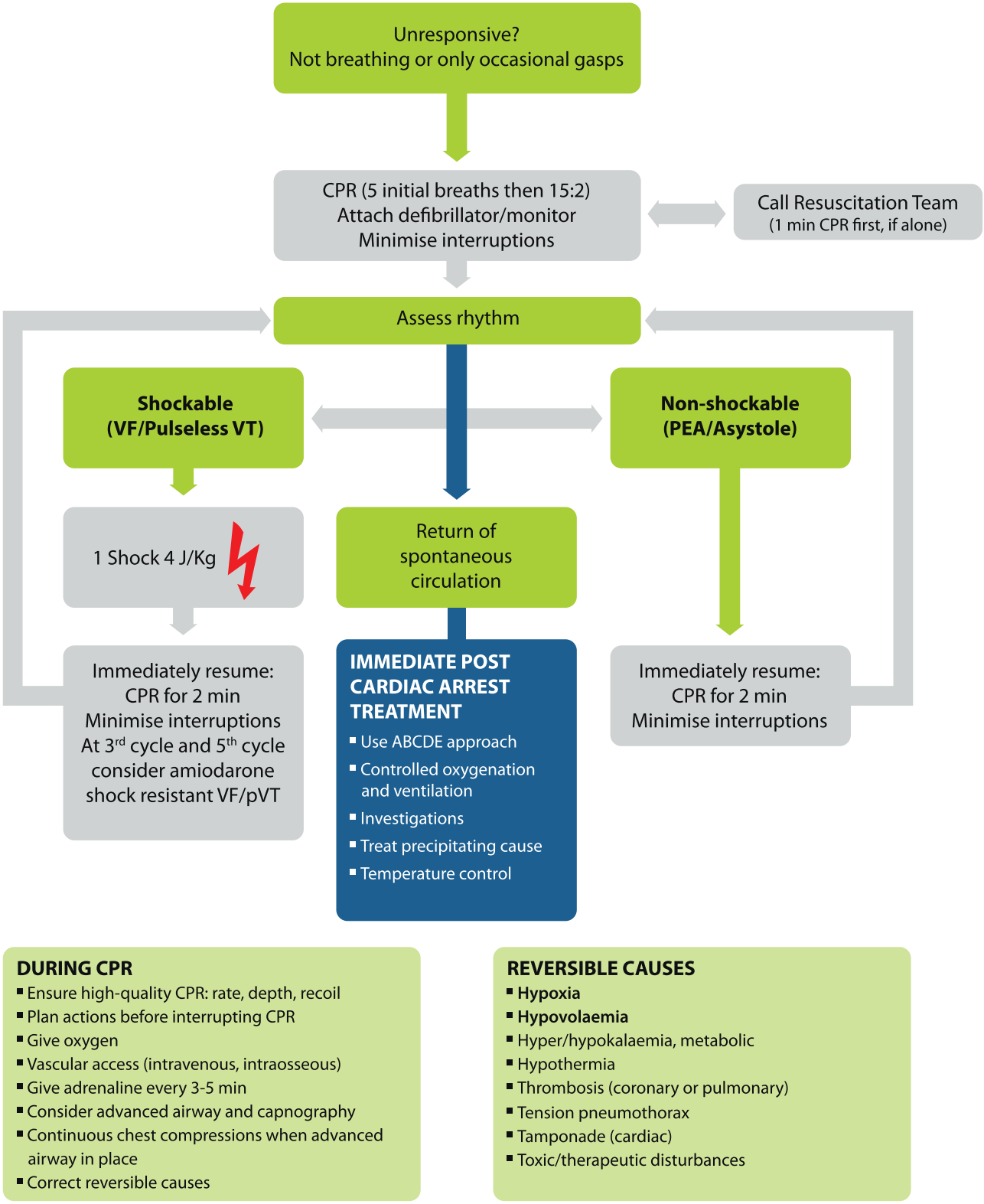


Fig. 1.25. Paediatric advanced life support algorithm.

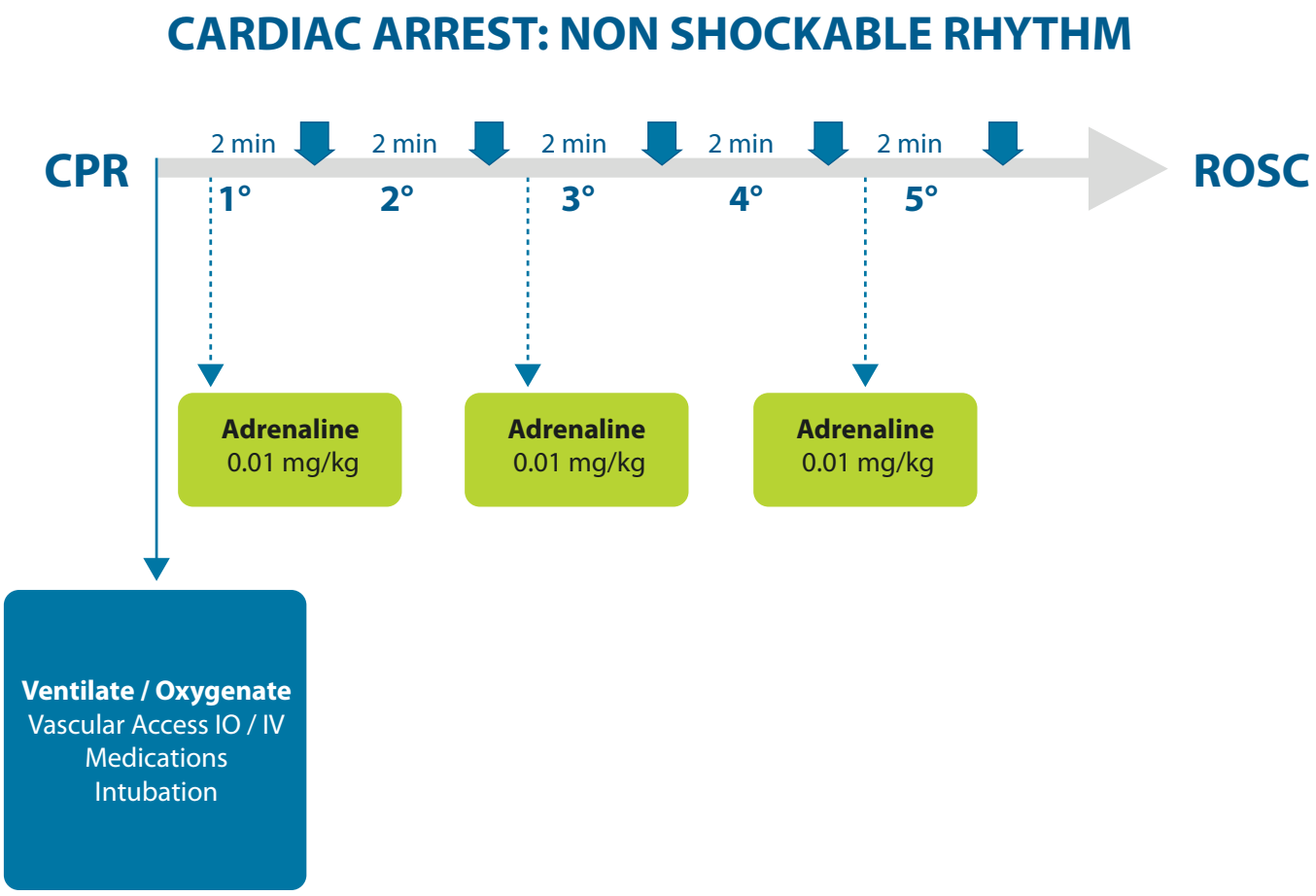


Fig. 1.26. Paediatric algorithm for non-shockable rhythm.

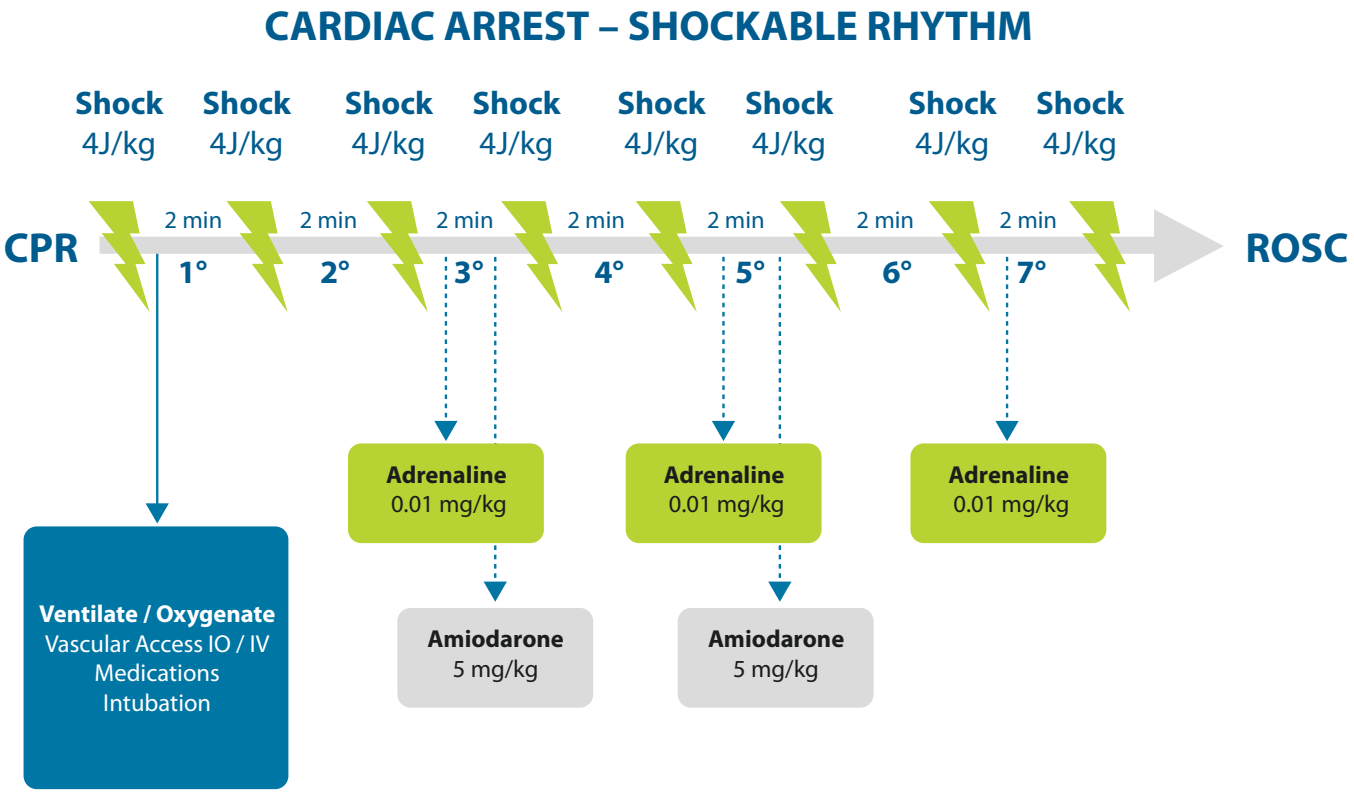


Fig. 1.27. Paediatric algorithm for shockable rhythm.

Bradycardia

Bradycardia is caused commonly by hypoxia, acidosis and/or severe hypotension; it may progress to cardiopulmonary arrest. Give 100% oxygen, and positive pressure ventilation if required, to any child presenting with bradyarrhythmia and circulatory failure. If a child with decompensated circulatory failure has a heart rate <60 beats min^{-1}, and they do not respond rapidly to ventilation with oxygen, start chest compressions and give adrenaline.

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.[636]

Tachycardia

Narrow complex tachycardia. If supraventricular tachycardia (SVT) is the likely rhythm, vagal manoeuvres (Valsalva or diving reflex) may be used in haemodynamically stable children. They can also be used in haemodynamically unstable children, but only if they do not delay chemical or electrical cardioversion.

Adenosine is usually effective in converting SVT into sinus rhythm. It is given by rapid, intravenous injection as close as practicable to the heart, and followed immediately by a bolus of normal saline. If the child has signs of decompensated shock with depressed conscious level, omit vagal manoeuvres and adenosine and attempt electrical cardioversion immediately.

Electrical cardioversion (synchronised with R wave) is also indicated when vascular access is not available, or when adenosine has failed to convert the rhythm. The first energy dose for electrical cardioversion of SVT is 1 J kg^{-1} and the second dose is 2 J kg^{-1}. If unsuccessful, give amiodarone or procainamide under guidance from a paediatric cardiologist or intensivist before the third attempt. Verapamil may be considered as an alternative therapy in older children but should not be routinely used in infants.

Wide complex tachycardia. In children, wide-QRS complex tachycardia is uncommon and more likely to be supraventricular than ventricular in origin.[637] Nevertheless, in haemodynamically unstable children, it must be considered to be VT until proven otherwise. Ventricular tachycardia occurs most often in the child with underlying heart disease (e.g., after cardiac surgery, cardiomyopathy, myocarditis, electrolyte disorders, prolonged QT interval, central intracardiac catheter). Synchronised cardioversion is the treatment of choice for unstable VT with signs of life. Consider anti-arrhythmic therapy if a second cardioversion attempt is unsuccessful or if VT recurs.

Stable arrhythmias

Whilst maintaining the child's airway, breathing and circulation, contact an expert before initiating therapy. Depending on the child's clinical history, presentation and ECG diagnosis, a child with stable, wide-QRS complex tachycardia may be treated for SVT and be given vagal manoeuvres or adenosine.

Special circumstances

Life support for blunt or penetrating trauma

Cardiac arrest from major (blunt or penetrating) trauma is associated with a very high mortality.[292,638–643] Consider the 4Ts and 4Hs as potentially reversible causes. There is little evidence to support any additional specific interventions that are different from the routine management of cardiac arrest; however, the use of resuscitative thoracotomy may be considered in children with penetrating injuries.[644,645]

Extracorporeal membrane oxygenation (ECMO)

For infants and children with a cardiac diagnosis and an in-hospital arrest, ECMO should be considered as a useful rescue strategy if expertise, adequate resources and systems are equally available. There is insufficient evidence to suggest for or against the use of ECMO in non-cardiac arrest or for children with myocarditis or cardiomyopathy who are not in arrest.[512]

Pulmonary hypertension

There is an increased risk of cardiac arrest in children with pulmonary hypertension.[646,647] 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.[648]

Post resuscitation care

Post cardiac arrest care must be a multidisciplinary activity and include all the treatments needed for complete neurological recovery.

Myocardial dysfunction

Myocardial dysfunction is common after cardiopulmonary resuscitation.[366,649–652] Parenteral fluids and vasoactive drugs (adrenaline, dobutamine, dopamine and noradrenaline) may improve the child's post-arrest haemodynamic status and should be titrated to maintain a systolic blood pressure of at least >5th centile for age.[512]

Goals for oxygenation and ventilation

Aim for a normal PaO_2 range (normoxaemia) post-ROSC once a patient is stabilised.[559,653–655] There is insufficient paediatric evidence to suggest a specific $PaCO_2$ target, however, $PaCO_2$ should be measured post-ROSC and adjusted according to patient characteristics and needs.[397,512,559,656] It is sensible to aim in general for normocapnia, although this decision might be in part influenced by context and disease.

Temperature control and management post ROSC

Mild hypothermia has an acceptable safety profile in adults[446,450] and neonates.[657] Recently the THAPCA out of hospital study showed that both hypothermia (32–34 °C) and controlled normothermia (36–37.5 °C) could be used in children.[658] The study did not show a significant difference for the primary outcome (neurologic status at one year) with either approach. After ROSC, a strict control of the temperature must be maintained to avoid hyperthermia (>37.5 °C) and severe hypothermia (<32 °C).[512]

Glucose control

Both hyper- and hypoglycaemia may impair outcome of critically ill adults and children and should be avoided,[659–661] but tight glucose control may also be harmful.[662] Monitor blood glucose and avoid hypoglycaemia and hyperglycaemia.[366,663,664]

Prognosis of cardiopulmonary arrest

Although several factors are associated with outcome after cardiopulmonary arrest and resuscitation there are no simple guidelines to determine when resuscitative efforts become futile.[512,656] The relevant considerations in the decision to continue the resuscitation include the duration of CPR, cause of arrest, pre-existing medical conditions, age, site of arrest, whether the arrest was witnessed,[519,665] the duration of untreated cardiopulmonary arrest ('no flow' time) the presence of a shockable rhythm as the first or subsequent rhythm, and associated special circumstances (e.g., icy water drowning[666,667] exposure to toxic drugs). The role

of the EEG as a prognostic factor is still unclear. Guidance on the termination of resuscitation attempts is discussed in the chapter on ethics in resuscitation and end-of-life decisions.[10]

Parental presence

In some Western societies, the majority of parents want to be present during the resuscitation of their child. Families who are present at their child's death show better adjustment and undergo a better grieving process.[668] Evidence about parental presence during resuscitation comes from selected countries and can probably not be generalised to all of Europe, where there may be different socio-cultural and ethical considerations.[669,670]

Resuscitation and support of transition of babies at birth

The guidelines that follow do not define the only way that resuscitation at birth should be achieved; they do, however, represent a widely accepted view of how resuscitation at birth can be carried out both safely and effectively.

Preparation

A minority of infants require resuscitation at birth, but a few more have problems with this perinatal transition, which, if no support is given, might subsequently result in a need for resuscitation. Of those needing any help, the overwhelming majority will require only assisted lung aeration. A tiny minority may need a brief period of chest compressions in addition to lung aeration.[671–673] In deliveries with a known increased risk of problems, specially trained personnel should be present with at least one person experienced in tracheal intubation. Each institution should have a protocol in place for rapidly mobilising a team with competent resuscitation skills for any birth.

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 assessment, stabilisation or resuscitation at birth. 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

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 and subsequent ventilation of an appropriate size for the newborn, warm dry towels and blankets, a sterile instrument for cutting and clamping the umbilical cord and clean gloves for the attendant and assistants.

Timing of clamping the umbilical cord

A systematic review on delayed cord clamping and cord milking in preterm infants found improved stability in the immediate postnatal period, including higher mean blood pressure and haemoglobin on admission, compared to controls.[674] Delaying umbilical cord clamping for at least one minute is recommended for newborn infants not requiring resuscitation. A similar delay should be applied to preterm babies not requiring immediate resuscitation after birth. Until more evidence is available, infants who are not breathing or crying may require the umbilical cord to be clamped, so that resuscitation measures can commence promptly.

Temperature control

Naked, wet, newborn babies cannot maintain their body temperature in a room that feels comfortably warm for adults. The association between hypothermia and mortality has been known for more than a century,[675] and the admission temperature of newborn non-asphyxiated infants is a strong predictor of mortality at all gestations and in all settings.[676] Preterm infants are especially vulnerable. Maintain the temperature of newly born non-asphyxiated infants at between 36.5 °C and 37.5 °C after birth. Whilst maintenance of a baby's temperature is important, this should be monitored in order to avoid hyperthermia (>38.0 °C).

Initial assessment

The Apgar score was not designed to be assembled and ascribed in order to then identify babies in need of resuscitation.[677,678] However, individual components of the score, namely respiratory rate, heart rate and tone, if assessed rapidly, can identify babies needing resuscitation.[677] 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

Immediately after birth the heart rate is assessed to evaluate the condition of the baby and subsequently is the most sensitive indicator of a successful response to interventions. Heart rate is initially most rapidly and accurately assessed by listening to the apex beat with a stethoscope[679] or by using an electrocardiograph.[680–682] Feeling the pulse in the base of the umbilical cord is often effective but can be misleading, cord pulsation is only reliable if found to be more than 100 beats per minute (bpm)[679] and clinical assessment may underestimate the heart rate.[679,683,684] For babies requiring resuscitation and/or continued respiratory support, a modern pulse oximeter can give an accurate heart rate.[681]

Colour

Colour is a poor means of judging oxygenation,[685] 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. If a baby appears blue check preductal 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}

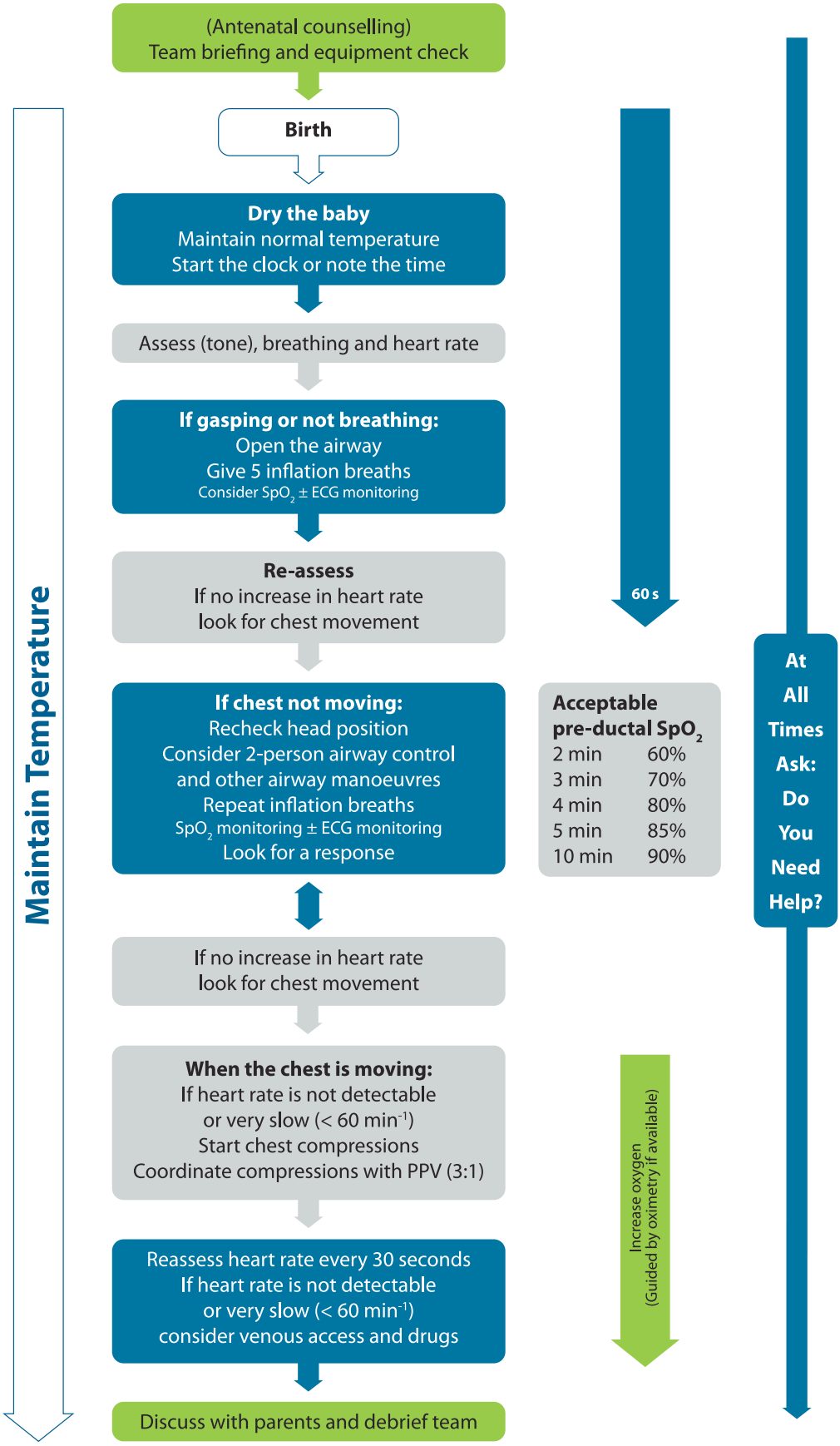


Fig. 1.28. Newborn life support algorithm (SpO_2: transcutaneous pulse oximetry, ECG: electrocardiograph, PPV: positive pressure ventilation).

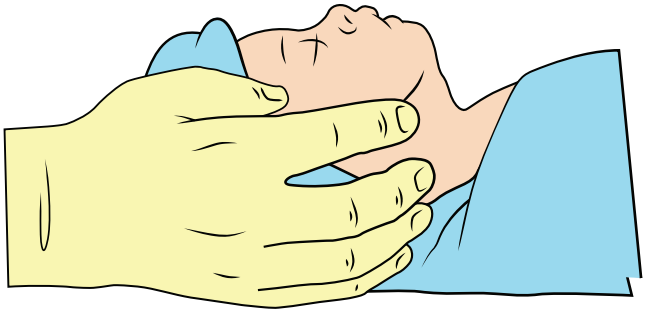


Fig. 1.29. Newborn with head in neutral position.

There is no need for immediate clamping of the cord. This baby requires no intervention other than drying, wrapping in a warm towel and, where appropriate, handing to the mother.

2. Breathing inadequately or apnoeic, normal or reduced tone, heart rate less than 100 min^{-1}

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

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.

   Preterm babies may be breathing and showing signs of respiratory distress in which case they should be supported initially with CPAP.

Newborn life support

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

Airway

Place the baby on his or her back with the head in a neutral position (Fig. 1.29). A 2 cm thickness of the blanket or towel placed under the baby's shoulder may be helpful in maintaining proper head position. In floppy babies application of jaw thrust or the use of an appropriately sized oropharyngeal airway may be essential in opening the airway. The supine position for airway management is traditional but side-lying has also been used for assessment and routine delivery room management of term newborns.[686] There is no need to remove lung fluid from the oropharynx routinely.[687] Suction is needed only if the airway is obstructed.

Meconium

Lightly meconium stained liquor is common and does in general not give rise to much difficulty with transition. The much less common finding of very thick meconium stained liquor at birth is an indicator of perinatal distress and should alert to the potential need for resuscitation. Intrapartum suctioning and routine intubation and suctioning of vigorous infants born through meconium stained liquor are not recommended. The presence of thick, viscous meconium in a non-vigorous baby is the only indication for initially considering visualising the oropharynx and suctioning material, which might obstruct the airway. Tracheal intubation should not be routine in the presence of meconium and should only be performed for suspected tracheal obstruction.[688–692] The emphasis

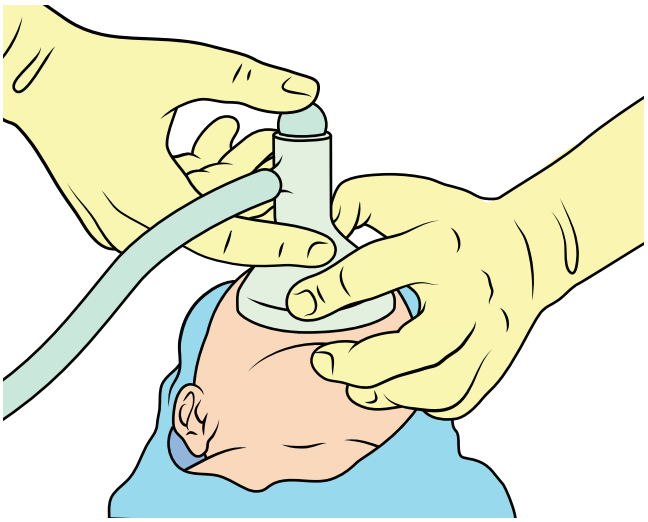


Fig. 1.30. Mask ventilation of newborn.

should be on initiating ventilation within the first minute of life in non-breathing or ineffectively breathing infants and this should not be delayed.

Initial breaths and assisted ventilation

After initial steps at birth, if breathing efforts are absent or inadequate, lung aeration is the priority and must not be delayed (Figs. 1.28 and 1.30). In term babies, respiratory support should start with air.[693] The primary measure of adequate initial lung inflation is a prompt improvement in heart rate. If the heart rate is not improving assess the chest wall movement. For the first five positive pressure inflations maintain the initial inflation pressure for 2–3 s. This will usually help lung expansion.[694,695] Most babies needing respiratory support 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. Without adequate lung aeration, chest compressions will be ineffective; therefore, confirm lung aeration and ventilation 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.

Air/oxygen

Term babies. In term infants receiving respiratory support at birth with positive pressure ventilation (PPV), it is best to begin with air (21%) as opposed to 100% oxygen. If, despite effective ventilation, there is no increase in heart rate or oxygenation (guided by oximetry wherever possible) remains unacceptable, use a higher concentration of oxygen to achieve an adequate preductal oxygen saturation.[696,697] High concentrations of oxygen are associated with an increased mortality and delay in time of onset of spontaneous breathing,[698] therefore, if increased oxygen concentrations are used they should be weaned as soon as possible.[693,699]

Preterm babies. Resuscitation of preterm infants less than 35 weeks gestation at birth should be initiated in air or low concentration oxygen (21–30%).[6,693,700,701] Titrate the administered oxygen concentration to achieve acceptable pre-ductal oxygen saturations

Table 1.3
Oral tracheal tube lengths by gestation.

Gestation (weeks)	ETT at lips (cm)
23–24	5.5
25–26	6.0
27–29	6.5
30–32	7.0
33–34	7.5
35–37	8.0
38–40	8.5
41–43	9.0

approximating to the 25th percentile in healthy term babies immediately after birth.[696,697]

Pulse oximetry

Modern pulse oximetry, using neonatal probes, provides reliable readings of heart rate and transcutaneous oxygen saturation within 1–2 min of birth.[702,703] Uncompromised babies born at term at sea level have SpO_2 ~60% during labour,[704] which increases to >90% by 10 min.[696] The 25th percentile is approximately 40% at birth and increases to ~80% at 10 min.[697] Use pulse oximetry to avoid excessive use of oxygen (Fig. 1.28). Transcutaneous oxygen saturations above the acceptable levels should prompt weaning of any supplemental oxygen.

Positive end expiratory pressure

All term and preterm babies who remain apnoeic despite initial steps must receive positive pressure ventilation after initial lung inflation. Provide positive end expiratory pressure (PEEP) of ~5 cm H_2O for preterm newborn babies receiving PPV.[676]

Assisted ventilation devices

Effective ventilation can be achieved with a self-inflating bag or with a T-piece mechanical device designed to regulate pressure.[705,706] However, self-inflating bags are the only devices, which can be used in the absence of compressed gas but cannot deliver continuous positive airway pressure (CPAP) and may not be able to achieve PEEP even with a PEEP valve in place.[707]

Laryngeal mask airway

The LMA may be considered as an alternative to a facemask or to tracheal intubation for positive pressure ventilation among newborns weighing more than 2000 g or delivered ≥34 weeks gestation.[708,709] The laryngeal mask airway has not been evaluated in the setting of meconium stained fluid, during chest compressions, or for the administration of emergency intra-tracheal medications.

Tracheal tube placement

Tracheal intubation may be considered at several points during neonatal resuscitation:

- When suctioning the lower airways to remove a presumed tracheal blockage
- When, after correction of mask technique and/or the baby's head position, bag-mask ventilation is ineffective or prolonged
- When chest compressions are performed
- Special circumstances (e.g. congenital diaphragmatic hernia or to give tracheal surfactant)

The use and timing of tracheal intubation will depend on the skill and experience of the available resuscitators. Appropriate tube lengths based on gestation are shown in Table 1.3.[710] It should be recognised that vocal cord guides, as marked on tracheal tubes by different manufacturers to aid correct placement, vary considerably.[711]

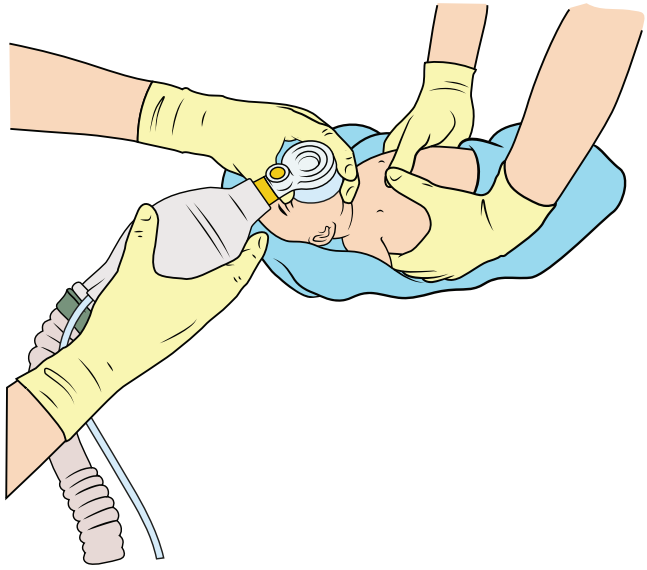


Fig. 1.31. Ventilation and chest compression of newborn.

Tracheal tube placement must be assessed visually during intubation, and positioning confirmed. Following tracheal intubation and intermittent positive-pressure, a prompt increase in heart rate is a good indication that the tube is in the tracheobronchial tree.[712] Exhaled CO_2 detection is effective for confirmation of tracheal tube placement in infants, including VLBW infants[713–716] and neonatal studies suggest that it confirms tracheal intubation in neonates with a cardiac output more rapidly and more accurately than clinical assessment alone.[715–717] Failure to detect exhaled CO_2 strongly suggests oesophageal intubation[713,715] but false negative readings have been reported during cardiac arrest [713] and in VLBW infants.[718] Detection of exhaled carbon dioxide in addition to clinical assessment is recommended as the most reliable method to confirm tracheal placement in neonates with spontaneous circulation.

Continuous positive airways pressure

Initial respiratory support of all spontaneously breathing preterm infants with respiratory distress may be provided by continuous positive airways pressure (CPAP), rather than intubation.[719–721] There are few data to guide the appropriate use of CPAP in term infants at birth and further clinical studies are required.[722,723]

Circulatory support

Give chest compressions if the heart rate is less than 60 beats min^{-1} despite adequate ventilation. As ventilation is the most effective and important intervention in newborn resuscitation, and may be compromised by compressions, it is vital to ensure that effective ventilation is occurring before commencing chest compressions.

The most effective technique for providing chest compressions is with two thumbs over the lower third of the sternum with the fingers encircling the torso and supporting the back (Fig. 1.31).[724] This technique generates higher blood pressures and coronary artery perfusion with less fatigue than the previously used two-finger technique.[725–728] 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.[729–732]

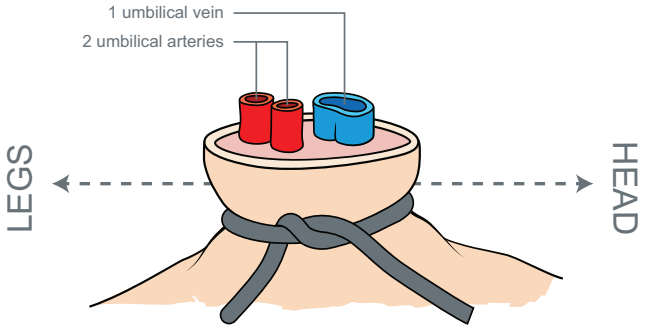


Fig. 1.32. Newborn umbilical cord showing the arteries and veins.

Use a 3:1 compression to ventilation ratio, aiming to achieve approximately 120 events per minute, i.e. approximately 90 compressions and 30 ventilations.[733–738] Co-ordinate compressions and ventilations to avoid simultaneous delivery.[739] A 3:1 compression to ventilation ratio is used for resuscitation at birth where compromise of gas exchange is nearly always the primary cause of cardiovascular collapse, but rescuers may consider using higher ratios (e.g., 15:2) if the arrest is believed to be of cardiac origin. When delivering chest compressions it would appear sensible to increase the supplementary oxygen concentration towards 100%. 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 a centrally positioned umbilical venous catheter (Fig. 1.32).

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 an initial dose of 10 micrograms kg^{-1} (0.1 ml kg^{-1} of 1:10,000 adrenaline) intravenously as soon as possible with subsequent intravenous doses of 10–30 micrograms kg^{-1} (0.1–0.3 ml kg^{-1} of 1:10,000 adrenaline) if required.[6,693,700] Do not use the tracheal route.

Bicarbonate. There are insufficient data to recommend routine use of bicarbonate in resuscitation of the newly born. If it is used during prolonged arrests unresponsive to other therapy, give a dose of 1–2 mmol kg^{-1} 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.[740] This is a rare event. In the absence of suitable blood, give a bolus of isotonic crystalloid of 10 ml kg^{-1} initially. If successful it may need to be repeated to maintain an improvement. When resuscitating preterm infants volume is rarely needed and has been associated with intraventricular and pulmonary haemorrhages when large volumes are infused rapidly.

Withholding or discontinuing resuscitation

Mortality and morbidity for newborns varies according to region and to availability of resources.[741] Opinions vary amongst providers, parents and societies about the balance of benefits and disadvantages of using aggressive therapies in such babies.[742,743]

Discontinuing resuscitation

Local and national committees will define recommendations for stopping resuscitation. If the heart rate of a newly born baby is not detectable and remains undetectable for 10 min, it may be appropriate to consider stopping resuscitation. The decision should be individualised. 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 and firm guidance cannot be given.

Withholding resuscitation

It is possible to identify conditions associated with high mortality and poor outcome, where withholding resuscitation may be considered reasonable, particularly when there has been the opportunity for discussion with parents.[744–746] There is no evidence to support the prospective use of any particular delivery room prognostic score presently described, over gestational age assessment alone, in preterm infants <25 weeks gestation. When withdrawing or withholding resuscitation, care should be focused on the comfort and dignity of the baby and family.

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 the routine local plan and, if possible, hand the baby to the mother at the earliest opportunity. If resuscitation is required inform the parents of the procedures undertaken and why they were required. Parents' wishes to be present during resuscitation should be supported where possible.[747]

Post-resuscitation care

Babies who have required resuscitation may later deteriorate. Once adequate ventilation and circulation are established, the infant should be maintained in or transferred to an environment in which close monitoring and anticipatory care can be provided.

Glucose

The range of blood glucose concentration that is associated with the least brain injury following asphyxia and resuscitation cannot be defined based on available evidence. Infants who require significant resuscitation should be monitored and treated to maintain glucose in the normal range.

Induced hypothermia

Newly born infants born at term or near-term with evolving moderate to severe hypoxic – ischaemic encephalopathy should, where possible, be offered therapeutic hypothermia.[748,749] Whole body cooling and selective head cooling are both appropriate strategies. There is no evidence in human newborns that cooling is effective if started more than 6 h after birth.

Prognostic tools

Although widely used in clinical practice, for research purposes and as a prognostic tool,[750] the applicability of the APGAR score has been questioned due to large inter- and intra-observer variations. These are partly explained by a lack of agreement on how to score infants receiving medical interventions or being born

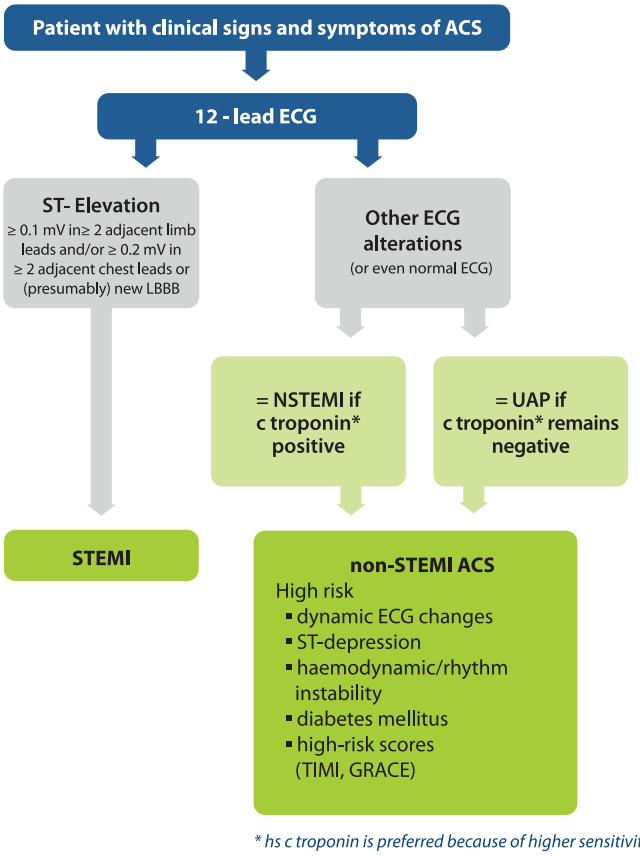


Fig. 1.33. Definitions of acute coronary syndromes (ACS); ECG, electrocardiogram; LBBB, left bundle branch block; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation acute myocardial infarction; c troponin, cardiac troponin; UAP, unstable angina pectoris; TIMI, thrombolysis in acute myocardial infarction; GRACE, global registry of acute coronary events.

preterm. Therefore a development of the score was recommended as follows: all parameters are scored according to the conditions regardless of the interventions needed to achieve the condition and considering whether being appropriate for gestational age. In addition, the interventions needed to achieve the condition have to be scored as well. This Combined-Apgar has been shown to predict outcome in preterm and term infants better than the conventional score.[751,752]

Briefing/debriefing

Prior to resuscitation it is important to discuss the responsibilities of each member of the team. After the management in the delivery room a team debrief of the event using positive and constructive critique techniques should be conducted and personal bereavement counselling offered to those with a particular need.

Initial management of acute coronary syndromes

The term acute coronary syndrome (ACS) encompasses three different entities of the acute manifestation of coronary heart disease (Fig. 1.33): ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction 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.[753] Electrocardiographic (ECG) characteristics (absence or presence of ST elevation) differentiate STEMI from non-STEMI ACS. The latter may present with ST segment depression, nonspecific ST segment wave abnormalities, or even a normal ECG. In the absence of ST elevation, an increase in the plasma concentration of cardiac biomarkers, particularly troponin T or I as the most specific markers of myocardial cell necrosis, indicates non-STEMI.

Acute coronary syndromes are the commonest cause of malignant arrhythmias leading to sudden cardiac death. The therapeutic goals are to treat acute life-threatening conditions, such as ventricular fibrillation (VF) or extreme bradycardia, and to preserve left ventricular function and prevent heart failure by minimising the extent of myocardial damage. The current guidelines address the first hours after onset of symptoms. Out-of-hospital treatment and initial therapy in the emergency department (ED) may vary according to local capabilities, resources and regulations. These recommendations are consistent with the guidelines for the diagnosis and treatment of ACS with and without ST elevation published by the European Society of Cardiology and the American College of Cardiology/American Heart Association.[424,754]

Diagnosis and risk stratification in acute coronary syndromes

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. None of these signs and symptoms of ACS can be used alone for the diagnosis of ACS. A reduction in chest pain after nitroglycerin administration can be misleading and is not recommended as a diagnostic manoeuvre.[755] Symptoms may be more intense and last longer in patients with STEMI but are not reliable for discriminating between STEMI and non-STEMI-ACS.[424,756–758]

12-lead ECG

When an ACS is suspected, a 12-lead-ECG should be acquired and interpreted as soon as possible after first patient contact, to facilitate early diagnosis and triage.[754,756,758] STEMI is typically diagnosed when, ST-segment elevation, measured at the J point, fulfilling specific voltage criteria in the absence of left ventricular (LV) hypertrophy or left bundle branch block (LBBB).[424] In patients with clinical suspicion of ongoing myocardial ischaemia with new or presumed new LBBB, consider prompt reperfusion therapy, preferably using primary PCI (PPCI). Right precordial leads should be recorded in all patients with inferior STEMI in order to detect right ventricular MI.

Recording of a 12-lead ECG out-of-hospital enables advanced notification to the receiving facility and expedites treatment decisions after hospital arrival. In many studies, using pre-hospital 12-lead ECG, the time from hospital admission to initiating reperfusion therapy is reduced by 10 to 60 minutes. This is associated with shorter times to reperfusion and improved patient survival in both patients with PCI and those undergoing fibrinolysis.[759–767]

Trained EMS personnel (emergency physicians, paramedics and nurses) can identify STEMI with a high specificity and sensitivity comparable to diagnostic accuracy in the hospital.[768,769] It is thus reasonable that paramedics and nurses be trained to diagnose STEMI without direct medical consultation, as long as there is strict concurrent provision of quality assurance. If interpretation of the pre-hospital ECG is not available on-site, computer interpretation[770,771] or field transmission of the ECG is reasonable.[762,770–777]

Biomarkers, rules for early discharge and chest pain observation protocols

In the absence of ST elevation on the ECG, the presence of a suggestive history and elevated concentrations of biomarkers (troponins, CK and CKMB) characterise non-STEMI and distinguish it from STEMI and unstable angina respectively. Highly sensitive

(ultrasensitive) cardiac troponin assays can increase sensitivity and accelerate diagnosis of MI in patients with symptoms suspicious of cardiac ischaemia.[778] Cardiac biomarker testing should be part of the initial evaluation of all patients presenting to the ED with symptoms suggestive of cardiac ischaemia. However, the delay in release of biomarkers from damaged myocardium prevents their use in diagnosing myocardial infarction in the first hours after the onset of symptoms. For patients who present within 6 h of symptom onset, and have an initial negative cardiac troponin, biomarkers should be measured again between 2–3 and up to 6 h later for hs-cTn (12 h with regular troponin).

In patients suspected of an ACS the combination of an unremarkable past history and physical examination with negative initial ECG and biomarkers cannot be used to exclude ACS reliably. Therefore a follow up period is mandatory in order to reach a diagnosis and make therapeutic decisions. At some point after AMI is excluded, the evaluation of the patient should be complemented by either a non-invasive evaluation for anatomical coronary disease or provocative testing for inducible myocardial ischaemia.

Imaging techniques

Effective screening of patients with suspected ACS, but with negative ECG and negative cardiac biomarkers, remains challenging. Non invasive imaging techniques (CT angiography,[779] cardiac magnetic resonance, myocardial perfusion imaging,[780] and echocardiography[781]) have been evaluated as means of screening these low-risk patients and identifying subgroups that can be discharged home safely.[782–785] Echocardiography should be routinely available in the ED, and used in all patients with suspected ACS.

Multi-detector computer tomography coronary angiography (MDCTCA) has been recently proposed in the management acute chest pain in the ED. In a recent meta-analysis, MDCTCA demonstrated a high sensitivity and a low negative likelihood ratio of 0.06, and was effective in ruling out the presence of ACS in low to intermediate risk patients presenting to the ED with acute chest pain.[786] But the inability of anatomical findings to prove the presence of ischaemia, the cancer risk induced by radiation exposure and potential overuse still raise concerns about the relevance of this strategy.

Treatment of acute coronary syndromes – symptoms

Nitrates

Glyceryl trinitrate may be considered if the systolic blood pressure (SBP) is above 90 mmHg and the patient has ongoing ischaemic chest pain (Fig. 1.34). Glyceryl trinitrate can also be useful in the treatment of acute pulmonary congestion. Do not use nitrates in patients with hypotension (SBP $\leq$ 90 mmHg), particularly if combined with bradycardia, and in patients with inferior infarction and suspected right ventricular involvement. Give glyceryl trinitrate 0.4 mg sublingual or equivalent every 5 min up to 3 doses as SBP allows. Begin IV dosing at 10 μg min^{-1} for persistent pain or pulmonary oedema; titrate to desired BP effect.

Analgesia

Morphine is the analgesic of choice for nitrate-refractory pain and also has calming effects on the patient making sedatives unnecessary in most cases. Since morphine is a dilator of venous capacitance vessels, it may have additional benefit in patients with pulmonary congestion. Give morphine in initial doses of 3–5 mg intravenously and repeat every few minutes until the patient is pain-free. Avoid non-steroidal anti-inflammatory drugs (NSAIDs) for analgesia because they have pro-thrombotic effects.[787]

Oxygen

Evidence is accumulating about the questionable role of supplemental oxygen in cardiac arrest, after ROSC and in ACS. Patients with acute chest pain with presumed ACS do not need supplemental oxygen unless they present with signs of hypoxia, dyspnoea or heart failure. There is increasing evidence suggesting that hyperoxia may be harmful in patients with uncomplicated myocardial infarction.[393,788–790] During cardiac arrest, use 100% oxygen. After ROSC, titrate the inspired oxygen concentration to achieve arterial blood oxygen saturation in the range of 94–98%, or 88–92 in chronic obstructive pulmonary disease.[424,791]

Treatment of acute coronary syndromes – cause

Inhibitors of platelet aggregation

Platelet activation and aggregation following atherosclerotic plaque rupture are central pathophysiologic mechanisms of acute coronary syndromes and antiplatelet therapy is a pivotal treatment of ACS whether with or without ST segment elevation, with or without reperfusion and with or without revascularisation.

Acetylsalicylic acid (ASA). Large randomised controlled trials indicate decreased mortality when ASA (75–325 mg) is given to hospitalised patients with ACS independent of the reperfusion or revascularisation strategy.

ADP receptor inhibitors. The inhibition of the platelet ADP receptor by the thienopyridines clopidogrel and prasugrel (irreversible inhibition) and the cyclo-pentyl-triazolo-pyrimidine ticagrelor (reversible inhibition) leads to further inhibition of platelet aggregation in addition to that produced by ASA.

Glycoprotein (Gp) IIB/IIIA inhibitors. Glycoprotein (Gp) IIB/IIIA receptor activation 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 receptor blockers in patients with STEMI or non-STEMI-ACS. Do not give Gp IIB/IIIA receptor blockers before coronary anatomy is known.

Antithrombins

Unfractionated heparin (UFH) is an indirect inhibitor of thrombin, which in combination with ASA is used as an adjunct with fibrinolytic therapy or PPCI and is an important part of treatment of unstable angina and STEMI. Alternatives are characterised by a more specific factor Xa activity (low molecular weight heparins [LMWH], fondaparinux) or are direct thrombin inhibitors (bivalirudin). Rivaroxaban, apixaban and other oral direct thrombin antagonists may have an indication after stabilisation in specific patient groups but not in the initial treatment of ACS.[792] Details on the use of antithrombins are given in Section 8 Initial Management of Acute Coronary Syndromes.[7]

Reperfusion strategy in patients presenting with STEMI

Reperfusion therapy in patients with STEMI is the most important advance in the treatment of myocardial infarction in the last 30 years. Reperfusion may be achieved with fibrinolysis, with PPCI, or a combination of both. Efficacy of reperfusion therapy is profoundly dependent on the time interval from symptom onset to reperfusion. Fibrinolysis is effective specifically in the first 2–3 h after symptom onset; PPCI is less time sensitive.

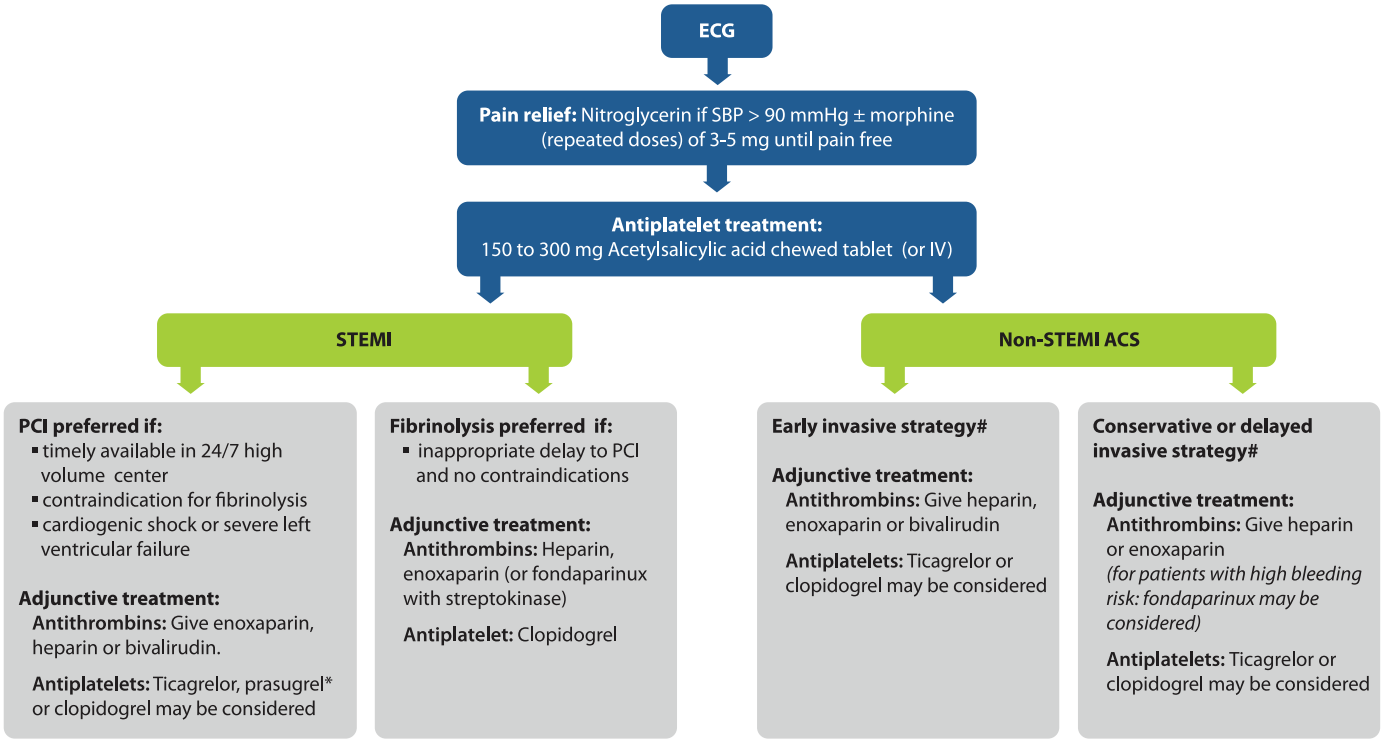


Fig. 1.34. Treatment algorithm for acute coronary syndromes; ECG, electrocardiogram; SBP, systolic blood pressure; STEMI, ST-elevation myocardial infarction; non-STEMI-ACS, non-ST-elevation acute coronary syndrome; PCI, percutaneous coronary intervention.

Fibrinolysis

Giving fibrinolytics to out-of-hospital to patients with STEMI or signs and symptoms of an ACS with presumed new LBBB is beneficial. The efficacy is greatest early after onset of symptoms. 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. The real advantage of prehospital fibrinolysis is where there are long transport times, i.e. >30–60 min.

Healthcare professionals who give fibrinolytic therapy must be aware of its contraindications and risks. Patients with large AMIs (e.g. indicated by extensive ECG changes) are likely to gain most from fibrinolytic therapy. Benefits of fibrinolytic therapy are less impressive in inferior wall infarctions than in anterior infarctions.

Primary percutaneous intervention

Coronary angioplasty with or without stent placement has become the first-line treatment for patients with STEMI. PPCI performed with a limited delay to first balloon inflation after first medical contact, at a high-volume centre, by an experienced operator who maintains an appropriate expert status, is the preferred treatment as it improves morbidity and mortality as compared with immediate fibrinolysis.[793]

Fibrinolysis versus primary PCI

Primary PCI has been limited by access to catheter laboratory facilities, appropriately skilled clinicians and delay to first balloon inflation. Fibrinolysis therapy is a widely available reperfusion strategy. Both treatment strategies are well established and have been the subject of large randomised multicentre trials over the last decades. Time from onset of symptoms and PPCI related delay (diagnosis to balloon interval minus the diagnosis to needle interval) are key in selecting the most appropriate revascularisation strategy. Fibrinolytic therapy is most effective in patients presenting within 2–3 h from onset of ischaemic symptoms. It compares favourably with PPCI when started within 2 h from symptom onset and is combined with rescue or delayed PCI. In early presenters, patients of younger age and large anterior infarctions, PPCI related delays of 60 min may be unacceptable while in late presenters (>3 h from the onset of symptoms) PPCI related delays of up to 120 min may be acceptable.[794]

Improving the systems of care may significantly shorten time delay to PPCI[795,796]:

- A pre-hospital ECG should be acquired as soon as possible and interpreted for the diagnosis of STEMI. This can reduce mortality in both patients planned for PPCI and fibrinolytic therapy.
- STEMI recognition may be accomplished by ECG transmission or onsite interpretation by physicians, or highly trained nurses or paramedics, with or without the aid of computer ECG interpretation.
- When PPCI is the planned strategy, pre-hospital activation of catheterisation laboratory for PPCI will contribute to a mortality benefit.[797]

Additional elements for an effective system of care include:

- Requiring the catheterisation laboratory to be ready within 20 min available 24/7.
- Providing real-time data feedback on the real time course from symptom onset to PCI

For those patients with a contraindication to fibrinolysis, PCI should still be pursued despite the delay, rather than not providing reperfusion therapy at all. For those STEMI patients presenting in shock, primary PCI (or coronary artery bypass surgery) is the

preferred reperfusion treatment. Fibrinolysis should only be considered if there is a substantial delay to PCI.

Triage and inter-facility transfer for primary PCI

The majority of patients with an ongoing STEMI will be first diagnosed either in the pre-hospital environment or in the setting of the ED of a non-PCI capable hospital. When PCI can be performed within a time limit of 60–90 min, then direct triage and transport for PCI is preferred to pre-hospital fibrinolysis.[797–801] For adult patients presenting with STEMI in the ED of a non-PCI capable hospital emergent transfer without fibrinolysis to a PCI centre should be considered provided that PPCI can be performed within acceptable time delays.

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.[794] 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 and maintain coronary blood flow and myocardial perfusion. Routine immediate angiography post fibrinolytic therapy is associated with increased ICH and major bleeding without offering any benefit in terms of mortality or reinfarction.[802–806] It is reasonable to perform angiography and PCI in patients with failed fibrinolysis according to clinical signs and/or insufficient ST-segment resolution.[807] In case of clinically successful fibrinolysis (evidenced by clinical signs and ST-segment resolution >50%), angiography delayed by several hours after fibrinolysis (the pharmaco-invasive approach) has been shown to improve outcome. This strategy includes early transfer for angiography and PCI if necessary after fibrinolytic treatment.

Special situations

Cardiogenic shock. Acute coronary syndrome (ACS) is the most common cause of cardiogenic shock, mainly through a large zone of myocardial ischaemia or a mechanical complication of myocardial infarction. Although uncommon, the short-term mortality of cardiogenic shock is up to 40%[808] contrasting with a good quality of life in patients discharged alive. An early invasive strategy (i.e. primary PCI, PCI early after fibrinolysis) is indicated for those patients who are suitable for revascularisation.[809] Observational studies suggest that this strategy could be also beneficial in elderly patients (over 75 years). Even if commonly used in clinical practice, there is no evidence supporting the use of IABP in cardiogenic shock.[808]

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

Reperfusion after successful CPR. The invasive management of patients with return of spontaneous circulation (ROSC) after cardiac arrest (i.e. early coronary angiography (CAG) followed by immediate PCI if deemed necessary), particularly patients after prolonged resuscitation and having nonspecific ECG changes, has been controversial due to the lack of specific evidence and significant implications on resource utilisation (including transfer of patients to PCI centres).

PCI following ROSC with ST-elevation. The highest prevalence of acute coronary lesion is observed in patients with ST segment elevation (STE) or left bundle branch block (LBBB) on post-ROSC electrocardiogram (ECG). There is no randomised study but as many observational studies reported a benefit regarding survival and neurological outcome, it is highly probable that this early invasive management is a strategy associated with a clinically relevant benefit in patients with ST segment elevation. A recent meta-analysis indicates that early angiography is associated with reduction of hospital mortality [OR 0.35 (0.31–0.41)] and increased neurologically favourable survival [OR 2.54 (2.17–2.99)].[797]

Based on the available data, emergent cardiac catheterisation lab evaluation (and immediate PCI if required) should be performed in selected adult patients with ROSC after OHCA of suspected cardiac origin with ST segment elevation on ECG.[810]

Observational studies also indicate that optimal outcomes after OHCA are achieved with a combination of targeted temperature management and PCI, which can be combined in a standardised post-cardiac-arrest protocol as part of an overall strategy to improve neurologically intact survival in this patient group.

PCI following ROSC without ST-elevation. In patients with ROSC after cardiac arrest but without ST elevation, data are conflicting regarding the potential benefit of an emergent cardiac catheterisation lab evaluation, all coming from observational studies,[410,412] or subgroup analysis.[413] It is reasonable to discuss an emergent cardiac catheterisation lab evaluation after ROSC in patients with the highest risk of coronary cause of CA. A variety of factors such as patient age, duration of CPR, haemodynamic instability, presenting cardiac rhythm, neurologic status upon hospital arrival, and perceived likelihood of cardiac aetiology can influence the decision to undertake the intervention. In patients who present in a non-PCI centre transfer for angiography and PPCI if indicated should be considered on an individual basis, weighing the expected benefits from early angiography against the risks from patient transport.

First aid

First aid is defined as the helping behaviours and initial care provided for an acute illness or injury. First aid can be initiated by anyone in any situation. A first aid provider is defined as someone trained in first aid who should:

- recognise, assess and prioritise the need for first aid
- provide care using appropriate competencies
- recognise limitations and seek additional care when needed

The goals of first aid are to preserve life, alleviate suffering, prevent further illness or injury, and promote recovery. This 2015 definition for first aid, as created by the ILCOR First Aid Task Force, addresses the need to recognise injury and illness, the requirement to develop a specific skill base and the need for first aid providers to simultaneously provide immediate care and to activate emergency medical services or other medical care as required.[811] First aid assessments and interventions should be medically sound and based on scientific evidence-based medicine or, in the absence of such evidence, on expert medical consensus. The scope of first aid is not purely scientific, as both training and regulatory requirements will influence it. Because the scope of first aid varies between countries, states and provinces, the guidelines contained herein may need to be refined according to circumstances, need, and regulatory constraints.

First aid for medical emergencies

Positioning of a breathing but unresponsive victim

Several different side-lying recovery positions have been compared but overall no significant differences between the positions have been identified.[812–814]

Position individuals who are unresponsive but breathing normally into a lateral, side-lying recovery position as opposed to leaving them supine (lying on back). In certain situations such as resuscitation-related agonal respirations or trauma, it may not be appropriate to move the individual into a recovery position.

Optimal position for a shock victim

Place individuals with shock into the supine (lying on back) position. Where there is no evidence of trauma use passive leg raising to provide a further transient improvement in vital signs[815–817]; the clinical significance of this transient improvement is uncertain.

Oxygen administration for first aid

There are no direct indications for the use of supplemental oxygen by first aid providers.[818–821] Supplemental oxygen might have potential adverse effects that complicate the disease course or even worsen clinical outcomes. If used, supplemental oxygen should be administered only by first aid providers who have been properly trained in its use and if they can monitor its effects.

Bronchodilator administration

The administration of a bronchodilator in asthma has been shown to decrease the time to resolution of symptoms in children and to reduce the time for the subjective improvement of dyspnoea in young adult asthma sufferers.[822,823] Assist individuals with asthma who are experiencing difficulty in breathing with their bronchodilator administration. First aid providers must be trained in the various methods of administering a bronchodilator.[824–826]

Stroke recognition

Stroke is a non-traumatic, focal vascular-induced injury of the central nervous system and typically results in permanent damage in the form of cerebral infarction, intracerebral haemorrhage and/or subarachnoid haemorrhage.[827] Early admission to a stroke centre and early treatment greatly improves stroke outcome and highlights the need for first aid providers to quickly recognise stroke symptoms.[828,829] There is good evidence that the use of a stroke-screening tool improves the time to definitive treatment.[830–833] Use a stroke assessment system to decrease the time to recognition and definitive treatment for individuals with suspected acute stroke. First aid providers must be trained in the use of FAST (Face, Arm, Speech Tool) or CPSS (Cincinnati Pre-hospital Stroke Scale) to assist in the early recognition of stroke.

Aspirin administration for chest pain

The early administration of aspirin in the pre-hospital environment, within the first few hours of the onset of chest pain due to suspected myocardial infarction, reduces cardiovascular mortality.[834,835] In the pre-hospital environment, administer 150-300 mg chewable aspirin early to adults with chest pain due to suspected myocardial infarction (ACS/AMI). There is a relatively low risk of complications particularly anaphylaxis and serious bleeding.[836–840] Aspirin should not be administered to patients who have a known allergy or contraindication to aspirin. Do not administer aspirin to adults with chest pain of unclear aetiology. The early administration of aspirin should never delay the transfer of the patient to a hospital for definitive care.

Second dose of adrenaline for anaphylaxis

Anaphylaxis is a potentially fatal, allergic reaction that requires immediate recognition and intervention. Adrenaline reverses the pathophysiological manifestations of anaphylaxis and remains the most important drug, especially if it is given within the first few minutes of a severe allergic reaction.[287,841,842] In the pre-hospital setting, adrenaline is administered via prefilled auto-injectors, which contain one dose of 300 μg of adrenaline (adult dose) for intramuscular self-administration or assisted by a trained first aid provider. Administer a second intramuscular dose of adrenaline to individuals in the pre-hospital environment with anaphylaxis that has not been relieved within 5–15 minutes by an initial intramuscular auto-injector dose of adrenaline.[843–852] A second intramuscular dose of adrenaline may also be required if symptoms re-occur.

Hypoglycaemia treatment

Hypoglycaemia in diabetes patients is usually a sudden and life-threatening event with the typical symptoms of hunger, headache, agitation, tremor, sweating, psychotic behaviour (frequently resembling drunkenness) and loss of consciousness. It is most important that these symptoms are recognised as hypoglycaemia as the victim requires rapid first aid treatment. Treat conscious patients with symptomatic hypoglycaemia with glucose tablets equating to glucose 15–20 g. If glucose tablets are not available, use other dietary forms of sugar.[853–855] If the patient is unconscious or unable to swallow then oral treatment should be withheld due to the risk of aspiration, and the emergency medical services should be called.

Exertion-related dehydration and rehydration therapy

First aid providers are often called upon to assist at "hydration stations" for sporting events. Use 3–8% oral carbohydrate–electrolyte (CE) beverages for rehydration of individuals with simple exercise-induced dehydration.[856–864] Alternative acceptable beverages for rehydration include water, 12% CE solution,[856] coconut water,[857,863,864] 2% milk,[861] or tea with or without carbohydrate electrolyte solution added.[858,865] Oral hydration may not be appropriate for individuals with severe dehydration associated with hypotension, hyperpyrexia or mental status changes. Such individuals should receive care by an advanced medical provider capable of administering intravenous fluids.

Eye injury from chemical exposure

For an eye injury due to exposure to a chemical substance, take immediate action by irrigating the eye using continuous, large volumes of clean water. Irrigation with large volumes of water was more effective at improving corneal pH as compared to using low volumes or saline irrigation.[866] Refer the individual for emergency professional review.

First aid for trauma emergencies

Control of bleeding

Apply direct pressure, with or without a dressing, to control external bleeding where possible. Do not try to control major external bleeding by the use of proximal pressure points or elevation of an extremity. However it may be beneficial to apply localised cold therapy, with or without pressure, for minor or closed extremity bleeding.[867,868] Where bleeding cannot be controlled by direct pressure it may be possible to control bleeding by the use of a haemostatic dressing or a tourniquet (see below).

Haemostatic dressings

Haemostatic dressings are commonly used to control bleeding in the surgical and military settings especially when the wound is in a non-compressible area such as the neck, abdomen, or groin.[869–873] Use a haemostatic dressing when direct pressure cannot control severe external bleeding or the wound is in a position where direct pressure is not possible.[874–877] Training is required to ensure the safe and effective application of these dressings.

Use of a tourniquet

Haemorrhage from vascular injured extremities may result in life-threatening exsanguination and is one of the leading causes of preventable death on the battlefield and in the civilian setting.[878,879] Tourniquets have been used in military settings for severe external limb bleeding for many years.[880,881] The application of a tourniquet has resulted in a decrease in mortality.[880–889] Use a tourniquet when direct wound pressure cannot control severe external bleeding in a limb. Training is required to ensure the safe and effective application of a tourniquet.

Straightening an angulated fracture

Fractures, dislocations, sprains and strains are extremity injuries commonly cared for by first aid providers. Do not straighten an angulated long bone fracture.

Protect the injured limb by splinting the fracture. Realignment of fractures should only be undertaken by those specifically trained to perform this procedure.

First aid treatment for an open chest wound

The correct management of an open chest wound is critical, as the inadvertent sealing of these wounds by the incorrect use of occlusive dressings or device or the application of a dressing that becomes occlusive may result in the potential life-threatening complication of a tension pneumothorax.[890] Leave an open chest wound exposed to freely communicate with the external environment without applying a dressing, or cover the wound with a non-occlusive dressing if necessary. Control localised bleeding with direct pressure.

Spinal motion restriction

In suspected cervical spine injury it has been routine to apply cervical collars to the neck, in order to avoid further injury from spinal movement. However, this intervention has been based on consensus and opinion rather than on scientific evidence.[891,892] Furthermore, clinically significant adverse effects such as raised intracranial pressure have been shown to occur following the application of a cervical collar.[893–897] The routine application of a cervical collar by a first aid provider is no longer recommended. In suspected cervical spine injury, manually support the head in a position limiting angular movement until experienced healthcare provision is available.

Recognition of concussion

Although a concussion scoring system would greatly assist first aid providers in the recognition of concussion,[898] there is no simple validated scoring system in use in current practice. An individual with a suspected concussion should be evaluated by a professional.

Cooling of burns

Immediate active cooling of thermal burns, defined as any method undertaken to decrease local tissue temperature, is a common first aid recommendation for many years. Cooling thermal burns will minimise the resulting depth of the burn[899,900] and possibly decrease the number of patients that will eventually require hospital admission for treatment.[901] The other perceived benefits of cooling are pain relief and reduction of oedema, reduced infection rates and a faster wound healing process.

Actively cool thermal burns as soon as possible for a minimum of 10 min duration using water. Care must be taken when cooling large thermal burns or burns in infants and small children so as not to induce hypothermia.

Burn dressings

A broad range of burn wound dressings are available,[902] but no scientific evidence was found to determine which type of dressings, wet or dry, is most effective. Subsequent to cooling, burns should be dressed with a loose sterile dressing.

Dental avulsion

Following a fall or accident involving the face, a tooth can be injured or avulsed. Immediate re-implantation is the intervention of choice but it is often not possible for first aid providers to re-implant the tooth due to a lack of training or skills in that procedure. If a tooth cannot be immediately re-implanted, store it in Hank's Balanced Salt Solution. If this is not available use Propolis, egg white, coconut water, ricetral, whole milk, saline or Phosphate Buffered Saline (in order of preference) and refer the individual to a dentist as soon as possible.

Education in first aid

First aid education programmes, public health campaigns and formal first aid training are recommended in order to improve prevention, recognition and management of injury and illness.[901,903,904]

Principles of education in resuscitation

The chain of survival[13] was extended to the formula of survival[11] because it was realised that the goal of saving more lives relies not only on solid and high quality science but also on the effective education of lay people and healthcare professionals.[905] Ultimately, those who are engaged in the care of cardiac arrest victims should be able to implement resource efficient systems that can improve survival after cardiac arrest.

Basic level training

Who to train and how to train

Basic life support (BLS) is the cornerstone of resuscitation and it is well established that bystander CPR is critical to survival in out-of-hospital cardiac arrests. Chest compressions and early defibrillation are the main determinants of survival from out-of-hospital cardiac arrest and there is some evidence that the introduction of training for lay people has improved survival at 30 days and 1 year.[906,907]

There is evidence that training lay people in BLS is effective in improving the number of people willing to undertake BLS in a real situation.[908–910] For high-risk populations (e.g. areas where there is high risk of cardiac arrest and low bystander response), recent evidence shows that specific factors can be identified which will enable targeted training based on the community's unique characteristics.[911,912] There is evidence that likely rescuers in these populations are unlikely to seek training on their own but that they gain competency in BLS skills and/or knowledge after training.[913–915] They are willing to be trained and are likely to share training with others.[913,914,916–918]

One of the most important steps in increasing the rate of bystander resuscitation and improving survival worldwide is to educate all school children. This can be achieved easily by teaching children for just two hours per year, beginning at the age of 12 years old.[919] At that age, school children have a positive attitude towards learning resuscitation and both medical professionals and teachers require special training to achieve these results with children.[920]

It has been shown that well trained EMS dispatchers are able to improve bystander CPR and patient outcomes.[921] However there are concerns with their ability to recognise cardiac arrest particularly in relation to agonal breathing.[50] Consequently training of EMS dispatchers should include a focus on the identification and the significance of agonal breathing,[52] and the importance of

seizures as aspects of cardiac arrest. In addition EMS dispatchers need to be taught simplified scripts for instructing bystanders in CPR.[52]

BLS/AED curricula should be tailored to the target audience and kept as simple as possible. Increasing access to different modalities of training (e.g. the use of digital media, on-line, instructor-led teaching) and self-directed learning, offer alternative means of teaching both lay and professional providers. Self-instruction programmes with synchronous or asynchronous hands-on practice (e.g., video, DVD, on-line training, computer giving feedback during training) appear to be an effective alternative to instructor-led courses for laypeople and healthcare providers learning BLS skills.[922–926]

All citizens should be taught how to perform chest compressions as a minimum requirement. 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, public campaigns, internet-based viral videos), it should focus on compression-only CPR. Local communities may want to consider their approach based on their local population epidemiology, cultural norms and bystander response rates. For those initially trained in compression-only CPR, ventilation may be covered in subsequent training. 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 carers, should be taught standard CPR i.e. chest compressions and ventilation.

Most studies show that CPR skills decay within three to six months after initial training.[924,927–930] AED skills are retained for longer than BLS skills alone.[931,932] There is some evidence that higher frequency, short burst training could potentially enhance BLS training and reduce skill decay.[928,930–932] A systematic appraisal of the literature determined that audiovisual feedback devices during resuscitation resulted in rescuers providing chest compression parameters closer to recommendations but no evidence was found that this translates into improved patient outcomes.[933]

Advanced level training

Advanced level courses cover the knowledge, skills and attitudes needed to function as part of (and ultimately lead) a resuscitation team. Supportive evidence has emerged for blended learning models (independent electronic learning coupled with a reduced duration instructor-led course). Simulation training is an integral part of resuscitation training and showed improvement in knowledge and skill performance compared to training without simulation.[934] Evidence that participants in ALS courses learn more or better CPR by using high-fidelity manikins is lacking. With this in mind, high-fidelity manikins can be used but if they are not available, the use of low-fidelity manikins is acceptable for standard advanced life support training.

Training of non-technical skills (NTS) including leadership and team training to improve CPR outcome

An increase in hospital survival from paediatric cardiac arrest and in surgical patients was found after implementation of team training programmes.[935,936] Resuscitation team performance has been shown to improve in actual cardiac arrest or simulated in-hospital advanced life support scenarios, when specific team or leadership training is added to advanced level courses.[937–941] If the simulated scenario training is followed by debriefing then learning will occur, as opposed to scenario training without debriefing.[942] Studies have failed to show a difference between debriefing with and without the use of video clips.[943,944] There is emerging evidence that frequent manikin-based refresher training in the form of low-dose in-situ training may save costs, reduce the total time for retraining, and it seems to be preferred by the learners.[945,946] Refresher training is invariably required to maintain knowledge and skills; however, the optimal frequency for refresher training is unclear.[945,947–949]

Implementation and change management

The formula for survival concludes with 'local implementation'.[11] The combination of medical science and educational efficiency is not sufficient to improve survival if there is poor or absent implementation.

Impact of guidelines

In each country, resuscitation practice is largely based on the implementation of internationally agreed resuscitation guidelines. Studies about the impact of international resuscitation guidelines suggest a positive effect on CPR performance,[906,950] return of spontaneous circulation[105,906,950–953] and survival to hospital discharge.[105,906,950–954]

Use of technology and social media

The prevalence of smartphones and tablet devices has led to the generation of numerous approaches to implementation through the use of 'apps' and also social media.

Measuring performance of resuscitation systems

As systems evolve to improve the outcomes from cardiac arrest, we need to accurately assess their impact. Measuring performance and implementing quality improvement initiatives will further enhance systems to deliver optimal results.[939,955–960]

Debriefing after resuscitation in the clinical setting

Feedback to members of an in-hospital cardiac arrest team about their performance in an actual cardiac arrest (as opposed to the training environment) can lead to improved outcomes. This can either be real-time and data-driven (e.g. use of feedback devices on cardiac compression metrics) or in a structured post-event performance focused debriefing.[939,961]

Medical emergency teams (MET) for adults

When considering the chain of survival for cardiac arrest,[13] the first link is the early recognition of the deteriorating patient and prevention of cardiac arrest. We recommend the use of a MET because they have been associated with a reduced incidence of cardiac/respiratory arrest[962–968] and improved survival rates.[963,965–968,962,969] The MET is one part of a rapid response system (RRS), which includes staff education about the signs of patient deterioration, appropriate and regular vital signs monitoring of patients, clear guidance (e.g. via calling criteria or early warning scores) to assist staff in the early detection of patient deterioration, a clear uniform system of calling for assistance and a clinical response to calls for assistance.

Training in resource limited settings

There are many different techniques for teaching ALS and BLS in resource limited settings. These include simulation, multimedia learning, self-directed learning, limited instruction, and

self-directed computer-based learning. Some of these techniques are less expensive and require less instructor resources enabling wider dissemination of ALS and BLS training.

The ethics of resuscitation and end-of-life decisions

The principle of patient autonomy

Respect for autonomy refers to a physician's obligation to respect a patient's preferences and to make decisions that accord with a patient's values and beliefs. Patient-centred healthcare places the patient at the centre of the decision-making process, rather than as a recipient of a medical decision. Applying this principle during cardiac arrest where the patient is often unable to communicate preferences is challenging.[970–973]

The principle of beneficence

Beneficence implies that interventions must benefit the patient after assessing relevant risk and benefit. Evidence-based clinical guidelines exist to assist healthcare professionals in deciding which treatment approaches are most appropriate.[11,974,975]

The principle of non-maleficence

CPR has become the norm for most patients with acute, life-threatening conditions.[976,977] CPR is, however, an invasive procedure with a low likelihood of success. CPR should, therefore, not be performed in futile cases. It is difficult to define futility in a way that is precise, prospective and applicable to the majority of cases.

The principle of justice and equitable access

Justice implies that health resources are distributed equally and fairly, irrespective of the patient's social status, in the absence of discrimination, with the right for each individual to receive the current standard of care.

Medical futility

Resuscitation is considered futile when the chances of good quality survival are minimal.[978] The decision not to attempt resuscitation does not require the consent of the patient or of those close to him, who often have unrealistic expectations.[979,980] Decision makers have a duty to consult the patient or a representative if the patient lacks capacity, in accordance with a "clear and accessible policy".[981–983]

Some countries allow prospective decisions to withhold CPR whilst in others countries or religions withholding CPR is not allowed or considered illegal. There is a lack of consistency in terms such as 'do not attempt resuscitation (DNAR)', 'do not attempt cardiopulmonary resuscitation (DNACPR)' or 'allow natural death (AND)'. This confusing use of acronyms may generate misunderstandings in national legislation and jurisdiction.[984,985]

Advance directives

Advance directives are decisions about treatment provided prospectively by an individual in case they are unable to participate directly in medical decision-making at some point in the future.[986] Periodic reviews of directives are required to ensure patients' current wishes and circumstances are accurately reflected.[979,987,988]

The legal status of advance directives in the national legislation of European countries is very disparate.[989]

Patient-centred care

The increasing centricity of the patient within healthcare demands that we seek to understand the perspective of the survivor of cardiac arrest. This requires a further commitment to work together with the public, with the survivors of cardiac arrest and their families as partners in this process.[990]

In-hospital cardiac arrest

Following in-hospital cardiac arrest (IHCA), the default position is to start resuscitation unless a decision was made to withhold CPR. Resuscitation decisions should be reviewed. Determining when CPR is likely to be unsuccessful or futile, is difficult. Prediction studies are particularly dependent on system factors such as time to start of CPR and time to defibrillation. The total study cohort but may not be applicable to an individual case. Decisions should not be made based on a single element, such as age.[991] There will remain grey areas where judgement is required for individual patients.

Out-of-hospital cardiac arrest

The decision to start or discontinue CPR is challenging outside a hospital because of the lack of sufficient information about a patient's wishes and values, comorbidities and baseline health status.[992,993]

Withholding or withdrawing CPR

Transport to hospital with ongoing CPR

Healthcare professionals should consider withholding or withdrawing CPR in children and adults when:

- the safety of the provider can no be assured;
- there is obvious mortal injury or irreversible death;
- a valid advance directive becomes available;
- there is other strong evidence that further CPR would be against patient's values and preferences or is considered futile;
- asystole for more than 20 min despite ongoing ALS, in the absence of a reversible cause.

After stopping CPR, the possibility of ongoing support of the circulation and transport to a dedicated centre with the perspective of organ donation should be considered.

Healthcare professionals should consider transport to hospital with ongoing CPR when, in the absence of the above CPR withdrawal criteria, there is one or more of the following present:

- EMS witnessed arrest
- ROSC at any moment
- VT/VF as presenting rhythm
- Presumed reversible cause (e.g. cardiac, toxic, hypothermia)

This decision should be considered early in the process e.g. after 10 min of ALS without ROSC and in view of the circumstances e.g. distance, CPR delay and presumed CPR quality in view of patient characteristics

Paediatric cardiac arrest

Despite differences in pathophysiology and aetiology, the ethical framework for decision-making in paediatric cardiac arrest does not differ much.

In most countries, legal authorities are involved in cases of sudden unexplained or accidental death. In some countries systematic

review of all child deaths is organised to get a better understanding and knowledge for the prevention of future children's deaths.[994]

Provider safety

Infectious disease epidemics have raised concerns about the safety of healthcare providers involved in the care of cardiac arrest patients. When attempting CPR in infectious patients healthcare professionals must use proper protective equipment and be sufficiently trained in its use.[995,996]

Organ donation

The primary goal of resuscitation is to save the patient's life.[997] Nonetheless, resuscitation efforts may result in brain death. In these cases, the aim of resuscitation could change to the preservation of organs for possible donation.[998] The duty of resuscitation teams for the living patient should not be confused with the duty of physicians for the dead donors, where the organs are preserved to save other people's lives. All European countries should enhance their efforts to maximise the possibility of organ donation from cardiac arrest patients who became brain dead or after stopping resuscitation in case of CPR failure.[999]

Variability in ethical CPR practices in Europe

Representatives of 32 European countries where the activities of the European Resuscitation Council are organised, have responded to questions regarding local ethical legislation and practice of resuscitation, and organisation of out-of-hospital and in-hospital resuscitation services. Equal access to emergency care and to early defibrillation is now well established. The principle of patient autonomy is now legally supported in the majority of countries. However in less than half the countries are family members usually allowed to be present during CPR. At this time euthanasia and physician-assisted suicide are controversial subjects in many European countries and the discussion is ongoing in several European countries. Healthcare professionals should know and apply the established national and local legislation and policies.

Family presence during resuscitation

The ERC supports relatives being offered the choice of being present during a resuscitation attempt whilst cultural and social variations must be understood and appreciated with sensitivity. DNAR decisions and discussions relating to DNAR should be recorded clearly in the patient's notes.[1001–1004] Over time the situation or the perspectives of patients might change and DNAR orders should be revised accordingly.[1005]

Training health care professionals about DNAR issues

Healthcare professionals should receive training about the legal and ethical basis of DNAR decisions and about how to communicate effectively with patients, relatives or next of kin. Quality of life, supportive care and end-of-life decisions need to be explained as an integrative part of the medical and nursing practice.[1006]

Practicing procedures on the recently dead

As there is wide diversity in opinion about practicing procedures on the recently dead, healthcare students and teaching professionals are advised to learn and follow the established legal, regional and local hospital policies.

Research and informed consent

Research in the field of resuscitation is necessary to test commonly interventions with uncertain efficacy or new potentially beneficial treatments.[1007,1008] To include participants in a study, informed consent must be obtained. In emergencies, there is often insufficient time to obtain informed consent. Deferred consent or exception to informed consent with prior community consultation, are considered ethically acceptable alternatives for respecting autonomy.[1009,1010] Following 12 years of ambiguity, a new European Union (EU) Regulation permitting deferred consent is expected to harmonise and foster emergency research across Member States.[1008,1009,1011,1012]

Audit of in-hospital cardiac arrests and registry analyses

Local CPR management can be improved through post-CPR debriefing to ensure a PDCA (plan-do-check-act) circle of quality improvement. Debriefing enables identification of CPR quality errors and prevents their repetition.[939,961,1013] Resuscitation team-based infrastructure and multilevel institutional audit,[1014] accurate reporting[1015] of resuscitation attempts at national audit level and/or multinational registry level, and subsequent data analysis and feedback from reported results may contribute to continuous improvement of in-hospital CPR quality and cardiac arrest outcomes.[362,1016–1019]

Conflict of interest policy for the 2015 ERC Guidelines

All authors of these ERC Guidelines 2015 have signed COI declarations (Appendix 2).

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Appendix 2. Conflicts of interest

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Nikolaos Nikolaou			Research grant Fourier trial-AMGEN
Gavin D. Perkins			Editor Resuscitation
Jasmeet Soar			Editor Resuscitation
Anatolij Truhlar			No conflict of interest reported
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Jasmeet Soar			Editor Resuscitation
Koen Monsieurs			No conflict of interest reported
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Michael Smyth			No conflict of interest reported
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European Resuscitation Council Guidelines for Resuscitation 2015 Section 2. Adult basic life support and automated external defibrillation

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Introduction

This chapter contains guidance on the techniques used during the initial resuscitation of an adult cardiac arrest victim. This includes basic life support (BLS: airway, breathing and circulation support without the use of equipment other than a protective device) and the use of an automated external defibrillator (AED). Simple techniques used in the management of choking (foreign body airway obstruction) are also included. Guidelines for the use of manual defibrillators and starting in-hospital resuscitation are found in the section Advanced Life Support Chapter.[1] A summary of the recovery position is included, with further information provided in the First Aid Chapter.[2]

The guidelines are based on the ILCOR 2015 Consensus on Science and Treatment Recommendations (CoSTR) for BLS/AED.[3] The ILCOR review focused on 23 key topics leading to 32 treatment recommendations in the domains of early access and cardiac arrest prevention, early, high-quality CPR, and early defibrillation. For these ERC guidelines the ILCOR recommendations were supplemented by focused literature reviews undertaken by Writing Group members in areas not reviewed by ILCOR. The writing group were cognisant of the costs and potential confusion created by changing guidance from 2010, and therefore sought to limit changes to those judged to be essential and supported by new evidence. Guidelines were drafted by Writing Group members, then reviewed by the full writing group and national resuscitation councils before final approval by the ERC Board.

Summary of changes since the ERC 2010 guidelines

Guidelines 2015 highlights the critical importance of the interactions between the emergency medical dispatcher, the bystander who provides CPR and the timely deployment of an automated external defibrillator. An effective, co-ordinated community response that draws these elements together is key to improving survival from out-of-hospital cardiac arrest (Fig. 2.1).

The emergency medical dispatcher plays an important role in the early diagnosis of cardiac arrest, the provision of dispatcher-assisted CPR (also known as telephone CPR), and the location and dispatch of an automated external defibrillator. The sooner the emergency services are called, the earlier appropriate treatment can be initiated and supported.

The knowledge, skills and confidence of bystanders will vary according to the circumstances, of the arrest, level of training and prior experience.

The ERC recommends that the bystander who is trained and able should assess the collapsed victim rapidly to determine if the victim

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Fig. 2.1. The interactions between the emergency medical dispatcher, the bystander who provides CPR and the timely use of an automated external defibrillator are the key ingredients for improving survival from out of hospital cardiac arrest.

is unresponsive and not breathing normally and then immediately alert the emergency services.

Whenever possible, alert the emergency services without leaving the victim.

The victim who is unresponsive and not breathing normally is in cardiac arrest and requires CPR. Immediately following cardiac arrest blood flow to the brain is reduced to virtually zero, which may cause seizure-like episodes that may be confused with epilepsy. Bystanders and emergency medical dispatchers should be suspicious of cardiac arrest in any patient presenting with seizures and carefully assess whether the victim is breathing normally.

The writing group endorses the ILCOR recommendation that all CPR providers should perform chest compressions for all victims in cardiac arrest. CPR providers trained and able to perform rescue breaths should combine chest compressions and rescue breaths. The addition of rescue breaths may provide additional benefit for children, for those who sustain an asphyxial cardiac arrest, or where the emergency medical service (EMS) response interval is prolonged. Our confidence in the equivalence between chest compression-only and standard CPR is not sufficient to change current practice.

High quality cardiopulmonary resuscitation remains essential to improving outcomes. The ERC 2015 guideline for chest compression depth is the same as the 2010 guideline. CPR providers should ensure chest compressions of adequate depth (at least 5 cm but not more than 6 cm) with a rate of 100–120 compressions per minute. Allow the chest to recoil completely after each compression and minimise interruptions in compressions. When providing rescue breaths/ventilations spend approximately 1 s inflating the chest with sufficient volume to ensure the chest rises visibly. The ratio of chest compressions to ventilations remains 30:2. Do not interrupt chest compressions for more than 10 s to provide ventilations.

Defibrillation within 3–5 min of collapse can produce survival rates as high as 50–70%. Early defibrillation can be achieved through CPR providers using public access and on-site AEDs. Public access AED programmes should be actively implemented in public places that have a high density of citizens, such as airports, railway stations, bus terminals, sport facilities, shopping malls, offices and casinos. It is here that cardiac arrests are often witnessed, and trained CPR providers can be on-scene quickly. Placing AEDs in areas where one cardiac arrest per 5 years can be expected is considered cost-effective, and the cost per added life-year is comparable to other medical interventions. Past experience of the number of cardiac arrests in a certain area, as well as the neighbourhood characteristics, may help guide AED placement. Registration of public access AEDs allows dispatchers to direct CPR providers to a nearby AED and may help to optimise response.

The adult CPR sequence can be used safely in children who are unresponsive and not breathing normally. For CPR providers with additional training a modified sequence which includes providing 5 initial rescue breaths before starting chest compressions and delaying going for help in the unlikely situation that the rescuer is alone is even more suitable for the child and drowning victim. Chest compression depths in children should be at least one third of the depth of the chest (for infants that is 4 cm, for children 5 cm).

A foreign body causing severe airway obstruction is a medical emergency. It almost always occurs whilst the victim is eating or drinking and requires prompt treatment. Start by encouraging the victim to cough. If the victim has severe airway obstruction or begins to tire, give back blows and, if that fails to relieve the obstruction, abdominal thrusts. If the victim becomes unresponsive, start CPR immediately whilst help is summoned.

Cardiac arrest

Sudden cardiac arrest (SCA) is one of the leading causes of death in Europe. Depending how SCA is defined, about 55–113 per 100,000 inhabitants a year or 350,000–700,000 individuals a year are affected in Europe.[4–6] On initial heart-rhythm analysis, about 25–50% of SCA victims have ventricular fibrillation (VF), a percentage that has declined over the last 20 years.[7–13] 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 emergency medical service personnel their rhythm has deteriorated to asystole.[14,15] When the rhythm is recorded soon after collapse, in particular by an on-site AED, the proportion of victims in VF can be as high as 76%.[16,17] More victims of SCA survive if bystanders act immediately while VF is still present. Successful resuscitation is less likely once the rhythm has deteriorated to asystole.

The recommended treatment for VF cardiac arrest is immediate bystander CPR and early electrical defibrillation. Most cardiac arrests of non-cardiac origin have respiratory causes, such as drowning (among them many children) and asphyxia. Rescue breaths as well as chest compressions are critical for successful resuscitation of these victims.

The chain of survival

The Chain of Survival summarises the vital links needed for successful resuscitation (Fig. 2.2). Most of these links apply to victims of both primary cardiac and asphyxial arrest.[18]

Early recognition and call for help

Chest pain should be recognised as a symptom of myocardial ischaemia. Cardiac arrest occurs in a quarter to a third of patients with myocardial ischaemia within the first hour after onset of chest pain.[19] Recognising the cardiac origin of chest pain, and calling the emergency services before a victim collapses, enables the emergency medical service to arrive sooner, hopefully before cardiac arrest has occurred, thus leading to better survival.[20–23]

Once cardiac arrest has occurred, early recognition is critical to enable rapid activation of the EMS and prompt initiation of bystander CPR. The key observations are **unresponsiveness** and **not breathing normally**. Emergency medical dispatchers can improve recognition by focusing on these keywords.

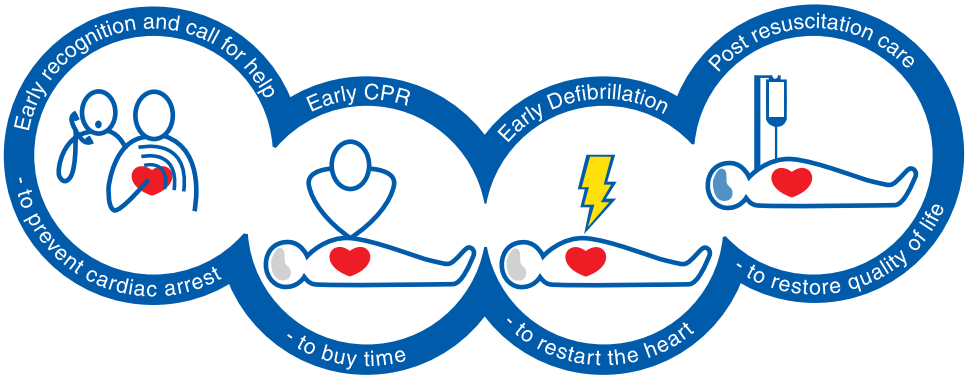


Fig. 2.2. The chain of survival.

Early bystander CPR

The immediate initiation of CPR can double or quadruple survival from cardiac arrest.[20,24–28] If able, bystanders with CPR training should give chest compressions together with ventilations. When a bystander has not been trained in CPR, the emergency medical dispatcher should instruct him or her to give chest-compression-only CPR while awaiting the arrival of professional help.[29–31]

Early defibrillation

Defibrillation within 3–5 min of collapse can produce survival rates as high as 50–70%. This can be achieved by public access and onsite AEDs.[13,17,32,33] Each minute of delay to defibrillation reduces the probability of survival to discharge by 10–12%. The links in the chain work better together: when bystander CPR is provided, the decline in survival is more gradual and averages 3–4% per minute delay to defibrillation.[20,24,34]

Early advanced life support and standardised post-resuscitation care

Advanced life support with airway management, drugs and correcting causal factors may be needed if initial attempts at resuscitation are un-successful. The quality of treatment during the post-resuscitation phase affects outcome and are addressed in the adult advanced life support and post resuscitation care chapters.[1,35]

The critical need for bystanders to act

In most communities, the median time from emergency call to emergency medical service arrival (response interval) is 5–8 min,[16,36–38] or 8–11 min to a first shock.[13,27] During this time the victim's survival depends on bystanders who initiate CPR and use an automated external defibrillator (AED).

Victims of cardiac arrest need immediate CPR. This provides a small but critical blood flow to the heart and brain. It also increases the likelihood that the heart will resume an effective rhythm and pumping power. Chest compressions are especially important if a shock cannot be delivered within the first few minutes after collapse.[39] After defibrillation, if the heart is still viable, its pacemaker activity resumes and produces an organised rhythm followed by mechanical contraction. In the first minutes after termination of VF, the heart rhythm may be slow, and the force of contractions weak; chest compressions must be continued until adequate cardiac function returns.

Use of an AED by lay CPR providers increases survival from cardiac arrest in public places.[16] AED use in residential areas is also increasing.[40] An AED uses voice prompts to guide the CPR provider, analyse the cardiac rhythm and instruct the CPR provider to deliver a shock if VF or rapid ventricular tachycardia (VT) is detected. They are accurate and will deliver a shock only when VF (or rapid VT) is present.[41,42]

Recognition of cardiac arrest

Recognising cardiac arrest can be challenging. Both bystanders and emergency call handlers (emergency medical dispatchers) have to diagnose cardiac arrest promptly in order to activate the chain of survival. Checking the carotid pulse (or any other pulse) has proved to be an inaccurate method for confirming the presence or absence of circulation.[43–47]

Agonal breaths are slow and deep breaths, frequently with a characteristic snoring sound. They originate from the brain stem, the part of the brain that remains functioning for some minutes even when deprived of oxygen. The presence of agonal breathing can be erroneously interpreted as evidence that there is a circulation and CPR is not needed. Agonal breathing may be present in up to 40% of victims in the first minutes after cardiac arrest, and if responded to as a sign of cardiac arrest, is associated with higher survival rates.[48] The significance of agonal breathing should be emphasised during basic life support training.[49,50] Bystanders should suspect cardiac arrest and start CPR if the victim is **unresponsive** and **not breathing normally**.

Immediately following cardiac arrest, blood flow to the brain is reduced to virtually zero, which may cause seizure-like episodes that can be confused with epilepsy. Bystanders should be suspicious of cardiac arrest in any patient presenting with seizures.[51,52] Although bystanders who have witnessed cardiac arrest events report changes in the victims' skin colour, notably pallor and bluish changes associated with cyanosis, these changes are not diagnostic of cardiac arrest.[51]

Role of the emergency medical dispatcher

The emergency medical dispatcher plays a critical role in the diagnosis of cardiac arrest, the provision of dispatcher assisted CPR (also known as telephone CPR), the location and dispatch of an automated external defibrillator and dispatch of a high priority EMS response. The sooner the emergency services are called, the earlier appropriate treatment can be initiated and supported.

Dispatcher recognition of cardiac arrest

Confirmation of cardiac arrest, at the earliest opportunity is critical. If the dispatcher recognises cardiac arrest, survival is more likely because appropriate measures can be taken.[53,54]

Enhancing dispatcher ability to identify cardiac arrest, and optimising emergency medical dispatcher processes, may be cost-effective solutions to improve outcomes from cardiac arrest.

Use of scripted dispatch protocols within emergency medical communication centres, including specific questions to improve cardiac arrest recognition may be helpful. Patients who are **unresponsive** and **not breathing normally** should be presumed to be in cardiac arrest. Adherence to such protocols may help improve cardiac arrest recognition,[9,55–57] whereas failure to adhere to protocols reduces rates of cardiac arrest recognition by dispatchers as well as the provision of telephone-CPR.[58–60]

Obtaining an accurate description of the victim's breathing pattern is challenging for dispatchers. Agonal breathing is often present, and callers may mistakenly believe the victim is still breathing normally.[9,60–68] Offering dispatchers additional education, specifically addressing the identification and significance of agonal breathing, can improve cardiac arrest recognition, increase the provision of telephone-CPR,[67,68] and reduce the number of missed cardiac arrest cases.[64]

Asking questions regarding the regularity or pattern of breathing may help improve recognition of abnormal breathing and thus identification of cardiac arrest. If the initial emergency call is for a person suffering seizures, the call taker should be highly suspicious of cardiac arrest, even if the caller reports that the victim has a prior history of epilepsy.[61,69]

Dispatcher assisted CPR

Bystander CPR rates are low in many communities. Dispatcher-assisted CPR (telephone-CPR) instructions have been demonstrated to improve bystander CPR rates,[9,56,70–72] reduce the time to first CPR,[56,57,68,72,73] increase the number of chest compressions delivered[70] and improve patient outcomes following out-of-hospital cardiac arrest (OHCA) in all patient groups.[9,29–31,57,71,74]

Dispatchers should provide telephone-CPR instructions in all cases of suspected cardiac arrest unless a trained provider is already delivering CPR. Where instructions are required for an adult victim, dispatchers should provide chest-compression-only CPR instructions.

If the victim is a child, dispatchers should instruct callers to provide both ventilations and chest compressions. Dispatchers should therefore be trained to provide instructions for both techniques.

Adult BLS sequence

The sequence of steps for the initial assessment and treatment of the unresponsive victim are summarised in Fig. 2.3. The sequence of steps takes the reader through recognition of cardiac arrest, calling EMS, starting CPR and using an AED. The number of steps has been reduced to focus on the key actions. The intent of the revised algorithm is to present the steps in a logical and concise manner that is easy for all types of rescuers to learn, remember and perform.

Fig. 2.4 presents the detailed step-by-step sequence for the trained provider. It continues to highlight the importance of ensuring rescuer, victim and bystander safety. Calling for additional help (if required) is incorporated in the alerting emergency services step below. For clarity the algorithm is presented as a linear sequence of steps. It is recognised that the early steps of checking response, opening the airway, checking for breathing and calling the emergency medical dispatcher may be accomplished simultaneously or in rapid succession.

Those who are not trained to recognise cardiac arrest and start CPR would not be aware of these guidelines and therefore require dispatcher assistance whenever they make the decision

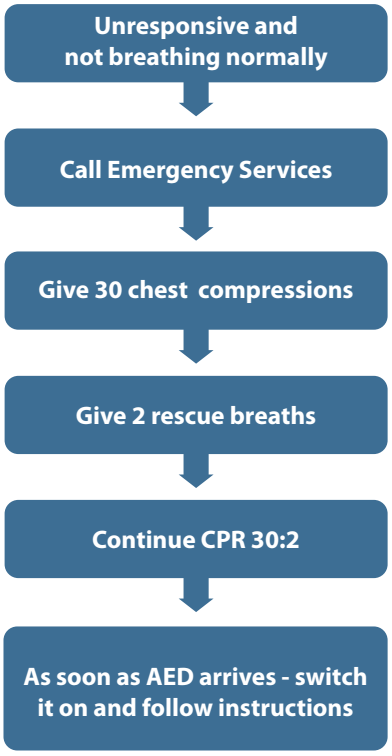


Fig. 2.3. The BLS/AED Algorithm.

to call 112. These guidelines do not therefore include specific recommendations for those who are not trained to recognise cardiac arrest and start CPR.

The remainder of this section provides supplemental information on some of the key steps within the overall sequence.

Opening the airway and checking for breathing

The trained provider should assess the collapsed victim rapidly to determine if they are responsive and breathing normally.

Open the airway using the head tilt and chin lift technique whilst assessing whether the person is breathing normally. Do not delay assessment by checking for obstructions in the airway. The jaw thrust and finger sweep are no longer recommended for the lay provider. Check for breathing using the techniques described in Fig. 2.4 noting the critical importance of recognising agonal breathing described above.

Alerting emergency services

112 is the European emergency phone number, available everywhere in the EU, free of charge. It is possible to call 112 from fixed and mobile phones to contact any emergency service: an ambulance, the fire brigade or the police. Some European countries provide an alternative direct access number to emergency medical services, which may save time. Bystanders should therefore follow national guidelines on the optimal phone number to use.

Early contact with the emergency services will facilitate dispatcher assistance in the recognition of cardiac arrest, telephone instruction on how to perform CPR, emergency medical service/first responder dispatch, and on locating and dispatching of an AED.[75–78]

If possible, stay with the victim while calling the emergency services. If the phone has a speaker facility switch it to speaker as this will facilitate continuous dialogue with the dispatcher including (if required) CPR instructions.[79] It seems reasonable that CPR

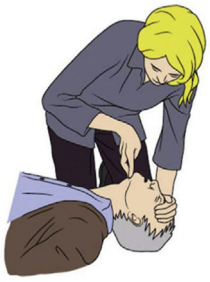
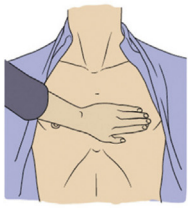
SEQUENCE / Action		Technical description
SAFETY **Make sure you, the victim and any bystanders are safe**		
RESPONSE **Check the victim for a response**		Gently shake his shoulders and ask loudly: “Are you all right?" If he responds leave him in the position in which you find him, provided there is no further danger; try to find out what is wrong with him and get help if needed; reassess him regularly
AIRWAY **Open the airway**		Turn the patient onto his back if necessary 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
BREATHING **Look, listen and feel for normal breathing**		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 seconds to determine whether the victim is breathing normally. If you have any doubt whether breathing is normal, act as if it is they are not breathing normally and prepare to start CPR
UNRESPONSIVE AND NOT BREATHING NORMALLY **Alert emergency services**		Ask a helper to call the emergency services (112) if possible otherwise call them yourself Stay with the victim when making the call if possible Activate speaker function on phone to aid communication with dispatcher
SEND FOR AED **Send someone to get AED**		Send someone to find and bring an AED if available. If you are on your own, do not leave the victim, start CPR

Fig. 2.4. Step by step sequence of actions for use by the BLS/AED trained provider to treat the adult cardiac arrest victim.

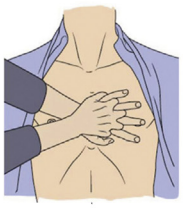
CIRCULATION

Start chest compressions



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))

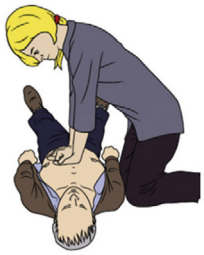


Place the heel of your other hand on top of the first hand

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 bony sternum (breastbone)



Position yourself vertically above the victim's chest and press down on the sternum approximately 5 cm (but not more than 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 100-120 min^{-1}

IF TRAINED AND ABLE

Combine chest compressions 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 second 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. Do not interrupt compressions by more than 10 seconds to deliver two breaths. Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions

Fig. 2.4. (*Continued*).

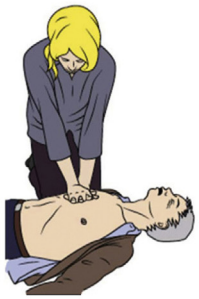
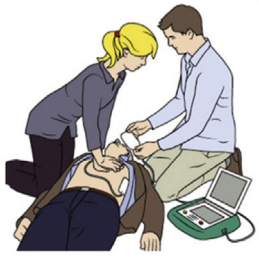
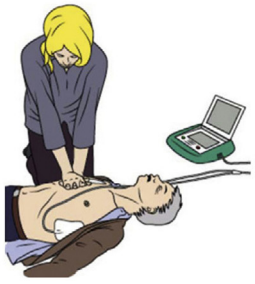
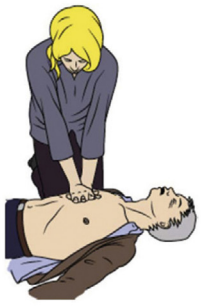
		Continue with chest compressions and rescue breaths in a ratio of 30:2
IF UNTRAINED OR UNABLE TO DO RESCUE BREATHS **Continue compression only CPR**		Give chest compressions only CPR (continuous compressions at a rate of 100-120 min^{-1})
WHEN AED ARRIVES **Switch on the AED and attach the electrode pads**		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		Ensure that nobody is touching the victim while the AED is analysing the rhythm
If a shock is indicated, **deliver shock**		Ensure that nobody is touching the victim Push shock button as directed (fully automatic AEDs will deliver the shock automatically) Immediately restart CPR 30:2 Continue as directed by the voice / visual prompts
If no shock is indicated, continue CPR		Immediately resume CPR. Continue as directed by the voice/visual prompts

Fig. 2.4. (*Continued*).

IF NO AED IS AVAILABLE CONTINUE CPR

Continue CPR



Do not interrupt resuscitation until:

- a health professional tells you to stop
- the victim is definitely waking up moving, opening eyes and breathing normally
- you become exhausted

IF UNRESPONSIVE BUT BREATHING NORMALLY

If you are certain the victim is breathing normally but is still unresponsive, place in the recovery position (see First aid chapter).

It is rare for CPR alone to restart the heart. Unless you are certain the person has recovered continue CPR

Signs the victim has recovered

- waking up
- moving
- opens eyes
- normal breathing

Be prepared to restart CPR immediately if patient deteriorates

Fig. 2.4. (*Continued*).

training should include how to activate the speaker phone.[80] Additional bystanders may be used to help call the emergency services.

Starting chest compressions

In adults needing CPR, there is a high probability of a primary cardiac cause. When blood flow stops after cardiac arrest, the blood in the lungs and arterial system remains oxygenated for some minutes. To emphasise the priority of chest compressions, it is recommended that CPR should start with chest compressions rather than initial ventilations. Manikin studies indicate that this is associated with a shorter time to commencement of CPR.[81–84]

When providing manual chest compressions:

1. Deliver compressions 'in the centre of the chest'.
2. Compress to a depth of at least 5 cm but not more than 6 cm.
3. Compress the chest at a rate of 100–120 min^{-1} with as few interruptions as possible.
4. Allow the chest to recoil completely after each compression; do not lean on the chest.

Hand position

Experimental studies show better haemodynamic responses when chest compressions are performed on the lower half of the sternum.[85–87] It is recommended that this location be taught in a simplified way, such as, "place the heel of your hand in the centre of the chest with the other hand on top". This instruction should be accompanied by a demonstration of placing the hands on the lower half of the sternum.[88,89]

Chest compressions are most easily delivered by a single CPR provider kneeling by the side of the victim, as this facilitates movement between compressions and ventilations with minimal interruptions. Over-the-head CPR for single CPR providers and straddle-CPR for two CPR providers may be considered when it is not possible to perform compressions from the side, for example when the victim is in a confined space.[90,91]

Compression depth

Fear of doing harm, fatigue and limited muscle strength frequently result in CPR providers compressing the chest less deeply than recommended. Four observational studies, published after the 2010 Guidelines, suggest that a compression depth range of 4.5–5.5 cm in adults leads to better outcomes than all other compression depths during manual CPR.[92–95] Based on an analysis of 9136 patients, compression depths between 40 and 55 mm with a peak at 46 mm, were associated with highest survival rates.[94] There is also evidence from one observational study suggesting that a compression depth of more than 6 cm is associated with an increased rate of injury in adults when compared with compression depths of 5–6 cm during manual CPR.[96] The ERC endorses the ILCOR recommendation that it is reasonable to aim for a chest compression of approximately 5 cm but not more than 6 cm in the average sized adult. In making this recommendation the ERC recognises that it can be difficult to estimate chest compression depth and that compressions that are too shallow are more harmful than compressions that are too deep. The ERC therefore decided to retain the 2010 guidance that chest compressions should be at least 5 cm but not more than 6 cm. Training should continue to prioritise achieving adequate compression depth.

Compression rate

Chest compression rate is defined as the actual rate of compressions being given at any one time. It differs from the number of chest compressions in a specific time period, which takes into account any interruptions in chest compressions.

Two studies, with a total of 13,469 patients, found higher survival among patients who received chest compressions at a rate of 100–120 min^{-1}, compared to >140, 120–139, <80 and 80–99 min^{-1}. Very high chest compression rates were associated with declining chest compression depths.[97,98] The ERC recommends, therefore, that chest compressions should be performed at a rate of 100–120 min^{-1}.

Minimising pauses in chest compressions

Delivery of rescue breaths, shocks, ventilations and rhythm analysis lead to pauses in chest compressions. Pre- and post-shock pauses of less than 10 s, and chest compression fractions >60% are associated with improved outcomes.[99–103] Pauses in chest compressions should be minimised, by ensuring CPR providers work effectively together.

Firm surface

CPR should be performed on a firm surface whenever possible. Air-filled mattresses should be routinely deflated during CPR.[104] The evidence for the use of backboards is equivocal.[105–109] If a backboard is used, take care to avoid interrupting CPR and dislodging intravenous lines or other tubes during board placement.

Chest wall recoil

Leaning on the chest preventing full chest wall recoil is common during CPR.[110,111] Allowing complete recoil of the chest after each compression results in better venous return to the chest and may improve the effectiveness of CPR.[110,112–114] CPR providers should, therefore, take care to avoid leaning after each chest compression.

Duty cycle

Optimal duty cycle (ratio of the time the chest is compressed to the total time from one compression to the next) has been studied in animal models and simulation studies with inconsistent results.[115–123] A recent human observational study has challenged the previously recommended duty cycle of 50:50 by suggesting compression phases >40% might not be feasible, and may be associated with decreased compression depth.[124] For CPR providers, the duty cycle is difficult to adjust, and is largely influenced by other chest compression parameters.[119,124] In reviewing the evidence, the ERC acknowledges there is very little evidence to recommend any specific duty cycle and, therefore, insufficient new evidence to prompt a change from the currently recommended ratio of 50%.

Feedback on compression technique

The use of CPR feedback and prompt devices during CPR in clinical practice is intended to improve CPR quality as a means of increasing the chances of ROSC and survival.[125,126] The forms of feedback include voice prompts, metronomes, visual dials, numerical displays, waveforms, verbal prompts, and visual alarms.

The effect of CPR feedback or prompt devices has been studied in two randomised trials[92,127] and 11 observational studies.[128–138] None of these studies demonstrated improved survival to discharge with feedback, and only one found a significantly higher ROSC rate in patients where feedback was used. However, in this study feedback was activated at the discretion of the physician and no details of the decision-making process to activate or not activate feedback were provided.[136] The use of CPR feedback or prompt devices during CPR should only be considered as part of a broader system of care that should include comprehensive CPR quality improvement initiatives,[138,139] rather than as an isolated intervention.

Rescue breaths

In non-paralysed, gasping pigs with unprotected, unobstructed airways, continuous-chest-compression CPR without artificial ventilation resulted in improved outcome.[140] Gasping may be present early after the onset of cardiac arrest in about one third of humans, thus facilitating gas exchange.[48] During CPR in intubated humans, however, the median tidal volume per chest compression was only about 40 mL, insufficient for adequate ventilation.[141] In witnessed cardiac arrest with ventricular fibrillation, immediate continuous chest compressions tripled survival.[142] Accordingly, continuous chest compressions may be most beneficial in the early, 'electric' and 'circulatory' phases of CPR, while additional ventilation becomes more important in the later, 'metabolic' phase.[39]

During CPR, systemic blood flow, and thus blood flow to the lungs, is substantially reduced, so lower tidal volumes and respiratory rates than normal can maintain effective oxygenation and ventilation.[143–146] When the airway is unprotected, a tidal volume of 1 L produces significantly more gastric inflation than a tidal volume of 500 mL.[147] Inflation durations of 1 s are feasible without causing excessive gastric insufflation.[148] Inadvertent hyperventilation during CPR may occur frequently, especially when using manual bag-valve-mask ventilation in a protected airway. While this increased intrathoracic pressure[149] and peak airway pressure,[150] a carefully controlled animal experiment revealed no adverse effects.[151]

From the available evidence we suggest that during adult CPR tidal volumes of approximately 500–600 mL (6–7 mL kg^{-1}) are delivered. Practically, this is the volume required to cause the chest to rise visibly.[152] CPR providers should aim for an inflation duration of about 1 s, with enough volume to make the victim's chest rise, but avoid rapid or forceful breaths. The maximum interruption in chest compression to give two breaths should not exceed 10 s.[153] These recommendations apply to all forms of ventilation during CPR when the airway is unprotected, including mouth-to-mouth and bag-mask ventilation, with and without supplementary oxygen.

Mouth-to-nose ventilation

Mouth-to-nose ventilation is an acceptable alternative to mouth-to-mouth ventilation.[154] It may be considered if the victim's mouth is seriously injured or cannot be opened, the CPR provider is assisting a victim in the water, or a mouth-to-mouth seal is difficult to achieve.

Mouth-to-tracheostomy ventilation

Mouth-to-tracheostomy ventilation may be used for a victim with a tracheostomy tube or tracheal stoma who requires rescue breathing.[155]

Compression–ventilation ratio

Animal data support a ratio of compression to ventilation of greater than 15:2.[156–158] A mathematical model suggests that a ratio of 30:2 provides the best compromise between blood flow and oxygen delivery.[159,160] A ratio of 30:2 was recommended in Guidelines 2005 and 2010 for the single CPR provider attempting resuscitation of an adult. This decreased the number of interruptions in compression and the no-flow fraction,[161,162] and reduced the likelihood of hyperventilation.[149,163] Several observational studies have reported slightly improved outcomes after implementation of the guideline changes, which included switching from a compression ventilation ratio of 15:2–30:2.[161,162,164,165] The ERC continues, therefore, to recommend a compression to ventilation ratio of 30:2.

Compression-only CPR

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.[140,166] Animal and mathematical model studies of chest-compression-only CPR have also shown that arterial oxygen stores deplete in 2–4 min.[158,167] If the airway is open, occasional gasps and passive chest recoil may provide some air exchange.[48,141,168–170]

Observational studies, classified mostly as very low-quality evidence, have suggested equivalence of chest-compression-only CPR and chest compressions combined with rescue breaths in adults with a suspected cardiac cause for their cardiac arrest.[26,171–182]

The ERC has carefully considered the balance between potential benefit and harm from compression-only CPR compared to standard CPR that includes ventilation. Our confidence in the equivalence between chest-compression-only and standard CPR is not sufficient to change current practice. The ERC, therefore, endorses the ILCOR recommendations that all CPR providers should perform chest compressions for all patients in cardiac arrest. CPR providers trained and able to perform rescue breaths should perform chest compressions and rescue breaths as this may provide additional benefit for children and those who sustain an asphyxial cardiac arrest[175,183,184] or where the EMS response interval is prolonged.[179]

Use of an automated external defibrillator

AEDs are safe and effective when used by laypeople with minimal or no training.[185] AEDs make it possible to defibrillate many minutes before professional help arrives. CPR providers should continue CPR with minimal interruption of chest compressions while attaching an AED and during its use. CPR providers should concentrate on following the voice prompts immediately when they are spoken, in particular resuming CPR as soon as instructed, and minimizing interruptions in chest compression. Indeed, pre-shock and post-shock pauses in chest compressions should be as short as possible.[99,100,103,186] Standard AEDs are suitable for use in children older than 8 years.[187–189]

For children between 1 and 8 years paediatric pads should be used, together with an attenuator or a paediatric mode if available; if these are not available, the AED should be used as it is. There are a few case reports of successful use of AEDS in children ages less than 1 year.[190,191] The incidence of shockable rhythms in infants is very low except when there is cardiac disease.[187–189,192–195] In these rare cases, if an AED is the only defibrillator available, its use should be considered (preferably with a dose attenuator).

CPR before defibrillation

The importance of immediate defibrillation has always been emphasised in guidelines and during teaching, and is considered to have a major impact on survival from ventricular fibrillation. This concept was challenged in 2005 because evidence suggested that a period of up to 180 s of chest compression before defibrillation might improve survival when the EMS response time exceeded 4–5 min.[196,197] Three more recent trials have not confirmed this survival benefit.[198–200] An analysis of one randomised trial suggested a decline in survival to hospital discharge by a prolonged period of CPR (180 s) and delayed defibrillation in patients with a shockable initial rhythm.[200] Yet, for EMS agencies with higher baseline survival-to-hospital discharge rates (defined as >20% for an initial shockable rhythm), 180 s of CPR prior to defibrillation was more beneficial compared to a shorter period of CPR (30–60 s).[201] The ERC recommends that CPR should be continued while a defibrillator or AED is being brought on-site and applied, but defibrillation should not be delayed any longer.

Interval between rhythm checks

The 2015 ILCOR Consensus on Science reported that there are currently no studies that directly address the question of optimal intervals between rhythm checks, and their effect on survival: ROSC; favourable neurological or functional outcome; survival to discharge; coronary perfusion pressure or cardiac output.

In accordance with the ILCOR recommendation, and for consistency with previous guidelines, the ERC recommends that chest compressions should be paused every two minutes to assess the cardiac rhythm.

Voice prompts

It is critically important that CPR providers pay attention to AED voice prompts and follow them without any delay. Voice prompts are usually programmable, and it is recommended that they be set in accordance with the sequence of shocks and timings for CPR given above. These should include at least:

1. minimise pauses in chest compressions for rhythm analysis and charging;
2. a single shock only, when a shockable rhythm is detected;
3. a voice prompt for immediate resumption of chest compression after the shock delivery;
4. a period of 2 min of CPR before the next voice prompt to reanalyse the rhythm.

Devices measuring CPR quality may in addition provide real-time CPR feedback and supplemental voice/visual prompts.

The duration of CPR between shocks, as well as the shock sequence and energy levels are discussed further in the Advanced Life Support Chapter.[1]

In practice, AEDs are used mostly by trained rescuers, where the default setting of AED prompts should be for a compression to ventilation ratio of 30:2.

If (in an exception) AEDs are placed in a setting where such trained rescuers are unlikely to be available or present, the owner or distributor may choose to change the settings to compression only.

Fully-automatic AEDs

Having detected a shockable rhythm, a fully automatic AED will deliver a shock without further action from the CPR provider. One manikin study showed that untrained nursing students committed fewer safety errors using a fully automatic AED compared with a semi-automatic AED.[202] A simulated cardiac arrest scenario on a manikin showed that safety was not compromised when untrained lay CPR providers used a fully automatic AED rather than a semi-automatic AED.[203] There are no human data to determine whether these findings can be applied to clinical use.

Public access defibrillation (PAD) programmes

The conditions for successful resuscitation in residential areas are less favourable than in public areas: fewer witnessed arrests, lower bystander CPR rates and, as a consequence, fewer shockable rhythms than in public places. This limits the effectiveness of AED use for victims at home.[204] Most studies demonstrating a survival benefit from AED use were conducted with AEDs in

public places.[32,205–208] More recent data from nationwide studies in Japan and the USA confirmed that when an AED was available, victims were defibrillated much sooner and with a better chance of survival.[16,209] However, an AED delivered a shock in only 3.7%[209] or 1.2%[16] of all cardiac arrests. There was a clear inverse relationship in the Japanese study between the number of AEDs available per square km and the interval between collapse and the first shock, leading to a positive relationship with survival.

Public access AED programmes should, therefore, be actively implemented in public places with a high density and movement of citizens such as airports, railway stations, bus terminals, sport facilities, shopping malls, offices and casinos where cardiac arrests are usually witnessed and trained CPR providers can quickly be on scene. The density and location of AEDs required for a sufficiently rapid response is not well established, especially when cost-effectiveness is a consideration. Factors such as expected incidence of cardiac arrest, expected number of life-years gained, and reduction in response time of AED-equipped CPR providers compared to that of traditional EMS should inform this decision. Placement of AEDs in areas where one cardiac arrest per 5 years can be expected is considered cost-effective and comparable to other medical interventions.[210–212] For residential areas, past experience may help guide AED placement, as may neighbourhood characteristics.[213,214] Registration of AEDs for public access, so that dispatchers can direct CPR providers to a nearby AED, may also help to optimise response.[215] Cost saving is also possible, as early defibrillation and on-site AED defibrillation may result in lower in-hospital cost.[216,217]

The full potential of AEDs has not yet been achieved, because they are mostly used in public settings, yet 60–80% of cardiac arrests occur at home. The proportion of patients found in VF is lower at home than in public places, however the absolute number of potentially treatable patients is higher at home.[204] Public access defibrillation (PAD) rarely reaches victims at home.[208] Different strategies, therefore, are required for early defibrillation in residential areas. Dispatched first responders, such as police and fire fighters will, in general, have longer response times, but they have the potential to reach the whole population.[17,36] The logistic problem for first responder programmes is that the CPR provider needs to arrive, not just earlier than the traditional ambulance, but within 5–6 min of the initial call, to enable attempted defibrillation in the electrical or circulatory phase of cardiac arrest.[39] With longer delays, the survival benefit decreases: a few minutes gain in time will have less impact when a first responder arrives more than 10 min after the call.[34,218] Dispatched lay CPR providers, local to the victim and directed to a nearby AED, may improve bystander CPR rates[33] and help reduce the time to defibrillation.[40]

When implementing an AED programme, community and programme leaders should consider factors such as development of a team with responsibility for monitoring, maintaining the devices, training and retraining individuals who are likely to use the AED, and identification of a group of volunteer individuals who are committed to using the AED for victims of cardiac arrest.[219] Funds must be allocated on a permanent basis to maintain the programme.

Programmes that make AEDs available in residential areas have only been evaluated for response time, not for survival benefit.[40] The acquisition of an AED for individual use at home, even for those considered at high risk of sudden cardiac arrest is not effective.[220]

The special circumstances chapter provides the evidence underpinning the ERC recommendation that AEDs should be mandatory on board all commercial aircraft in Europe, including regional and low-cost carriers.[221]

Universal AED signage

When a victim collapses an AED must be obtained rapidly: simple and clear signage indicating the location of an AED and the fastest way to it is important. ILCOR has designed such an AED sign that may be recognised worldwide and this is recommended.[222]

In-hospital use of AEDs

There are no published randomised trials comparing in-hospital use of AEDs with manual defibrillators. Two older, observational 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.[223,224] A more recent observational study showed that an AED could be used successfully before the arrival of the hospital resuscitation team.[225] Three observational studies showed no improvements in survival to hospital discharge for in-hospital adult cardiac arrest when using an AED compared with manual defibrillation.[226–228] In one of these studies,[226] patients in the AED group with non-shockable rhythms had a lower survival-to-hospital discharge rate compared with those in the manual defibrillator group (15% vs. 23%; $P = 0.04$). Another large observational study of 11,695 patients from 204 hospitals also showed that in-hospital AED use was associated with a lower survival-to-discharge rate compared with no AED use (16.3% vs. 19.3%; adjusted rate ratio [RR], 0.85; 95% confidence interval [CI], 0.78–0.92; $P < 0.001$).[229] For non-shockable rhythms, AED use was associated with lower survival (10.4% vs. 15.4%; adjusted RR, 0.74; 95% CI, 0.65–0.83; $P < 0.001$), and a similar survival rate for shockable rhythms, (38.4% vs. 39.8%; adjusted RR, 1.00; 95% CI, 0.88–1.13; $P = 0.99$). This suggests that AEDs may cause harmful delays in starting CPR, or interruptions in chest compressions in patients with non-shockable rhythms.[230] Only a small proportion (less than 20%) of in-hospital cardiac arrests have an initial shockable rhythm.[229,231,232]

We recommend the use of AEDs in those areas of the hospital where there is a risk of delayed defibrillation,[233] because it will take several minutes for a resuscitation team to arrive, and first responders do not have skills in manual defibrillation. The goal is to attempt defibrillation within 3 min of collapse. In hospital areas where there is rapid access to manual defibrillation, either from trained staff or a resuscitation team, manual defibrillation should be used in preference to an AED. Whichever defibrillation technique is chosen (and some hospitals may choose to have defibrillators that offer both an AED and manual mode) an effective system for training and retraining should be in place.[232,234] Sufficient healthcare providers should be trained to enable the goal of providing the first shock within 3 min of collapse anywhere in the hospital. Hospitals should monitor collapse-to-first shock intervals and audit resuscitation outcomes.

Risks to the CPR provider and recipients of CPR

Risks to the victim who receives CPR who is not in cardiac arrest

Many CPR providers do not initiate CPR because they are concerned that delivering chest compressions to a victim who is not in cardiac arrest will cause serious complications. Three studies have investigated the risk of CPR in persons not in cardiac arrest.[235–237] Pooled data from these three studies, encompassing 345 patients, found an incidence of bone fracture (ribs and clavicle) of 1.7% (95% CI 0.4–3.1%), pain in the area of chest compression 8.7% (95% CI 5.7–11.7%), and no clinically relevant visceral injury. Bystander CPR extremely rarely leads to serious harm in victims who are eventually found not to be in cardiac arrest. CPR providers should not,

therefore, be reluctant to initiate CPR because of concern of causing harm.

Risks to the victim who receives CPR who is in cardiac arrest

A systematic review of skeletal injuries after manual chest compression reports an incidence of rib fractures ranging from 13% to 97%, and of sternal fractures from 1% to 43%.[238] Visceral injuries (lung, heart, abdominal organs) occur less frequently and may or may not be associated with skeletal injury.[239] Injuries are more common when the depth of chest compression exceeds 6 cm in the average adult.[96]

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

Observational studies of training or actual CPR performance and case reports have described rare occurrences of muscle strain, back symptoms, shortness of breath, hyperventilation, pneumothorax, chest pain, myocardial infarction and nerve injury.[240,241] The incidence of these events is very low, and CPR training and actual performance is safe in most circumstances.[242] Individuals undertaking CPR training should be advised of the nature and extent of the physical activity required during the training programme. Learners and CPR providers who develop significant symptoms (e.g. chest pain or severe shortness of breath) during CPR training should be advised to stop.

CPR provider fatigue

Several manikin studies have found that chest compression depth can decrease as soon as two minutes after starting chest compressions.[243] An in-hospital patient study showed that, even while using real-time feedback, the mean depth of compression deteriorated between 1.5 and 3 min after starting CPR.[244] It is therefore recommended that CPR providers change over about every two minutes to prevent a decrease in compression quality due to CPR provider fatigue. Changing CPR providers should not interrupt chest compressions.

Risks during defibrillation

Many studies of public access defibrillation showed that AEDs can be used safely by laypeople and first responders.[185] A systematic review identified eight papers that reported a total of 29 adverse events associated with defibrillation.[245] The causes included accidental or intentional defibrillator misuse, device malfunction and accidental discharge during training or maintenance procedures. Four single-case reports described shocks to CPR providers from discharging implantable cardioverter defibrillators (ICDs), in one case resulting in a peripheral nerve injury. No studies were identified which reported harm to CPR providers from attempting defibrillation in wet environments.

Although injury to the CPR provider from a defibrillator shock is extremely rare, it has been shown that standard surgical gloves do not provide adequate protection.[246–249] CPR providers, therefore, should not continue manual chest compressions during shock delivery, and victims should not be touched during ICD discharge. Direct contact between the CPR provider and the victim should be avoided when defibrillation is performed.

Psychological effects

One large, prospective trial of public access defibrillation reported few adverse psychological effects associated with CPR or AED use that required intervention.[242] Two large, retrospective, questionnaire-based studies found that bystanders who performed CPR regarded their intervention as a positive experience.[250,251] Family members witnessing a resuscitation attempt may also derive psychological benefit.[252–254] The rare occurrences of adverse psychological effects in CPR providers after performing CPR should be recognised and managed appropriately.

Disease transmission

The risk of disease transmission during training and actual CPR performance is extremely low.[255–257] Wearing gloves during CPR is reasonable, but CPR should not be delayed or withheld if gloves are not available.

Barrier devices for use with rescue breaths

Three studies showed that barrier devices decrease transmission of bacteria during rescue breathing in controlled laboratory settings.[258,259] No studies were identified which examined the safety, effectiveness or feasibility of using barrier devices (such as a face shield or face mask) to prevent victim contact when performing CPR. Nevertheless if the victim is known to have a serious infection (e.g. HIV, tuberculosis, hepatitis B or SARS) a barrier device is recommended.

If a barrier device is used, care should be taken to avoid unnecessary interruptions in CPR. Manikin studies indicate that the quality of CPR is superior when a pocket mask is used compared to a bag-valve mask or simple face shield.[260–262]

Foreign body airway obstruction (choking)

Foreign body airway obstruction (FBAO) is an uncommon but potentially treatable cause of accidental death.[263] As most choking events are associated with eating, they are commonly witnessed. As victims initially are conscious and responsive, there are often opportunities for early interventions which can be life saving.

Recognition

Because recognition of airway obstruction is the key to successful outcome, it is important not to confuse this emergency with fainting, myocardial infarction, seizure or other conditions that may cause sudden respiratory distress, cyanosis or loss of consciousness. FBAO usually occurs while the victim is eating or drinking. People at increased risk of FBAO include those with reduced conscious levels, drug and/or alcohol intoxication, neurological impairment with reduced swallowing and cough reflexes (e.g. stroke, Parkinson's disease), respiratory disease, mental impairment, dementia, poor dentition and older age.[264]

Fig. 2.5 presents the treatment algorithm for the adult with FBAO. Foreign bodies may cause either mild or severe airway obstruction. It is important to ask the conscious victim "Are you choking?" The victim that is able to speak, cough and breathe has mild obstruction. The victim that is unable to speak, has a weakening cough, is struggling or unable to breathe, has severe airway obstruction.

Treatment for mild airway obstruction

Coughing generates high and sustained airway pressures and may expel the foreign body. Aggressive treatment with back blows, abdominal thrusts and chest compressions, may cause harm and can worsen the airway obstruction. These treatments should be reserved for victims who have signs of severe airway obstruction. Victims with mild airway obstruction should remain under continuous observation until they improve, as severe airway obstruction may subsequently develop.

Treatment for severe airway obstruction

The clinical data on choking are largely retrospective and anecdotal. For conscious adults and children over one year of age

Action		Technical description
SUSPECT CHOKING **Be alert to choking particularly if victim is eating**		
ENCOURAGE TO COUGH **Instruct victim to cough**		
GIVE BACK BLOWS **If cough becomes ineffective give up to 5 back blows**		If the victim shows signs of severe airway obstruction and is conscious apply five back blows Stand to the side and slightly behind the victim Support the chest with one hand and lean the victim well forwards so that when the obstructing object is dislodged it comes out of the mouth rather than goes further down the airway Give five sharp blows between the shoulder blades with the heel of your other hand
GIVE ABDOMINAL THRUSTS **If back blows are ineffective give up to 5 abdominal thrutsts**		If five back blows fail to relieve the airway obstruction, give up to five abdominal thrusts as follows: Stand behind the victim and put both arms round the upper part of the abdomen Lean the victim forwards Clench your fist and place it between the umbilicus (navel) and the ribcage Grasp this hand with your other hand and pull sharply inwards and upwards Repeat up to five times If the obstruction is still not relieved, continue alternating five back blows with five abdominal thrusts

Fig. 2.5. Step by step sequence of actions for the treatment of the adult victim with foreign body airway obstruction.

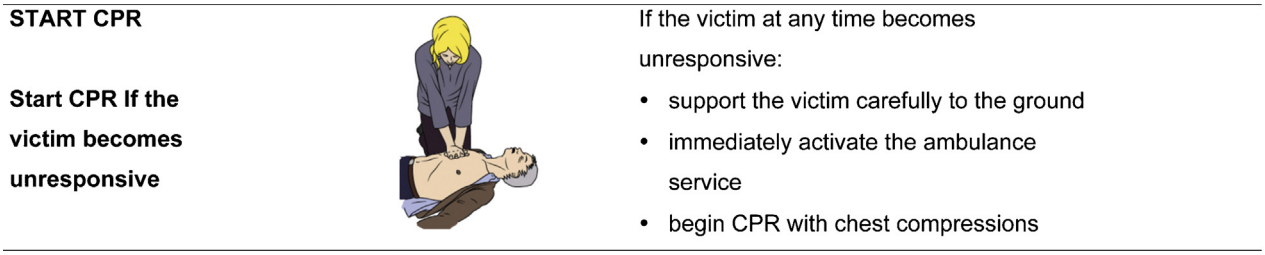


Fig. 2.5. (*Continued*).

with complete FBAO, case reports have demonstrated the effectiveness of back blows or 'slaps', abdominal thrusts and chest thrusts.[265] Approximately 50% of episodes of airway obstruction are not relieved by a single technique.[266] The likelihood of success is increased when combinations of back blows or slaps, and abdominal and chest thrusts are used.[265]

Treatment of foreign body airway obstruction in an unresponsive victim

A randomised trial in cadavers[267] and two prospective studies in anaesthetised volunteers[268,269] showed that higher airway pressures can be generated using chest thrusts compared with abdominal thrusts. Bystander initiation of chest compression for unresponsive or unconscious victims of FBAO was independently associated with good neurological outcome (odds ratio, 10.57; 95% CI, 2.472–65.059, $P < 0.0001$).[270] Chest compressions should, therefore, be started promptly if the victim becomes unresponsive or unconscious. After 30 compressions attempt 2 rescue breaths, and continue CPR until the victim recovers and starts to breathe normally.

Aftercare and referral for medical review

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

Resuscitation of children (see also Recognition of Cardiac Arrest section) and victims of drowning (see also The Chain of Survival section)

Many children do not receive resuscitation because potential CPR providers fear causing harm if they are not specifically trained in resuscitation for children. This fear is unfounded: it is far better to use the adult BLS sequence for resuscitation of a child than to do nothing. For ease of teaching and retention, laypeople should be taught that the adult sequence may also be used for children who are not responsive and not breathing normally. The following minor modifications to the adult sequence will make it even more suitable for use in children:

- Give 5 initial rescue breaths before starting chest compressions.
- Give CPR for 1 min before going for help in the unlikely event the CPR provider is alone.
- Compress the chest by at least one third of its depth; use 2 fingers for an infant under one year; use 1 or 2 hands for a child over 1 year as needed to achieve an adequate depth of compression.

The same modifications of 5 initial breaths and 1 min of CPR by the lone CPR provider before getting help, may improve outcome for victims of drowning. This modification should be taught only to those who have a specific duty of care to potential drowning victims (e.g. lifeguards).

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Conflicts of interest

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European Resuscitation Council Guidelines for Resuscitation 2015 Section 3. Adult advanced life support

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Introduction

Adult advanced life support (ALS) includes advanced interventions after basic life support has started and when appropriate an automated external defibrillator (AED) has been used. Adult basic life support (BLS) and use of AEDs is addressed in Section 2. The transition between basic and advanced life support should be seamless as BLS will continue during and overlap with ALS interventions. This section on ALS includes the prevention of cardiac arrest, specific aspects of prehospital ALS, starting in-hospital resuscitation, the ALS algorithm, manual defibrillation, airway management during CPR, drugs and their delivery during CPR, and the treatment of peri-arrest arrhythmias. There are two changes in the presentation of these guidelines since European Resuscitation Council (ERC) Guidelines 2010.[1] There is no longer a separate section on electrical therapies[2] and the ALS aspects are now part of this section. Post-resuscitation care guidelines are presented in a new section (Section 5) that recognises the importance of the final link in the Chain of Survival.[3]

These Guidelines are based on the International Liaison Committee on Resuscitation (ILCOR) 2015 Consensus on Science and Treatment Recommendations (CoSTR) for ALS.[4] The 2015 ILCOR review focused on 42 topics organised in the approximate sequence of ALS interventions: defibrillation, airway, oxygenation and ventilation, circulatory support, monitoring during CPR, and drugs during CPR. For these Guidelines the ILCOR recommendations were supplemented by focused literature reviews undertaken by the ERC ALS Writing Group for those topics not reviewed in the 2015 ILCOR CoSTR. Guidelines were drafted and agreed by the ALS Writing Group members before final approval by the ERC General Assembly and ERC Board.

Summary of changes since 2010 Guidelines

The 2015 ERC ALS Guidelines have a change in emphasis aimed at improved care and implementation of these guidelines in order to improve patient focused outcomes.[5] The 2015 ERC ALS Guidelines do not include any major changes in core ALS interventions since the previous ERC guidelines published in 2010.[1,2] The key changes since 2010 are:

- Continuing emphasis on the use of rapid response systems for care of the deteriorating patient and prevention of in-hospital cardiac arrest.
- Continued emphasis on minimally interrupted high-quality chest compressions throughout any ALS intervention: chest

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compressions are paused briefly only to enable specific interventions. This includes minimising interruptions in chest compressions to attempt defibrillation.
- Keeping the focus on the use of self-adhesive pads for defibrillation and a defibrillation strategy to minimise the preshock pause, although we recognise that defibrillator paddles are used in some settings.
- There is a new section on monitoring during ALS with an increased emphasis on the use of waveform capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of return of spontaneous circulation (ROSC).
- There are a variety of approaches to airway management during CPR and a stepwise approach based on patient factors and the skills of the rescuer is recommended.
- The recommendations for drug therapy during CPR have not changed, but there is greater equipoise concerning the role of drugs in improving outcomes from cardiac arrest.
- The routine use of mechanical chest compression devices is not recommended, but they are a reasonable alternative in situations where sustained high-quality manual chest compressions are impractical or compromise provider safety.
- Peri-arrest ultrasound may have a role in identifying reversible causes of cardiac arrest.
- Extracorporeal life support techniques may have a role as a rescue therapy in selected patients where standard ALS measures are not successful.

3a – 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.[3] Once cardiac arrest occurs, only about 20% of patients who have an in-hospital cardiac arrest will survive to go home.[6,7]

The key recommendations for the prevention of in-hospital cardiac arrest are unchanged since the previous guidance in 2010.[1] We suggest an approach to prevention of in-hospital cardiac arrest that includes staff education, monitoring of patients, recognition of patient deterioration, a system to call for help and an effective response – the chain of prevention.[8]

The problem

Cardiac arrest in patients in unmonitored ward areas is not usually a sudden unpredictable event.[9] Patients often have slow and progressive physiological deterioration, involving hypoxaemia and hypotension that is unnoticed or poorly managed by ward staff.[10–12] The initial cardiac arrest rhythm is usually non-shockable[6,7] and survival to hospital discharge is poor, particularly in patients with preceding signs of respiratory depression or shock.[7,13] Early and effective treatment might prevent some cardiac arrests, deaths and unanticipated ICU admissions. Studies conducted in hospitals with traditional cardiac arrest teams have shown that patients attended by the team but who were found not to have a cardiac arrest, have a high morbidity and mortality.[14–16] Registry data from the US suggests that hospitals with lowest incidence of IHCA also have the highest CA survival.[17]

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

These include infrequent, late or incomplete vital signs assessments; lack of knowledge of normal vital signs values; poor design of vital signs charts; poor sensitivity and specificity of 'track and trigger' systems; failure of staff to increase monitoring or escalate care, and staff workload.[18–26] Problems with assessing and treating airway, breathing and circulation abnormalities as well organisational problems such as poor communication, lack of teamwork and insufficient use of treatment limitation plans are not infrequent.[10,27,28]

Education in acute care

Several studies show that medical and nursing staff lack knowledge and skills in acute care,[29–37] e.g. oxygen therapy,[30] fluid and electrolyte balance,[31] analgesia,[32] issues of consent,[33] pulse oximetry,[30,34,35] and drug doses.[36] Staff education is an essential part of implementing a system to prevent cardiac arrest but to date, randomised controlled studies addressing the impact of specific educational interventions are lacking.[37]

In one 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.[38,39] Rapid response teams, such as METs, play a role in educating and improving acute care skills of ward personnel.[37,40] The introduction of specific, objective calling criteria,[41] referral tools[42] and feedback to caregivers[43] has resulted in improved MET use and a significant reduction in cardiac arrests. Another study found that the number of cardiac arrest calls decreased while pre-arrest calls increased after implementing a standardised educational programme[44] in two hospitals[45]; this was associated with a decrease in CA incidence and improved CA survival. Other research suggests that multiprofessional education did not alter the rate of mortality or staff awareness of patients at risk on general wards.[46]

Monitoring and recognition of the critically ill patient

Clinical signs of acute illness are similar whatever the underlying process, as they reflect failing respiratory, cardiovascular and neurological systems. Alterations in physiological variables, singly or in combination are associated with, or can be used to predict the occurrence of cardiac arrest,[12,47–50] hospital death[20,21,51–68] and unplanned ICU admission,[47,66,69,70] and with increasing magnitude and number of derangements the likelihood of death is increased.[18,47,48,63,71–79] Even though abnormal physiology is common on general wards,[80] the measurement and documentation of vital signs is suboptimal.[9,11,22,49,81–83] To assist in the early detection of critical illness, each patient should have a documented plan for vital signs monitoring including which physiological measurements needs no be undertaken and frequency.[24,84]

Many hospitals use early warning scores (EWS) or calling criteria to identify ward patients needing escalation of care,[22,49,82,85–89] and this increases vital signs monitoring.[82,88,89] These calling criteria or 'track and trigger' systems include single-parameter systems, multiple-parameter systems, aggregate weighted scoring systems or combination systems.[90] Aggregate weighted track and trigger systems offer a graded escalation of care, whereas single parameter track and trigger systems provide an all-or-nothing response. Simpler systems may have advantages over more complex ones.[91,92] Nurse concern may also be an important predictor of patient deterioration.[93–95]

The use of an aggregate score based on a number of vital sign abnormalities appears more important than abnormalities in a single criteria.[96,97] Aggregate-weighted scoring systems vary in their performance and in which endpoint they predict.[20,70,98] In older (>65 year) patients, who represent the largest group of IHCA patients,[99] signs of deterioration before cardiac arrest are often blunted, and the predictive value of the Modified Early Warning Score (MEWS) progressively decreases with increasing patient age.[100]

The design of vital signs charts[19,101] or the use of technology[102–104] may have an important role in the detection of

deterioration and the escalation of care, but these require further study. Possible benefits include increased vital signs recording,[105] improved identification of signs of deterioration,[19,101,104] reduced time to team activation[103] and improved patient outcomes.[103,106]

Calling for help and the response to critical illness

Nursing staff and junior doctors often find it difficult to ask for help or escalate treatment as they feel their clinical judgement may be criticised.[107–110] In addition, there is a common belief, especially amongst younger staff, that the patient's primary team should be capable of dealing with problems close to their area of specialty.[110] It is logical that hospitals should ensure all staff are empowered to call for help and also trained to use structured communication tools such as RSVP (reason-story-vital signs-plan)[111] or SBAR (situation-background-assessment-recommendation)[112] tools to ensure effective inter-professional communication. However, recent research suggests that structured communication tools are rarely used in clinical practice.[113]

The response to patients who are critically ill or who are at risk of becoming critically ill is now usually provided by a medical emergency team (MET), rapid response team (RRT), or critical care outreach team (CCOT).[114–117] These 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, who respond to specific calling criteria. Any member of the healthcare team can initiate a MET/RRT/CCOT call. In some hospitals, the patient, and their family and friends, are also encouraged to activate the team.[118–120] Team interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids.[121–125] However, *post-hoc* analysis of the MERIT study data suggests that nearly all MET calls required 'critical care-type' interventions.[126] The MET, RRT or CCOT is often also involved in discussions regarding 'do not attempt cardiopulmonary resuscitation' (DNACPR) or end-of-life plans.[127–133] Recently, attempts have been made to develop a screening tool to identify patients at the end of life and quantify the risk of death in order to minimise prognostic uncertainty and avoid potentially harmful and futile treatments.[134]

Studying the effect of the MET/RRT/CCOT systems on patient outcomes is difficult because of the complex nature of the intervention. During the period of most studies of rapid response teams, there has been a major international focus on improving other aspects of patient safety, e.g. hospital acquired infections, earlier treatment of sepsis and better medication management, all of which have the potential to influence patient deterioration and may have a beneficial impact on reducing cardiac arrests and hospital deaths. Most studies on RRT/MET systems to date originate from the USA and Australia and the systems effectiveness in other health care systems in not clear.[135]

A well-designed, cluster-randomised controlled trial of the MET system (MERIT study) involving 23 hospitals[22] did not show a reduction in cardiac arrest rate after introduction of a MET when analysed on an intention-to-treat basis. Both the control and MET groups demonstrated improved outcome compared to baseline. *Post hoc* analysis of the MERIT study showed there was a decrease in cardiac arrest and unexpected mortality rate with increased activation of the MET system.[136] The evidence from predominantly single centre observational studies is inconclusive, with some studies showing reduced numbers of cardiac arrests after MET/RRT implementation[38,41,123,137–159] and some studies failing to show a reduction[121,122,124,125,160–163]. However, systematic reviews, meta-analyses and multicentre studies do suggest that RRT/MET systems reduce rates of cardiopulmonary arrest and lower hospital mortality rates.[164–166] Concern has been expressed about MET activity leading to potential adverse events resulting from staff leaving normal duties to attend MET calls. Research suggests that although MET calls may cause disruption to normal hospital routines and inconvenience to staff, no major patient harm follows.[167]

Appropriate placement of patients

Ideally, the sickest patients should be admitted to an area that can provide the greatest supervision and the highest level of organ support and nursing care. International organisations have offered definitions of levels of care and produced admission and discharge criteria for high dependency units (HDUs) and ICUs.[168,169]

Staffing levels

Hospital staffing tends to be at its lowest during the night and at weekends, which may influence patient monitoring, treatment and outcome. Data from the US National Registry of CPR Investigators shows that survival rates from in-hospital cardiac arrest are lower during nights and weekends.[170] Outcomes for patients admitted to hospital and those discharged from the ICU are worse after hours and at weekends.[171–174] Studies show that higher nurse staffing is associated with lower rates of failure-to-rescue, and reductions in rates of cardiac arrest rates, pneumonia, shock and death.[23,175–177]

Resuscitation decisions

The decision to start, continue and terminate resuscitation efforts is based on the balance between the risks, benefits and burdens these interventions place on patients, family members and healthcare providers. There are circumstances where resuscitation is inappropriate and should not be provided. Consider a 'do not attempt cardiopulmonary resuscitation' (DNACPR) decision when the patient:

- does not wish to have CPR
- is very unlikely to survive cardiac arrest even if CPR is attempted.

There is wide variation in DNACPR decision-making practice throughout Europe particularly with respect to involvement of patients in decision-making.[178–181] Improved knowledge, training and DNACPR decision-making should improve patient care and prevent futile CPR attempts.[182,183] The section on ethics in the ERC Guidelines provides further information.[184]

Guidelines for prevention of in-hospital cardiac arrest

Hospitals should provide a system of care that includes: (a) staff education regarding the signs of patient deterioration and the rationale for rapid response to illness, (b) appropriate, and frequent monitoring of patients' vital signs, (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 help.[8] 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. Frequency of measurement should relate to the patient's severity of illness, and 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.[24,84]

(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. The charting system should facilitate easy identification of signs of deterioration.
(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/day and seven days per week. The team must include staff with the appropriate skills. The patient's primary clinical team should also be involved at an early stage in decision-making.
(7) Train all clinical staff in the recognition, monitoring and management of the critically ill patient. Include advice on clinical management while awaiting the arrival of more experienced staff. Ensure that staff know their role(s) in the rapid response system.
(8) Hospitals must empower staff of all disciplines to call for help when they identify a patient at risk of deterioration or cardiac arrest. Staff should be trained in the use of structured communication tools to ensure effective handover of information between doctors, nurses and other healthcare professions.
(9) Identify patients for whom cardiopulmonary arrest is an anticipated terminal event and in whom CPR is inappropriate, and patients who do not wish to be treated with CPR. Hospitals should have a DNACPR policy, based on national guidance, which is understood by all clinical staff.
(10) Ensure accurate audit of cardiac arrest, deteriorating patients, unexpected deaths and unanticipated ICU admissions using common datasets. Also audit 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 in older people. Inherited abnormalities (e.g. Brugada syndrome, hypertrophic cardiomyopathy), congenital heart disease, myocarditis and substance abuse are predominant causes in the young.

Most SCD victims have a history of cardiac disease and warning signs, most commonly chest pain, in the hour before cardiac arrest.[185] In patients with a known diagnosis of cardiac disease, syncope (with or without prodrome – particularly recent or recurrent) is an independent risk factor for increased risk of death.[186–196] Chest pain on exertion only, and palpitations associated with syncope only, are associated with hypertrophic cardiomyopathy, coronary abnormalities, Wolff–Parkinson–White, and arrhythmogenic right ventricular cardiomyopathy.

Apparently healthy children and young adults who suffer SCD can also have signs and symptoms (e.g. syncope/pre-syncope, chest pain and palpitations) that should alert healthcare professionals to seek expert help to prevent cardiac arrest.[197–206]

Children and young adults presenting with characteristic symptoms of arrhythmic syncope should have a specialist cardiology assessment, which should include an ECG and in most cases an echocardiogram and exercise test. Characteristics of arrhythmic syncope include: syncope in the supine position, occurring during or after exercise, with no or only brief prodromal symptoms, repetitive episodes, or in individuals with a family history of sudden death. In addition, non-pleuritic chest pain, palpitations associated with syncope, seizures (when resistant to treatment, occurring at night or precipitated by exercise, syncope, or loud noise) and drowning in a competent swimmer should raise suspicion of increased risk. Systematic evaluation in a clinic specialising in the care of those at risk for SCD is recommended in family members of young victims of SCD or those with a known cardiac disorder resulting in an increased risk of SCD.[186,207–211] A family history of syncope or SCD, palpitations as a symptom, supine syncope and syncope associated with exercise and emotional stress are more common in patients with long QT syndrome (LQTS).[212] In older adults[213,214] the absence of nausea and vomiting before syncope and ECG abnormalities is an independent predictor of arrhythmic syncope.

Inexplicable drowning and drowning in a strong swimmer may be due to LQTS or catecholaminergic polymorphic ventricular tachycardia (CPVT).[215] There is an association between LQTS and presentation with seizure phenotype.[216,217]

Guidance has been published for the screening of those at risk of sudden death including the screening of athletes. Screening programmes for athletes vary between countries.[218,219] Identification of individuals with inherited conditions and screening of family members can help prevent deaths in young people with inherited heart disorders.[220–222]

3b – Prehospital resuscitation

This section provides an overview of prehospital resuscitation. Many of the specific issues about prehospital resuscitation are addressed in sections covering ALS interventions, or are generic for both resuscitation for in-hospital and out-of-hospital cardiac arrest.[223] Adult BLS and automated external defibrillation contains guidance on the techniques used during the initial resuscitation of an adult cardiac arrest victim. In addition, many of the specific situations associated with cardiac arrest that are encountered in prehospital resuscitation are addressed in Section 4 – cardiac arrest in special circumstances.[224]

EMS personnel and interventions

There is considerable variation across Europe in the structure and process of emergency medical services (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. Although some studies have documented higher survival rates after cardiac arrest in EMS systems that include experienced physicians,[225–232] compared with those that rely on non-physician providers,[225,226,233,234] some other comparisons have found no difference in survival between systems using paramedics or physicians as part of the response.[235–237] Well-organised non-physician systems with highly trained paramedics have also reported high survival rates.[238] Given the inconsistent evidence, the inclusion or exclusion of physicians among prehospital personnel responding to cardiac arrests will depend largely on existing local policy.

Whether ALS interventions by EMS improve outcomes is also uncertain. A meta-analysis suggested that ALS care can increase survival in non-traumatic OHCA.[239] However, a recent large

observational study using propensity matching showed survival to hospital discharge and 90 day survival was greater among patients receiving BLS.[240] It is not possible to say whether this is a true difference or the result of unmeasured confounders.

CPR versus defibrillation first for out-of-hospital cardiac arrest

There is evidence that performing chest compressions while retrieving and charging a defibrillator improves the probability of survival.[241] One randomised controlled trial (RCT)[242] found increased ROSC, discharge- and one-year survival in patients with longer arrest times (>5 min). However, we have to keep in mind that this, and a large before-after study from Seattle[243] that showed better outcomes with 90 s of CPR before a shock when the response interval was >4 min), are from a time when 3 stacked-shocks were used and shorter periods of CPR between shocks (1 min). Evidence from five RCTs[242,244–247] and another study[248] suggests that among unmonitored patients with OHCA and an initial rhythm of VF/pVT, there is no benefit in a period of CPR of 90–180 s before defibrillation when compared with immediate defibrillation with CPR being performed while the defibrillator equipment is being applied.

A sub-analysis in one RCT[245] showed no difference in survival to hospital discharge with a prolonged period of CPR (180 s) and delayed defibrillation in patients with a shockable initial rhythm who received bystander CPR. Yet, for those EMS agencies with a higher baseline survival to hospital discharge (defined as >20% for an initial shockable rhythm), 180 s of CPR prior to defibrillation was more beneficial compared with a shorter period of CPR (30–60 s).

EMS personnel should provide high-quality CPR while a defibrillator is retrieved, applied and charged. Defibrillation should not be delayed longer than needed to establish the need for defibrillation and charging. The routine delivery of a pre-specified period of CPR (e.g. 2 or 3 min) before rhythm analysis and a shock is delivered is not recommended.

Termination of resuscitation rules

The 'basic life support termination of resuscitation rule' is predictive of death when applied by defibrillation-only emergency medical technicians.[249] The rule recommends termination when there is no ROSC, no shocks are administered and EMS personnel do not witness the arrest. Several studies have shown external generalisability of this rule.[250–256] More recent studies show that EMS systems providing ALS interventions can also use this BLS rule and therefore termed it the 'universal' termination of resuscitation rule.[251,257,258]

Additional studies have shown associations with futility of certain variables such as no ROSC at scene; non-shockable rhythm; unwitnessed arrest; no bystander CPR, call response time and patient demographics.[259–267]

Termination of resuscitation rules for in-hospital cardiac arrest are less reliable although EMS rules may be useful for those with out-of-hospital cardiac arrest who have ongoing resuscitation in the emergency department.[268–271]

Prospectively validated termination of resuscitation rules can be used to guide termination of prehospital CPR in adults; however, these must be validated in an EMS system similar to the one in which implementation is proposed. Termination of resuscitation rules may require integration with guidance on suitability for extracorporeal CPR (eCPR) or organ donation.[272] Organ donation is specifically addressed in Section 5 – Post-resuscitation care.[273,274]

3c – In-hospital resuscitation

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

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

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

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

Location

Patients who have monitored arrests are usually diagnosed rapidly. Ward patients may have had a period of deterioration and an unwitnessed arrest.[9,11] Ideally, all patients who are at high risk of cardiac arrest should be cared for in a monitored area where facilities for immediate resuscitation are available.

Training of first responders

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

Number of responders

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

Equipment available

All clinical areas should have immediate access to resuscitation equipment and drugs to facilitate rapid resuscitation of the patient in cardiopulmonary arrest. Ideally, the equipment used for CPR (including defibrillators) and the layout of equipment and drugs should be standardised throughout the hospital.[275–277] Equipment should be checked regularly, e.g. daily, to ensure its readiness for use in an emergency.

Resuscitation team

The resuscitation team may take the form of a traditional cardiac arrest team, which is called only when cardiac arrest is recognised. Alternatively, hospitals may have strategies to recognise patients at risk of cardiac arrest and summon a team (e.g. MET or RRT) before cardiac arrest occurs. The term 'resuscitation team' reflects the range of response teams. In hospital cardiac arrests are rarely sudden or unexpected. A strategy of recognising patients at risk of cardiac arrest may enable some of these arrests to be prevented, or

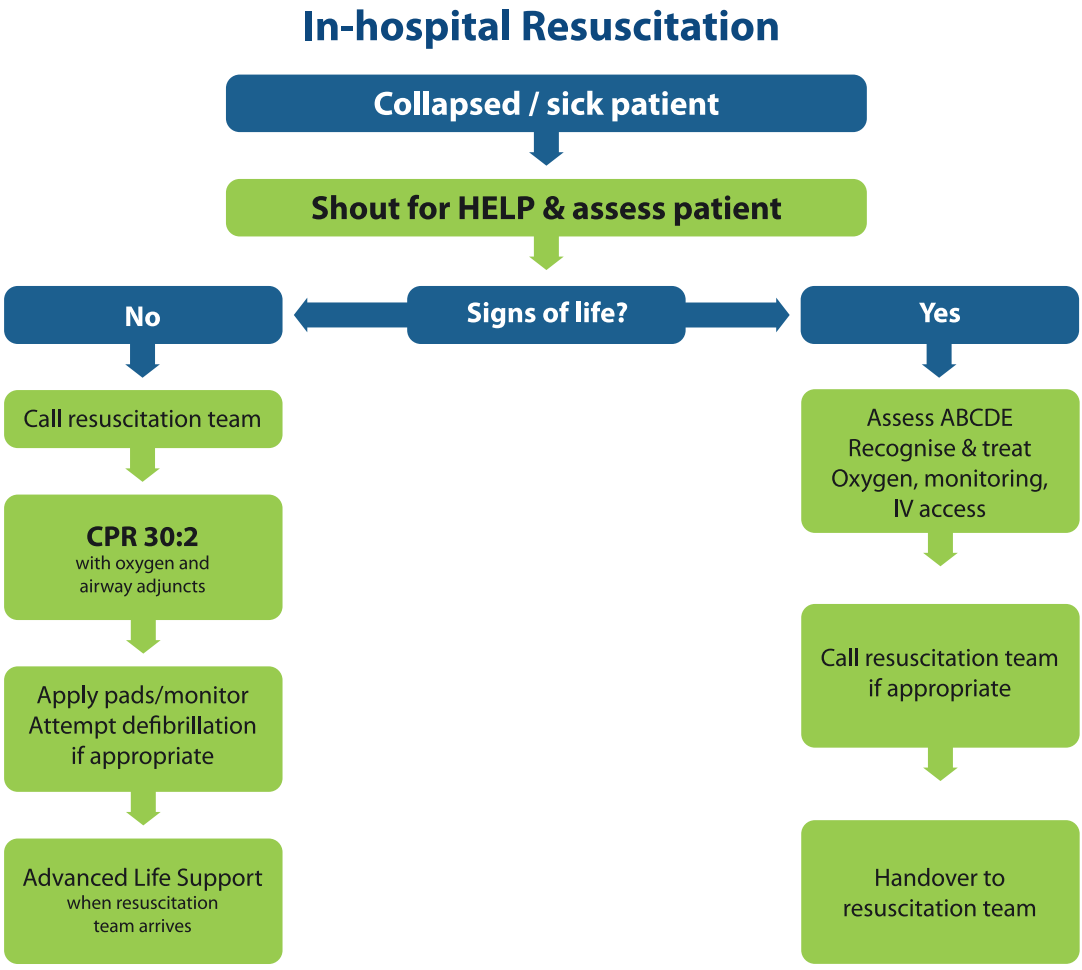


Fig. 3.1. In-hospital resuscitation algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure; IV – intravenous; CPR – cardiopulmonary resuscitation

may prevent futile resuscitation attempts in those who are unlikely to benefit from CPR.

Immediate actions for a collapsed patient in a hospital

An algorithm for the initial management of in-hospital cardiac arrest is shown in Fig. 3.1.

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

The responsive patient

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

The unresponsive patient

The exact sequence will depend on the training of staff and experience in assessment of breathing and circulation. Trained healthcare staff cannot assess the breathing and pulse sufficiently reliably to confirm cardiac arrest.[278–287]

Agonal breathing (occasional gasps, slow, laboured or noisy breathing) is common in the early stages of cardiac arrest and is a sign of cardiac arrest and should not be confused as a sign of life.[288–291] Agonal breathing can also occur during chest compressions as cerebral perfusion improves, but is not indicative of ROSC. Cardiac arrest can cause an initial short seizure-like episode that can be confused with epilepsy.[292,293] Finally changes in skin colour, notably pallor and bluish changes associated with cyanosis are not diagnostic of cardiac arrest.[292]

- Shout for help (if not already)
- Turn the victim on to his back and then open the airway:
- Open airway and check breathing:
  - Open the airway using a head tilt chin lift
  - Keeping the airway open, look, listen and feel for normal breathing (an occasional gasp, slow, laboured or noisy breathing is not normal):
    - Look for chest movement
    - Listen at the victim's mouth for breath sounds
    - Feel for air on your cheek
  - Look, listen and feel for no more than 10 s to determine if the victim is breathing normally.
- Check for signs of a circulation:
  - It may be difficult to be certain that there is no pulse. If the patient has no signs of life (consciousness, purposeful

movement, normal breathing, or coughing), or if there is doubt, start CPR immediately until more experienced help arrives or the patient shows signs of life.
  - Delivering chest compressions to a patient with a beating heart is unlikely to cause harm.[294] However, delays in diagnosing cardiac arrest and starting CPR will adversely effect survival and must be avoided.
  - Only those experienced in ALS should try to assess the carotid pulse whilst simultaneously looking for signs of life. This rapid assessment should take no more than 10 s. Start CPR if there is any doubt about the presence or absence of a pulse.
- If there are signs of life, urgent medical assessment is required. Depending on the local protocols, this may take the form of a resuscitation team. While awaiting this team, give the patient oxygen, attach monitoring and insert an intravenous cannula. When a reliable measurement of oxygen saturation of arterial blood (e.g. pulse oximetry (SpO_2)) can be achieved, titrate the inspired oxygen concentration to achieve a SpO_2 of 94–98%.
- If there is no breathing, but there is a pulse (respiratory arrest), ventilate the patient's lungs and check for a circulation every 10 breaths. Start CPR if there is any doubt about the presence or absence of a pulse.

Starting in-hospital CPR

The key steps are listed here. Supporting evidence can be found in the sections on specific interventions that follow.

- 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.
- Compress to a depth of at least 5 cm but not more than 6 cm.
- Perform chest compressions should be performed at a rate of 100–120 min^{-1}.
- Allow the chest to recoil completely after each compression; do not lean on the chest.
- Minimise interruptions and ensure high-quality compressions.
- Undertaking high-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. Pocket mask ventilation or two-rescuer bag-mask ventilation, which can be supplemented with an oral airway, should be started. Alternatively, use a supraglottic airway device (SGA) and self-inflating bag. Tracheal intubation should be attempted only by those who are trained, competent and experienced in this skill.
- Waveform capnography must be used for confirming tracheal tube placement and monitoring ventilation rate. Waveform capnography can also be used with a bag-mask device and SGA. The further use of waveform capnography to monitor CPR quality and potentially identify ROSC during CPR is discussed later in this section.[295]
- Use an inspiratory time of 1 s and give enough volume to produce a normal chest rise. Add supplemental oxygen to give the highest feasible inspired oxygen as soon as possible.[4]
- Once the patient's trachea has been intubated or a SGA has been inserted, continue uninterrupted chest compressions (except for defibrillation or pulse checks when indicated) at a rate of 100–120 min^{-1} and ventilate the lungs at approximately 10 breaths min^{-1}. Avoid hyperventilation (both excessive rate and tidal volume).
- 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 unable to do this, do chest compressions until help or airway equipment arrives. The ALS Writing Group recognises that there can be good clinical reasons to avoid mouth-to-mouth ventilation in clinical settings, and it is not commonly used in clinical settings, but there will be situations where giving mouth-to-mouth breaths could be life-saving.
- When the defibrillator arrives, apply self-adhesive defibrillation pads to the patient whilst chest compressions continue and then briefly analyse the rhythm. If self-adhesive defibrillation pads are not available, use paddles. The use of self-adhesive electrode pads or a 'quick-look' paddles technique will enable rapid assessment of the heart rhythm compared with attaching ECG electrodes.[296] Pause briefly to assess the heart rhythm. With a manual defibrillator, if the rhythm is VF/pVT charge the defibrillator while another rescuer continues chest compressions. Once the defibrillator is charged, pause the chest compressions and then give one shock, and immediately resume chest compressions. Ensure no one is touching the patient during shock delivery. Plan and ensure safe defibrillation before the planned pause in chest compressions.
- If using an automated external defibrillator (AED) follow the AED's audio-visual prompts, and similarly aim to minimise pauses in chest compressions by rapidly following prompts.
- The ALS Writing Group recognises that in some settings where self-adhesive defibrillation pads are not available, alternative defibrillation strategies using paddles are used to minimise the preshock pause.
- The ALS writing group is aware that in some countries a defibrillation strategy that involves charging the defibrillator towards the end of every 2 min cycle of CPR in preparation for the pulse check is used.[297,298] If the rhythm is VF/pVT a shock is given and CPR resumed. Whether this leads to any benefit is unknown, but it does lead to defibrillator charging for non-shockable rhythms.
- Restart chest compressions immediately after the defibrillation attempt. Minimise interruptions to chest compressions. When 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.
- 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).[111,112] Locate the patient's records.
- The quality of chest compressions during in-hospital CPR is frequently sub-optimal.[299,300] The importance of uninterrupted chest compressions cannot be over emphasised. Even short interruptions to chest compressions are disastrous for outcome and every effort must be made to ensure that continuous, effective chest compression is maintained throughout the resuscitation attempt. Chest compressions should commence at the beginning of a resuscitation attempt and continue uninterrupted unless they are paused briefly for a specific intervention (e.g. rhythm check). Most interventions can be performed without interruptions to chest compressions. The team leader should monitor the quality of CPR and alternate CPR providers if the quality of CPR is poor.
- Continuous $ETCO_2$ monitoring during CPR can be used to indicate the quality of CPR, and a rise in $ETCO_2$ can be an indicator of ROSC during chest compressions.[295,301–303]
- If possible, the person providing chest compressions should be changed every 2 min, but without pauses in chest compressions.

3d – ALS treatment algorithm

Introduction

Heart rhythms associated with cardiac arrest are divided into two groups: shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)) and non-shockable rhythms (asystole and pulseless electrical activity (PEA)). The principal difference in the treatment of these two groups of arrhythmias is the need for attempted defibrillation in those patients with VF/pVT. Other interventions, including high-quality chest compressions with minimal interruptions, airway management and ventilation, venous access, administration of adrenaline and the identification and correction of reversible causes, are common to both groups.

Although the ALS cardiac arrest algorithm (Fig. 3.2) is applicable to all cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances (see Section 4).[224]

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander basic life support (BLS), uninterrupted, high-quality chest compressions and early defibrillation for VF/pVT. The use of adrenaline has been shown to increase ROSC but not survival to discharge. Furthermore there is a possibility that it causes worse long-term neurological survival. Similarly, the evidence to support the use of advanced airway interventions during ALS remains limited.[4,304–311]

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 an indicator of equipoise for many ALS interventions at the time of writing these guidelines, three large RCTs (adrenaline versus placebo [ISRCTN73485024], amiodarone versus lidocaine versus placebo[312] [NCT01401647] and SGA versus tracheal intubation [ISRCTN No: 08256118]) are currently ongoing.

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 injected every 3–5 min until ROSC is achieved – the timing of the initial dose of adrenaline is described below. In VF/pVT, a single dose of amiodarone 300 mg is indicated after a total of three shocks and a further dose of 150 mg can be considered after five shocks. The optimal CPR cycle time is not known and algorithms for longer cycles (3 min) exist which include different timings for adrenaline doses.[313]

Duration of resuscitation attempt

The duration of any individual resuscitation attempt should be based on the individual circumstances of the case and is a matter of clinical judgement, taking into consideration the circumstances and the perceived prospect of a successful outcome. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing, as long as the patient remains in VF/pVT, or there is a potentially reversible cause than can be treated. The use of mechanical compression devices and extracorporeal CPR techniques make prolonged attempts at resuscitation feasible in selected patients.

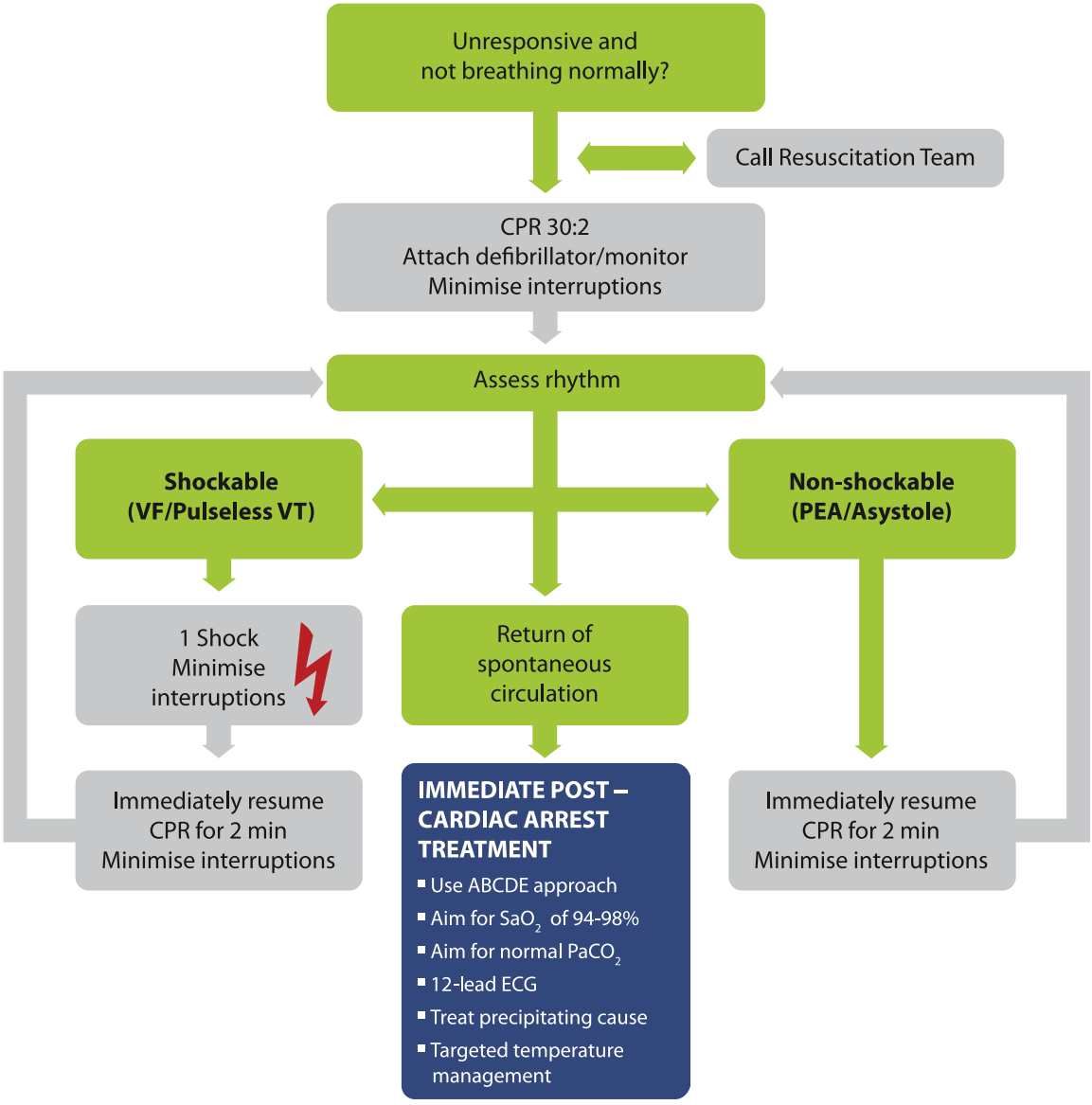
In a large observational study of patients with IHCA, the median duration of resuscitation was 12 min (IQR 6–21 min) in those with ROSC compared with 20 min (IQR 14–30 min) for those with no ROSC.[314] Hospitals with the longest resuscitation attempts (median 25 min [IQR 25–28 min]) had a higher risk-adjusted rate of ROSC and survival to discharge compared with a shorter median duration of resuscitation attempt.[314,315] It is generally accepted that asystole for more than 20 min in the absence of a reversible cause and with ongoing ALS constitutes a reasonable ground for stopping further resuscitation attempts.[316] The ethical principles of starting and stopping CPR are addressed in Section 11, the Ethics of resuscitation and end-of-life decisions.[184]

Shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia)

The first monitored rhythm is VF/pVT in approximately 20% both for in-hospital[317,7,318,319] and out-of-hospital cardiac arrests.[320] The incidence of VF/pVT may be decreasing,[321–324] and can vary according to bystander CPR rates. Ventricular fibrillation/pulseless ventricular tachycardia will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA.[317,325] Having confirmed cardiac arrest, summon help (including the request for a defibrillator) and start CPR, beginning with chest compressions, with a compression: ventilation (CV) ratio of 30:2. When the defibrillator arrives, continue chest compressions while applying the defibrillation electrodes. Identify the rhythm and treat according to the ALS algorithm.

- If VF/pVT 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.
- Defibrillation shock energy levels are unchanged from the 2010 guidelines.[2] For biphasic waveforms (rectilinear biphasic or biphasic truncated exponential), use an initial shock energy of at least 150 J. For pulsed biphasic waveforms, begin at 120–150 J. The shock energy for a particular defibrillator should be based on the manufacturer's guidance. It is important that those using manual defibrillators are aware of the appropriate energy settings for the type of device used. Manufacturers should consider labelling their manual defibrillators with energy level instructions, but in the absence of this and if appropriate energy levels are unknown, for adults use the highest available shock energy for all shocks. With manual defibrillators it is appropriate to consider escalating the shock energy if feasible, after a failed shock and for patients where refibrillation occurs.[326,327]
- Minimise the delay between stopping chest compressions and delivery of the shock (the preshock pause); even a 5–10 s delay will reduce the chances of the shock being successful.[328–331]
- Without pausing to reassess the rhythm or feel for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions to limit the post-shock pause and the total peri-shock pause.[330,331] Even if the defibrillation attempt is successful in restoring a perfusing rhythm, it takes time to establish a post shock circulation[332] and it is very rare for a pulse to be palpable immediately after defibrillation.[333] In one study, after defibrillation attempts, most patients having ALS remained pulseless for over 2 min and the duration of asystole before ROSC was longer than 2 min beyond the shock in as many as 25%.[334] If a shock has been successful immediate resumption of chest compressions does not increase the risk of VF recurrence.[335] Furthermore, the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.[336]
- Continue CPR for 2 min, then pause briefly to assess the rhythm; if still VF/pVT, give a second shock (150–360 J biphasic). Without pausing to reassess the rhythm or feel 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/pVT, give a third shock (150–360 J biphasic). Without reassessing the rhythm or feeling for a pulse, resume CPR (CV

Advanced Life Support



DURING CPR

- Ensure high quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Continuous compressions when advanced airway in place
- Vascular access (intravenous or intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks

TREAT REVERSIBLE CAUSES

Hypoxia	Thrombosis – coronary or pulmonary
Hypovolaemia	Tension pneumothorax
Hypo-/hyperkalaemia/metabolic	Tamponade – cardiac
Hypothermia/hyperthermia	Toxins

CONSIDER

- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR

Fig. 3.2. Advanced life support algorithm. CPR – cardiopulmonary resuscitation; VF/Pulseless VT – ventricular fibrillation/pulseless ventricular tachycardia; PEA – pulseless electrical activity; ABCDE – Airway, Breathing Circulation, Disability, Exposure; SaO_2 – oxygen saturation; $PaCO_2$ – partial pressure carbon dioxide in arterial blood; ECG – electrocardiogram.

ratio 30:2) immediately after the shock, starting with chest compressions.
- If IV/IO access has been obtained, during the next 2 min of CPR give adrenaline 1 mg and amiodarone 300 mg.[337]
- The use of waveform capnography may enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. Several human studies have shown that there is a significant increase in end-tidal CO_2 when ROSC occurs.[295,301–303,338,339] If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- If ROSC has not been achieved with this 3rd shock, the adrenaline may improve myocardial blood flow and 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 and the maximum effect on coronary perfusion pressure is achieved around the same time (70 s).[340] Importantly, high-quality chest compressions are needed to circulate the drug to achieve these times.
- Timing of adrenaline dosing can cause confusion amongst ALS providers and this aspect needs to be emphasised during training.[341] Training should emphasise that giving drugs must not lead to interruptions in CPR and delay interventions such as defibrillation. Human data suggests drugs can be given without affecting the quality of CPR.[305]
- 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 feel a pulse. Ensure that rhythm checks are brief, and pulse checks are 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, immediately resume CPR. If ROSC has been achieved, begin post-resuscitation care.

During treatment of VF/pVT, healthcare providers must practice efficient coordination between CPR and shock delivery whether using a manual defibrillator or an AED. When VF is present for more than a few minutes, the myocardium is depleted of oxygen and metabolic substrates. A brief period of chest compressions will deliver oxygen and energy substrates and increase the probability of restoring a perfusing rhythm after shock delivery.[342] Analyses of VF waveform characteristics predictive of shock success indicate that the shorter the time between chest compression and shock delivery, the more likely the shock will be successful.[342,343] Reduction in the peri-shock pause (the interval between stopping compressions to resuming compressions after shock delivery) by even a few seconds can increase the probability of shock success.[328–331] Moreover, continuing high-quality CPR however may improve the amplitude and frequency of the VF and improve the chance of successful defibrillation to a perfusing rhythm.[344–346]

Regardless of the arrest rhythm, after the initial adrenaline dose has been given, give further doses of adrenaline 1 mg every 3–5 min until ROSC is achieved; in practice, this will be about once every two cycles of the algorithm. If signs of life return during CPR (purposeful movement, normal breathing or coughing), or there is an increase in $ETCO_2$, check the monitor; if an organised rhythm is present, check for a pulse. If a pulse is palpable, start post-resuscitation care. If no pulse is present, continue CPR.

Witnessed, monitored VF/pVT

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:
- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and, if appropriate, ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

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

If this initial three-shock strategy is unsuccessful for a monitored VF/pVT cardiac arrest, the ALS algorithm should be followed and these three-shocks treated as if only the first single shock has been given.

The first dose of adrenaline should be given after another 2 shock attempts if VF persists, i.e. Give 3 shocks, then 2 min CPR, then shock attempt, then 2 min CPR, then shock attempt, and then consider adrenaline during this 2 min of CPR. We recommend amiodarone is given after three defibrillation attempts irrespective of whether they are consecutive shocks, or interrupted by CPR and non-shockable rhythms.

Specific guidance concerning the need for resternotomy, and drug timing if the initial stacked shocks are unsuccessful when cardiac arrest occurs after cardiac surgery is addressed in Section 4 – cardiac arrest in special circumstances.[224]

Persistent ventricular fibrillation/pulseless ventricular tachycardia

In VF/pVT persists, consider changing the position of the pads/paddles.[2] Review all potentially reversible causes using the 4 H and 4 T approach (see below) and treat any that are identified. Persistent VF/pVT may be an indication for percutaneous coronary intervention (PCI) – in these cases, a mechanical chest compression device can be used to maintain high-quality chest compressions for transport and PCI.[347] The use of extracorporeal CPR (see below) should also be considered to support the circulation whilst a reversible cause it treated.

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm.[348–352] Its routine use is therefore not recommended. It may be appropriate therapy only when used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest.[353] Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, then retract the fist immediately to create an impulse-like stimulus. There are rare reports of a precordial thump converting a perfusing to a non-perfusing rhythm.[354]

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. 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. Tracheal intubation must not delay defibrillation attempts. 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 be less than 5 s. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt

may be deferred until ROSC. No RCTs 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 100–120 min^{-1} without pausing during ventilation. A pause in the chest compressions causes the coronary perfusion pressure to fall substantially. On resuming compressions, there is some delay before the original coronary perfusion pressure is restored, thus chest compressions that are not interrupted for ventilation (or any reason) result in a substantially higher mean coronary perfusion pressure.

In the absence of personnel skilled in tracheal intubation, a supraglottic airway (SGA) (e.g. laryngeal mask airway, laryngeal tube or i-gel) is an acceptable alternative. Once a SGA has been inserted, attempt to deliver continuous chest compressions, uninterrupted by ventilation.[355] 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). Airway interventions for cardiac arrest and the evidence supporting them are described in Section 3f.

Intravenous access and drugs

Peripheral versus central venous drug delivery. Establish intravenous access if this has not already been achieved. Although peak drug concentrations are higher and circulation times are shorter when drugs are injected into a central venous catheter compared with a peripheral cannula,[356] insertion of a central venous catheter requires interruption of CPR and can be technically challenging and associated with complications. Peripheral venous cannulation is quicker, easier to perform and safer. Drugs injected peripherally must be followed by a flush of at least 20 ml of fluid and elevation of the extremity for 10–20 s to facilitate drug delivery to the central circulation.

Intraosseous route. If intravenous access is difficult or impossible, consider the IO route. This is now established as an effective route in adults.[357–365] Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a vein.[366,367] Animal studies suggest that adrenaline reaches a higher concentration and more quickly when it is given intravenously as compared with the intraosseous route, and that the sternal intraosseous route more closely approaches the pharmacokinetic of IV adrenaline.[368] The recent availability of mechanical IO devices has increased the ease of performing this technique.[369] There are a number of intraosseous devices available as well as a choice of insertion sites including the humerus, proximal or distal tibia, and sternum. We have not done a formal review of devices or insertion sites as part of the 2015 Guidelines process. The decision concerning choice of device and insertion site should be made locally and staff adequately trained in its use.

Adrenaline for initial VF/pVT arrest. On the basis of expert consensus, for VF/pVT 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. The use of waveform capnography may enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. If ROSC is suspected during CPR, withhold adrenaline and continue CPR. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

Despite the widespread use of adrenaline during resuscitation, 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. Further information concerning the role of adrenaline in cardiac arrest is addressed in Section 3g – drugs and fluids during CPR.

Anti-arrhythmic drugs. We recommend that amiodarone should be given after three defibrillation attempts irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest. Give amiodarone 300 mg intravenously; a further dose of 150 mg may be given after five defibrillation attempts. 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. Further information concerning the role of amiodarone in cardiac arrest is addressed in Section 3g – drugs and fluid during CPR.

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity (other than ventricular tachyarrhythmia) that would normally be associated with a palpable pulse.[370] These patients often have some mechanical myocardial contractions, but these are too weak to produce a detectable pulse or blood pressure – this is sometimes described as 'pseudo-PEA' (see below). PEA is often caused by reversible conditions, and can be treated if those conditions are identified and corrected. Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively.

If the initial monitored rhythm is PEA or asystole, start CPR 30:2. If asystole is displayed, without stopping CPR, check 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 as soon as venous or intraosseous access is achieved, and repeat every alternate CPR cycle (i.e. about every 3–5 min). If a pulse is present, begin post-resuscitation care. If signs of life return during CPR, check the rhythm and check for a pulse. If ROSC is suspected during CPR withhold adrenaline and continue CPR. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves, because this may respond to cardiac pacing. There is no benefit in attempting to pace true asystole. In addition, if there is doubt about whether the rhythm is asystole or extremely fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Continuing high-quality CPR however may improve the amplitude and frequency of the VF and improve the chance of successful defibrillation to a perfusing rhythm.[344–346]

The optimal CPR time between rhythm checks may vary according to the cardiac arrest rhythm and whether it is the first or subsequent loop.[371] Based on expert consensus, for the treatment of asystole or PEA, following a 2-min cycle of CPR, if the rhythm has changed to VF, follow the algorithm for shockable rhythms. Otherwise, continue CPR and give adrenaline every 3–5 min following the failure to detect a palpable pulse with the pulse check. If VF is identified on the monitor midway through a 2-min cycle of CPR, complete the cycle of CPR before formal rhythm and shock delivery if appropriate – this strategy will minimise interruptions in chest compressions.

Potentially reversible causes

Potential causes or aggravating factors for which specific treatment exists must be considered during any cardiac arrest. For ease

of memory, these are divided into two groups of four, based upon their initial letter: either H or T. More details on many of these conditions are covered in Section 4 – special circumstances.[224]

The four 'Hs'

Minimise the risk of hypoxia by ensuring that the patient's lungs are ventilated adequately with the maximal possible inspired oxygen during CPR. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described in Section 3f, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by hypovolaemia is due usually to severe haemorrhage. This may be precipitated by trauma (Section 4),[224] gastrointestinal bleeding or rupture of an aortic aneurysm. Intravascular volume should be restored rapidly with warmed fluid, coupled with urgent surgery to stop the haemorrhage.

Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests (usually by using a blood gas analyser) or suggested by the patient's medical history, e.g. renal failure (Section 4).[224] Intravenous calcium chloride is indicated in the presence of hyperkalaemia, hypocalcaemia and calcium channel-blocker overdose.

Hypothermia should be suspected based on the history such as cardiac arrest associated with drowning (Section 4).[224]

The four 'Ts'

Coronary thrombosis associated with an acute coronary syndrome or ischaemic heart disease is the most common cause of sudden cardiac arrest. An acute coronary syndrome is usually diagnosed and treated after ROSC is achieved. If an acute coronary syndrome is suspected, and ROSC has not been achieved, urgent coronary angiography should be considered when feasible and if required percutaneous coronary intervention. Mechanical chest compression devices and extracorporeal CPR can help facilitate this.

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. The treatment of cardiac arrest with known or suspected pulmonary embolism is addressed in Section 4 including the role of fibrinolysis, surgical or mechanical thrombectomy and extracorporeal CPR.[224]

A tension pneumothorax may be the primary cause of PEA and may be associated with trauma or follow attempts at central venous catheter insertion. The diagnosis is made clinically or by ultrasound. Decompress rapidly by thoracostomy or needle thoracocentesis, and then insert a chest drain. In the context of cardiac arrest from major trauma, consider bilateral thoracostomies for decompression of a suspected tension pneumothorax (Section 4).[224]

Cardiac tamponade is difficult to diagnose because the typical signs of distended neck veins and hypotension are usually obscured by the arrest itself. Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for resuscitative thoracotomy (Section 4).[224] The use of ultrasound will make the diagnosis of cardiac tamponade much more reliable.

In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or toxic substances may be revealed only by laboratory investigations (Section 4).[224] Where available, the appropriate antidotes should be used, but most often treatment is supportive and standard ALS protocols should be followed.

Use of ultrasound imaging during advanced life support

Several studies have examined the use of ultrasound during cardiac arrest to detect potentially reversible causes.[372–374] 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. Specific protocols for ultrasound evaluation during CPR may help to identify potentially reversible causes (e.g. cardiac tamponade, pulmonary embolism, hypovolaemia, pneumothorax) and identify pseudo-PEA.[373,375–382] 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.[375,381,383] 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 although sensitivity and specificity has not been reported.[384–387]

Monitoring during advanced life support

There are a number of methods and emerging technologies to monitor the patient during CPR and potentially help guide ALS interventions. These include:

- Clinical signs such as breathing efforts, movements and eye opening can occur during CPR. These can indicate ROSC and require verification by a rhythm and pulse check, but can also occur because CPR can generate a sufficient circulation to restore signs of life including consciousness.[388]
- The use of CPR feedback or prompt devices during CPR is addressed in Section 2 – basic life support.[223] The use of CPR feedback or prompt devices during CPR should only be considered as part of a broader system of care that should include comprehensive CPR quality improvement initiatives [389,390] rather than an isolated intervention.
- Pulse checks when there is an ECG rhythm compatible with an output can be used to identify ROSC, but may not detect pulses in those with low cardiac output states and a low blood pressure.[391] The value of attempting to feel arterial pulses during chest compressions to assess the effectiveness of chest compressions is unclear. A pulse that is felt in the femoral triangle may indicate venous rather than arterial blood flow. There are no valves in the inferior vena cava and retrograde blood flow into the venous system can produce femoral vein pulsations.[392] Carotid pulsation during CPR does not necessarily indicate adequate myocardial or cerebral perfusion.
- ECG monitoring of heart rhythm. Monitoring heart rhythm through pads, paddles or ECG electrodes is a standard part of ALS. Motion artefacts prevent reliable heart rhythm assessment during chest compressions forcing rescuers to stop chest compressions to assess the rhythm, and preventing early recognition of recurrent VF/pVT. Some modern defibrillators have filters that remove artefact from compressions but there are no human studies showing improvements in patient outcomes from their use. We suggest against the routine use of artefact-filtering algorithms for analysis of ECG rhythm during CPR unless as part of a research programme.[393]
- End-tidal carbon dioxide with waveform capnography. The use of waveform capnography during CPR has a greater emphasis in Guidelines 2015 and is addressed in more detail below.
- Blood sampling and analysis during CPR can be used to identify potentially reversible causes of cardiac arrest. Avoid finger prick samples in critical illness because they may not be reliable; instead, use samples from veins or arteries.
- Blood gas values are difficult to interpret during CPR. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state.[394] Analysis of central venous blood may provide a better estimation of tissue pH.

Central venous oxygen saturation monitoring during ALS is feasible but its role in guiding CPR is not clear.

- Invasive cardiovascular monitoring in critical care settings, e.g. continuous arterial blood pressure and central venous pressure monitoring. Invasive arterial pressure monitoring will enable the detection of low blood pressure values when ROSC is achieved. Consider aiming for an aortic diastolic pressure of greater than 25 mmHg during CPR by optimising chest compressions.[395] In practice this would mean measuring an arterial diastolic pressure. Although haemodynamic-directed CPR showed some benefit in experimental studies[396–399] there is currently no evidence of improvement in survival with this approach in humans.[4]
- Ultrasound assessment is addressed above to identify and treat reversible causes of cardiac arrest, and identify low cardiac output states ('pseudo-PEA'). Its use has been discussed above.
- Cerebral oximetry using near-infrared spectroscopy measures regional cerebral oxygen saturation (rSO2) non-invasively.[400–402] This remains an emerging technology that is feasible during CPR. Its role in guiding CPR interventions including prognostication during and after CPR is yet to be established.[403]

Waveform capnography during advanced life support

End-tidal carbon dioxide is the partial pressure of carbon dioxide (CO_2) at the end of an exhaled breath. It reflects cardiac output and pulmonary blood flow, as CO_2 is transported by the venous system to the right side of the heart and then pumped to the lungs by the right ventricle, as well as the ventilation minute volume. During CPR, end-tidal CO_2 values are low, reflecting the low cardiac output generated by chest compression. Waveform capnography enables continuous real time end-tidal CO_2 to be monitored during CPR. It works most reliably in patients who have a tracheal tube, but can also be used with a supraglottic airway device or bag mask. There is currently no evidence that use of waveform capnography during CPR results in improved patient outcomes, although the prevention of unrecognised oesophageal intubation is clearly beneficial. The role of waveform capnography during CPR includes:

- Ensuring tracheal tube placement in the trachea (see below for further details).
- Monitoring ventilation rate during CPR and avoiding hyperventilation.
- Monitoring the quality of chest compressions during CPR. End-tidal CO_2 values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value.[404] Whether this can be used to guide care and improve outcome requires further study.[295] (Fig. 3.3)
- Identifying ROSC during CPR. An increase in end-tidal CO_2 during CPR may indicate ROSC and prevent unnecessary and potentially harmful dosing of adrenaline in a patient with ROSC.[295,301,338,339] If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- Prognostication during CPR. Lower end-tidal CO_2 values may indicate a poor prognosis and less chance of ROSC.[4] Precise values of end-tidal CO_2 depend on several factors including the cause of cardiac arrest, bystander CPR, chest compression quality, ventilation rate and volume, time from cardiac arrest and the use of adrenaline. Values are higher after an initial asphyxial arrest, with bystander CPR and decline over time after cardiac arrest.[295,302,405] Low end-tidal CO_2 values during CPR have been associated with lower ROSC rates and increased mortality, and high values with better ROSC and survival.[295,406,407] Failure to achieve an end-tidal CO_2 value >1.33 kPa (10 mmHg) after 20 min of CPR is associated with a poor outcome in observational studies.[4] In addition it has been used as a criterion for withholding extracorporeal life support in patients with refractory cardiac arrest.[408] The inter-individual differences and influence of cause of cardiac arrest, the problem with self-fulfilling prophecy in studies, our lack of

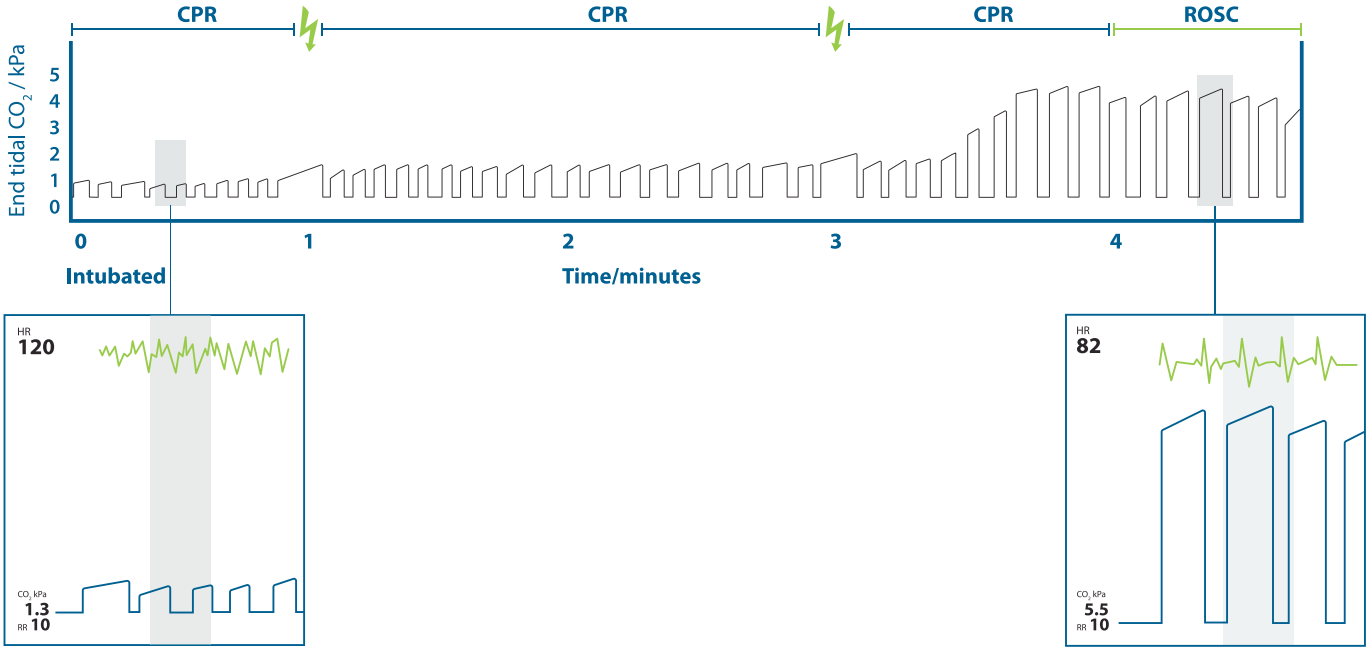


Fig. 3.3. *Waveform capnography* showing changes in the end-tidal carbon dioxide during CPR and after ROSC. The boxes show examples of monitor displays at the times indicated. In this example the patient's trachea is intubated at zero minutes. The patient is then ventilated at 10 breaths min^{-1} and given chest compressions (indicated by CPR) at about two per second. A minute after tracheal intubation, there is pause in chest compressions and ventilation followed by a defibrillation attempt, and chest compressions and ventilation then continue. Higher-quality chest compressions lead to an increased end-tidal carbon dioxide value. There is a further defibrillation attempt after two minutes of chest compressions. There are then further chest compressions and ventilation. There is a significant increase in the end-tidal carbon dioxide value during chest compressions and the patient starts moving and eye opening. Chest compressions are stopped briefly and there is a pulse indicating ROSC. Ventilation continues at 10 breaths min^{-1}. CPR – cardiopulmonary resuscitation; ROSC – return of spontaneous circulation; End tidal CO_2 – end-tidal carbon dioxide; HR – heart rate; RR – respiratory rate.

confidence in the accuracy of measurement during CPR, and the need for an advanced airway to measure end-tidal CO_2 reliably limits our confidence in its use for prognostication. Thus, we recommend that a specific end-tidal CO_2 value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO_2 values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

Extracorporeal cardiopulmonary resuscitation (eCPR)

Extracorporeal CPR (eCPR) should be considered as a rescue therapy for those patients in whom initial ALS measures are unsuccessful and, or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI) or pulmonary thrombectomy for massive pulmonary embolism).[409,410] There is an urgent need for randomised studies of eCPR and large eCPR registries to identify the circumstances in which it works best, establish guidelines for its use and identify the benefits, costs and risks of eCPR.[411,412]

Extracorporeal techniques require vascular access and a circuit with a pump and oxygenator and can provide a circulation of oxygenated blood to restore tissue perfusion. This has the potential to buy time for restoration of an adequate spontaneous circulation, and treatment of reversible underlying conditions. This is commonly called extracorporeal life support (ECLS), and more specifically extracorporeal CPR (eCPR) when used during cardiac arrest. These techniques are becoming more commonplace and have been used for both in-hospital and out-of-hospital despite limited observational data in select patient groups. Observational studies suggest eCPR for cardiac arrest is associated with improved survival when there is a reversible cause for cardiac arrest (e.g. myocardial infarction, pulmonary embolism, severe hypothermia, poisoning), there is little comorbidity, the cardiac arrest is witnessed, the individual receives immediate high-quality CPR, and eCPR is implemented early (e.g. within 1 h of collapse) including when instituted by emergency physicians and intensivists.[413–419] The implementation of eCPR requires considerable resource and training. When compared with manual or mechanical CPR, eCPR has been associated with improve survival after IHCA in selected patients.[413,415] After OHCA outcomes with both standard and eCPR are less favourable.[420] The duration of standard CPR before eCPR is established and patient selection are important factors for success.[409,413,417,419,421–423]

3e – Defibrillation

This section predominantly addresses the use of manual defibrillators. Guidelines concerning the use of an automated external defibrillator (AED) are addressed in Section 2 – Basic Life Support.[223] The defibrillation strategy for the 2015 European Resuscitation Council (ERC) guidelines has changed little from the former guidelines:

- The importance of early, uninterrupted chest compressions remains emphasised throughout these guidelines, together with minimising the duration of pre-shock and post-shock pauses.
- Continue chest compressions during defibrillator charging, deliver defibrillation with an interruption in chest compressions of no more than 5 s and immediately resume chest compressions following defibrillation.
- Self-adhesive defibrillation pads have a number of advantages over manual paddles and should always be used in preference when they are available.
- CPR should be continued while a defibrillator or automated external defibrillator (AED) is retrieved and applied but defibrillation should not be delayed longer than needed to establish the need for defibrillation and charging.
- The use of up to three-stacked shocks may be considered if initial VF/pVT occurs during a witnessed, monitored arrest with a defibrillator immediately available e.g. cardiac catheterisation.
- Although it is recognised that some geographic areas continue to use the older monophasic waveforms, they are not considered in this chapter. When possible, biphasic waveforms should be used in preference to the older monophasic waveform for the treatment of both atrial and ventricular arrhythmias. Defibrillation recommendations in these guidelines apply only to biphasic waveforms. For those using monophasic defibrillators, please refer to Guidelines 2010.[2]
- Defibrillation shock energy levels are unchanged from the 2010 guidelines.[2] For biphasic waveforms (rectilinear biphasic or biphasic truncated exponential), deliver the first shock with an energy of at least 150 J. For pulsed biphasic waveforms, begin at 120–150 J. The shock energy for a particular defibrillator should be based on the manufacturer's guidance. It is important that those using manual defibrillators are aware of the appropriate energy settings for the type of device used. Manufacturers should consider labelling their manual defibrillators with energy level instructions, but in the absence of this and if appropriate energy levels are unknown, for adults use the highest available shock energy for all shocks. With manual defibrillators it is appropriate to consider escalating the shock energy if feasible, after a failed shock and for patients where refibrillation occurs.[326,327]

There are no high-quality clinical studies to indicate the optimal strategies within any given waveform and between different waveforms.[4] Knowledge gaps include the minimal acceptable first-shock energy level; the characteristics of the optimal biphasic waveform; the optimal energy levels for specific waveforms; and the best shock strategy (fixed versus escalating). It is becoming increasingly clear that selected energy is a poor comparator with which to assess different waveforms as impedance-compensation and subtleties in waveform shape result in significantly different transmyocardial current between devices for any given selected energy. The optimal energy levels may ultimately vary between different manufacturers and associated waveforms. Manufacturers are encouraged to undertake high-quality clinical trials to support their defibrillation strategy recommendations.

Strategies for 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.[328–331,424,425] The pre-shock pause can 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.[297,426] The safety check to avoid rescuer contact with the patient at the moment of defibrillation should be undertaken rapidly but efficiently. The post shock pause is minimised by resuming chest compressions immediately after shock delivery (see below). The entire process of manual defibrillation should be achievable with less than a 5 s interruption to chest compressions.

Hands-on defibrillation

By allowing continuous chest compressions during the delivery of the defibrillation shock, hands-on defibrillation can minimise peri-shock pause and allow continuation of chest compressions during defibrillation. The benefits of this approach are not proven and further studies are required to assess the safety and efficacy of this technique. A recent study did not observe a benefit when

shocks were delivered without pausing manual or mechanical chest compressions.[427] Standard clinical examination gloves (or bare hands) do not provide a safe level of electrical insulation for hands-on defibrillation.[428]

Safe use of oxygen during defibrillation

In an oxygen-enriched atmosphere, sparking from poorly applied defibrillator paddles can cause a fire and significant burns to a patient.[429–434] The absence of case reports of fires caused by sparking where defibrillation was delivered using self-adhesive defibrillation pads suggests that the latter minimise the risk of electrical arcing and should always be used when possible.

- The risk of fire during attempted defibrillation can be minimised by taking the following precautions:
- Take off any oxygen mask or nasal cannulae and place them at least 1 m away from the patient's chest.
- Leave the ventilation bag connected to the tracheal tube or supraglottic airway, ensuring that there is no residual PEEP remaining in the circuit.
- If the patient is connected to a ventilator, for example in the operating room or critical care unit, leave the ventilator tubing (breathing circuit) connected to the tracheal tube unless chest compressions prevent the ventilator from delivering adequate tidal volumes. In this case, the ventilator is usually substituted by a ventilation bag, which can itself be left connected. If not in use, switch off the ventilator to prevent venting large volumes of oxygen into the room or alternatively connect it to a test lung. During normal use, when connected to a tracheal tube, oxygen from a ventilator in the critical care unit will be vented from the main ventilator housing well away from the defibrillation zone. Patients in the critical care unit may be dependent on positive end expiratory pressure (PEEP) to maintain adequate oxygenation; during cardioversion, when the spontaneous circulation potentially enables blood to remain well oxygenated, it is particularly appropriate to leave the critically ill patient connected to the ventilator during shock delivery.

The technique for electrode contact with the chest

The techniques described below aim to place external defibrillation electrodes (self-adhesive pads) in an optimal position using techniques that minimise transthoracic impedance.

Electrode position

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

More patients are presenting with implantable medical devices (e.g. permanent pacemaker, implantable cardioverter defibrillator (ICD)). Medic alert bracelets are recommended for these patients. These devices may be damaged during defibrillation if current is discharged through electrodes placed directly over the device.[435,436] Place the electrode away from the device (at least 8 cm) or use an alternative electrode position (anterior–lateral, anterior–posterior) as described below.[435]

Placement for ventricular arrhythmias and cardiac arrest. Place electrodes (either pads or paddles) in the conventional sternal–apical position. The right (sternal) electrode is placed to the right of the sternum, below the clavicle. The apical paddle is placed in the left mid-axillary line, approximately level with the V6 ECG electrode. This position should be clear of any breast tissue.[437] It is important that this electrode is placed sufficiently laterally. Other acceptable pad positions include

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

It does not matter which electrode (apex/sternum) is placed in either position. The long axis of the apical paddle should be orientated in a cranio-caudal direction to minimise transthoracic impedance.[438]

Placement for atrial arrhythmias. Atrial fibrillation is maintained by functional re-entry circuits anchored in the left atrium. As the left atrium is located posteriorly in the thorax, electrode positions that result in a more posterior current pathway may theoretically be more effective for atrial arrhythmias. Although some studies have shown that antero-posterior electrode placement is more effective than the traditional antero-apical position in elective cardioversion of atrial fibrillation,[439,440] the majority have failed to demonstrate any clear advantage of any specific electrode position.[441–444] Efficacy of cardioversion may be less dependent on electrode position when using biphasic impedance-compensated waveforms.[443–445] The following electrode positions all appear safe and effective for cardioversion of atrial arrhythmias:

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

Respiratory phase

Transthoracic impedance varies during respiration, being minimal at end-expiration. If possible, defibrillation should be attempted at this phase of the respiratory cycle. Positive end expiratory pressure (PEEP) increases transthoracic impedance and should be minimised during defibrillation. Auto-PEEP (gas trapping) may be particularly high in asthmatics and may necessitate higher than usual energy levels for defibrillation.[446]

Fibrillation waveform analysis

It is possible to predict, with varying reliability, the success of defibrillation from the fibrillation waveform.[342,343,447–467] If optimal defibrillation waveforms and the optimal timing of shock delivery can be determined in prospective studies, it should be possible to prevent the delivery of unsuccessful high energy shocks and minimise myocardial injury. This technology is under active development and investigation but current sensitivity and specificity is insufficient to enable introduction of VF waveform analysis into clinical practice.

CPR versus defibrillation as the initial treatment

This aspect has been dealt with in detail above in 4b – prehospital resuscitation. Rescuers should provide high-quality CPR while a defibrillator is retrieved, applied and charged. Do not delay defibrillation longer than needed to establish the need for defibrillation and charging. The routine delivery of a pre-specified period of CPR (e.g. 2 or 3 min) before rhythm analysis and a shock is delivered is not recommended.

One shock versus three stacked shock sequence

In 2010, it was recommended that when defibrillation was required, a single shock should be provided with immediate resumption of chest compressions after the shock.[468,469] This recommendation was made for two reasons. Firstly in an attempt to minimise peri-shock interruptions to chest compressions and secondly because it was felt that with the greater efficacy of biphasic shocks, if a biphasic shock failed to defibrillate, a further period of chest compressions could be beneficial.

Studies since 2010 have not shown that any specific shock strategy is of benefit for any survival end-point.[470,471] There is no conclusive evidence that a single shock strategy is of benefit for ROSC or recurrence of VF compared with three stacked shocks, but in view of the evidence suggesting that outcome is improved by minimising interruptions to chest compressions, we continue to recommend single shocks for most situations.

When defibrillation is warranted, give a single shock and resume chest compressions immediately following the shock. Do not delay CPR for rhythm reanalysis or a pulse check immediately after a shock. Continue CPR (30 compressions: 2 ventilations) for 2 min until rhythm reanalysis is undertaken and another shock given (if indicated). Even if the defibrillation attempt is successful, it takes time until the post shock circulation is established[332] and it is very rare for a pulse to be palpable immediately after defibrillation.[333] Patients can remain pulseless for over 2 min and the duration of asystole before ROSC can be longer than 2 min in as many as 25% of successful shocks.[334]

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

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and if appropriate ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

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

Waveforms

Biphasic waveforms, are now well established as a safe and effective waveform for defibrillation. Biphasic defibrillators compensate for the wide variations in transthoracic impedance by electronically adjusting the waveform magnitude and duration to ensure optimal current delivery to the myocardium, irrespective of the patient's size (impedance compensation). There are two main types of biphasic waveform: the biphasic truncated exponential (BTE) and rectilinear biphasic (RLB). A pulsed biphasic waveform is also in clinical use, in which the current rapidly oscillates between baseline and a positive value before inverting in a negative pattern. It may have a similar efficacy as other biphasic waveforms, but the single clinical study of this waveform was not performed with an impedance compensating waveform, which is used in the commercially available product.[472,473]

We recommend that a biphasic waveform is used for cardioversion of both atrial and ventricular arrhythmias in preference to a monophasic waveform. We place a high value on the reported higher first shock success rate for termination of fibrillation with a biphasic waveform, the potential for less post shock myocardial dysfunction and the existing 2010 Guidelines.[1,2,468,469] We acknowledge that many emergency medical services (EMS) systems and hospitals continue to use older monophasic devices. For those using monophasic defibrillators, please refer to Guidelines 2010.[2]

Energy levels

Defibrillation requires the delivery of sufficient electrical energy to defibrillate a critical mass of myocardium, abolish the wavefronts of VF and enable restoration of spontaneous synchronised electrical activity in the form of an organised rhythm. The optimal energy for defibrillation is that which achieves defibrillation whilst causing the minimum of myocardial damage.[474] Selection of an appropriate energy level also reduces the number of repetitive shocks, which in turn limits myocardial damage.[475]

Optimal energy levels for defibrillation are unknown. The recommendations for energy levels are based on a consensus following careful review of the current literature. Although delivered energy levels are selected for defibrillation, it is the transmyocardial current flow that achieves defibrillation. Current correlates well with successful defibrillation and cardioversion.[476] Defibrillation shock energy levels are unchanged from the 2010 guidelines.[2]

First shock

Relatively few studies have been published in the past five years on which to refine the 2010 guidelines. There is no evidence that one biphasic waveform or device is more effective than another. First shock efficacy of the BTE waveform using 150–200 J has been reported as 86–98%.[477–481] First shock efficacy of the RLB waveform using 120 J is up to 85%.[327] First shock efficacy of a new pulsed biphasic waveform at 130 J showed a first shock success rate of 90%.[472] Two studies have suggested equivalence with lower and higher starting energy biphasic defibrillation.[482,483] Although human studies have not shown harm (raised biomarkers, ECG changes, ejection fraction) from any biphasic waveform up to 360 J,[482,484] several animal studies have suggested the potential for harm with higher energy levels.[485–488]

The initial biphasic shock should be no lower than 120 J for RLB waveforms and at least 150 J for BTE waveforms. Ideally, the initial biphasic shock energy should be at least 150 J for all waveforms. Manufacturers should display the effective waveform dose range on the face of the biphasic defibrillator. If the rescuer is unaware of the recommended energy settings of the defibrillator, use the highest setting for all shocks.

Second and subsequent shocks

The 2010 guidelines recommended either a fixed or escalating energy strategy for defibrillation. Several studies demonstrated that although an escalating strategy reduces the number of shocks required to restore an organised rhythm compared with fixed-dose biphasic defibrillation, and may be needed for successful defibrillation,[326,489] rates of ROSC or survival to hospital discharge are not significantly different between strategies.[482,483] Conversely, a fixed-dose biphasic protocol demonstrated high cardioversion rates (>90%) with a three-shock fixed dose protocol but the small number of cases did not exclude a significant lower ROSC rate for recurrent VF.[490] Several in-hospital studies using an escalating shock energy strategy have demonstrated improvement in cardioversion rates (compared with fixed dose protocols) in non-arrest

rhythms with the same level of energy selected for both biphasic and monophasic waveforms.[491–496]

Animal studies, case reports and small case series have documented the use of two defibrillators to deliver a pair of shocks at the same time ('dual sequential defibrillation') to patients in refractory shockable states.[497–501] Given the very limited evidence, the routine use of dual sequential defibrillation' cannot be recommended.

There remains no evidence to support either a fixed or escalating energy protocol, although an escalating protocol may be associated with a lower incidence of refibrillation (see below). Both strategies are acceptable; however, if the first shock is not successful and the defibrillator is capable of delivering shocks of higher energy it is reasonable to increase the energy for subsequent shocks.

Recurrent ventricular fibrillation (refibrillation). Refibrillation is common and occurs in the majority of patients following initial first-shock termination of VF. Refibrillation was not specifically addressed in 2010 guidelines. Distinct from refractory VF, defined as 'fibrillation that persists after one or more shocks', recurrence of fibrillation is usually defined as 'recurrence of VF during a documented cardiac arrest episode, occurring after initial termination of VF while the patient remains under the care of the same providers (usually out-of-hospital).' Two studies showed termination rates of subsequent refibrillation were unchanged when using fixed 120 J or 150 J shock protocols respectively,[490,502] but a larger study showed termination rates of refibrillation declined when using repeated 200 J shocks, unless an increased energy level (360 J) was selected.[326] In a retrospective analysis, termination rate of VF into a pulse generating rhythm was higher if the VF appeared after a pulse generating rhythm, than after PEA or asystole.[503]

In view of the larger study suggesting benefit from higher subsequent energy levels for refibrillation,[326] we recommend that if a shockable rhythm recurs after successful defibrillation with ROSC, and the defibrillator is capable of delivering shocks of higher energy it is reasonable to increase the energy for subsequent shocks.

Other related defibrillation topics

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.[504] Synchronisation can be difficult in VT because of the wide-complex and variable forms of ventricular arrhythmia. Inspect the synchronisation marker carefully for consistent recognition of the R wave. If needed, choose another lead and/or adjust the amplitude. If synchronisation fails, give unsynchronised shocks to the unstable patient in VT to avoid prolonged delay in restoring sinus rhythm. Ventricular fibrillation or pulseless VT requires unsynchronised shocks. Conscious patients require anaesthesia or sedation, and analgesia before attempting synchronised cardioversion.

Atrial fibrillation. Optimal electrode position has been discussed previously, but anterolateral and anteroposterior are both acceptable positions.[443] Biphasic waveforms are more effective than monophasic waveforms for cardioversion of AF[493,494,505,506]; and cause less severe skin burns.[507] More data are needed before specific recommendations can be made for optimal biphasic energy levels and different biphasic waveforms. Biphasic rectilinear and biphasic truncated exponential waveform show similar high efficacy in the elective cardioversion of atrial fibrillation.[508] Commencing at high energy levels has not shown to result in more successful cardioversion rates compared to lower energy levels.[494,509–514] An initial synchronised shock of 120–150 J, escalating if necessary is a reasonable strategy based on current data.

Atrial flutter and paroxysmal supraventricular tachycardia. Atrial flutter and paroxysmal SVT generally require less energy than atrial fibrillation for cardioversion.[513] Give an initial shock of 70–120 J biphasic. Give subsequent shocks using stepwise increases in energy.[476]

Ventricular tachycardia. The energy required for cardioversion of VT depends on the morphological characteristics and rate of the arrhythmia.[515] Ventricular tachycardia with a pulse responds well using biphasic energy levels of 120–150 J for the initial shock. Consider stepwise increases if the first shock fails to achieve sinus rhythm.[515]

Pacing

Consider pacing in patients with symptomatic bradycardia refractory to anti-cholinergic drugs or other second line therapy. Immediate pacing is indicated especially when the block is at or below the His-Purkinje level. If transthoracic pacing is ineffective, consider transvenous pacing. Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because this will likely respond to cardiac pacing. The use of epicardial wires to pace the myocardium following cardiac surgery is effective and discussed elsewhere. Do not attempt pacing for asystole unless P waves are present; it does not increase short or long-term survival in- or out-of-hospital.[516–524] For haemodynamically unstable, conscious patients with bradyarrhythmias, percussion pacing as a bridge to electrical pacing may be attempted, although its effectiveness has not been established.[525,526]

Implantable cardioverter defibrillators

Implantable cardioverter defibrillators (ICDs) are becoming increasingly common as the devices are implanted more frequently as the population ages. They are implanted because a patient is considered to be at risk from, or has had, a life-threatening shockable arrhythmia and are usually embedded under the pectoral muscle below the left clavicle (in a similar position to pacemakers, from which they cannot be immediately distinguished). More recently, extravascular devices can be implanted subcutaneously in the left chest wall, with a lead running to the left of the sternum.

On sensing a shockable rhythm, an ICD will discharge approximately 40 J (approximately 80 J for subcutaneous devices) through an internal pacing wire embedded in the right ventricle. On detecting VF/pVT, ICD devices will discharge no more than eight times, but may reset if they detect a new period of VF/pVT. Patients with fractured ICD leads may suffer repeated internal defibrillation as the electrical noise is mistaken for a shockable rhythm; in these circumstances, the patient is likely to be conscious, with the ECG showing a relatively normal rate. A magnet placed over the ICD will disable the defibrillation function in these circumstances.

Discharge of an ICD may cause pectoral muscle contraction in the patient, and shocks to the rescuer have been documented.[527] In view of the low energy levels discharged by conventional ICDs, it is unlikely that any harm will come to the rescuer, but minimising contact with the patient whilst the device is discharging is prudent. Surface current from subcutaneous ICDs is currently under investigation. Cardioverter and pacing function should always be re-evaluated following external defibrillation, both to check the device itself and to check pacing/defibrillation thresholds of the device leads.

Pacemaker spikes generated by devices programmed to unipolar pacing may confuse AED software and emergency personnel,

and may prevent the detection of VF.[528] The diagnostic algorithms of modern AEDs can be insensitive to such spikes.

3f – Airway management and ventilation

Introduction

The optimal strategy for managing the airway has yet to be determined. Several observational studies have challenged the premise that advanced airway interventions (tracheal intubation or supraglottic airways) improve outcomes.[529] Options for airway management and ventilation during CPR include: no airway and no ventilation (compression-only CPR), compression-only CPR with the airway held open (with or without supplementary oxygen), mouth-to-mouth breaths, mouth-to-mask, bag-mask ventilation with simple airway adjuncts, supraglottic airways (SGAs), and tracheal intubation (inserted with the aid of direct laryngoscopy or videolaryngoscopy, or via a SGA). In practice a combination of airway techniques will be used stepwise during a resuscitation attempt.[530] The best airway, or combination of airway techniques will vary according to patient factors, the phase of the resuscitation attempt (during CPR, after ROSC), and the skills of rescuers.[311] A stepwise approach to airway and ventilation management using a combination of techniques is therefore suggested. Compression-only CPR and use of ventilation during basic life support is addressed in Section 2 – Basic Life Support.[223]

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

Airway obstruction

Causes of airway obstruction

Obstruction of the airway may be partial or complete. It may occur at any level, from the nose and mouth down to the trachea. In the unconscious patient, the commonest site of airway obstruction is at the soft palate and epiglottis.[531,532] Obstruction may also be caused by vomit or blood (regurgitation of gastric contents or trauma), or by foreign bodies. Laryngeal obstruction may be caused by oedema from burns, inflammation or anaphylaxis. Upper airway stimulation may cause laryngeal spasm. Obstruction of the airway below the larynx is less common, but may arise from excessive bronchial secretions, mucosal oedema, bronchospasm, pulmonary oedema or aspiration of gastric contents.

Recognition of airway obstruction

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

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

In partial airway obstruction, air entry is diminished and usually noisy. Inspiratory stridor is caused by obstruction at the laryngeal level or above. Expiratory wheeze implies obstruction of the lower airways, which tend to collapse and obstruct during expiration. In a patient who is making respiratory efforts, complete airway obstruction causes paradoxical chest and abdominal movement, often described as 'see-saw' breathing. During airway obstruction, other accessory muscles of respiration are used, with the neck and the shoulder muscles contracting to assist movement of the thoracic cage.

Basic airway management

There are three manoeuvres that may improve the patency of an airway obstructed by the tongue or other upper airway structures: head tilt, chin lift, and jaw thrust.

Head tilt and chin lift

The rescuer's hand is placed on the patient's forehead and the head gently tilted back; the fingertips of the other hand are placed under the point of the patient's chin, which is lifted gently to stretch the anterior neck structures.[533–538]

Jaw thrust

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

Airway management in patients with suspected cervical spine injury

When there is a risk of cervical spine injury, establish a clear upper airway by using jaw thrust or chin lift in combination with manual in-line stabilisation (MILS) of the head and neck by an assistant.[539,540] If life-threatening airway obstruction persists despite effective application of jaw thrust or chin lift, add head tilt in small increments until the airway is open; establishing a patent airway takes priority over concerns about a potential cervical spine injury.

Adjuncts to basic airway techniques

Despite a total lack of published data on the use of nasopharyngeal and oropharyngeal airways during CPR, they are often helpful, and sometimes essential, to maintain an open airway, particularly when resuscitation is prolonged. The position of the head and neck is maintained to keep the airway aligned. Oropharyngeal and nasopharyngeal airways overcome backward displacement of the soft palate and tongue in an unconscious patient, but head tilt and jaw thrust may also be required.

Oropharyngeal airways. Oropharyngeal airways are available in sizes suitable for the newborn to large adults. An estimate of the size required is obtained by selecting an airway with a length corresponding to the vertical distance between the patient's incisors and the angle of the jaw. The most common sizes are 2, 3 and 4 for small, medium and large adults, respectively.

Nasopharyngeal airways. In patients who are not deeply unconscious, a nasopharyngeal airway is tolerated better than an oropharyngeal airway. The nasopharyngeal airway may be life saving in patients with clenched jaws, trismus or maxillofacial injuries, when insertion of an oral airway is impossible. The tubes are sized in millimetres according to their internal diameter and the length increases with diameter. Sizes of 6–7 mm are suitable for adults.

Oxygen during CPR

During CPR, give the maximal feasible inspired oxygen concentration. A self-inflating bag can be connected to a facemask, tracheal tube or supraglottic airway (SGA). Without supplementary oxygen,

the self-inflating bag ventilates the patient's lungs with ambient air (21% oxygen). The delivered oxygen concentration can be increased to about 85% by using a reservoir system and attaching oxygen at a flow 10 l min^{-1}. There are no data to indicate the optimal arterial blood oxygen saturation (SaO_2) during CPR, and no trials comparing different inspired oxygen concentrations. In one observational study of patients receiving 100% inspired oxygen via a tracheal tube during CPR, a higher measured PaO_2 value during CPR was associated with ROSC and hospital admission.[541] The worse outcomes associated with a low PaO_2 during CPR could however be an indication of illness severity. Animal data and observational clinical data indicate an association between high SaO_2 after ROSC and worse outcome (Section 5 – Post-resuscitation care).[273,542–544]

After ROSC, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%. Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration. This is addressed in further detail in Section 5 – post resuscitation care.[273]

Suction

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

Choking

The initial management of foreign body airway obstruction (choking) is addressed in Section 2 – basic life support.[223] In an unconscious patient with suspected foreign body airway obstruction if initial basic measures are unsuccessful use laryngoscopy and forceps to remove the foreign body under direct vision. To do this effectively requires training.

Ventilation

Advanced Life Support providers should give artificial ventilation as soon as possible for any patient in whom spontaneous ventilation is inadequate or absent. Expired air ventilation (rescue breathing) is effective, but the rescuer's expired oxygen concentration is only 16–17%, so it must be replaced as soon as possible by ventilation with oxygen-enriched air. The pocket resuscitation mask is similar to an anaesthetic facemask, and enables mouth-to-mask ventilation. It has a unidirectional valve, which directs the patient's expired air away from the rescuer. The mask is transparent so that vomit or blood from the patient can be seen. Some masks have a connector for the addition of oxygen. When using masks without a connector, supplemental oxygen can be given by placing the tubing underneath one side and ensuring an adequate seal. Use a two-hand technique to maximise the seal with the patient's face.

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

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

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

Inadvertent hyperventilation during CPR is common. While this increased intrathoracic pressure[545] and peak airway pressures[546] in small case series in humans, a carefully controlled animal experiment revealed no adverse effects.[547] We suggest a ventilation rate of 10 min^{-1} during continuous chest compressions with an advanced airway based on very limited evidence.[4]

Self-inflating bag

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

Although a bag-mask enables ventilation with high concentrations of oxygen, its use by a single person requires considerable skill. When used with a face mask, it is often difficult to achieve a gas-tight seal between the mask and the patient's face, and to maintain a patent airway with one hand while squeezing the bag with the other. Any significant leak will cause hypoventilation and, if the airway is not patent, gas may be forced into the stomach.[548,549] This will reduce ventilation further and greatly increase the risk of regurgitation and aspiration.[550] The two-person technique for bag-mask ventilation is preferable. Several recent observational studies and a meta-analysis have documented better outcomes with use of bag-mask ventilation compared with more advanced airways (SGA or tracheal tube).[529,551–554] However, these observation studies are subject to significant bias caused by confounders such as advanced airways not being required in those patients who achieve ROSC and awaken early.

Once a tracheal tube or a SGA has been inserted, ventilate the lungs at a rate of 10 breaths min^{-1} and continue chest compressions without pausing during ventilations. The laryngeal seal achieved with a SGA may not be good enough to prevent at least some gas leaking when inspiration coincides with chest compressions. Moderate gas leakage is acceptable, particularly as most of this gas will pass up through the patient's mouth. If excessive gas leakage results in inadequate ventilation of the patient's lungs, chest compressions will have to be interrupted to enable ventilation, using a compression–ventilation ratio of 30:2.

Passive oxygen delivery

In the presence of a patent airway, chest compressions alone may result in some ventilation of the lungs.[555] Oxygen can be delivered passively, either via an adapted tracheal tube (Boussignac tube),[556,557] or with the combination of an oropharyngeal airway and standard oxygen mask with non-rebreather reservoir.[558] In theory, a SGA can also be used to deliver oxygen passively but this has yet to be studied. One study has shown higher neurologically favourable survival with passive oxygen delivery (oral airway and oxygen mask) compared with bag-mask ventilation after out-of-hospital VF cardiac arrest, but this was a retrospective analysis and is subject to numerous confounders.[558] Until further data are available, passive oxygen delivery without ventilation is not recommended for routine use during CPR.

Alternative airway devices

The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest.[309] There is evidence that, without adequate training and experience, the incidence of complications, such as unrecognised

oesophageal intubation (2.4–17% in several studies involving paramedics)[559–563] and dislodgement, is unacceptably high.[564] Prolonged attempts at tracheal intubation are harmful; the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Several alternative airway devices have been used 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), the i-gel, and the LMA Supreme (LMAS) 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 SGAs are easier to insert than a tracheal tube and,[565] unlike tracheal intubation, can generally be inserted without interrupting chest compressions.[566]

There are no data supporting the routine use of any specific approach to airway management during cardiac arrest. The best technique is dependent on the precise circumstances of the cardiac arrest and the competence of the rescuer. It is recognised that during cardiac arrest a stepwise approach to airway management is commonly used, which implies that multiple devices may be used during a single resuscitation attempt.

Laryngeal mask airway (LMA)

The original LMA (classic LMA [cLMA]), which is reusable, has been studied during CPR, but none of these studies has compared it directly with the tracheal tube. Although the cLMA remains in common use in elective anaesthetic practice, it has been superseded by several 2nd generation SGAs that have more favourable characteristics, particularly when used for emergency airway management.[567] Most of these SGAs are single use and achieve higher oropharyngeal seal pressures than the cLMA, and some incorporate gastric drain tubes.

Combitube

The Combitube is a double-lumen tube introduced blindly over the tongue, and provides a route for ventilation whether the tube has passed into the oesophagus. There are many studies of the Combitube in CPR and successful ventilation was achieved in 79–98% of patients.[568–576] Two RCTs of the Combitube versus tracheal intubation for out-of-hospital cardiac arrest showed no difference in survival.[575,576] Use of the Combitube is waning and in many parts of the world it is being replaced by other devices such as the LT.

Laryngeal tube

The laryngeal tube (LT) was introduced in 2001; it is known as the King LT airway in the United States. After just 2 h of training, nurses successfully inserted a laryngeal tube and achieved ventilation in 24 of 30 (80%) of OHCAs.[577] In five observational studies, a disposable version of the laryngeal tube (LT-D) was inserted successfully by prehospital personnel in 85–100% of OHCAs (number of cases ranged from 92 to 347).[578–582] Although some studies are supportive of the use of the LT during cardiac arrest several other studies have reported that insertion problems are common; these include problems with positioning and leakage.[580,583]

i-gel

The cuff of the i-gel is made of thermoplastic elastomer gel and does not require inflation; the stem of the i-gel incorporates a bite block and a narrow oesophageal drain tube. It is very easy to insert, requiring only minimal training and a laryngeal seal pressure of 20–24 cmH_2O can be achieved.[584,585] The ease of insertion of the i-gel and its favourable leak pressure make it theoretically very attractive as a resuscitation airway device for those inexperienced in tracheal intubation. In observational studies insertion success rates for the i-gel were 93% ($n = 98$) when used by paramedics for OHCA[586] and 99% ($n = 100$) when used by doctors and nurses for IHCA.[587]

LMA supreme (LMAS). The LMAS is a disposable version of the Proseal LMA, which is used in anaesthetic practice. In an observational study, paramedics inserted the LMAS successfully and were able to ventilate the lungs of 33 (100%) cases of OHCA.[588]

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.[309] It should be used only when trained personnel are available to carry out the procedure with a high level of skill and confidence. A systematic review of randomised controlled trials (RCTs) of tracheal intubation versus alternative airway management in acutely ill and injured patients identified just three trials[589]: two were RCTs of the Combitube versus tracheal intubation for out-of-hospital cardiac arrest,[575,576] which showed no difference in survival. The third study was a RCT of prehospital tracheal intubation versus management of the airway with a bag-mask in children requiring airway management for cardiac arrest, primary respiratory disorders and severe injuries.[590] There was no overall benefit for tracheal intubation; on the contrary, of the children requiring airway management for a respiratory problem, those randomised to intubation had a lower survival rate that those in the bag-mask group.

The perceived advantages of tracheal intubation over bag-mask ventilation include: enabling ventilation without interrupting chest compressions[591]; enabling effective ventilation, particularly when lung and/or chest compliance is poor; minimising gastric inflation and therefore the risk of regurgitation; protection against pulmonary aspiration of gastric contents; and the potential to free the rescuer's hands for other tasks. Use of the bag-mask is more likely to cause gastric distension that, theoretically, is more likely to cause regurgitation with risk of aspiration. However, there are no reliable data to indicate that the incidence of aspiration is any more in cardiac arrest patients ventilated with bag-mask versus those that are ventilated via tracheal tube.

The perceived disadvantages of tracheal intubation over bag-valve-mask ventilation include:

- The risk of an unrecognised misplaced tracheal tube – in patients with out-of-hospital cardiac arrest the reliably documented incidence ranges from 0.5% to 17%: emergency physicians–0.5%;[592] paramedics – 2.4%,[559] 6%,[560,561] 9%,[562] 17%.[563]
- A prolonged period without chest compressions while intubation is attempted – in a study of prehospital intubation by paramedics during 100 cardiac arrests the total duration of the interruptions in CPR associated with tracheal intubation attempts was 110 s (IQR 54–198 s; range 13–446 s) and in 25% the interruptions were more than 3 min.[593] Tracheal intubation attempts accounted for almost 25% of all CPR interruptions.
- A comparatively high failure rate. Intubation success rates correlate with the intubation experience attained by individual paramedics.[594] Rates for failure to intubate are as high as 50% in prehospital systems with a low patient volume and providers who do not perform intubation frequently.[595,596]
- Tracheal intubation is a difficult skill to acquire and maintain. In one study, anaesthesia residents required about 125 intubations in the operating room setting before they were able to achieve and intubation success rate of 95%.[597]

Only one study has prospectively compared tracheal intubation with insertion of a SGA in OHCA and this was a feasibility study that is not powered to show differences in survival.[530] A secondary analysis of the North American Resuscitation Outcomes Consortium (ROC) PRIMED study that compared tracheal intubation (n = 8487) with SGAs (LT, Combitube, or LMA; n = 1968) showed that successful tracheal intubation was associated with increased neurologically favourable survival to hospital discharge (adjusted OR 1.40, 95% CI 1.04–1.89) when compared with successful SGA insertion.[598] In a Japanese OHCA study, tracheal intubation (n = 16,054) was compared with the LMA (n = 34,125) and the oesophageal obturator airway (n = 88,069) over a 3-year period.[599] Adjusted ORs for favourable one-month survival were lower for the LMA (0.77, 95% CI 0.64–0.94) and the oesophageal obturator airway (0.81, 95% CI 0.68–0.96) in comparison with tracheal intubation. Even though the data from these two observational studies are risk-adjusted, it is likely that hidden confounders account for the findings.

Healthcare personnel who undertake prehospital intubation should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills. Rescuers must weigh the risks and benefits of intubation against the need to provide effective chest compressions. The intubation attempt may require some interruption of chest compressions but, once an advanced airway is in place, ventilation will not require interruption of chest compressions. Personnel skilled in advanced airway management should be able to undertake laryngoscopy without stopping chest compressions; a brief pause in chest compressions will be required only as the tube is passed through the vocal cords. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until ROSC[558,600]; this strategy is being studied in a large prehospital randomised trial.[601] The intubation attempt should interrupt chest compressions for less than 5 s; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After intubation, tube placement must be confirmed and the tube secured adequately.

Videolaryngoscopy

Videolaryngoscopes are being used increasingly in anaesthetic and critical care practice.[602,603] In comparison with direct laryngoscopy, they enable a better view of the larynx and improve the success rate of intubation. Preliminary studies indicate that use of videolaryngoscopes improve laryngeal view and intubation success rates during CPR[604–606] but further data are required before recommendations can be made for wider use during CPR.

Confirmation of correct placement of the tracheal tube

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine use of primary and secondary techniques to confirm correct placement of the tracheal tube should reduce this risk.

Clinical assessment. Primary assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and adequate) and over the epigastrium (breath sounds should not be heard). Clinical signs of correct tube placement (condensation in the tube, chest rise, breath sounds on auscultation of lungs, and inability to hear gas entering the stomach) are not reliable. The reported sensitivity (proportion of tracheal intubations correctly identified) and specificity (proportion of oesophageal intubations correctly identified) of clinical assessment varies: sensitivity 74–100%; specificity 66–100%.[592,607–610]

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

Oesophageal detector device. The oesophageal detector device creates a suction force at the tracheal end of the tracheal tube, either by pulling back the plunger on a large syringe or releasing a compressed flexible bulb. Air is aspirated easily from the lower airways through a tracheal tube placed in the cartilage-supported rigid trachea. When the tube is in the oesophagus, air cannot be aspirated because the oesophagus collapses when aspiration is attempted. The oesophageal detector device may be misleading in patients with morbid obesity, late pregnancy or severe asthma or when there are copious tracheal secretions; in these conditions the trachea may collapse when aspiration is attempted. Detection of correct placement of a tracheal tube during CPR has been documented in five observational studies[561,611–614] that included 396 patients, and in one randomised study[615] that included 48 patients.[4] The pooled specificity was 92% (95% CI 84–96%), the pooled sensitivity was 88% (95% CI 84–192%), and the false positive rate was 0.2% (95% CI, 0–0.6%). One observational study showed no statistically significant difference between the performance of a bulb (sensitivity 71%, specificity 100%) and a syringe (sensitivity 73%, specificity 100%) type oesophageal detection devices in the detection of tracheal placement of a tracheal tube.[615]

Thoracic impedance. There are smaller changes in thoracic impedance with oesophageal ventilations than with ventilation of the lungs.[616–618] Changes in thoracic impedance may be used to detect ventilation[619] and oesophageal intubation[591,620] during cardiac arrest. It is possible that this technology can be used to measure tidal volume during CPR. The role of thoracic impedance as a tool to detect tracheal tube position and adequate ventilation during CPR is undergoing further research but is not yet ready for routine clinical use.

Ultrasound for tracheal tube detection. Three observational studies including 254 patients in cardiac arrest have documented the use of ultrasound to detect tracheal tube placement.[621–623] The pooled specificity was 90% (95% CI 68–98%), the sensitivity was 100% (95% CI 98–100%), and the FPR was 0.8% (95% CI 0.2–2.6%).

Carbon dioxide detectors. Carbon dioxide (CO_2) detector devices measure the concentration of exhaled carbon dioxide from the lungs. The persistence of exhaled CO_2 after six ventilations indicates placement of the tracheal tube in the trachea or a main bronchus.[592] Confirmation of correct placement above the carina will require auscultation of the chest bilaterally in the mid-axillary lines. Broadly, there three types of carbon dioxide detector device:

(1) Disposable colorimetric end-tidal carbon dioxide ($ETCO_2$) detectors use a litmus paper to detect CO_2, and these devices generally give readings of purple ($ETCO_2$ < 0.5%), tan ($ETCO_2$ 0.5–2%) and yellow ($ETCO_2$ > 2%). In most studies, tracheal placement of the tube is considered verified if the tan colour persists after a few ventilations. Seven observational studies[592,614,624–628] including 1119 patients have evaluated the diagnostic accuracy of colorimetric CO_2 devices in cardiac arrest patients.[4] The specificity was 97% (95% CI 84–99%), the sensitivity was 87% (95% CI 85–89%), and the FPR was 0.3% (0–1%). Although colorimetric CO_2 detectors identify placement in patients with good perfusion quite well, these devices are less accurate than clinical assessment in cardiac arrest patients

because pulmonary blood flow may be so low that there is insufficient exhaled carbon dioxide. Furthermore, if the tracheal tube is in the oesophagus, six ventilations may lead to gastric distension, vomiting and aspiration.

(2) Non-waveform electronic digital $ETCO_2$ devices generally measure $ETCO_2$ using an infrared spectrometer and display the results with a number; they do not provide a waveform graphical display of the respiratory cycle on a capnograph. Five studies of these devices for identification of tracheal tube position in cardiac arrest document 70–100% sensitivity and 100% specificity.[592,609,614,627,629,630]

(3) End-tidal CO_2 detectors that include a waveform graphical display (capnographs) are the most reliable for verification of tracheal tube position during cardiac arrest. Two studies of waveform capnography to verify tracheal tube position in victims of cardiac arrest demonstrate 100% sensitivity and 100% specificity in identifying correct tracheal tube placement.[592,631] One observational study showed that the use of waveform capnography compared with no waveform capnography in 153 critically-ill patients (51 with cardiac arrest) decreased the occurrence of unrecognised oesophageal intubation on hospital arrival from 23% to 0% (OR 29; 95% CI 4–122).[631] Three observational studies with 401 patients[592,607,613] and one randomised study[615] including 48 patients showed that the specificity for waveform capnography to detect correct tracheal placement was 100% (95% CI 87–100%). The sensitivity was 100% in one study when waveform capnography was used in the pre-hospital setting immediately after intubation, and oesophageal intubation was less common than the average (1.5%).[592,607] The sensitivity was between 65% to 68% in the other three studies when the device was used in OHCA patients after intubation in the emergency department (ED).[607,613,615] The difference may be related to prolonged resuscitation with compromised or non-existent pulmonary blood flow. Based on the pooled sensitivity/specificity from these studies and assumed oesophageal intubation prevalence of 4.5%, the false positive rate (FPR) of waveform capnography was 0% (95% CI 0–0.6%).

Based on the available data, the accuracy of colorimetric CO_2 detectors, oesophageal detector devices and non-waveform capnometers does not exceed the accuracy of auscultation and direct visualisation 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 must supplement clinical assessment (auscultation and visualisation of tube through cords). Waveform capnography will not discriminate between tracheal and bronchial placement of the tube – careful auscultation is essential. Existing portable monitors make capnographic initial confirmation and continuous monitoring of tracheal tube position feasible in almost all settings, including out-of-hospital, emergency department and in-hospital locations where intubation is performed.

The ILCOR ALS Task Force recommends using waveform capnography to confirm and continuously monitor the position of a tracheal tube during CPR in addition to clinical assessment (strong recommendation, low quality evidence). Waveform capnography is given a strong recommendation as it may have other potential uses during CPR (e.g. monitoring ventilation rate, assessing quality of CPR). The ILCOR ALS Task Force recommends that if waveform capnography is not available, a non-waveform carbon dioxide detector, oesophageal detector device or ultrasound in addition to clinical assessment is an alternative (strong recommendation, low quality evidence).

Cricoid pressure

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

In non-arrest patients cricoid pressure may offer some measure of protection to the airway from aspiration but it may also impede ventilation or interfere with intubation. The role of cricoid pressure during cardiac arrest has not been studied. Application of cricoid pressure during bag-mask ventilation reduces gastric inflation.[632–635]

Studies in anaesthetised patients show that cricoid pressure impairs ventilation in many patients, increases peak inspiratory pressures and causes complete obstruction in up to 50% of patients depending on the amount of cricoid pressure (in the range of recommended effective pressure) that is applied.[632,633,636–641]

Securing the tracheal tube

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

Cricothyroidotomy

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

Surgical cricothyroidotomy provides a definitive airway that can be used to ventilate the patient's lungs until semi-elective intubation or tracheostomy is performed. Needle cricothyroidotomy is a much more temporary procedure providing only short-term oxygenation. It requires a wide-bore, non-kinking cannula, a high-pressure oxygen source, runs the risk of barotrauma and can be particularly ineffective in patients with chest trauma. It is also prone to failure because of kinking of the cannula, and is unsuitable for patient transfer. In the 4th National Audit Project of the UK Royal College of Anaesthetists and the Difficult Airway Society NAP4, 60% of needle cricothyroidotomies attempted in the intensive care unit (ICU), and elsewhere, failed.[642] In contrast, all surgical cricothyroidotomies achieved access to the trachea. While there may be several underlying causes, these results indicate a need for more training in surgical cricothyroidotomy and this should include regular manikin-based training using locally available equipment.[643]

Summary of airway management for cardiac arrest

The ILCOR ALS Task Force has suggested using either an advanced airway (tracheal intubation or SGA) or a bag-mask for airway management during CPR.[4] This very broad recommendation is made because of the total absence of high quality data to indicate which airway strategy is best.

The type of airway used may depend on the skills and training of the healthcare provider. In comparison with bag-mask ventilation and use of a SGA, tracheal intubation requires considerably more training and practice and may result in unrecognised oesophageal intubation and increased hands-off time. A bag-mask, a SGA and a tracheal tube are frequently used in the same patient as part of a stepwise approach to airway management but this has not been formally assessed. Patients who remain comatose after initial

resuscitation from cardiac arrest will ultimately require tracheal intubation regardless of the airway technique used during cardiac arrest. Anyone attempting tracheal intubation must be well trained and equipped with waveform capnography. In the absence of these prerequisites, consider use of bag-mask ventilation and/or an SGA until appropriately experience and equipped personnel are present.

There are very few data relating to airway management during in-hospital cardiac arrest and it is necessary to extrapolate from data derived from out-of-hospital cardiac arrest. On this basis, the principles discussed above apply equally to in-hospital cardiac arrest.

3g – Drugs and fluids for cardiac arrest

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

There are three groups of drugs relevant to the management of cardiac arrest that were reviewed during the 2015 Consensus Conference: vasopressors, anti-arrhythmics and other drugs.[4] The systematic reviews found insufficient evidence to comment on critical outcomes such as survival to discharge and survival to discharge with good neurological outcome with either vasopressors or anti-arrhythmic drugs. There was also insufficient evidence to comment on the best time to give drugs to optimise outcome. Thus, although drugs are still included among ALS interventions, they are of secondary importance to high-quality uninterrupted chest compressions and early defibrillation. As an indicator of equipoise regarding the use of drugs during ALS, two large RCTs (adrenaline versus placebo [ISRCTN73485024], and amiodarone versus lidocaine versus placebo[312] [NCT01401647] are currently ongoing.

Vasopressors

Despite the continued widespread use of adrenaline and the use of vasopressin during resuscitation in some countries, there is no placebo-controlled study that shows that the routine use of any vasopressor during human cardiac arrest increases survival to hospital discharge, although improved short-term survival has been documented.[305,306,308] The primary goal of CPR is to re-establish blood flow to vital organs until the restoration of spontaneous circulation. Despite the lack of data from cardiac arrest in humans, vasopressors continue to be recommended as a means of increasing cerebral and coronary perfusion pressure during CPR.

Adrenaline (epinephrine) versus no adrenaline

One randomised, placebo-controlled trial on patients with out-of-hospital cardiac arrest from all rhythms showed that administration of standard-dose adrenaline was associated with significantly higher rates of prehospital ROSC (relative risk [RR] 2.80 [95% CI 1.78–4.41], $p < 0.00001$) and survival to hospital admission (RR 1.95 [95% CI 1.34–2.84], $p = 0.0004$) when compared to placebo.[308] There was no difference in survival to hospital discharge (RR 2.12 [95% CI 0.75–6.02], $p = 0.16$) or good neurological outcome, defined as Cerebral Performance Categories (CPC) 1 or 2 (RR 1.73, [95% CI 0.59–5.11], $p = 0.32$). The trial, however, was stopped early and included only 534 subjects.

Another trial randomised 851 patients with out-of-hospital cardiac arrest to receive advanced life support with or without intravenous drug administration. Results of this trial showed that administration of intravenous drugs was associated with significantly higher rates of prehospital ROSC (40% vs. 25%; $p < 0.001$) and admission to hospital (43% vs. 29%; $p < 0.001$).[305] However, the rates of survival to hospital discharge did not differ (10.5 vs.9.2; $p = 0.61$). The effect on ROSC was most prominent and only significant in the non-shockable group.[305] In a post-hoc analysis comparing patients who were given adrenaline vs. not given adrenaline, the OR of being admitted to hospital was higher with adrenaline, but the likelihood of being discharged from hospital alive and surviving with favourable neurological outcome was reduced [OR for adrenaline vs. no-adrenaline were 2.5 (95% CI 1.9–3.4), 0.5 (95% CI 0.3–0.8) and 0.4 (95% CI 0.2–0.7) respectively].[644]

A series of observational studies on large cohorts of out-of-hospital cardiac arrest patients have compared the outcomes of patients who were administered adrenaline with those of patients who did not receive adrenaline. Adjustments were made using logistic regression and propensity matching. A study conducted in Japan which included a total of 417,188 patients (13,401 of whom were propensity-matched) showed that use of prehospital adrenaline was significantly associated with increased odds of ROSC before hospital arrival (adjusted OR 2.36 [95% CI 2.22–2.50]) but decreased chance of survival (0.46 [95%CI 0.42–0.51]) and good functional outcome (0.31 [95% CI 0.26–0.36]) at one month after the arrest.[645] Conversely, another Japanese study conducted on 11,048 propensity-matched, bystander-witnessed arrests showed that prehospital administration of adrenaline was associated with significantly higher rates of overall survival and, for patients with non-shockable rhythms, it was also associated with significantly higher odds of neurologically intact survival (adjusted OR 1.57 [95% CI 1.04–2.37]).[646] However, the absolute increase of neurologically intact survival in this last group of patients was minimal (0.7% vs. 0.4%). Finally, in a recent study in France on 1556 cardiac arrest patients who achieved ROSC and were admitted to hospital, administration of adrenaline was associated with significantly lower odds of neurologically intact survival.[647]

There is an increasing concern about the potential detrimental effects of adrenaline. While its alpha-adrenergic, vasoconstrictive effects cause systemic vasoconstriction, which increases macrovascular coronary and cerebral perfusion pressures, its beta-adrenergic actions (inotropic, chronotropic) may increase coronary and cerebral blood flow, but with concomitant increases in myocardial oxygen consumption, ectopic ventricular arrhythmias (particularly when the myocardium is acidotic), transient hypoxaemia from pulmonary arteriovenous shunting, impaired microcirculation,[648] and worse post-cardiac arrest myocardial dysfunction.[649,650] Experimental evidence suggests that epinephrine also impairs cerebral microcirculation.[651] In retrospective secondary analyses, adrenaline use is associated with more rhythm transitions during ALS, both during VF[652] and PEA.[325]

Two systematic reviews of adrenaline for OHCA indicate rates of ROSC are increased with adrenaline but good long-term survival (survival to discharge and neurological outcome) is either no better, or worse.[653,654]

The optimal dose of adrenaline is not known, and there are no human data supporting the use of repeated doses. In fact, increasing cumulative dose of epinephrine during resuscitation of patients with asystole and PEA is an independent risk factor for unfavourable functional outcome and in-hospital mortality.[655]

Our current recommendation is to continue the use of adrenaline during CPR as for Guidelines 2010. We have considered the benefit in short-term outcomes (ROSC and admission to hospital) and our uncertainty about the benefit or harm on survival to discharge and neurological outcome given the limitations of the observational studies.[4,653,654] We have decided not to change current practice until there is high-quality data on long-term outcomes. Dose response and placebo-controlled efficacy trials are needed to evaluate the use of adrenaline in cardiac arrest. We are aware of an ongoing randomised study of adrenaline vs.

placebo for OHCA in the UK (PARAMEDIC 2: The Adrenaline Trial, ISRCTN73485024).

Adrenaline (epinephrine) versus vasopressin

The potentially deleterious beta-effects of adrenaline have led to exploration of alternative vasopressors. Vasopressin is a naturally occurring antidiuretic hormone. In very high doses it is a powerful vasoconstrictor that acts by stimulation of smooth muscle V1 receptors. Vasopressin has neither chronotropic nor inotropic effects on the heart. In comparison with adrenaline it has a longer half-life (10–20 min vs. 4 min) and it is potentially more effective during acidosis.[656,657] Vasopressin has been proposed as an alternative to adrenaline in cardiac arrest, based on the finding that its levels were significantly higher in successfully resuscitated patients than in patients who died.[658] However, a trial comparing up to four doses of either 40 IU vasopressin or 1 mg adrenaline every 5–10 min in patients with out of hospital cardiac arrest did not demonstrate any significant difference in terms of survival to hospital discharge or neurological outcome between the two study arms.[659] This trial had serious methodological issues and included a small number of patients.

A series of randomised controlled trials[660–664] demonstrated no difference in outcomes (ROSC, survival to discharge, or neurological outcome) with vasopressin versus adrenaline as a first line vasopressor in cardiac arrest. Other studies comparing adrenaline alone or in combination with vasopressin also demonstrated no difference in ROSC, survival to discharge or neurological outcome.[665–667] There are no alternative vasopressors that provide survival benefit during cardiac arrest resuscitation when compared with adrenaline.

We suggest vasopressin should not be used in cardiac arrest instead of adrenaline. Those healthcare professionals working in systems that already use vasopressin may continue to do so because there is no evidence of harm from using vasopressin when compared to adrenaline.[4]

Steroids

Two studies suggest that a bundled regimen of adrenaline, vasopressin and methylprednisolone improved survival after in-hospital cardiac arrest. In a single-centre randomised, placebo-controlled trial in patients with in-hospital cardiac arrest, a combination of vasopressin 20 IU and adrenaline 1 mg per CPR cycle for the first 5 CPR cycles *plus* methylprednisolone 40 mg at the first CPR cycle *plus* hydrocortisone 300 mg in case of post-resuscitation shock was associated with significantly higher rates of ROSC (39/48 [81%] vs. 27 of 52 [52%]; $p = 0.003$) and survival to hospital discharge (9 [19%] vs. 2 [4%]; $p = 0.02$) than conventional treatment.[668] These results were confirmed by a subsequent three-centre trial including a total of 300 patients from the same group of investigators.[669] This last trial also showed significantly higher odds of survival with good neurological outcome (CPC = 1–2) (OR 3.28, 95% CI 1.17–9.20; $p = 0.02$).

The population in these studies had very rapid advanced life support, a high incidence of asystolic cardiac arrest and low baseline survival compared to other in-hospital studies. Thus the findings of these studies are not generalisable to all cardiac arrests and we suggest that steroids are not used routinely for cardiac arrest.[4]

Adrenaline

Indications. Adrenaline is:

- the first drug used in cardiac arrest of any cause: it is included in the ALS algorithm for use every 3–5 min of CPR (alternate cycles).
- preferred in the treatment of anaphylaxis (Section 4).[224]
- a second-line treatment for cardiogenic shock.

Dose during CPR. During cardiac arrest, the initial IV/IO dose of adrenaline is 1 mg. There are no studies showing improvement in survival or neurological outcomes with higher doses of adrenaline for patients in refractory cardiac arrest.[4]

Following ROSC, even small doses of adrenaline (50–100 μg) may induce tachycardia, myocardial ischaemia, VT and VF. Once a perfusing rhythm is established, if further adrenaline is deemed necessary, titrate the dose carefully to achieve an appropriate blood pressure. Intravenous doses of 50 μg are usually sufficient for most hypotensive patients.

Use. Adrenaline is available most commonly in two dilutions:

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

Both these dilutions are used routinely in Europe.

Anti-arrhythmics

As with vasopressors, the evidence that anti-arrhythmic drugs are of benefit in cardiac arrest is limited. No anti-arrhythmic drug given during human cardiac arrest has been shown to increase survival to hospital discharge, although amiodarone has been shown to increase survival to hospital admission.[670,671] Despite the lack of human long-term outcome data, the balance of evidence is in favour of the use anti-arrhythmic drugs for the management of arrhythmias in cardiac arrest. There is an ongoing trial comparing amiodarone to lidocaine and to placebo designed and powered to evaluate for functional survival.[312]

Amiodarone

Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Atrioventricular conduction is slowed, and a similar effect is seen with accessory pathways. Amiodarone has a mild negative inotropic action and causes peripheral vasodilation through non-competitive alpha-blocking effects. The hypotension that occurs with intravenous amiodarone is related to the rate of delivery and is due more to the solvent (Polysorbate 80 and benzyl alcohol), which causes histamine release, rather than the drug itself.[672] A premixed formulation of intravenous amiodarone (PM101) that does not contain Polysorbate 80 and uses a cyclodextrin to maintain amiodarone in the aqueous phase is available in the United States.[673]

Following three initial shocks, amiodarone in shock-refractory VF improves the short-term outcome of survival to hospital admission compared with placebo[670] or lidocaine.[671] Amiodarone also appears to improve the response to defibrillation when given to humans or animals with VF or haemodynamically unstable ventricular tachycardia.[674–678] There is no evidence to indicate the optimal time at which amiodarone should be given when using a single-shock strategy. In the clinical studies to date, the amiodarone was given if VF/pVT persisted after at least three shocks. For this reason, and in the absence of any other data, amiodarone 300 mg is recommended if VF/pVT persists after three shocks.

Indications. Amiodarone is indicated in:

- refractory VF/pVT
- haemodynamically stable ventricular tachycardia (VT) and other resistant tachyarrhythmias (Section 11).

Dose during CPR. We recommend that an initial intravenous dose of 300 mg amiodarone, diluted in 5% glucose (or other suitable solvent) to a volume of 20 ml (or from a pre-filled syringe) should

be given after three defibrillation attempts irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest. A further dose of 150 mg may be given after five defibrillation attempts. Amiodarone can cause thrombophlebitis when injected into a peripheral vein; use a central vein if a central venous catheter is *in situ* but, if not, use a large peripheral vein or the IO route followed by a generous flush.

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

Lidocaine

Lidocaine is recommended for use during ALS when amiodarone is unavailable.[671] Lidocaine is a membrane-stabilising anti-arrhythmic drug that acts by increasing the myocyte refractory period. It decreases ventricular automaticity, and its local anaesthetic action suppresses ventricular ectopic activity. Lidocaine suppresses activity of depolarised, arrhythmogenic tissues while interfering minimally with the electrical activity of normal tissues. Therefore, it is effective in suppressing arrhythmias associated with depolarisation (e.g. ischaemia, digitalis toxicity) but is relatively ineffective against arrhythmias occurring in normally polarised cells (e.g. atrial fibrillation/flutter). Lidocaine raises the threshold for VF.

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

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

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

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

Magnesium

We recommend magnesium is not routinely used for the treatment of cardiac arrest. Studies in adults in and out of hospital have failed to demonstrate any increase in the rate of ROSC when magnesium is given routinely during CPR.[679–684]

Magnesium is an important constituent of many enzyme systems, especially those involved with ATP generation in muscle. It plays a major role in neurochemical transmission, where it decreases acetylcholine release and reduces the sensitivity of the motor endplate. Magnesium also improves the contractile response of the stunned myocardium, and limits infarct size by a mechanism that has yet to be fully elucidated.[685] The normal plasma range of magnesium is 0.8–1.0 mmol l^{-1}.

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

Give an initial intravenous dose of 2 g (4 ml (8 mmol)) of 50% magnesium sulphate); it may be repeated after 10–15 min. Preparations of magnesium sulphate solutions differ among European countries.

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

Calcium

Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. There is no data supporting any beneficial action for calcium after most cases of cardiac arrest[686–691]; conversely, other studies have suggested a possible adverse effect when given routinely during cardiac arrest (all rhythms).[692,693] High plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may impair cerebral recovery. Give calcium during resuscitation only when indicated specifically, i.e. in pulseless electrical activity caused by:

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

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

Buffers

Cardiac arrest results in combined respiratory and metabolic acidosis because pulmonary gas exchange ceases and cellular metabolism becomes anaerobic. The best treatment of acidaemia in cardiac arrest is CPR. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state[394]; analysis of central venous blood may provide a better estimation of tissue pH. Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. It has the following effects.

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

Mild acidaemia causes vasodilation and can increase cerebral blood flow. Therefore, full correction of the arterial blood pH may theoretically reduce cerebral blood flow at a particularly critical time. As the bicarbonate ion is excreted as carbon dioxide via the lungs, ventilation needs to be increased.

Several animal and clinical studies have examined the use of buffers during cardiac arrest. Clinical studies using Tribonate® [694] or sodium bicarbonate as buffers have failed to demonstrate any advantage.[694–701] Two studies have found that EMS systems using sodium bicarbonate earlier and more frequently had significantly higher ROSC and hospital discharge rates and better long-term neurological outcome.[702,703]. Animal studies have generally been inconclusive, but some have shown benefit in giving sodium bicarbonate to treat cardiovascular toxicity (hypotension, cardiac arrhythmias) caused by tricyclic antidepressants and other fast sodium channel blockers (Section 4).[224,704,705] Giving sodium bicarbonate routinely during cardiac arrest and CPR or after ROSC is not recommended. Consider sodium bicarbonate for:

- life-threatening hyperkalaemia
- cardiac arrest associated with hyperkalaemia
- tricyclic overdose.

Give 50 mmol (50 ml of an 8.4% solution) or 1 mmol kg^{-1} of sodium bicarbonate intravenously. Repeat the dose as necessary, but use acid/base analysis (either arterial, central venous or marrow aspirate from IO needle) to guide therapy. Severe tissue damage may be caused by subcutaneous extravasation of concentrated sodium bicarbonate. The solution is incompatible with calcium salts as it causes the precipitation of calcium carbonate.

Fibrinolysis during CPR

Fibrinolytic drugs may be used when pulmonary embolism is the suspected or known cause of cardiac arrest. Thrombus formation is a common cause of cardiac arrest, most commonly due to acute myocardial ischaemia following coronary artery occlusion by thrombus, but occasionally due to a dislodged venous thrombus causing a pulmonary embolism. The use of fibrinolytic drugs to break down coronary artery and pulmonary artery thrombus has been the subject of several studies. Fibrinolytics have also been demonstrated in animal studies to have beneficial effects on cerebral blood flow during cardiopulmonary resuscitation,[706,707] and a clinical study has reported less anoxic encephalopathy after fibrinolytic therapy during CPR.[708]

Several studies have examined the use of fibrinolytic therapy given during non-traumatic cardiac arrest unresponsive to standard therapy.[709–715] and some have shown non-significant improvements in survival to hospital discharge,[709,712] and greater ICU survival.[708] A small series of case reports also reported survival to discharge in three cases refractory to standard therapy with VF or PEA treated with fibrinolytics.[716] Conversely, two large clinical trials[717,718] failed to show any significant benefit for fibrinolytics in out-of-hospital cardiac arrest unresponsive to initial interventions.

Results from the use of fibrinolytics in patients suffering cardiac arrest from suspected pulmonary embolism have been variable. A meta-analysis, which included patients with pulmonary embolism as a cause of the arrest, concluded that fibrinolytics increased the rate of ROSC, survival to discharge and long-term neurological function.[719] Several other studies have demonstrated an improvement in ROSC and admission to hospital or the intensive care unit, but not in survival to neurologically intact hospital discharge.[709–712,714,715,720–723]

Although several relatively small clinical studies[709,710,712,721] and case series[708,716,724–726] have not demonstrated any increase in bleeding complications with thrombolysis during CPR in non-traumatic cardiac arrest, a recent large study[718] and meta-analysis[719] have shown an increased risk of intracranial bleeding associated with the routine use of fibrinolytics during non-traumatic cardiac arrest. Successful fibrinolysis during cardiopulmonary resuscitation is usually associated with good neurological outcome.[719,721,722]

Fibrinolytic therapy should not be used routinely in cardiac arrest. Consider fibrinolytic therapy when cardiac arrest is caused by proven or suspected acute pulmonary embolism. 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.[727–729] Ongoing CPR is not a contraindication to fibrinolysis. Treatment of pulmonary embolism is addressed in Section 4 including the role of extracorporeal CPR, and surgical or mechanical thrombectomy.[224]

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 balanced crystalloid solutions, Hartmann's solution or 0.9% sodium chloride. Avoid glucose, which is redistributed away from the intravascular space rapidly and causes hyperglycaemia, and may worsen neurological outcome after cardiac arrest.[730–738]

Whether fluids should be infused routinely during cardiac arrest is controversial. There are no published human studies specifically aimed to evaluate the advantages of routine fluid use compared to no fluids during normovolaemic cardiac arrest. Three animal studies show that the increase in right atrial pressure produced by infusion of fluids during CPR decreases coronary perfusion pressure,[739–741] and another animal study[742] shows that the coronary perfusion pressure rise with adrenaline during CPR is not improved with the addition of a fluid infusion. In a clinical trial which randomised patients to rapid prehospital cooling, accomplished by infusing up to 2 L of 4 °C normal saline immediately after ROSC, the incidence of re-arrest and pulmonary oedema on first chest X-ray was significantly higher in the intervention group.[743] This was not confirmed by a similar study in which patients received a median of 1 L of cold saline before hospital admission.[744] Results of a further study on rapid prehospital cooling (NCT01173393) are awaited.

One animal study shows that hypertonic saline improves cerebral blood flow during CPR.[745] However, one small clinical study[746] and one randomised trial[747] have not shown any benefit with hypertonic fluid during CPR. One retrospective matched pair analysis of a German OHCA registry showed that use of hypertonic saline with 6% hydroxyethyl starch was associated with increased rates of survival to hospital admission.[748] However there are concerns regarding the use of colloids and starch solutions in particular in critically ill patients.[749]

Ensure normovolaemia, but in the absence of hypovolaemia, infusion of an excessive volume of fluid is likely to be harmful.[750] Use intravenous fluid to flush peripherally injected drugs into the central circulation.

3h – CPR techniques and devices

At best, standard manual CPR produces coronary and cerebral perfusion that is just 30% of normal.[751] Several CPR techniques and devices aim to improve haemodynamics and survival when used by 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.[752] It is prudent that rescuers are well-trained and that if a circulatory adjunct is used, a programme 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,[753–755] no adjunct has consistently been shown to be superior to conventional manual CPR.

Mechanical chest compression devices

Providing high-quality manual chest compressions can be challenging and there is evidence that CPR quality deteriorates with time. Automated mechanical chest compression devices may enable the delivery of high quality compressions especially in circumstances where this may not be possible with manual compressions – CPR in a moving ambulance where safety is at risk, prolonged CPR (e.g. hypothermic arrest), and CPR during certain procedures (e.g. coronary angiography or preparation for extracorporeal CPR).[347,390,414,756–761] Data from the US American CARES (Cardiac Arrest Registry to Enhance Survival) registry shows that 45% of participating EMS services use mechanical devices.[762]

Since Guidelines 2010 there have been three large RCTs enrolling 7582 patients that have shown no clear advantage from the routine use of automated mechanical chest compression devices for OHCA.[763–765] Ensuring high-quality chest compressions with adequate depth, rate and minimal interruptions, regardless of whether they are delivered by machine or human is important.[766,767] In addition mechanical compressions usually follow a period of manual compressions.[768] The transition from manual compressions to mechanical compressions whilst minimising interruptions to chest compression and avoiding delays in defibrillation is therefore an important aspect of using these devices.

We suggest that automated mechanical chest compression devices are not used routinely to replace manual chest compressions. We suggest that automated mechanical chest compression devices are a reasonable alternative to high-quality manual chest compressions in situations where sustained high-quality manual chest compressions are impractical or compromise provider safety.[4] Interruptions to CPR during device deployment should be avoided. Healthcare personnel who use mechanical CPR should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills.

The experience from the three large RCTs suggests that use of mechanical devices requires initial and on-going training and quality assurance to minimise interruptions in chest compression when transitioning from manual to mechanical compressions and preventing delays in defibrillation. The use of training drills and 'pit-crew' techniques for device deployment are suggested to help minimise interruptions in chest compression.[769–771]

Our recommendation is generic for automated chest compression devices. Although there may be some device specific differences, they have not been directly compared in RCTs, and the three large RCTs[763–765] did not suggest a difference between the two most studied devices [the AutoPulse (Zoll Circulation, Chelmsford, Massachusetts, USA) and LUCAS-2 (Physio-Control Inc/Jolife AB, Lund, Sweden)] for critical and important patient outcomes when compared with the use of manual chest compressions alone.[4]

Information concerning the routine use of mechanical devices for IHCA is limited.[772] One small RCT of 150 IHCA patients showed improved survival with mechanical chest compressions delivered by a piston device [Thumper 1007 CCV device (Michigan Instruments, Grand Rapids, Michigan, USA)] when compared with manual compressions (OR 2.81, 95% CI 1.26–6.24).[773]

Lund University cardiac arrest system (LUCAS) CPR

The LUCAS delivers chest compression and active decompression through a piston system with suction cup. The current model is a battery driven device that delivers 100 compressions min^{-1} to a depth of 40–50 mm. There have been two large RCTs of the LUCAS device since the 2010 Guidelines.[764,765]

The LINC (LUCAS in cardiac arrest) RCT included 2589 adult OHCA patients and compared a modified CPR algorithm, which included mechanical chest compressions with a standard resuscitation algorithm which included manual chest compressions.[764] In the intention to treat analysis, there was no improvement in the primary outcome of 4-h survival (mechanical CPR 23.6% vs. manual CPR 23.7%, treatment difference −0.05%, 95% CI 3.3–3.2%; $p > 0.99$), 1 month survival (survival: 8.6% vs. 8.5%, treatment difference 0.16%, 95% CI 2.0–2.3%) and favourable neurological survival (8.1% vs. 7.3%, treatment difference 0.78%, 95% CI 1.3–2.8%). A follow-up study reported that patients who received LUCAS CPR were more likely to sustain injury (OR 3.4, 95% CI 1.55–7.31%), including rib fractures (OR 2.0, 95% CI 1.11–3.75%).[774]

The PaRAMeDIC trial (Prehospital Randomised Assessment of a Mechanical Compression Device) trial was cluster RCT that randomised ambulance vehicles to LUCAS or control and included 4471 patients (1652 LUCAS, 2819 manual chest compressions).[765] The intention-to-treat analysis found no improvement in the primary outcome of 30-day survival (LUCAS CPR 6% vs. manual CPR 7%, adjusted OR 0.86, 95% CI 0.64–1.15). Survival with a favourable neurological outcome at three months was lower amongst patients randomised to LUCAS CPR (5% vs. 6%, adjusted OR 0.72, 95% CI 0.52–0.99). In addition, in patients with VF/pVT, there was a lower 30-day survival with LUCAS CPR (OR 0.71, 95% CI 0.52–0.98). Delays in attempted defibrillation caused by device deployment may have caused this.

A meta-analysis of the three LUCAS RCTs that included 7178 patients with OHCA was included in the PARAMEDIC publication.[764,765,775] and reported similar initial and long-term survival (survived event OR 1.00, 95% CI 0.90–1.11; survival to discharge/30-days OR 0.96, 95% CI 0.80–1.15). Meta-analysis from the two larger RCTs noted significant heterogeneity ($I^2 = 69\%$) but no overall difference in neurological outcomes between LUCAS and manual chest compressions (random effects model OR 0.93, 95% CI 0.64–1.33).[764,765]

Load-distributing band CPR (AutoPulse)

The load-distributing band (LDB) is a battery-powered device consisting of a large backboard and a band that encircles the patient's chest. Compressions are delivered at a rate of 80 min^{-1} by tightening of the band. The evidence from clinical trials considered for the LDB in 2010 was conflicting. Evidence from one OHCA multi-centre RCT showed no improvement in 4-h survival and worse neurological outcome with LDB-CPR.[776] A further study showed lower odds of 30-day survival (OR 0.4) but subgroup analysis showed an increased rate of ROSC in LDB-CPR treated patients.[777] Non-randomised trials reported increased rates of sustained ROSC,[778,779] increased survival to discharge following

OHCA[779] and improved haemodynamics following failed resuscitation from in-hospital cardiac arrest.[780]

A recent large RCT showed similar outcomes for LDB and manual CPR.[763] The CIRC (Circulation Improving Resuscitation Care) trial, an equivalence RCT, randomised 4753 adult OHCA patients to the LDB or manual chest compressions. After a predefined adjustment for covariates and multiple interim analyses the adjusted OR was 1.06 (95% CI 0.83–1.37) and within the pre-defined region of equivalence for the primary outcome of survival to discharge (manual CPR vs. LDB CPR 11.0% vs. 9.4%). Good neurological survival to hospital discharge was similar (mechanical CPR 44.4% vs. manual CPR 48.1%, adjusted OR 0.80, 95% CI 0.47–1.37).

Open-chest CPR

Open-chest CPR produces better coronary perfusion coronary pressure than standard CPR and may be indicated for patients with cardiac arrest caused by trauma,[781] in the early postoperative phase after cardiothoracic surgery[782,783] (see Section 4 – special circumstances[224]) or when the chest or abdomen is already open (transdiaphragmatic approach), for example, in trauma surgery.[784]

Active compression-decompression CPR (ACD-CPR)

ACD-CPR is achieved with a hand-held device equipped with a suction cup to lift the anterior chest actively during decompression. Decreasing intrathoracic pressure during the decompression phase increases venous return to the heart and increases cardiac output and subsequent coronary and cerebral perfusion pressures during the compression phase.[785–788] Results of ACD-CPR have been mixed. In some clinical studies ACD-CPR improved haemodynamics compared with standard CPR,[786,788–790] but in another study it did not.[791] In three randomised studies,[790,792,793] ACD-CPR improved long-term survival after out-of-hospital cardiac arrest; however, in five other randomised studies, ACD-CPR made no difference to outcome.[794–798] The efficacy of ACD-CPR may be highly dependent on the quality and duration of training.[799]

A meta-analysis of 10 trials of out-of-hospital cardiac arrest and two of in-hospital cardiac arrest showed no early or late survival benefit to ACD-CPR over conventional CPR[234,800] and this is confirmed by another recent meta-analysis.[801] Two post-mortem studies have shown more rib and sternal fractures after ACD-CPR compared with conventional CPR,[802,803] but another found no difference.[804]

Impedance threshold device (ITD)

The impedance threshold device (ITD) is a valve that limits air entry into the lungs during chest recoil between chest compressions; this decreases intrathoracic pressure and increases venous return to the heart. When used with a cuffed tracheal tube and active compression–decompression (ACD),[805–807] the ITD is thought to act synergistically to enhance venous return during active decompression. The ITD has also been used during conventional CPR with a tracheal tube or facemask.[808] If rescuers can maintain a tight face-mask seal, the ITD may create the same negative intrathoracic pressure as when used with a tracheal tube.[808]

An RCT of the ITD with standard CPR compared to standard CPR alone with 8718 OHCA patients failed to show any benefit with ITD use in terms of survival and neurological outcome.[809] We therefore recommend that the ITD is not used routinely with standard CPR.

Two RCTs did not show a benefit in terms of survival to hospital discharge of the ITD with active compression decompression CPR when compared with active compression decompression CPR alone.[805,810]

Results of a large trial of a combination of ITD with active compression decompression CPR (ACD CPR) compared to standard CPR was reported in two publications. The primary publication reported the results from 2470 patients with OHCA[811] whereas the secondary publication reported results from those with non-traumatic cardiac arrest ($n = 27380$).[812] This study did detect a statistically significant difference in neurologically favourable survival at discharge, and survival at 12 months but no difference for survival to discharge and neurologically favourable survival at 12 months.[4] After consideration of the number needed to treat a decision was made not to recommend the routine use of the ITD and ACD.[4]

3i – Peri-arrest arrhythmias

The correct identification and treatment of arrhythmias in the critically ill patient may prevent cardiac arrest from occurring or reoccurring after successful initial resuscitation. The treatment algorithms described in this section have been designed to enable the non-specialist ALS provider to treat the patient effectively and safely in an emergency; for this reason, they have been kept as simple as possible. If patients are not acutely ill there may be several other treatment options, including the use of drugs (oral or parenteral) that will be less familiar to the non-expert. In this situation there will be time to seek advice from cardiologists or other senior doctors with the appropriate expertise.

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

Principles of treatment

The initial assessment and treatment of a patient with an arrhythmia should follow the ABCDE approach. Key elements in this process include assessing for adverse signs; oxygen if indicated and guided by pulse oximetry; 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 mmHg).
2. Syncope – loss of consciousness, which occurs as a consequence of reduced cerebral blood flow
3. Heart failure – arrhythmias compromise myocardial performance by reducing coronary artery blood flow. In acute situations this is manifested by pulmonary oedema (failure of the

left ventricle) and/or raised jugular venous pressure, and hepatic engorgement (failure of the right ventricle).

4. Myocardial ischaemia – this occurs when myocardial oxygen consumption exceeds delivery. Myocardial ischaemia may present with chest pain (angina) or may occur without pain as an isolated finding on the 12-lead ECG (silent ischaemia). The presence of myocardial ischaemia is especially important if there is underlying coronary artery disease or structural heart disease because it may cause further life-threatening complications including cardiac arrest.

Treatment options

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

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

Tachycardias

If the patient is unstable

If the patient is unstable and deteriorating, with any of the adverse signs and symptoms described above being caused by the tachycardia, attempt synchronised cardioversion immediately (Fig. 3.4). 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.

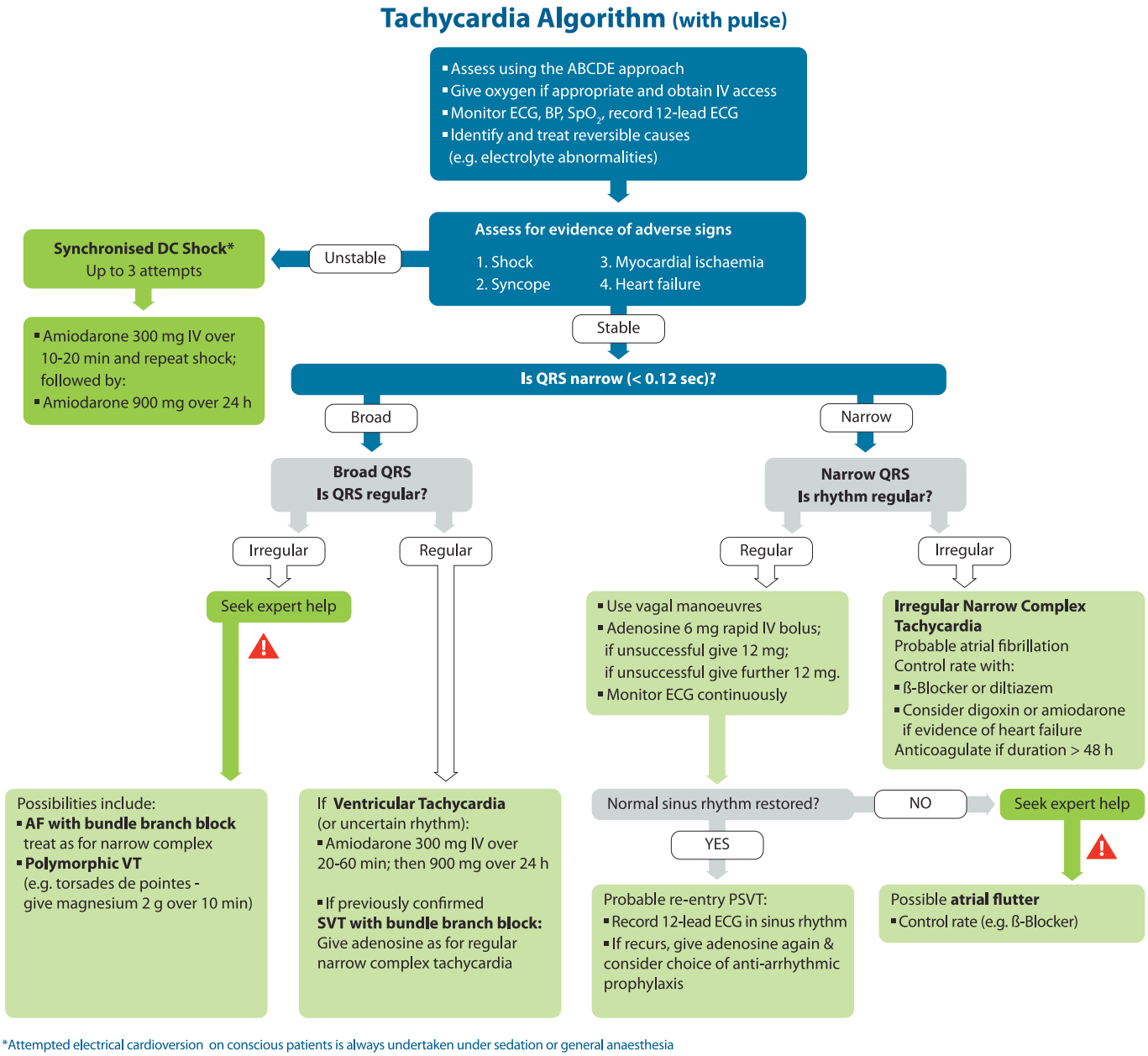


Fig. 3.4. Tachycardia algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure; IV – intravenous; SpO_2 – oxygen saturation measured by pulse oximetry; BP – blood pressure; ECG – electrocardiogram; DC – direct current; AF – atrial fibrillation; VT – ventricular tachycardia; SVT – supraventricular tachycardia; PSVT – paroxysmal supraventricular tachycardia.

The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 h.

Repeated attempts at electrical cardioversion are not appropriate for recurrent (within hours or days) paroxysms (self-terminating episodes) of atrial fibrillation. This is relatively common in critically ill patients who may have ongoing precipitating factors causing the arrhythmia (e.g. metabolic disturbance, sepsis). Cardioversion does not prevent subsequent arrhythmias. If there are recurrent episodes, treat them with drugs.

Synchronised electrical cardioversion. If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronised with the R wave of the ECG rather than with the T wave.[813] By avoiding the relative refractory period in this way, the risk of inducing ventricular fibrillation is minimised. Conscious patients must be anaesthetised or sedated before synchronised cardioversion is attempted. For a broad-complex tachycardia and AF, start with 120–150 J biphasic and increase in increments if this fails. Atrial flutter and paroxysmal supraventricular tachycardia (SVT) will often convert with lower energies: start with 70–120-J biphasic.

If the patient is stable

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

All anti-arrhythmic treatments – physical manoeuvres, drugs, or electrical treatment – can also be pro-arrhythmic, so that clinical deterioration may be caused by the treatment rather than lack of effect. The use of multiple anti-arrhythmic drugs or high doses of a single drug can cause myocardial depression and hypotension. This may cause a deterioration of the cardiac rhythm. Expert help should be sought before using repeated doses or combinations of anti-arrhythmic drugs.

Broad-complex tachycardia

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

Regular broad complex tachycardia. A regular broad-complex tachycardia is likely to be ventricular tachycardia or SVT with bundle branch block. If there is uncertainty about the source of the arrhythmia, give intravenous adenosine (using the strategy described below) as it may convert the rhythm to sinus and help diagnose the underlying rhythm.

Stable ventricular tachycardia can be treated with amiodarone 300 mg intravenously over 20–60 min followed by an infusion of 900 mg over 24 h. Specialist advice should be sought before considering alternatives treatments such as procainamide, nifekalant or sotalol.

Irregular broad complex tachycardia. Irregular broad complex tachycardia is most likely to be AF with bundle branch block. Another possible cause is AF with ventricular pre-excitation (Wolff–Parkinson–White (WPW) syndrome). In this case there is more variation in the appearance and width of the QRS complexes than in AF with bundle branch block. A third possible cause is polymorphic VT (e.g. torsade de pointes), although this rhythm is relatively unlikely to be present without adverse features.

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

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

Narrow-complex tachycardia

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

The commonest regular narrow-complex tachycardias include:

- sinus tachycardia;
- AV nodal re-entry tachycardia (AVNRT, the commonest type of SVT);
- AV re-entry tachycardia (AVRT), which is associated with Wolff–Parkinson–White (WPW) syndrome;
- atrial flutter with regular AV conduction (usually 2:1).

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

Regular narrow-complex tachycardia.

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

AVNRT and AVRT (paroxysmal SVT). AVNRT is the commonest type of paroxysmal SVT, often seen in people without any other form of heart disease and is relatively uncommon in a peri-arrest setting.[814] It causes a regular narrow-complex tachycardia, often with no clearly visible atrial activity on the ECG. Heart rates are usually well above the typical range of sinus rates at rest (60–120 beats min^{-1}). It is usually benign, unless there is additional co-incidental structural heart disease or coronary disease.

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

Atrial flutter with regular AV conduction (often 2:1 block). Atrial flutter with regular AV conduction (often 2:1 block) produces a regular narrow-complex tachycardia in which it may be difficult to see atrial activity and identify flutter waves with confidence, so it may be indistinguishable initially from AVNRT and AVRT. When atrial flutter with 2:1 block or even 1:1 conduction is accompanied by bundle branch block, it produces a regular broad-complex tachycardia that will usually be very difficult to distinguish from VT. Treatment of this rhythm as if it were VT will usually be effective, or will lead to slowing of the ventricular response and identification of the rhythm. Most typical atrial flutter has an atrial rate of

about 300 beats min^{-1}, so atrial flutter with 2:1 block tends to produce a tachycardia of about 150 beats min^{-1}. Much faster rates are unlikely to be due to atrial flutter with 2:1 block.

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

- Start with vagal manoeuvres[814]: carotid sinus massage or the Valsalva manoeuvre will terminate up to a quarter of episodes of paroxysmal SVT. Carotid sinus massage stimulates baroreceptors, which increase vagal tone and reduces sympathetic drive, which slows conduction via the AV node. Carotid sinus massage is given by applying pressure over the carotid artery at the level of the cricoid cartilage. Massage the area with firm circular movements for about 5 s. If this does not terminate the arrhythmia, repeat on the opposite side. Avoid carotid massage if a carotid bruit is present: rupture of an atheromatous plaque could cause cerebral embolism and stroke. A Valsalva manoeuvre (forced expiration against a closed glottis) in the supine position may be the most effective technique. A practical way of achieving this without protracted explanation is to ask the patient to blow into a 20 ml syringe with enough force to push back the plunger. Record an ECG (preferably multi-lead) during each manoeuvre. If the rhythm is atrial flutter, slowing of the ventricular response will often occur and demonstrate flutter waves.
- If the arrhythmia persists and is not atrial flutter, use adenosine. Give 6 mg as a rapid intravenous bolus. Record an ECG (preferably multi-lead) during each injection. If the ventricular rate slows transiently but the arrhythmia then persists, look for atrial activity such as atrial flutter or other atrial tachycardia and treat accordingly. If there is no response to adenosine 6 mg, give a 12 mg bolus; if there is no response, give one further 12 mg-bolus. This strategy will terminate 90–95% of supraventricular arrhythmias.
- Successful termination of a tachyarrhythmia by vagal manoeuvres or adenosine indicates that it was almost certainly AVNRT or AVRT. Monitor the patients for further rhythm abnormalities. Treat recurrence either with further adenosine or with a longer-acting drug with AV nodal-blocking action (e.g. diltiazem or verapamil).
- If adenosine is contraindicated or fails to terminate a regular narrow-complex tachycardia without demonstrating that it is atrial flutter, give a calcium channel blocker (e.g. verapamil or diltiazem).

Irregular narrow-complex tachycardia

An irregular narrow-complex tachycardia is most likely to be AF with an uncontrolled ventricular response or, less commonly, atrial flutter with variable AV block. Record a 12-lead ECG to identify the rhythm. If the patient is unstable with adverse features caused by the arrhythmia, attempt synchronised electrical cardioversion as described above. The European Society of Cardiology provides detailed guidelines on the management of AF: www.escardio.org.

If there are no adverse features, treatment options include:

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

Obtain expert help to determine the most appropriate treatment for the individual patient. The longer a patient remains in AF, the greater is the likelihood of atrial clot developing. In general, patients who have been in AF for more than 48 h should not be treated by cardioversion (electrical or chemical) until they have received full anticoagulation or absence of atrial clot has been shown by transoesophageal echocardiography. If the clinical scenario dictates that cardioversion is required and the duration of AF is greater than 48 h (or the duration is unknown) discuss anticoagulation, choice of agent, and duration with a cardiologist.

If the aim is to control heart rate, the drugs of choice are beta-blockers and diltiazem. Digoxin and amiodarone may be used in patients with heart failure.

If the duration of AF is less than 48 h and rhythm control is considered appropriate, chemical cardioversion may be attempted. Seek expert help and consider, flecainide, propafenone, or ibutilide. Amiodarone (300 mg intravenously over 20–60 min followed by 900 mg over 24 h) may also be used but is less effective. Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

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

Bradycardia

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

Bradycardias are caused by reduced sinoatrial node firing or failure of the atrial–ventricular conduction system. Reduced sinoatrial node firing is seen in sinus bradycardia (caused by excess vagal tone), sinus arrest, and sick sinus syndrome. Atrioventricular (AV) blocks are divided into first, second, and third degrees and may be associated with multiple medications or electrolyte disturbances, as well as structural problems caused by acute myocardial infarction and myocarditis. A first-degree AV block is defined by a prolonged P-R interval (>0.20 s), and is usually benign. Second-degree AV block is divided into Mobitz types I and II. In Mobitz type I, the block is at the AV node, is often transient and may be asymptomatic. In Mobitz type II, the block is most often below the AV node at the bundle of His or at the bundle branches, and is often symptomatic, with the potential to progress to complete AV block. Third-degree heart block is defined by AV dissociation, which may be permanent or transient, depending on the underlying cause.

Initial assessment

Assess the patient with bradycardia using the ABCDE approach. Consider the potential cause of the bradycardia and look for the adverse signs. Treat any reversible causes of bradycardia identified in the initial assessment. If adverse signs are present start to treat the bradycardia. Initial treatments are pharmacological, with pacing being reserved for patients unresponsive to pharmacological treatments or with risks factors for asystole (Fig. 3.5).

Pharmacological treatment

If adverse signs are present, give atropine 500 μg, intravenously and, if necessary, repeat every 3–5 min to a total of 3 mg. Doses of atropine of less than 500 μg, paradoxically, may cause further slowing of the heart rate.[815] In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart

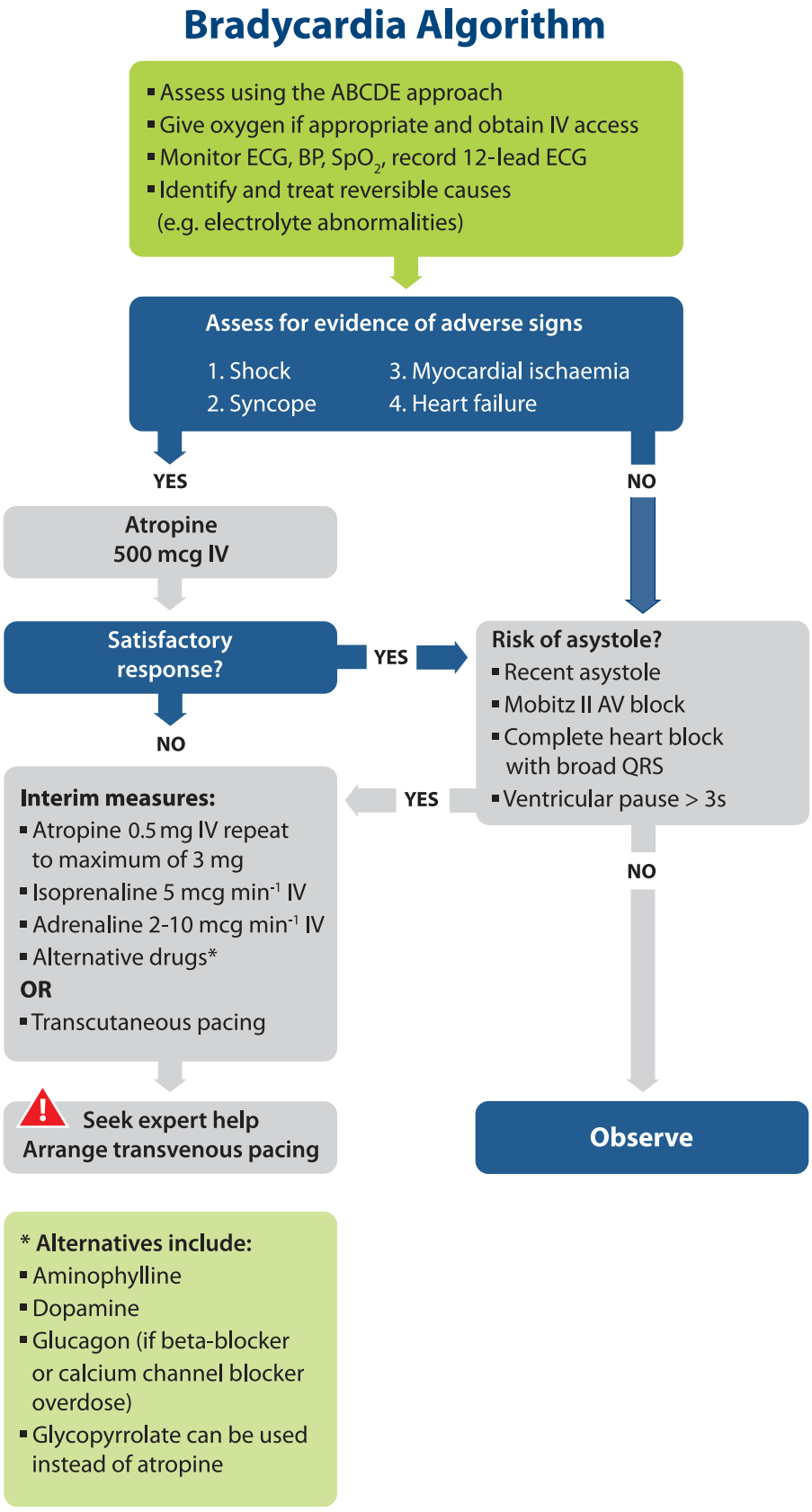


Fig. 3.5. Bradycardia algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure; IV – intravenous; SpO_2 – oxygen saturation measured by pulse oximetry; BP – blood pressure; ECG – electrocardiogram; AV – atrioventricular.

rate.[816] Use atropine cautiously in the presence of acute coronary ischaemia or myocardial infarction; increased heart rate may worsen ischaemia or increase the zone of infarction.

If treatment with atropine is ineffective, consider second line drugs. These include isoprenaline (5 μg min^{-1} starting dose), adrenaline (2–10 μg min^{-1}) and dopamine (2–10 μg kg^{-1} min^{-1}). Theophylline (100–200 mg slow intravenous injection) should be considered if the bradycardia is caused by inferior myocardial infarction, cardiac transplant or spinal cord injury. Consider giving intravenous glucagon if beta-blockers or calcium channel blockers are a potential cause of the bradycardia. Do not give atropine to patients with cardiac transplants – it can cause a high-degree AV block or even sinus arrest.[817]

Pacing

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

Transcutaneous pacing can be painful and may fail to produce effective mechanical capture. Verify mechanical capture and reassess the patient's condition. Use analgesia and sedation to control pain, and attempt to identify the cause of the bradyarrhythmia.

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

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

Antiarrhythmic drugs

Adenosine

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

Amiodarone

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

- Control of haemodynamically stable monomorphic VT, polymorphic VT and wide-complex tachycardia of uncertain origin.
- Paroxysmal SVT uncontrolled by adenosine, vagal manoeuvres or AV nodal blockade;
- to control rapid ventricular rate due to accessory pathway conduction in pre-excited atrial arrhythmias. In patients with pre-excitation and AF, digoxin, non-dihydropyridine calcium channel antagonists, or intravenous amiodarone should not be administered as they may increase the ventricular response and may result in VF.[818,819]
- Unsuccessful electrical cardioversion.

Give amiodarone, 300 mg intravenously, over 10–60 min depending on the circumstances and haemodynamic stability of the patient. This loading dose is followed by an infusion of 900 mg over 24 h. Additional infusions of 150 mg can be repeated as necessary for recurrent or resistant arrhythmias to a maximum manufacturer-recommended total daily dose of 2 g (this maximum licensed dose varies between different countries). In patients with severely impaired heart function, intravenous amiodarone is preferable to other anti-arrhythmic drugs for atrial and ventricular arrhythmias. Major adverse effects from amiodarone are hypotension and bradycardia, which can be prevented by slowing the rate of drug infusion. The hypotension associated with amiodarone is caused by vasoactive solvents (Polysorbate 80 and benzyl alcohol). An aqueous formulation of amiodarone does not contain these solvents and causes no more hypotension than lidocaine.[677] Whenever possible, intravenous amiodarone should be given via a central venous catheter; it causes thrombophlebitis when infused into a peripheral vein. In an emergency it can be injected into a large peripheral vein.

Calcium channel blockers: verapamil and diltiazem

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

- stable regular narrow-complex tachycardias uncontrolled or unconverted by adenosine or vagal manoeuvres;
- to control ventricular rate in patients with AF or atrial flutter and preserved ventricular function.

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

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

Beta-adrenergic blockers

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

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

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

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

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

Magnesium

Magnesium is the first line treatment for polymorphic ventricular tachycardia (torsades de pointes) and ventricular or supraventricular tachycardia associated with hypomagnesaemia. It may also reduce ventricular rate in atrial fibrillation. Give magnesium sulphate 2 g (8 mmol) over 10 min. This can be repeated once if necessary.

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European Resuscitation Council Guidelines for Resuscitation 2015 Section 4. Cardiac arrest in special circumstances

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Introduction

Irrespective of the cause of cardiac arrest, early recognition and calling for help, including appropriate management of the deteriorating patient, early defibrillation, high-quality cardiopulmonary resuscitation (CPR) with minimal interruption of chest compressions and treatment of reversible causes, are the most important interventions.

In certain conditions, however, advanced life support (ALS) guidelines require modification. The following guidelines for resuscitation in special circumstances are divided into three parts: special causes, special environments and special patients. The first part covers treatment of potentially reversible causes of cardiac arrest, for which specific treatment exists, and which must be identified or excluded during any resuscitation. For improving recall during ALS, these are divided into two groups of four, based upon their initial letter – either H or T – and are called the '4Hs and 4Ts': Hypoxia; Hypo-/hyperkalaemia and other electrolyte disorders; Hypo-/hyperthermia; Hypovolaemia; Tension pneumothorax; Tamponade (cardiac); Thrombosis (coronary and pulmonary); Toxins (poisoning). The second part covers cardiac arrest in special environments, where universal guidelines have to be modified due to specific locations or location-specific causes of cardiac arrest. The third part is focused on patients with specific conditions, and those with certain long-term comorbidities where a modified approach and different treatment decisions may be necessary.

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Summary of changes since 2010 Guidelines

The main changes in the ERC Guidelines 2015 in comparison with the Guidelines 2010[1] are summarised below:

Special causes

- Survival after an asphyxia-induced cardiac arrest is rare and survivors often have severe neurological impairment. During CPR, early effective ventilation of the lungs with supplementary oxygen is essential.
- A high degree of clinical suspicion and aggressive treatment can prevent cardiac arrest from electrolyte abnormalities. The new algorithm provides clinical guidance to emergency treatment of life-threatening hyperkalaemia.
- Hypothermic patients without signs of cardiac instability (systolic blood pressure $\geq$90 mmHg, absence of ventricular arrhythmias or core temperature $\geq$28 °C) can be rewarmed externally using minimally invasive techniques (e.g. with warm forced air and warm intravenous fluid). Patients with signs of cardiac instability should be transferred directly to a centre capable of extracorporeal life support (ECLS).
- Early recognition and immediate treatment with intramuscular adrenaline remains the mainstay of emergency treatment for anaphylaxis.
- The mortality from traumatic cardiac arrest (TCA) is very high. The most common cause of death is haemorrhage. It is recognised that most survivors do not have hypovolaemia, but instead have other reversible causes (hypoxia, tension pneumothorax, cardiac tamponade) that must be immediately treated. The new treatment algorithm for TCA was developed to prioritise the sequence of life-saving measures. Chest compressions should not delay the treatment of reversible causes. Cardiac arrests of non-traumatic origin leading to a secondary traumatic event should be recognised and treated with standard algorithms.
- There is limited evidence for recommending the routine transport of patients with continuing CPR after out-of-hospital cardiac arrest (OHCA) of suspected cardiac origin. Transport may be beneficial in selected patients where there is immediate hospital access to the catheterisation laboratory and an infrastructure providing prehospital and in-hospital teams experienced in mechanical or haemodynamic support and percutaneous coronary intervention (PCI) with ongoing CPR.
- Recommendations for administration of fibrinolytics when pulmonary embolism is the suspected cause of cardiac arrest remain unchanged. Routine use of surgical embolectomy or mechanical thrombectomy when pulmonary embolism is the suspected cause of cardiac arrest is not recommended. Consider these methods only when there is a known diagnosis of pulmonary embolism.
- Routine use of gastric lavage for gastrointestinal decontamination in poisoning is no longer recommended. Reduced emphasis is placed on hyperbaric oxygen therapy in carbon monoxide poisoning.

Special environments

- The special environments section includes recommendations for treatment of cardiac arrest occurring in specific locations. These locations are specialised healthcare facilities (e.g. operating theatre, cardiac surgery, catheterisation laboratory, dialysis unit, dental surgery), commercial airplanes or air ambulances, field of play, outside environment (e.g. drowning, difficult terrain, high altitude, avalanche burial, lightning strike and electrical injuries) or the scene of a mass casualty incident.
- Patients undergoing surgical procedures involving general anaesthesia, particularly in emergencies, are at risk from perioperative cardiac arrest. A new section covers the common causes and relevant modification to resuscitative procedures in this group of patients.
- Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase. Key to successful resuscitation is recognition of the need to perform emergency resternotomy, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective. Resternotomy should be performed within 5 min if other interventions have failed.
- Cardiac arrest from shockable rhythms (Ventricular Fibrillation (VF) or pulseless Ventricular Tachycardia (pVT)) during cardiac catheterisation should immediately be treated with up to three stacked shocks before starting chest compressions. Use of mechanical chest compression devices during angiography is recommended to ensure high-quality chest compressions and reduce the radiation burden to personnel during angiography with ongoing CPR.
- In dental surgery, do not move the patient from the dental chair in order to start CPR. Quickly recline the dental chair into a horizontal position and place a stool under the head of the chair to increase its stability during CPR.
- The in-flight use of AEDs aboard commercial airplanes can result in up to 50% survival to hospital discharge. AEDs and appropriate CPR equipment should be mandatory on board of all commercial aircraft in Europe, including regional and low-cost carriers. Consider an over-the-head technique of CPR if restricted access precludes a conventional method, e.g. in the aisle.
- The incidence of cardiac arrest on board helicopter emergency medical services (HEMS) and air ambulances is low. Importance of pre-flight preparation and use of mechanical chest compression devices are emphasised.
- Sudden and unexpected collapse of an athlete on the field of play is likely to be cardiac in origin and requires rapid recognition and early defibrillation.
- The duration of submersion is a key determinant of outcome from drowning. Submersion exceeding 10 min is associated with poor outcome. Bystanders play a critical role in early rescue and resuscitation. Resuscitation strategies for those in respiratory or cardiac arrest continue to prioritise oxygenation and ventilation.
- The chances of good outcome from cardiac arrest in difficult terrain or mountains may be reduced because of delayed access and prolonged transport. There is a recognised role of air rescue and availability of AEDs in remote but often-visited locations.
- The cut-off criteria for prolonged CPR and extracorporeal rewarming of avalanche victims in cardiac arrest are more stringent to reduce the number of futile cases treated with extracorpoereal life support (ECLS). ECLS is indicated if the duration of burial is >60 min (instead of >35 min), core temperature at extrication is <30 °C (instead of <32 °C), and serum potassium at hospital admission is $\leq$8 mmol L^{-1} (instead of $\leq$12 mmol L^{-1}); otherwise standard guidelines apply.
- Safety measures are emphasised when providing CPR to the victim of an electrical injury.
- Recommendations for management of multiple victims should prevent delay of treatment available for salvageable victims during mass casualty incidents (MCIs). Safety at scene is paramount. A triage system should be used to prioritise treatment and, if the number of casualties overwhelms healthcare resources, withhold CPR for those without signs of life.

Special patients

- The section on special patients gives guidance for CPR in patients with severe comorbidities (asthma, heart failure with

ventricular assist devices, neurological disease, obesity) and those with specific physiological conditions (pregnancy, elderly people).

- The first line treatment for acute asthma is inhaled beta-2 agonists while intravenous beta-2 agonists are suggested only for those patients in whom inhaled therapy cannot be used reliably. Inhaled magnesium is no longer recommended.
- In patients with ventricular assist devices (VADs), confirmation of cardiac arrest may be difficult. If during the first 10 days after surgery, cardiac arrest does not respond to defibrillation, perform resternotomy immediately.
- Patients with subarachnoid haemorrhage may have ECG changes that suggest an acute coronary syndrome (ACS). Whether a computed tomography (CT) brain scan is done before or after coronary angiography will depend on clinical judgement regarding the likelihood of a subarachnoid haemorrhage versus acute coronary syndrome.
- No changes to the sequence of actions are recommended in resuscitation of obese patients, although delivery of effective CPR may be challenging. Consider changing rescuers more frequently than the standard 2-min interval. Early tracheal intubation by an experienced provider is recommended.
- For the pregnant woman in cardiac arrest, high-quality CPR with manual uterine displacement, early ALS and delivery of the fetus if early return of spontaneous circulation (ROSC) is not achieved remain key interventions.

A – SPECIAL CAUSES

Hypoxia

Introduction

Cardiac arrest caused by pure hypoxaemia is uncommon. It is seen more commonly as a consequence of asphyxia, which accounts for most of the non-cardiac causes of cardiac arrest. There are many causes of asphyxial cardiac arrest (Table 4.1); although there is usually a combination of hypoxaemia and hypercarbia, it is the hypoxaemia that ultimately causes cardiac arrest.[2]

Pathophysiological mechanisms

If breathing is completely prevented by airway obstruction or apnoea, consciousness will be lost when oxygen saturation in the arterial blood reaches about 60%. The time taken to reach this concentration is difficult to predict, but is likely to be of the order 1–2 min.[3] Based on animal experiments of cardiac arrest caused by asphyxia, pulseless electrical activity (PEA) will occur in 3–11 min. Asystole will ensue several minutes later.[4] In comparison with simple apnoea, the exaggerated respiratory movements that frequently accompany airway obstruction will increase oxygen consumption resulting in more rapid arterial blood oxygen desaturation and a shorter time to cardiac arrest. According to Safar, complete airway obstruction after breathing air will result in PEA cardiac arrest in 5–10 min.[2] VF is rarely the first monitored rhythm after asphyxial cardiac arrest – in one of the largest series of hanging-associated out-of-hospital cardiac arrests (OHCAs), from Melbourne, Australia, just 7 (0.5%) of 1321 patients were in VF.[5]

Table 4.1
Causes of asphyxial cardiac arrest

Airway obstruction: soft tissues (coma), laryngospasm, aspiration
Anaemia
Asthma
Avalanche burial
Central hypoventilation – brain or spinal cord injury
Chronic obstructive pulmonary disease
Drowning
Hanging
High altitude
Impaired alveolar ventilation from neuromuscular disease
Pneumonia
Tension pneumothorax
Trauma
Traumatic asphyxia or compression asphyxia (e.g. crowd crush)

Treatment

Treating the cause of the asphyxia/hypoxaemia is the highest priority because this is a potentially reversible cause of the cardiac arrest. Effective ventilation with supplementary oxygen is a particular priority in these patients. The better outcomes for OHCA victims receiving compression-only CPR[6] is not the case for asphyxial cardiac arrests, which have much better survival rates with conventional CPR.[7] Follow the standard ALS algorithm when resuscitating these patients.

Outcome

Survival after cardiac arrest from asphyxia is rare and most survivors sustain severe neurological injury. Of five published series that included a total of 286 patients with cardiac arrest following hanging where CPR was attempted (this was attempted in only about 16% of cases), there were just six (2%) survivors with a full recovery; 11 other survivors all had severe permanent brain injury.[5,8–11] In one third (89; 31%) of these 286 patients, rescuers were able to achieve ROSC – thus when CPR is attempted, ROSC is not uncommon but subsequent neurologically intact survival is rare. Those who are unconscious but have not progressed to a cardiac arrest are much more likely to make a good neurological recovery.[11,12]

Hypo-/hyperkalaemia and other electrolyte disorders

Introduction

Electrolyte abnormalities can cause cardiac arrhythmias or cardiac arrest. Life-threatening arrhythmias are associated most commonly with potassium disorders, particularly hyperkalaemia, and less commonly with disorders of serum calcium and magnesium. Consider electrolyte disturbances in patient groups at risk – renal failure, severe burns, cardiac failure and diabetes mellitus.

The electrolyte values for definitions have been chosen as a guide to clinical decision-making. The precise values that trigger treatment decisions will depend on the patient's clinical condition and rate of change of electrolyte values. There is little or no evidence for the treatment of electrolyte abnormalities during cardiac arrest. Guidance during cardiac arrest is based on the strategies used in the non-arrest patient.

Prevention of electrolyte disorders

When possible, identify and treat life-threatening electrolyte abnormalities before cardiac arrest occurs. Monitor renal function in patients at risk and avoid combination of drugs that may exacerbate hyperkalaemia. Prevent recurrence of electrolyte disorders by removing any precipitating factors (e.g. drugs, diet).

Potassium disorders

Potassium homeostasis. Extracellular potassium concentration is regulated tightly between 3.5 and 5.0 mmol L^{-1}. A large concentration gradient normally exists between intracellular and extracellular fluid compartments. This potassium gradient across cell membranes contributes to the excitability of nerve and muscle cells, including the myocardium. Evaluation of serum potassium must take into consideration the effects of changes in serum pH. When serum pH decreases (acidaemia), serum potassium increases because potassium shifts from the cellular to the vascular space; a process that is reversed when serum pH increases (alkalaemia).

Hyperkalaemia. This is the most common electrolyte disorder associated with cardiac arrest. It is usually caused by impaired excretion by the kidneys, drugs or increased potassium release from cells and metabolic acidosis. Hyperkalaemia occurs in up to 10% of hospitalised patients.[13–15] Chronic kidney disease (CKD) is common in the general population and the incidence of hyperkalaemia increases from 2 to 42% as glomerular filtration rate (GFR) drops from 60 to 20 mL min^{-1}.[16] Patients with end-stage renal disease are particularly susceptible, particularly following an OHCA.[17] Prolonged hyperkalaemia is an independent risk factor for in-hospital mortality.[18] Acute hyperkalaemia is more likely than chronic hyperkalaemia to cause life-threatening cardiac arrhythmias or cardiac arrest.

Definition. There is no universal definition. We have defined hyperkalaemia as a serum potassium concentration higher than 5.5 mmol L^{-1}; in practice, hyperkalaemia is a continuum. As the potassium concentration increases above this value the risk of adverse events increases and the need for urgent treatment increases. Severe hyperkalaemia has been defined as a serum potassium concentration higher than 6.5 mmol L^{-1}.

Causes. The main causes of hyperkalaemia are:

- renal failure (i.e. acute kidney injury or chronic kidney disease);
- drugs (e.g. angiotensin converting enzyme inhibitors (ACE-I), angiotensin II receptor antagonists (ARB), potassium-sparing diuretics, non-steroidal anti-inflammatory drugs, beta-blockers, trimethoprim);
- tissue breakdown (e.g. rhabdomyolysis, tumour lysis, haemolysis);
- metabolic acidosis (e.g. renal failure, diabetic ketoacidosis);
- endocrine disorders (e.g. Addison's disease);
- diet (may be sole cause in patients with advanced chronic kidney disease) and
- spurious – pseudo-hyperkalaemia (suspect in cases with normal renal function, normal ECG and/or history of haematological disorder). Pseudo-hyperkalaemia describes the finding of a raised serum (clotted blood) K^+ value concurrently with a normal plasma (non-clotted blood) potassium value. The clotting process releases K^+ from cells and platelets, which increases the serum K^+ concentration by an average of 0.4 mmol/L. The most common cause of pseudo-hyperkalaemia is a prolonged transit time to the laboratory or poor storage conditions.[19,20]

The risk of hyperkalaemia is even greater when there is a combination of factors such as the concomitant use of angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers and potassium-sparing diuretics.

Recognition of hyperkalaemia. Exclude hyperkalaemia in all patients with an arrhythmia or cardiac arrest. Patients may present with weakness progressing to flaccid paralysis, paraesthesia, or depressed deep tendon reflexes. Alternatively, the clinical picture can be overshadowed by the primary illness causing hyperkalaemia. The first indicator of hyperkalaemia may also be the presence of ECG abnormalities, arrhythmias, or cardiac arrest. The use of a blood gas analyser to measure potassium can reduce delays in recognition.[21,22]

The effect of hyperkalaemia on the ECG depends on the absolute serum potassium as well as the rate of increase.[23] The reported frequency of ECG changes in severe hyperkalaemia is variable, but most patients appear to show ECG abnormalities at a serum potassium concentration higher than 6.7 mmol L^{-1}.[23,24] The presence of ECG changes strongly correlates with mortality.[25] In some cases, the ECG may be normal or show atypical changes including ST elevation.

The ECG changes associated with hyperkalaemia are usually progressive and include:

- first degree heart block (prolonged PR interval >0.2 s);
- flattened or absent P waves;
- tall, peaked (tented) T waves (i.e. T wave larger than R wave in more than 1 lead);
- ST-segment depression;
- S & T wave merging (sine wave pattern);
- widened QRS (>0.12 s);
- ventricular tachycardia;
- bradycardia;
- cardiac arrest (PEA, VF/pVT, asystole).

Treatment of hyperkalaemia. There are five key treatment strategies for hyperkalaemia[22]:

- cardiac protection;
- shifting potassium into cells;
- removing potassium from the body;
- monitoring serum potassium and blood glucose;
- prevention of recurrence.

When hyperkalaemia is strongly suspected, e.g. in the presence of ECG changes, start life-saving treatment even before laboratory results are available. The treatment strategy for hyperkalaemia has been reviewed extensively.[13,22,26] Follow the hyperkalaemia emergency treatment algorithm (Fig. 4.1).[22] Avoid salbutamol monotherapy, which may be ineffective. There is insufficient evidence to support the use of sodium bicarbonate to decrease serum potassium. Consider the need for early specialist or critical care referral.

The main risks associated with treatment of hyperkalaemia are:

- Hypoglycaemia following insulin-glucose administration (usually occurs within 1–3 h of treatment, but may occur up to 6 h after infusion).[27] Monitor blood glucose and treat hypoglycaemia promptly.
- Tissue necrosis secondary to extravasation of intravenous calcium salts. Ensure secure vascular access prior to administration.
- Intestinal necrosis or obstruction following use of potassium exchange resins. Avoid prolonged use of resins and give laxative.
- Rebound hyperkalaemia after the effect of drug treatment has worn off (i.e. within 4–6 h). Continue to monitor serum potassium for a minimum of 24 h after an episode.

Patient not in cardiac arrest
Assess patient:

- Use systematic ABCDE approach and correct any abnormalities, obtain IV access.
- Check serum potassium.
- Record an ECG.

Monitor cardiac rhythm in patients with severe hyperkalaemia. Treatment is determined according to severity of hyperkalaemia. Approximate values are provided to guide treatment. Follow hyperkalaemia emergency treatment algorithm (Fig. 4.1).

Mild elevation (5.5–5.9 mmol L^{-1}).

- Address cause of hyperkalaemia to correct and avoid further rise in serum potassium (e.g. drugs, diet).
- If treatment is indicated, remove potassium from the body: potassium exchange resins-calcium resonium 15–30 g, or sodium polystyrene sulfonate (Kayexalate) 15–30 g, given either orally or by retention enema/PR (per rectum) (onset in >4 h).

Moderate elevation (6.0–6.4 mmol L^{-1}) without ECG changes.

- Shift potassium intracellularly with glucose/insulin: 10 units short-acting insulin and 25 g glucose IV over 15–30 min (onset in 15–30 min; maximal effect at 30–60 min; duration of action 4–6 h; monitor blood glucose).
- Remove potassium from the body (see above; consider dialysis guided by clinical setting).

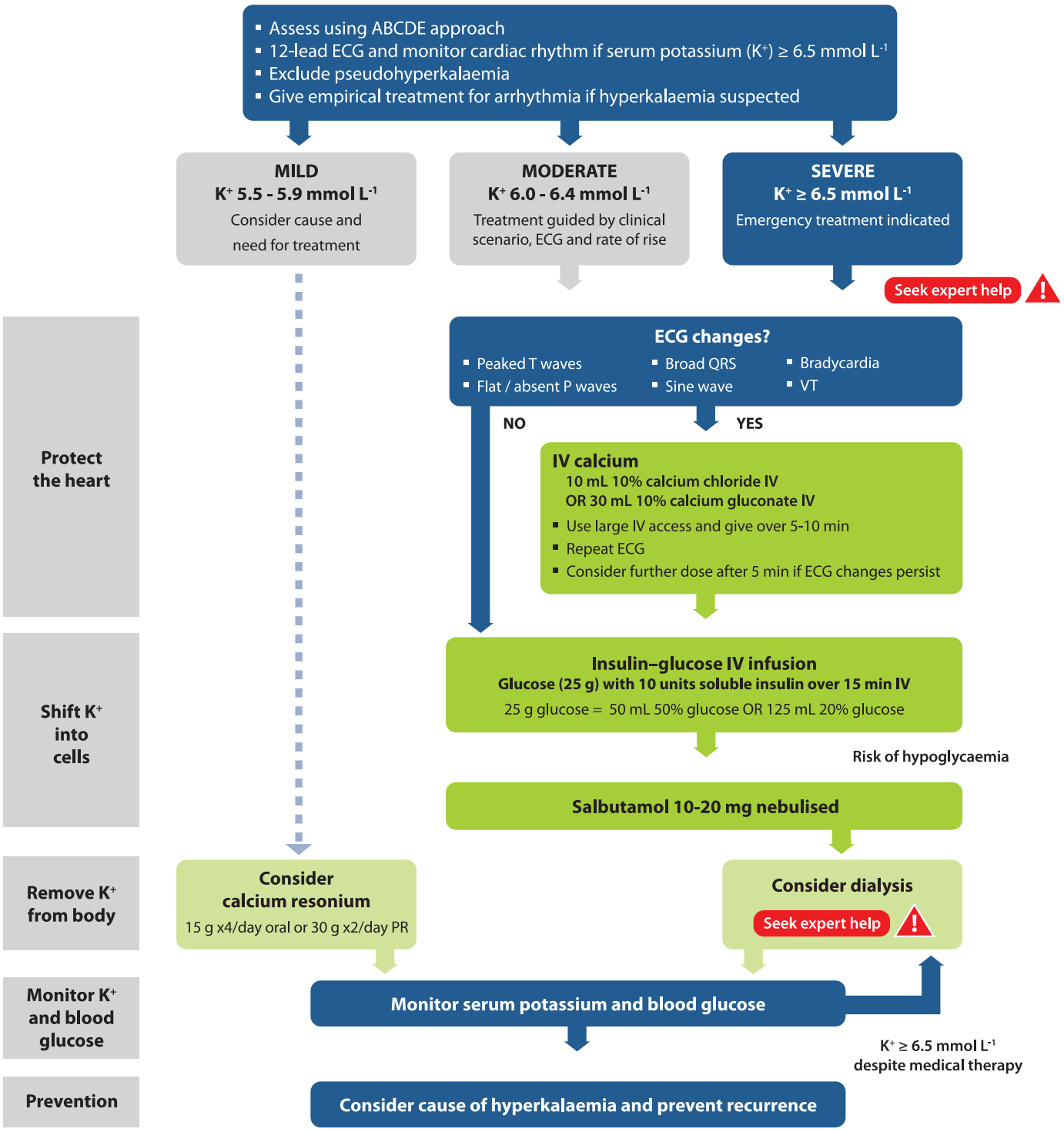

Fig. 4.1. Emergency treatment of hyperkalaemia. PR per rectum; ECG electrocardiogram; VT ventricular tachycardia.
Reproduced with permission from Renal Association and Resuscitation Council (UK).

Severe elevation (≥6.5 mmol L^{-1}) without ECG changes.

- Seek expert help.
- Give glucose/insulin (see above).
- Give salbutamol 10–20 mg nebulised (onset in 15–30 min; duration of action 4–6 h).
- Remove potassium from the body (consider dialysis).

Severe elevation (≥6.5 mmol L^{-1}) with toxic ECG changes.

- Seek expert help.
- Protect the heart with calcium chloride: 10 mL 10% calcium chloride IV over 2–5 min to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane. This protects the heart by reducing the risk of VF/pVT but does not lower serum potassium (onset in 1–3 min).
- Use shifting agents (glucose/insulin and salbutamol).
- Remove potassium from the body (consider dialysis at outset or if refractory to medical treatment).

Modifications to cardiopulmonary resuscitation. The following modifications to standard ALS guidelines are recommended in the presence of severe hyperkalaemia:

- Confirm hyperkalaemia using a blood gas analyser if available.
- Protect the heart: give 10 mL calcium chloride 10% IV by rapid bolus injection.
- Shift potassium into cells: Give glucose/insulin: 10 units short-acting insulin and 25 g glucose IV by rapid injection. Monitor blood glucose.
- Give sodium bicarbonate: 50 mmol IV by rapid injection (if severe acidosis or renal failure).
- Remove potassium from body: Consider dialysis for hyperkalaemic cardiac arrest resistant to medical treatment. Several dialysis modalities have been used safely and effectively in cardiac arrest, but this may only be available in specialist centres.[28] Consider use of a mechanical chest compression device if prolonged CPR is needed.

Indications for dialysis. The main indications for dialysis in patients with hyperkalaemia are:

- severe life-threatening hyperkalaemia with or without ECG changes or arrhythmia;
- hyperkalaemia resistant to medical treatment;
- end-stage renal disease;
- oliguric acute kidney injury (<400 mL day^{-1} urine output);
- marked tissue breakdown (e.g. rhabdomyolysis).

Special considerations for management of cardiac arrest in a dialysis unit are addressed in the section Special environments (see cardiac arrest in a dialysis unit).

Hypokalaemia. Hypokalaemia is the most common electrolyte disorder in clinical practice.[29] It is seen in up to 20% of hospitalised patients.[30] Hypokalaemia increases the incidence of arrhythmias and sudden cardiac death (SCD).[31] The risk is increased in patients with pre-existing heart disease and in those treated with digoxin.

Definition. Hypokalaemia is defined as a serum potassium level <3.5 mmol L^{-1}. Severe hypokalaemia is defined as a serum potassium level <2.5 mmol L^{-1} and may be associated with symptoms.

Causes. The main causes of hypokalaemia include:

- gastrointestinal loss (e.g. diarrhoea);
- drugs (e.g. diuretics, laxatives, steroids);
- renal losses (e.g. renal tubular disorders, diabetes insipidus, dialysis);
- endocrine disorders (e.g. Cushing's syndrome, hyperaldosteronism);
- metabolic alkalosis;
- magnesium depletion;
- poor dietary intake.

Treatment strategies used for hyperkalaemia may also induce hypokalaemia.

Recognition of hypokalaemia. Exclude hypokalaemia in every patient with an arrhythmia or cardiac arrest. In dialysis patients, hypokalaemia may occur at the end of a haemodialysis session or during treatment with peritoneal dialysis.

As serum potassium concentration decreases, the nerves and muscles are predominantly affected, causing fatigue, weakness, leg cramps, constipation. In severe cases (serum potassium <2.5 mmol L^{-1}), rhabdomyolysis, ascending paralysis and respiratory difficulties may occur.

ECG features of hypokalaemia are:

- U waves;
- T wave flattening;
- ST segment changes;
- arrhythmias, especially if patient is taking digoxin;
- cardiac arrest (PEA, VF/pVT, asystole).

Treatment. This depends on the severity of hypokalaemia and the presence of symptoms and ECG abnormalities. Gradual replacement of potassium is preferable, but in an emergency, intravenous potassium is required. The maximum recommended IV dose of potassium is 20 mmol h^{-1}, but more rapid infusion (e.g. 2 mmol min^{-1} for 10 min, followed by 10 mmol over 5–10 min) is indicated for unstable arrhythmias when cardiac arrest is imminent. Continuous ECG monitoring is essential during IV infusion and the dose should be titrated after repeated sampling of serum potassium levels.

Many patients who are potassium deficient are also deficient in magnesium. Magnesium is important for potassium uptake and for the maintenance of intracellular potassium values, particularly in the myocardium. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases of hypokalaemia.[32]

Calcium and magnesium disorders

The recognition and management of calcium and magnesium disorders is summarised in Table 4.2.

Hypo-/hyperthermia

Accidental hypothermia

Definition. Every year approximately 1500 people die of primary accidental hypothermia in the United States.[33] Accidental hypothermia is defined as an involuntary drop of the body core temperature <35 °C. The Swiss staging system is used to estimate core temperature at the scene. Its stages are based on clinical signs, which roughly correlate with the core temperature:

- hypothermia I; mild hypothermia (conscious, shivering, core temperature 35–32 °C);
- hypothermia II; moderate hypothermia (impaired consciousness without shivering, core temperature 32–28 °C);
- hypothermia III; severe hypothermia (unconscious, vitals signs present, core temperature 28–24 °C);
- hypothermia IV; cardiac arrest or low flow state (no or minimal vital signs, core temperature <24 °C);
- hypothermia V; death due to irreversible hypothermia (core temperature <13.7 °C).[34]

Diagnosis. Hypothermia is diagnosed in any patient with a core temperature <35 °C, or where measurement unavailable, a history of exposure to cold, or when the trunk feels cold.[33] Accidental hypothermia may be under-diagnosed in countries with a temperate climate. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia may follow a mild insult. The risk of hypothermia is increased by alcohol or drug ingestion, exhaustion, illness, injury or neglect especially when there is a decrease in the level of consciousness.

A low-reading thermometer is needed to measure the core temperature and confirm the diagnosis. The core temperature in the lower third of the oesophagus correlates well with heart temperature. Tympanic measurement using a thermistor technique is a reliable alternative but may be considerably lower than core temperature if the environment is very cold, the probe is not well insulated, or the external auditory canal is filled with snow or water[35,36] Widely available tympanic thermometers based on infrared technique do not seal the ear canal and are not designed for low core temperature readings.[37] The in-hospital core temperature measurement site should be the same throughout resuscitation and rewarming. Bladder and rectal temperatures lag behind core temperature;[38,39] for this reason, measurement of bladder and

Table 4.2
Calcium and magnesium disorders with associated clinical presentation, ECG manifestations and recommended treatment

Disorder	Causes	Presentation	ECG	Treatment
Hypercalcaemia				
Calcium > 2.6 mmol L^{-1}	Primary or tertiary hyperparathyroidism Malignancy Sarcoidosis Drugs	Confusion Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Short QT interval Prolonged QRS interval Flat T waves AV block Cardiac arrest	Fluid replacement IV Furosemide 1 mg kg^{-1} IV Hydrocortisone 200–300 mg IV Pamidronate 30–90 mg IV Treat underlying cause
Hypocalcaemia				
Calcium < 2.1 mmol L^{-1}	Chronic renal failure Acute pancreatitis Calcium channel blocker overdose Toxic shock syndrome Rhabdomyolysis Tumour lysis syndrome	Paraesthesia Tetany Seizures AV-block Cardiac arrest	Prolonged QT interval T wave inversion Heart block Cardiac arrest	Calcium chloride 10% 10–40 mL Magnesium sulphate 50% 4–8 mmol (if necessary)
Hypermagnesaemia				
Magnesium > 1.1 mmol L^{-1}	Renal failure Iatrogenic	Confusion Weakness Respiratory depression AV-block Cardiac arrest	Prolonged PR and QT intervals T wave peaking AV block Cardiac arrest	*Consider treatment when magnesium > 1.75 mmol L^{-1}* Calcium chloride 10% 5–10 mL repeated if necessary Ventilatory support if necessary Saline diuresis – 0.9% saline with furosemide 1 mg kg^{-1} IV Haemodialysis
Hypomagnesaemia				
Magnesium < 0.6 mmol L^{-1}	GI loss Polyuria Starvation Alcoholism Malabsorption	Tremor Ataxia Nystagmus Seizures Arrhythmias – torsade de pointes Cardiac arrest	Prolonged PR and QT intervals ST-segment depression T-wave inversion Flattened P waves Increased QRS duration Torsade de pointes	*Severe or symptomatic*: 2 g 50% magnesium sulphate (4 mL; 8 mmol) IV over 15 min *Torsade de pointes*: 2 g 50% magnesium sulphate (4 mL; 8 mmol) IV over 1–2 min *Seizure*: 2 g 50% magnesium sulphate (4 mL; 8 mmol) IV over 10 min

rectal temperature has been de-emphasised in patients with severe hypothermia.

Decision to resuscitate. Cooling of the human body decreases cellular oxygen consumption by about 6% per 1 °C decrease in core temperature.[40] At 28 °C, oxygen consumption is reduced by approximately 50% and at 22 °C by approximately 75%. At 18 °C the brain can tolerate cardiac arrest for up to 10 times longer than at 37 °C. This results in hypothermia exerting a protective effect on the brain and heart,[41] and intact neurological recovery may be possible even after prolonged cardiac arrest if deep hypothermia develops before asphyxia.

Beware of diagnosing death in a hypothermic patient because hypothermia itself may produce a very slow, small-volume, irregular pulse and unrecordable blood pressure. In a deeply hypothermic patient (hypothermia IV) signs of life may be so minimal that it is easy to overlook them. Therefore, look for signs of life for at least 1 min and use an ECG monitor to detect any electrical cardiac activity. Neurologically intact survival has been reported after hypothermic cardiac arrest with a core temperature as low as 13.7 °C[42] and CPR for as long as six and a half hours.[43]

Intermittent CPR, as rescue allows, may also be of benefit.[44] If continuous CPR cannot be delivered, a patient with hypothermic cardiac arrest and a core temperature <28 °C (or unknown), should receive 5 min of CPR, alternating with periods ≤5 min without CPR. Patients with a core temperature <20 °C, should receive 5 min of CPR, alternating with periods ≤10 min without CPR.[45]

In the prehospital setting, resuscitation should be withheld in hypothermic patients only if the cause of cardiac arrest is clearly attributable to a lethal injury, fatal illness, prolonged asphyxia, or if the chest is incompressible.[46] In all other hypothermic patients, the traditional guiding principle that 'no one is dead until warm and dead' should be considered. In remote areas, the impracticalities of achieving rewarming have to be considered. In the hospital setting involve senior doctors and use clinical judgement to determine when to stop resuscitating a hypothermic victim in cardiac arrest.

Modifications to cardiopulmonary resuscitation

- Do not delay careful tracheal intubation when it is indicated. The advantages of adequate oxygenation and protection from aspiration outweigh the minimal risk of triggering VF by performing tracheal intubation.[47]
- Check for signs of life for up to 1 min. Palpate a central artery and assess the cardiac rhythm (if ECG monitor available). Echocardiography, near-infrared spectroscopy or ultrasound with Doppler may be used to establish whether there is (an adequate) cardiac output or peripheral blood flow.[48,49] If there is any doubt, start CPR immediately.
- Hypothermia can cause stiffness of the chest wall, making ventilations and chest compressions difficult. Consider the use of mechanical chest compression devices.[50]
- Once CPR is under way, confirm hypothermia with a low-reading thermometer.
- 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 drug given.[51] The evidence for the efficacy of drugs in severe hypothermia is limited and based mainly on animal studies. For instance, in severe hypothermic cardiac arrest, the efficacy of amiodarone is reduced.[52] Adrenaline may be effective in increasing coronary perfusion pressure, but not

survival.[53,54] Vasopressors may also increase the chances of successful defibrillation, but with a core temperature <30 °C, sinus rhythm often degrades back into VF. Given that defibrillation and adrenaline may induce myocardial injury, it is reasonable to withhold adrenaline, other CPR drugs and shocks until the patient has been warmed to a core temperature ≥30 °C. Once 30 °C has been reached, the intervals between drug doses should be doubled when compared to normothermia (i.e. adrenaline every 6–10 min). As normothermia (≥35 °C) is approached, use standard drug protocols.

Treatment of arrhythmias. As core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation followed by VF and finally asystole.[55,56] Arrhythmias other than VF tend to revert spontaneously as core temperature increases, and usually do not require immediate treatment. Bradycardia is physiological in severe hypothermia. Cardiac pacing is not indicated unless bradycardia associated with haemodynamic compromise persists after rewarming. The temperature at which defibrillation should firstly be attempted, and how often it should be attempted in the severely hypothermic patient, has not been established. If VF is detected, defibrillate according to standard protocols. If VF persists after three shocks, delay further attempts until core temperature is ≥30 °C.[57] CPR and rewarming may have to be continued for several hours to facilitate successful defibrillation.

Insulation. General measures for all victims include removal from the cold environment, prevention of further heat loss and rapid transfer to hospital.[58] In the field, a patient with moderate or severe hypothermia (hypothermia ≥ II) should be immobilised and handled carefully, oxygenated adequately, monitored (including ECG and core temperature), and the whole body dried and insulated.[51]

Remove wet clothes while minimising excessive movement of the victim. Removal of wet clothing or use of a vapour barrier seems to be equally effective to limit heat loss.[59] Conscious victims (hypothermia I) can mobilise as exercise rewarms a person more rapidly than shivering.[60] Patients will continue cooling after removal from a cold environment (i.e. afterdrop), which may result in a life-threatening decrease in core temperature triggering a cardiac arrest during transport (i.e. 'rescue death'). Prehospitally, avoid prolonged investigations and treatment, as further heat loss is difficult to prevent. Patients who stop shivering (e.g. hypothermia II–IV, and sedated or anaesthetised patients) will cool faster.

Prehospital rewarming. Rewarming may be passive, active external, or active internal. In hypothermia I passive rewarming is appropriate as patients are still able to shiver. Passive rewarming is best achieved by full body insulation with wool blankets, aluminium foil, cap and a warm environment. In hypothermia II–IV the application of chemical heat packs to the trunk has been recommended. In conscious patients who are able to shiver, this improves thermal comfort but does not speed rewarming.[61] If the patient is unconscious and the airway is not secured, arrange the insulation around the patient lying in a recovery (lateral decubitus) position. Rewarming in the field with heated intravenous fluids and warm humidified gases is not feasible.[51] Intensive active rewarming must not delay transport to a hospital where advanced rewarming techniques, continuous monitoring and observation are available.

Transport. Transport patients with hypothermia stage I to the nearest hospital. In hypothermia stage II–IV, signs of prehospital cardiac instability (i.e. systolic blood pressure <90 mmHg, ventricular arrhythmia, core temperature <28 °C) should determine the choice of admitting hospital. If any signs of cardiac instability are present, transport the patient to an ECLS centre, contacting them well in advance to ensure that the hospital can accept the patient for extracorporeal rewarming. In hypothermia V, reasons for withholding or terminating CPR should be investigated (e.g. obvious signs of irreversible death, valid DNAR, conditions unsafe for rescuer, avalanche burial ≥60 min and airway packed with snow and asystole). In the absence of any of these signs, start CPR and transfer the patient to an ECLS centre.

In-hospital rewarming. Unless the patient goes into VF, rewarm using active external methods (i.e. with forced warm air) and minimally invasively methods (i.e. with warm IV infusions). With a core temperature <32 °C and potassium <8 mmol L^{-1}, consider ECLS rewarming.[33] Most ECLS rewarmings have been performed using cardiopulmonary bypass, but more recently, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) has become the preferred method due to its rapid availability, the need for less anticoagulation, and the potential to prolong cardiorespiratory support after rewarming.

If an ECLS centre is not available, rewarming may be attempted in hospital using a dedicated team and a combination of external and internal rewarming techniques (e.g. forced warm air, warm infusions, forced peritoneal lavage).[62]

Continuous haemodynamic monitoring and warm IV fluids are essential. Patients will require large volumes of fluids during rewarming, as vasodilation causes expansion of the intravascular space. Avoid hyperthermia during and after rewarming. Once ROSC has been achieved use standard post-resuscitation care.

Hyperthermia

Introduction. 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 is a continuum of heat-related conditions, starting with heat stress, progressing to heat exhaustion, then to heat stroke and finally to multiple organ dysfunction and cardiac arrest.[63]

Malignant hyperthermia is a rare disorder of skeletal muscle calcium homeostasis characterised by muscle contracture and life-threatening hypermetabolic crisis following exposure of genetically predisposed individuals to halogenated anaesthetics and depolarising muscle relaxants.[64,65]

Heat exhaustion

Definition. Heat exhaustion is a non-life-threatening clinical syndrome of weakness, malaise, nausea, syncope, and other non-specific symptoms caused by heat exposure. Thermoregulation is not impaired. Heat exhaustion is caused by water and electrolyte imbalance due to heat exposure, with or without exertion. Rarely, severe heat exhaustion after physical exertion may be complicated by rhabdomyolysis, myoglobinuria, acute renal failure, and disseminated intravascular coagulation (DIC).

Symptoms. Symptoms are often vague, and patients may not realise that heat is the cause. Symptoms may include weakness, dizziness, headache, nausea, and sometimes vomiting. Syncope due to standing for long periods in the heat (heat syncope) is common and may mimic cardiovascular disorders. On examination, patients appear tired and are usually sweaty and tachycardic. Mental status is typically normal, unlike in heatstroke. Temperature is usually normal and, when elevated, usually does not exceed 40 °C.

Diagnosis. Diagnosis is clinical and requires exclusion of other possible causes (e.g. hypoglycaemia, acute coronary syndrome, infections). Laboratory testing is required only if needed to rule out other disorders.

Treatment

Fluids and electrolyte replacement. Treatment involves removing patients to a cool environment, lying them flat, and giving IV fluids and electrolyte replacement therapy; oral rehydration may not be effective in rapidly replacing electrolytes, but may be a more practical treatment. Rate and volume of rehydration are guided by age, underlying disorders, and clinical response. Replacement of 1–2 L crystalloids at 500 mL h^{-1} is often adequate. External cooling measures are usually not required. Consider external cooling in patients with a core temperature of ≥40 °C.

Heat stroke

Definition. Heat stroke (HS) is defined as hyperthermia accompanied by a systemic inflammatory response with a core temperature >40 °C, accompanied by mental state change and varying levels of organ dysfunction.[63]

There are two forms of HS:

1. Classic (non-exertional) heat stroke (CHS) occurs during high environmental temperatures and often effects the elderly during heat waves.[66]
2. Exertional heat stroke (EHS) occurs during strenuous physical exercise in high environmental temperatures and/or high humidity and usually effects healthy young adults.[67]

Mortality from heat stroke ranges between 10 and 50%.[68]

Predisposing factors. The elderly are at increased risk for heat-related illness because of underlying illness, medication use, declining thermoregulatory mechanisms and limited social support. There are several risk factors: lack of acclimatisation, dehydration, obesity, alcohol, cardiovascular disease, skin conditions (psoriasis, eczema, scleroderma, burn, cystic fibrosis), hyperthyroidism, phaeochromocytoma and drugs (anticholinergics, diamorphine, cocaine, amphetamine, phenothiazines, sympathomimetics, calcium channel blockers, beta-blockers).

Symptoms. Heat stroke can resemble septic shock and may be caused by similar mechanisms.[69] A single centre case series reported 14 ICU deaths in 22 heat stroke patients admitted to ICU with multiple organ failure.[70] Features included:

- core temperature ≥40 °C;
- hot, dry skin (sweating present in about 50% of cases of exertional heat stroke);
- early signs and symptoms (e.g. extreme fatigue, headache, fainting, facial flushing, vomiting and diarrhoea);
- cardiovascular dysfunction including arrhythmias and hypotension[71];
- respiratory dysfunction including acute respiratory distress syndrome (ARDS)[72];
- central nervous system dysfunction including seizures and coma[73];
- liver and renal failure[74];
- coagulopathy;
- rhabdomyolysis.[75]

Other clinical conditions presenting with increased core temperature need to be considered, including drug toxicity, drug withdrawal syndrome, serotonin syndrome, neuroleptic malignant syndrome, sepsis, central nervous system infection, endocrine disorders (e.g. thyroid storm, phaeochromocytoma).

Treatment. The mainstay of treatment is supportive therapy and rapidly cooling the patient.[76–78] Start cooling in the prehospital setting if possible. Aim to rapidly reduce the core temperature to approximately 39 °C. Patients with severe heat stroke need to be managed in an ICU environment. Large volumes of fluids and correction of electrolyte abnormalities may be required (see hypo-/hyperkalaemia and other electrolyte disorders).

Cooling techniques. Several cooling methods have been described, but there are few formal trials to determine which is optimal. Simple cooling techniques include drinking cold fluids, fanning the completely undressed patient and spraying tepid water on the patient. Ice packs over areas where there are large superficial blood vessels (axillae, groins, neck) may also be useful. Surface cooling methods may cause shivering. In cooperative stable patients, immersion in cold water can be effective[79]; however, this may cause peripheral vasoconstriction, shunt blood away from the periphery and reduce heat dissipation. Immersion is also not practical in the sickest patients.

Further techniques to cool patients with hyperthermia are similar to those used for targeted temperature management after cardiac arrest (see post resuscitation care).[80] Cold intravenous fluids will decrease body temperature. Gastric, peritoneal,[81] pleural or bladder lavage with cold water will lower the core temperature. Intravascular cooling techniques include the use of cold IV fluids,[82] intravascular cooling catheters[83,84] and extracorporeal circuits,[85] e.g. continuous veno-venous haemofiltration or cardiopulmonary bypass.

Pharmacological treatment. There are no specific drug therapies in heat stroke that lower core temperature. There is no good evidence that antipyretics (e.g. non-steroidal anti-inflammatory drugs or paracetamol) are effective in heat stroke. Diazepam may be useful to treat seizures and facilitate cooling.[86] Dantrolene has not been shown to be beneficial.[87–89]

Malignant hyperthermia

Malignant hyperthermia is a life-threatening genetic sensitivity of skeletal muscles to halogenated volatile anaesthetics and depolarising neuromuscular blocking drugs, occurring during or after anaesthesia.[90] Stop triggering agents immediately; give oxygen, correct acidosis and electrolyte abnormalities. Start active cooling and give dantrolene.[91]

Other drugs such as 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines also cause a condition similar to malignant hyperthermia and the use of dantrolene may be beneficial.[92]

Modifications to cardiopulmonary resuscitation. There are no specific studies of cardiac arrest in hyperthermia. If cardiac arrest occurs, follow standard guidelines and continue cooling the patient. Use the same cooling techniques as for targeted temperature management after cardiac arrest (see Section 5 Post-resuscitation care).[80] Attempt defibrillation using standard energy levels. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest.[93,94] The risk of unfavourable neurological outcome increases by 2.26 (odds ratio) for each degree of body temperature >37 °C.[95]

Hypovolaemia

Introduction

Hypovolaemia is a potentially treatable cause of cardiac arrest that usually results from a reduced intravascular volume (i.e. haemorrhage), but relative hypovolaemia may also occur in patients with severe vasodilation (e.g. anaphylaxis, sepsis). Hypovolaemia from mediator-activated vasodilation and increased capillary permeability is a major factor causing cardiac arrest in severe anaphylaxis.[96] Hypovolaemia from blood loss, is a leading cause of death in traumatic cardiac arrest.[97] External blood loss is usually obvious, e.g. trauma, haematemesis, haemoptysis, but may be more challenging to diagnose when occult, e.g. gastrointestinal bleeding or rupture of an aortic aneurysm. Patients undergoing major surgery are at high-risk from hypovolaemia due to post-operative haemorrhage and must be appropriately monitored (see perioperative cardiac arrest).

Depending on the suspected cause, initiate volume therapy with warmed blood products and/or crystalloids, in order to rapidly restore intravascular volume. At the same time, initiate immediate intervention to control haemorrhage, e.g. surgery, endoscopy, endovascular techniques,[98] or treat the primary cause (e.g. anaphylactic shock). In the initial stages of resuscitation use any crystalloid solution that is immediately available. If there is a qualified sonographer able to perform ultrasound without interruption to chest compressions, e.g. during rhythm check or ventilations, it may be considered as an additional diagnostic tool in hypovolaemic cardiac arrest.

Treatment recommendations for cardiac arrest and periarrest situations in anaphylaxis and trauma are addressed in separate sections because of the need for specific therapeutic approaches.

Anaphylaxis

Definition. A precise definition of anaphylaxis is not important for its emergency treatment.[99] The European Academy of Allergy and Clinical Immunology Nomenclature Committee proposed the following broad definition:[100] 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.[1,96,101,102]

Epidemiology. Anaphylaxis is common and affects about 1 in 300 of the European population at some stage in their lives, with an incidence from 1.5 to 7.9 per 100,000 person-years.[103] Anaphylaxis can be triggered by any of a very broad range of triggers with food, drugs, stinging insects, and latex the most commonly identified triggers.[103] Food is the commonest trigger in children and drugs the commonest in adults.[104] Virtually any food or drug can be implicated, but certain foods (nuts) and drugs (muscle relaxants, antibiotics, nonsteroidal anti-inflammatory drugs and aspirin) cause most reactions.[105] A significant number of cases of anaphylaxis are idiopathic. Between 1992 and 2012 in the UK, admission and fatality rates for drug- and insect sting-induced anaphylaxis were highest in the group aged 60 years and older. In contrast, admissions due to food-triggered anaphylaxis were most common in young people, with a marked peak in the incidence of fatal food reactions during the second and third decades of life.[106]

The overall prognosis of anaphylaxis is good, with a case fatality ratio of less than 1% reported in most population-based studies. The European Anaphylaxis Registry reported that only 2% of 3333 cases were associated with cardiac arrest.[107] If intensive care unit admission is required, survival to discharge is over 90%. Over the period 2005–2009, there were 81 paediatric and 1269 adult admissions with anaphylaxis admitted to UK critical care units. Survival to discharge was 95% for children, and 92% for adults.[108]

Anaphylaxis and risk of death is increased in those with pre-existing asthma, particularly if the asthma is poorly controlled, severe or in asthmatics who delay treatment.[109,110] When anaphylaxis is fatal, death usually occurs very soon after contact with the trigger. From a case series, fatal food reactions cause respiratory arrest typically within 30–35 min; insect stings cause collapse from shock within 10–15 min; and deaths caused by intravenous medication occur most commonly within 5 min. Death never occurred more than 6 h after contact with the trigger.[101,111]

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

The European Academy of Allergy and Clinical Immunology's (EAACI) Taskforce on Anaphylaxis state that anaphylaxis is highly likely when any one of the following three criteria is fulfilled[96,112]:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g. generalised hives, pruritus or flushing, swollen lips–tongue–uvula) and at least one of the following:
   a. Respiratory compromise, e.g. dyspnoea, wheeze–bronchospasm, stridor, reduced peak expiratory flow (PEF), hypoxaemia.
   b. Reduced blood pressure or associated symptoms of end-organ dysfunction, e.g. hypotonia (collapse), syncope, incontinence.
2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):
   a. Involvement of the skin–mucosal tissue, e.g. generalised hives, itch-flush, swollen lips–tongue–uvula.
   b. Respiratory compromise, e.g. dyspnoea, wheeze–bronchospasm, stridor, reduced PEF, hypoxaemia.
   c. Reduced blood pressure or associated symptoms, e.g. hypotonia (collapse), syncope, incontinence.
   d. Persistent gastrointestinal symptoms, e.g. crampy abdominal pain, vomiting.
3. Reduced blood pressure after exposure to known allergen for that patient (minutes to several hours):
   a. Infants and children: low systolic blood pressure (<70 mmHg from 1 month to 1 year; <70 mmHg + (2 × age) from 1 year to 10 years; <90 mmHg from 11 to 17 years) or >30% decrease in systolic blood pressure.
   b. Adults: systolic blood pressure of <90 mmHg or >30% decrease from that person's baseline.

Treatment. The evidence supporting specific interventions for the treatment of anaphylaxis is limited.[113] A systematic ABCDE approach to recognise and treat anaphylaxis is recommended with immediate administration of intramuscular (IM) adrenaline (Fig. 4.2). Treat life-threatening problems as you find them. The basic principles of treatment are the same for all age groups. Monitor all patients who have suspected anaphylaxis as soon as possible (e.g. by ambulance crew, in the emergency department, etc.). Minimum monitoring includes pulse oximetry, non-invasive blood pressure and a 3-lead ECG.

Patient positioning. Patients with anaphylaxis can deteriorate and are at risk of cardiac arrest if made to sit up or stand up.[114] All patients should be placed in a comfortable position. Patients with airway and breathing problems may prefer to sit up, as this will make breathing easier. Lying flat with or without leg elevation is helpful for patients with a low blood pressure.

Remove the trigger (if possible). Stop any drug suspected of causing anaphylaxis. Remove the stinger after a bee/wasp sting. Early removal is more important than the method of removal.[115] Do not delay definitive treatment if removing the trigger is not feasible.

Cardiac arrest following anaphylaxis. Start CPR immediately and follow current guidelines. Prolonged CPR may be necessary. Rescuers should ensure that help is on its way as early ALS is essential.

Airway obstruction. Anaphylaxis can cause airway swelling and obstruction. This will make airway and ventilation interventions (e.g. bag-mask ventilation, tracheal intubation, cricothyroidotomy) difficult. Consider early tracheal intubation before airway swelling makes this difficult. Call for expert help early.

Adrenaline (first line treatment). Adrenaline is the most important drug for the treatment of anaphylaxis.[116,117] Although there are no randomised controlled trials,[118] adrenaline is a logical treatment and there is consistent anecdotal evidence supporting its use to ease bronchospasm and circulatory collapse. As an

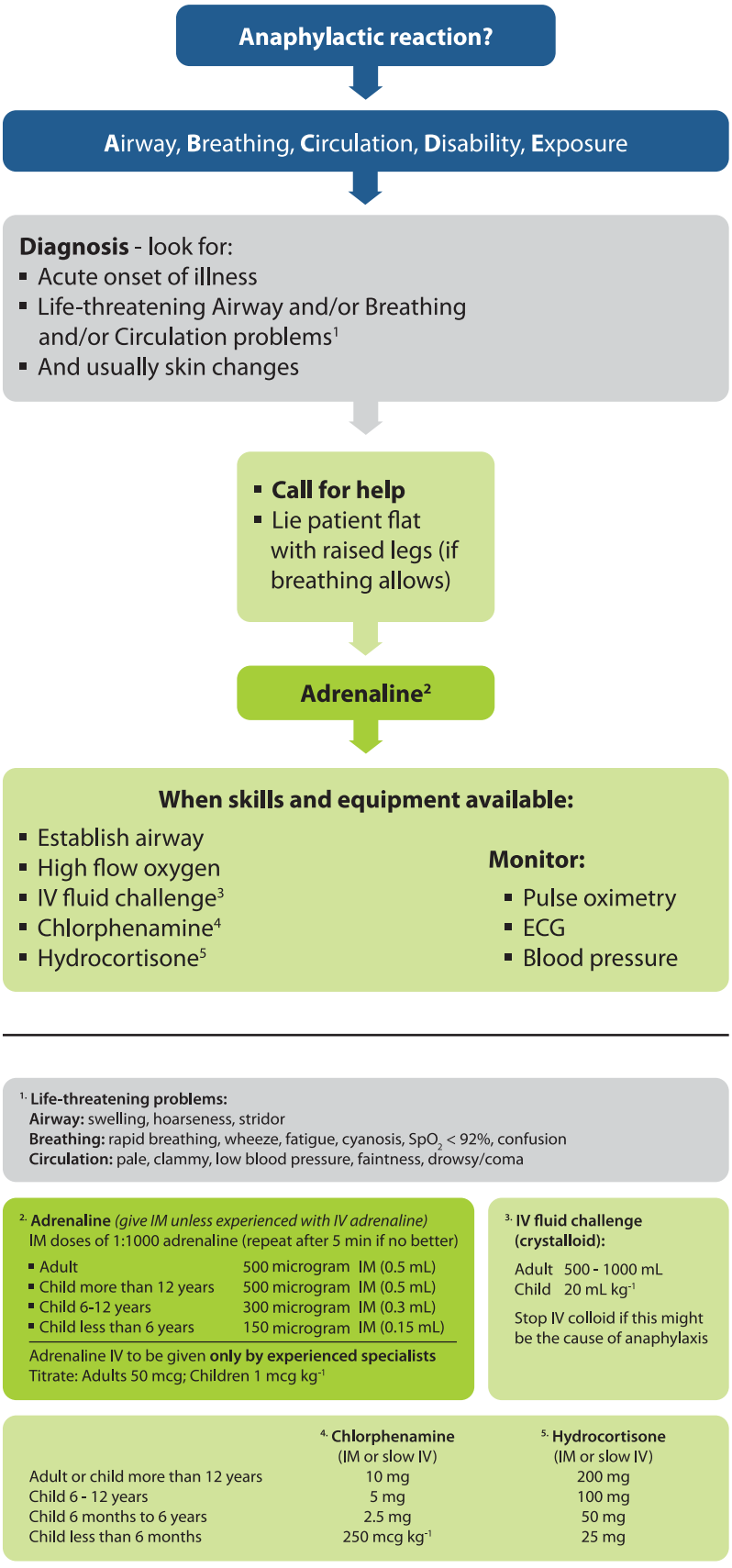

Fig. 4.2. Anaphylaxis treatment algorithm.[101]
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alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. Its beta-receptor activity dilates the bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release. Activation of beta-2 adrenergic receptors on mast cell surfaces inhibit their activation, and early adrenaline attenuates the severity of IgE-mediated allergic reactions. Adrenaline is most effective when given early after the onset of the reaction,[119] and adverse effects are extremely rare with correct IM doses.

Give adrenaline to all patients with life-threatening features. If these features are absent but there are other features of a systemic allergic reaction, the patient needs careful observation and symptomatic treatment using the ABCDE approach.

Intramuscular adrenaline. The intramuscular (IM) route is the best for most individuals who have to give adrenaline to treat anaphylaxis. Monitor the patient as soon as possible (pulse, blood pressure, ECG, pulse oximetry). This will help monitor the response to adrenaline. The IM route has several benefits:

- There is a greater margin of safety.
- It does not require intravenous access.
- The IM route is easier to learn.
- Patients with known allergies can self-administer IM adrenaline.

The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The needle for injection needs to be long enough to ensure that the adrenaline is injected into muscle.[120] The subcutaneous or inhaled routes for adrenaline are not recommended for the treatment of anaphylaxis because they are less effective than the IM route.[121–123]

Adrenaline intramuscular dose. The evidence for the recommended doses is limited. The EAACI suggests IM adrenaline ($1\ mg\ mL^{-1}$) should be given a dose of $10\ mcg\ kg^{-1}$ of body weight to a maximum total dose of 0.5 mg.[96]

The following doses are based on what is considered to be safe and practical to draw up and inject in an emergency (equivalent volume of 1:1000 adrenaline is shown in brackets):

>12 years and adults	500 microgram IM (0.5 mL)
>6–12 years	300 microgram IM (0.3 mL)
>6 months–6 years	150 microgram IM (0.15 mL)
<6 months	150 microgram IM (0.15 mL)

Repeat the IM adrenaline dose if there is no improvement in the patient's condition within 5 min. Further doses can be given at about 5-min intervals according to the patient's response.

Intravenous adrenaline (for specialist use only). There is a much greater risk of causing harmful side effects by inappropriate dosage or misdiagnosis of anaphylaxis when using intravenous (IV) adrenaline.[124] IV adrenaline should only be used by those experienced in the use and titration of vasopressors in their normal clinical practice (e.g. anaesthetists, emergency physicians, intensive care doctors). In patients with a spontaneous circulation, IV adrenaline can cause life-threatening hypertension, tachycardia, arrhythmias, and myocardial ischaemia. If IV access is not available or not achieved rapidly, use the IM route for adrenaline. Patients who are given IV adrenaline must be monitored – continuous ECG and pulse oximetry and frequent non-invasive blood pressure measurements as a minimum. Patients who require repeated IM doses of adrenaline may benefit from IV adrenaline. It is essential that these patients receive expert help early.

Adrenaline intravenous dose (for specialist use only).

- Adults: Titrate IV adrenaline using 50 microgram boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion.[125,126]
- Children: IM adrenaline is the preferred route for children having anaphylaxis. The IV route is recommended only in specialist paediatric settings by those familiar with its use (e.g. paediatric anaesthetists, paediatric emergency physicians, paediatric intensivists) and if the patient is monitored and IV access is already available. There is no evidence on which to base a dose recommendation – the dose is titrated according to response. A child may respond to a dose as small as $1\ mcg\ kg^{-1}$. This requires very careful dilution and checking to prevent dose errors.

Adrenaline intravenous/intraosseous dose (in cardiac arrest only). Cardiac arrest with suspected anaphylaxis should be treated with standard doses of IV or intraosseous (IO) adrenaline for cardiac arrest. If this is not feasible, consider IM adrenaline if cardiac arrest is imminent or has just occurred.

Oxygen (give as soon as available). Initially, give the highest concentration of oxygen possible using a mask with an oxygen reservoir.[127] Ensure high-flow oxygen (usually greater than $10\ L\ min^{-1}$ to prevent collapse of the reservoir during inspiration. If the patient's trachea is intubated, ventilate the lungs with high concentration oxygen using a self-inflating bag.

Fluids (give as soon as available). Large volumes of fluid may leak from the patient's circulation during anaphylaxis. There will also be vasodilation. If IV access has been gained, infuse IV fluids immediately. Give a rapid IV fluid challenge ($20\ mL\ kg^{-1}$) in a child or 500–1000 mL in an adult and monitor the response; give further doses as necessary. There is no evidence to support the use of colloids over crystalloids in this setting. Consider colloid infusion as a cause in a patient receiving a colloid at the time of onset of an anaphylaxis and stop the infusion. A large volume of fluid may be needed.

If IV access is delayed or impossible, the IO route can be used for fluids or drugs. Do not delay the administration of IM adrenaline while attempting IO access.

Antihistamines (give after initial resuscitation). Antihistamines are a second line treatment for anaphylaxis. The evidence to support their use is limited, but there are logical reasons for their use.[128] H_1-antihistamines help counter histamine-mediated vasodilation, bronchoconstriction, and particularly cutaneous symptoms. There is little evidence to support the routine use of an H_2-antihistamine (e.g. ranitidine, cimetidine) for the initial treatment of anaphylaxis.

Glucocorticosteroids (give after initial resuscitation). Corticosteroids may help prevent or shorten protracted reactions, although the evidence is limited.[129] In asthma, early corticosteroid treatment is beneficial in adults and children. There is little evidence on which to base the optimum dose of hydrocortisone in anaphylaxis.

Other drugs.

Bronchodilators. The presenting symptoms and signs of severe anaphylaxis and life-threatening asthma can be the same. Consider further bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), aminophylline (IV) or magnesium (IV) (see asthma). IV magnesium is a vasodilator and can make hypotension worse.

Cardiac drugs. Adrenaline remains the first line vasopressor for the treatment of anaphylaxis. There are animal studies and case reports describing the use of other vasopressors and inotropes (noradrenaline, vasopressin, terlipressin metaraminol, methoxamine, and glucagon) when initial resuscitation with adrenaline and fluids has not been successful.[130–142] Use these drugs only in specialist settings (e.g. ICU) where there is experience in their use. Glucagon can be useful to treat anaphylaxis in a patient taking a beta-blocker.[143] Some case reports of cardiac arrest suggest cardiopulmonary bypass[144,145] or mechanical chest compression devices may also be helpful.[146]

Investigations. Undertake the usual investigations appropriate for a medical emergency, e.g. 12-lead ECG, chest X-ray, urea and electrolytes, arterial blood gases, etc.

Mast cell tryptase. The specific test to help confirm a diagnosis of anaphylaxis is measurement of mast cell tryptase. Tryptase is the major protein component of mast cell secretory granules. In anaphylaxis, mast cell degranulation leads to markedly increased blood tryptase concentrations. Tryptase concentrations in the blood may not increase significantly until 30 min or more after the onset of symptoms, and peak 1–2 h after onset.[147] The half-life of tryptase is short (approximately 2 h), and concentrations may be back to normal within 6–8 h, so timing of any blood samples is very important. The time of onset of the anaphylaxis is the time when symptoms were first noticed.

(a) Minimum: one sample at 1–2 h after the start of symptoms.
(b) Ideally: Three timed samples:

- Initial sample as soon as feasible after resuscitation has started – do not delay resuscitation to take sample.
- Second sample at 1–2 h after the start of symptoms.
- Third sample either at 24 h or in convalescence (for example in a follow-up allergy clinic). This provides baseline tryptase levels – some individuals have an elevated baseline level.

Serial samples have better specificity and sensitivity than a single measurement in the confirmation of anaphylaxis.[148]

Discharge and follow-up. Patients who have had suspected anaphylaxis (i.e. an airway, breathing or circulation problem) should be treated and then observed in a clinical area with facilities for treating life-threatening ABC problems. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation. The exact incidence of biphasic reactions is unknown. Although studies quote an incidence of 1–20%, it is not clear whether all the patients in these studies actually had anaphylaxis or whether the initial treatment was appropriate.[149] There is no reliable way of predicting who will have a biphasic reaction. It is therefore important that decisions about discharge are made for each patient by an experienced clinician.

Before discharge from hospital, all patients must:

- Be reviewed by an allergy specialist and have a treatment plan based on their individual risk.
- Be given clear instructions to return to hospital if symptoms return.
- Be considered for an adrenaline auto-injector, or given a replacement[150–152] and ensured that appropriate training has been given.
- Have a plan for follow-up, including contact with the patient's general practitioner.

Patients need to know the allergen responsible (if identified) and how to avoid it. Patients need to be able to recognise the early symptoms of anaphylaxis, so that they can summon help quickly and prepare to use their emergency medication. Although there are no randomised clinical trials, there is evidence that individualised action plans for self-management should decrease the risk of recurrence.[153]

Traumatic cardiac arrest

Introduction. Traumatic cardiac arrest (TCA) carries a very high mortality, but in those where ROSC can be achieved, neurological outcome in survivors appears to be much better than in other causes of cardiac arrest.[154,155] The response to TCA is time-critical and success depends on a well-established chain of survival, including advanced prehospital and specialised trauma centre care. Immediate resuscitative efforts in TCA focus on simultaneous treatment of reversible causes, which takes priority over chest compressions.

Diagnosis. The diagnosis of traumatic cardiac arrest is made clinically. The patient presents with agonal or absent spontaneous respiration and absence of a central pulse.

A peri-arrest state is characterised by cardiovascular instability, hypotension, loss of peripheral pulses in uninjured regions and a deteriorating conscious level without obvious central nervous system (CNS) cause. If untreated, this state is likely to progress to cardiac arrest. Rapid focused ultrasound assessment may be helpful in the immediate diagnosis and management, but should not delay resuscitative interventions.[156]

It is vital that a medical cardiac arrest is not misdiagnosed as a TCA and must be treated with the universal ALS algorithm. Cardiac arrest or other causes of sudden loss of consciousness (e.g. hypoglycaemia, stroke, seizures) may cause a secondary traumatic event. Some observational studies have reported that about 2.5% of non-traumatic OHCAs occur in cars.[157–159] In these cases, shockable rhythms (VF/pVT) are more common.[97] The primary cause of the cardiac arrest can be elucidated from information about past medical history, events preceding the accident (if possible), and a systematic post-ROSC assessment, including a 12-lead ECG.

Prognostic factors and withholding resuscitation. There are no reliable predictors of survival for traumatic cardiac arrest. Factors that are associated with survival include the presence of reactive pupils, an organised ECG rhythm and respiratory activity.[159,160] Short duration of CPR and prehospital times have also been associated with positive outcomes.[161]

A large systematic review reported an overall survival rate of 3.3% in blunt and 3.7% in penetrating trauma, with good neurological outcome in 1.6% of all cases.[154] Outcome is age dependent, with children having a better prognosis than adults.[97,154] There is considerable variation in reported mortality (range 0–27%) reflecting heterogeneity in casemix and care in different systems. PEA, which in TCA may initially be a low output state, and asystole are the prevalent heart rhythms in TCA. Ventricular fibrillation (VF) is rare but carries the best prognosis.[97,155]

One study reported good neurological outcome in 36.4% of TCA patients presenting with VF, but only in 7% with PEA and 2.7% of those in asystole,[155] but other studies of patients in non-shockable rhythms have reported 100% mortality.[159,162,163] The American College of Surgeons and the National Association of EMS physicians recommend withholding resuscitation in situations where death is inevitable or established and in trauma patients presenting with apnoea, pulselessness and without organised ECG activity.[164] However, neurologically intact survivors initially presenting in this state have been reported.[155] We therefore recommend the following approach:

Consider withholding resuscitation in TCA in any of the following conditions:

- no signs of life within the preceeding 15 min;
- massive trauma incompatible with survival (e.g. decapitation, penetrating heart injury, loss of brain tissue).

We suggest termination of resuscitative efforts should be considered if there is:

- no ROSC after reversible causes have been addressed;
- no detectable ultrasonographic cardiac activity.

Trauma care systems throughout Europe vary considerably and we recommend establishing regional guidelines for treatment of TCA and tailoring patient pathways to infrastructure and resources.

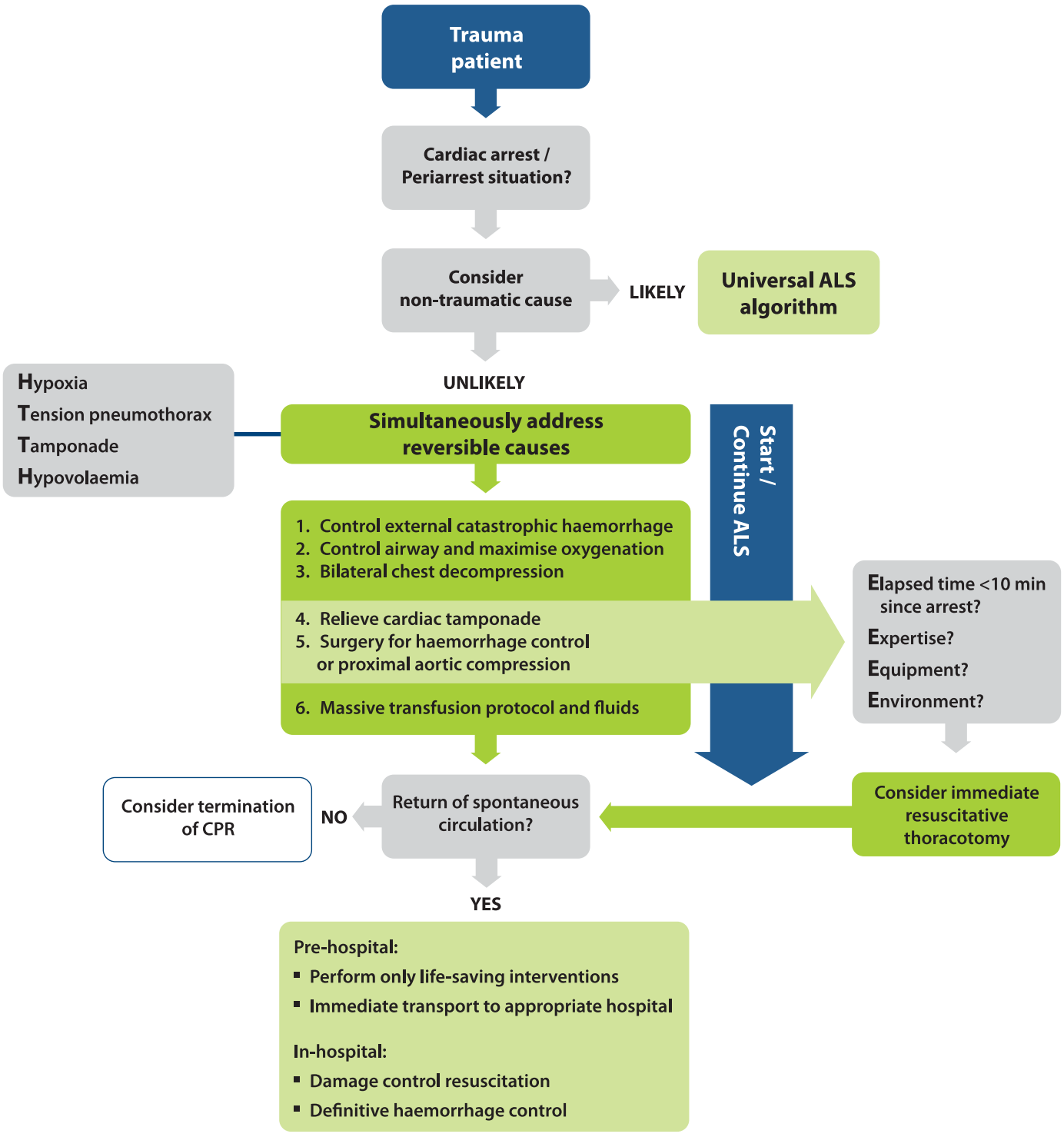

Fig. 4.3. Traumatic cardiac arrest algorithm.

Treatment. Emphasis on rapid treatment of all potentially reversible pathology is the basis of treatment guidelines. These principles are addressed in several treatment algorithms.[97,165–167] All algorithms attempt to rapidly address reversible causes of TCA in the prehospital and in-hospital phases of care. Fig. 4.3 shows a traumatic cardiac (peri-) arrest algorithm, which is based on the universal ALS algorithm.[168]

Effectiveness of chest compressions. Chest compressions are still the standard of care in patients with cardiac arrest, irrespective of aetiology. In cardiac arrest caused by hypovolaemia, cardiac tamponade or tension pneumothorax, chest compressions are unlikely to be as effective as in normovolaemic cardiac arrest.[169–172] Because of this fact, chest compressions take a lower priority than the immediate treatment of reversible causes, e.g. thoracotomy, controlling haemorrhage, etc. In an out-of-hospital setting, only essential life-saving interventions should be performed on scene followed by rapid transfer to the nearest appropriate hospital.

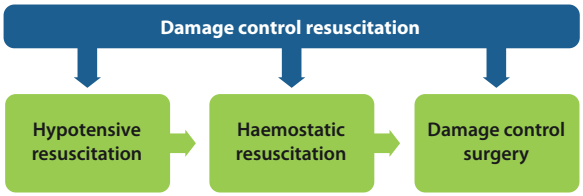

Fig. 4.4. Principles of damage control resuscitation in trauma.

Hypovolaemia. Uncontrolled haemorrhage is the cause of traumatic cardiac arrest in 48% of all TCA.[97] The treatment of severe hypovolaemic shock has several elements. The main principle is to achieve 'haemostasis without delay', usually with surgical or radiological intervention. Temporary haemorrhage control can be lifesaving:

- Treat compressible external haemorrhage with direct pressure (with or without a dressing), use tourniquets if needed and/or apply topical haemostatic agents.[173]
- Non-compressible haemorrhage is more difficult. Use splints (pelvic splint), blood products, intravenous fluids and tranexamic acid while moving the patient to surgical haemorrhage control.

Over the past 10 years the principle of 'damage control resuscitation' has been adopted in trauma resuscitation for uncontrolled haemorrhage. Damage control resuscitation combines permissive hypotension and haemostatic resuscitation with damage control surgery. Limited evidence[174] and general consensus have supported a conservative approach to intravenous fluid infusion, with permissive hypotension until surgical haemostasis is achieved. Permissive hypotension allows intravenous fluid administration to a volume sufficient to maintain a radial pulse.[175,176]

Haemostatic resuscitation is the very early use of blood products as primary resuscitation fluid to prevent exsanguination by trauma-induced coagulopathy.[177] The recommended ratio of packed red cells, fresh frozen plasma and platelets is 1:1:1.[178] Some services have also started using blood products in the prehospital phase of care.[179,180]

Simultaneous damage control surgery and haemostatic resuscitation using massive transfusion protocols (MTP)[173] are the principles of damage control resuscitation in patients with exsanguinating injuries (Fig. 4.4).[177]

Although the evidence for permissive hypotension during resuscitation is limited, particularly with regards to blunt trauma, permissive hypotension has been endorsed in both civilian and military care,[181] generally aiming for a systolic blood pressure of 80–90 mmHg. Caution is advised with this strategy in patients with traumatic brain injury were a raised intracranial pressure may require a higher cerebral perfusion pressure. The duration of hypotensive resuscitation should not exceed 60 min, because the risks of irreversible organ damage then exceed its intended benefits.[176]

Tranexamic acid (TXA) (loading dose 1 g over 10 min followed by infusion of 1 g over 8 h) increases survival from traumatic haemorrhage.[182] It is most effective when administered within the first hour and certainly within the first 3 h following trauma.[182] Give TXA in the prehospital setting when possible.

Hypoxaemia. Hypoxaemia due to airway obstruction and traumatic asphyxia has been reported as cause of traumatic cardiac arrest in 13% of all cases.[97] Effective airway management and ventilation can reverse hypoxic cardiac arrest and it is essential to establish and maintain oxygenation of trauma patients with a severely compromised airway. Tracheal intubation in trauma patients is a difficult procedure with a high failure rate if carried out by less experienced care providers.[183,184] Use basic airway manoeuvres and second-generation supraglottic airways to maintain oxygenation if tracheal intubation cannot be accomplished immediately.

Positive pressure ventilation worsens hypotension by impeding venous return to the heart, particularly in hypovolaemic patients.[185] Low tidal volumes and slow respiratory rates may help optimise cardiac preload. Monitor ventilation with continuous waveform capnography and adjust to achieve normocapnia.[173]

Tension pneumothorax. Thirteen percent of all cases of TCA are caused by tension pneumothorax.[97] To decompress the chest in TCA, perform bilateral thoracostomies in the 4th intercostal space, extending to a clamshell thoracotomy if required. In the presence of positive pressure ventilation, thoracostomies are likely to be more effective than needle thoracocentesis and quicker than inserting a chest tube (see tension pneumothorax).[186,187]

Cardiac tamponade and resuscitative thoracotomy. Cardiac tamponade is the underlying cause of approximately 10% of cardiac arrest in trauma.[97] Where there is TCA *and* penetrating trauma to the chest or epigastrium, immediate resuscitative thoracotomy (RT) via a clamshell incision[188] can be life saving.[189] The chance of survival is about 4 times higher in cardiac stab wounds than in gunshot wounds.[190]

Resuscitative thoracotomy is also applied for other life threatening injuries; the evidence was examined in 2012[191] and guidelines produced which recommend that, *after arrival in hospital*, the decision to proceed with RT should include the following criteria:

- blunt trauma patients with less than 10 min of prehospital CPR;
- penetrating torso trauma patients with less than 15 min of CPR.

These guidelines estimate survival rates for RT of approximately 15% for all patients with penetrating wounds and 35% for patients with a penetrating cardiac wound. In contrast, survival from RT following blunt trauma is dismal, with survival rates of 0–2% being reported.[191,192]

Successful RT is time critical. One UK service recommends that if surgical intervention cannot be accomplished within 10 min after loss of pulse in patients *with penetrating chest injury*, on scene RT should be considered.[10] Based on this approach, of 71 patients who underwent RT at scene, 13 patients survived and 11 of these made a good neurological recovery.

The prerequisites for a successful RT can be summarised as the 'four Es rule' (4E):

- *Expertise*: teams that perform RT must be led by a highly trained and competent healthcare practitioner. These teams must operate under a robust governance framework.
- *Equipment:* adequate equipment to carry out RT and to deal with the intrathoracic findings is mandatory.
- *Environment:* ideally RT should be carried out in an operating theatre. RT should not be carried out if there is inadequate physical access to the patient, or if the receiving hospital is not easy to reach.
- *Elapsed time*: the time from loss of vital signs to commencing a RT should not be longer than 10 min.

If any of the four criteria is not met, RT is futile and exposes the team to unnecessary risks.[193]

Needle aspiration of tamponade, with or without ultrasound guidance, is unreliable because the pericardium is commonly full of clotted blood.[194,195] If thoracotomy is not possible, however, consider ultrasound guided pericardiocentesis to treat TCA associated with suspected cardiac tamponade. Non-image guided pericardiocentesis is an alternative, only if ultrasound is not available. Placement of a pericardial drain may be beneficial in some patients.

Diagnostics. Ultrasonography should be used in the evaluation of the compromised trauma patient to target life-saving interventions if the cause of shock cannot be established clinically.[196,173] Haemoperitoneum, haemo- or pneumothorax and cardiac tamponade can be diagnosed reliably in minutes, even in the prehospital phase.[197] Early whole-body computed tomography (WBCT) scanning as part of the primary survey may improve outcome in major trauma.[198] WBCT is increasingly employed to identify the source of shock and to guide subsequent haemorrhage control.

Prehospital care. Short prehospital times are associated with increased survival rates for major trauma and TCA. The time elapsed between injury and surgical control of bleeding should therefore be minimised and the patient should be immediately transferred to a trauma centre for ongoing damage control resuscitation.[173] A 'scoop and run' concept for these patients may be life saving.

Tension pneumothorax

Introduction

Tension pneumothorax defined as haemodynamic compromise in a patient with an expanding intrapleural air mass is a treatable cause of cardiac arrest and should be excluded during CPR.[199] Tension pneumothorax can occur in a variety of clinical situations including trauma, asthma and other respiratory disease, but can also be iatrogenic following invasive procedures, e.g. attempts at central venous catheter insertion. It is more common and often more severe in patients undergoing positive pressure ventilation.[200] The incidence of tension pneumothorax is approximately 5% in major trauma patients treated in the prehospital setting (13% of those developing TCA), and less than 1% of adults admitted to ICU.[97,201,202]

Diagnosis

Diagnosis of tension pneumothorax in a patient with cardiac arrest or haemodynamic instability must be based on clinical examination. The symptoms include haemodynamic compromise (hypotension or cardiac arrest) in conjunction with signs suggestive of a pneumothorax (preceding respiratory distress, hypoxia, absent unilateral breath sounds on auscultation, subcutaneous emphysema) and mediastinal shift (tracheal deviation and jugular venous distention).[200] During CPR, presentation is not always classical, but when it is suspected in the presence of cardiac arrest or severe hypotension, chest decompression should be carried out immediately before radiographic confirmation.[201]

Treatment

Needle decompression. Needle chest decompression is rapid and within the skill set of most ambulance personnel but is of limited value.[203,204] A significant proportion of patients have chest wall thickness which makes needle decompression with a standard length 14-gauge cannula ineffective.[205] Cannulae are also prone to kinking and blockage.[206] Any attempt at needle decompression should be followed by insertion of a chest tube (see asthma).

Thoracostomy. Tracheal intubation, positive pressure ventilation and formal chest decompression effectively treats tension pneumothorax in patients with TCA. Simple thoracostomy is easy to perform and used routinely by several prehospital physician services.[187,207] This consists of the first stage of standard chest tube insertion – a simple incision and rapid dissection into the pleural space in the positive pressure ventilated patient (see traumatic cardiac arrest). Chest tube insertion is then carried out after the resuscitation phase. This requires additional equipment, takes longer to perform and creates a closed system that has the potential to re-tension. Chest drain tubes may become blocked with lung or blood clots and have the potential to kink.

Tamponade

Introduction

Cardiac tamponade occurs when the pericardial sac is filled with fluid under pressure, which leads to compromise of cardiac function and ultimately cardiac arrest. The condition most commonly occurs after penetrating trauma and cardiac surgery. Mortality is high and immediate decompression of the pericardium is required to give any chance of survival.

Treatment

Thoracotomy. The criteria and prerequisites for resuscitative thoracotomy in patients with penetrating trauma to the chest or epigastrium are described in section on traumatic cardiac arrest. Treatment of the tamponade following cardiac surgery is addressed in the section on cardiac arrest following cardiac surgery.

Pericardiocentesis. If thoracotomy is not possible, consider ultrasound-guided pericardiocentesis to treat cardiac arrest associated with suspected traumatic or non-traumatic cardiac tamponade. Non-image guided pericardiocentesis is an alternative, only if ultrasound is not available.

Thrombosis

Pulmonary embolism

Introduction. Cardiac arrest from acute pulmonary embolism is the most serious clinical presentation of venous thromboembolism, in most cases originating from a deep venous thrombosis (DVT).[208] The reported incidence of cardiac arrest caused by pulmonary embolism is 2–9% of all OHCAs,[209–212] and 5–6% of all in-hospital cardiac arrests.[213,214] but it is likely to be underestimated. Overall survival is low.[211,215] Specific treatments for cardiac arrest resulting from pulmonary embolism include administration of fibrinolytics, surgical embolectomy and percutaneous mechanical thrombectomy.

Diagnosis. Diagnosis of acute pulmonary embolism during cardiac arrest is difficult. One study has reported correct recognition of the underlying causes in up to 85% of all in-hospital resuscitation attempts,[214] but accurate prehospital diagnosis of acute pulmonary embolism is particularly challenging.[212,216]

The 2014 European Society of Cardiology Guidelines on the diagnosis and management of acute pulmonary embolism define 'confirmed pulmonary embolism' as a probability of pulmonary embolism high enough to indicate the need for specific treatment.[208]

Clinical history and assessment, capnography and echocardiography (if available) can all assist in the diagnosis of acute pulmonary embolism during CPR with varying degrees of specificity and sensitivity:

- Common symptoms preceding cardiac arrest are sudden onset of dyspnoea, pleuritic or substernal chest pain, cough, haemoptysis, syncope and signs of DVT in particular (unilateral low extremity swelling).[208] However, pulmonary embolism may not be symptomatic until it presents as sudden cardiac arrest.[217]
- Obtain information about past medical history, predisposing factors, and medication that may support diagnosis of pulmonary embolism, although none of these are specific, e.g.[208]
  - Previous pulmonary embolism or DVT
  - Surgery or immobilisation within the past four weeks
  - Active cancer

- Clinical signs of DVT
- Oral contraceptive use or hormone replacement therapy.
- Long-distance flights

In as many as 30% of the patients with pulmonary embolism, no risk factors are apparent.[218]

- If a 12-lead ECG can be obtained before onset of cardiac arrest, changes indicative of right ventricular strain may be found:
  - Inversion of T waves in leads V1–V4
  - QR pattern in V1
  - S1 Q3 T3 pattern (i.e. a prominent S wave in lead I, a Q wave and inverted T wave in lead III)
  - Incomplete or complete right bundle-branch block[208,219]
- Cardiac arrest commonly presents as PEA.[211]
- Low $ETCO_2$ readings (about 1.7 kPa/13 mmHg) while performing high quality chest compressions may support a diagnosis of pulmonary embolism, although it is a non-specific sign.[209]
- Consider emergency echocardiography performed by a qualified sonographer as an additional diagnostic tool to identify pulmonary embolism if it can be performed without interruptions to chest compressions, e.g. during rhythm check. Echocardiographic findings are evident after acute obstruction of more than 30% of the pulmonary arterial tree.[220] Common echocardiographic findings are an enlarged right ventricle with a flattened interventricular septum,[221,222] but absence of these features does not exclude pulmonary embolism.[223] Signs of right ventricular overload or dysfunction may also be caused by other cardiac or pulmonary disease.[224]
- More specific diagnostic methods, e.g. D-dimer testing, (computed tomographic) pulmonary angiography, lung scintigraphy, or magnetic resonance angiography, are not recommended for a cardiac arrest situation.

Modifications to cardiopulmonary resuscitation. A meta-analysis, which included patients with pulmonary embolism as a cause of cardiac arrest, concluded that fibrinolytics increased the rate of ROSC, survival to discharge and long-term neurological function.[225] A subgroup analysis of patients treated with thrombolytics compared with placebo in a randomised controlled trial[215] did not prove survival difference. However, this study was not designed for treatment of pulmonary embolism and not powered to reach significance in this small subgroup. Some other non-randomised studies have also documented use of thrombolytics in the treatment of cardiac arrest due to acute pulmonary embolism, but evidence for improved neurologically intact survival to hospital discharge is limited.[211,226]

In a cardiac arrest presumed to be caused by acute pulmonary embolism, follow the standard guidelines for ALS (see adult advanced life support).[168] The decision to treat for acute pulmonary embolism must be taken early, when a good outcome is still possible. The following treatment modifications are recommended:

- Consider administration of fibrinolytic therapy when acute pulmonary embolism is a known or suspected cause of cardiac arrest. Ongoing CPR is not a contraindication to fibrinolysis. Despite increased risk of severe bleeding, fibrinolysis may be an effective treatment, which can be initiated without delay, even outside specialised healthcare facilities. The potential benefit of fibrinolysis in terms of improved survival outweighs potential risks in a location where no alternative exists, e.g. in the prehospital setting.[211,227–231]
- Once a fibrinolytic drug is administered, continue CPR for at least 60–90 min before terminating resuscitation attempts.[227,232] Survival and good neurological outcome have been reported in cases requiring in excess of 100 min of CPR.[233]
- Consider the use of a mechanical chest compression device when maintenance of high quality chest compressions is needed for a prolonged time.

Extracorporeal CPR. Some observational studies suggest the use of extracorporeal life support (ECLS) if cardiac arrest is associated with pulmonary embolism.[234,235] The implementation of ECLS requires considerable resource and training. Its use should be considered as a rescue therapy for those patients in whom initial ALS measures are unsuccessful and/or to facilitate pulmonary thrombectomy.

Surgical embolectomy and mechanical thrombectomy. Survival of patients who underwent surgical embolectomy during CPR due to pulmonary embolism was reported as 13% and 71% in two case series,[229,236] but these results were not compared with standard treatment. Routine use of surgical embolectomy or mechanical thrombectomy for cardiac arrest from suspected pulmonary embolism is not recommended, but these methods may be considered when pulmonary embolism is the known cause of cardiac arrest.

Percutaneous pulmonary thrombectomy. In one case series, percutaneous pulmonary thrombectomy during CPR was successful in six of seven patients,[237,238] but larger studies are needed to validate this method.

Post-resuscitation care. In patients with sustained ROSC, exclude intra-abdominal and intra-thoracic CPR-related injuries, especially if a mechanical chest compression device was used simultaneously with administration of fibrinolytics.[239–241] Attempt to identify and treat the original cause of the pulmonary embolism. Evaluate the risks of a further pulmonary embolism and treat accordingly.

Coronary thrombosis

Coronary heart disease is the most frequent cause of OHCA. The peri-resuscitation management of acute coronary syndromes is addressed in a separate chapter (see Section 8 Initial management of acute coronary syndromes).[242] In cardiac arrest centres, coronary artery occlusion or high degree stenoses can be identified and treated. Of all patients in OHCA, however, at least half are not transported to hospital when ROSC is not achieved (see Section 10 Ethics of resuscitation and end-of-life decisions).[243] Although proper diagnosis of the cause may be difficult in a patient already in cardiac arrest, if the initial rhythm is VF it is most likely that the cause is coronary artery disease with an occluded large coronary vessel.

Consider transportation to hospital with ongoing CPR if treatment options are available that cannot be applied in the prehospital setting, such as immediate coronary angiography, primary percutaneous coronary intervention (PPCI) or other interventions such as (more rarely) pulmonary embolectomy (see pulmonary embolism). The decision to transport is complex and may depend on local circumstances. Prehospital initiation of extracorporeal cardiopulmonary life support (ECLS) requires specialised expertise and its feasibility on a wide-scale has not been established.[244–246] Mechanical chest compression devices maintain high quality CPR during transport and PCI (see cardiac arrest in HEMS and air ambulances).[247,248]

There is limited evidence for recommending routine transport to hospital with ongoing CPR. The decision will depend on patient selection, availability of optimal methods for mechanical or circulatory support during and after transport to the hospital, management of underlying pathology, treatment after ROSC, complication rate and outcome. There are no large outcome studies available, but small case series suggest benefit in selected cases.[249]

Before definitive recommendations can be made, controlled studies are needed.[250]

Transport with ongoing CPR and immediate access to the catheterisation laboratory may be considered if a prehospital and in-hospital infrastructure is available with teams experienced in mechanical or haemodynamic support and rescue PPCI with ongoing CPR. Excellent cooperation is required between prehospital and in-hospital teams. A decision to transport with ongoing CPR should take into consideration a realistic chance of survival (e.g. witnessed cardiac arrest with initial shockable rhythm (VF/pVT) and bystander CPR). Intermittent ROSC also strongly favours a decision to transport.[251]

Toxins

General considerations

Introduction. Overall, poisoning rarely causes cardiac arrest or death,[252] but hospital admissions are common, accounting for as many as 140,000 admissions each year in the UK.[252] 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 toxins. Evidence for treatment consists primarily of animal studies, case reports and small case series.[253–255]

Prevention of cardiac arrest. Assess the patient using systematic ABCDE approach. Airway obstruction and respiratory arrest secondary to a decreased conscious level is a common cause of death after self-poisoning (benzodiazepines, alcohol, opiates, tricyclics, barbiturates).[256,257] Early tracheal intubation of unconscious patients by trained personnel may decrease the risk of aspiration. Drug-induced hypotension usually responds to IV fluids, but occasionally vasopressor support (e.g. noradrenaline infusion) is required. Measure electrolytes (particularly potassium), blood glucose and arterial blood gases. Retain samples of blood and urine for analysis. Patients with severe poisoning should be cared for in a critical care setting.[257]

Modifications to resuscitation.

- Have a low threshold to ensure your personal safety where there is a suspicious cause or unexpected cardiac arrest. This is especially so when there is more than one casualty.
- Avoid mouth-to-mouth breathing 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 adult advanced life support).[168] This includes correction of electrolyte and acid-base abnormalities (see hypo-/hyperkalaemia and other electrolyte disorders).
- 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 hypo-/hyperthermia).
- Be prepared to continue resuscitation for a prolonged period, particularly in young patients, as the poison may be metabolised or excreted during extended resuscitation measures.
- Alternative approaches which may be effective in severely poisoned patients include: higher doses of medication than in standard protocols (e.g. high-dose insulin euglycemia)[258]; non-standard drug therapies (e.g. IV lipid emulsion)[259–262]; prolonged CPR, extracorporeal life support (ECLS),[263,264] and haemodialysis.
- 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 may be helpful: http://toxnet.nlm.nih.gov/.

Specific therapeutic measures

There are few specific therapeutic measures for poisoning that are useful immediately and improve outcomes: decontamination, enhancing elimination, and the use of specific antidotes.[265–267] Many of these interventions should be used only based on expert advice. For up-to-date guidance in severe or uncommon poisonings, seek advice from a poisons centre.

Decontamination. Decontamination is a process of removal of the toxin from the body determined by the route of exposure:

- For dermal exposures initial management consists of clothing removal and copious irrigation with water, except in case of reactive alkali metals that can ignite.
- Routine use of gastric lavage for gastrointestinal decontamination is no longer recommended. In the rare instances (e.g. lethal ingestion with recent exposure), it should only be performed by individuals with proper training and expertise. Gastric lavage may be associated with life-threatening complications, e.g. aspiration pneumonitis, aspiration pneumonia, esophageal or gastric perforation, fluid and electrolyte imbalances, arrhythmia. It is contraindicated if the airway is not protected and if a hydrocarbon with high aspiration potential or a corrosive substance has been ingested.[267,268]
- The preferred method of gastrointestinal decontamination in patients with an intact or protected airway is activated charcoal. It is most effective if given within 1 h of the time of the ingestion.[269] Activated charcoal does not bind lithium, heavy metals and toxic alcohols. Most common side effects are vomiting and constipation. The evidence that active charcoal improves outcome is limited.[257]
- Based mainly on volunteer studies, consider whole-bowel irrigation in potentially toxic ingestions of sustained-release or enteric-coated drugs particularly for those patients presenting later than 2 h after drug ingestion when activated charcoal is less effective. It may be also used for the removal of substantial amounts of iron, lithium, potassium, or packets of illicit drugs. Whole-bowel irrigation is contraindicated in patients with bowel obstruction, perforation, ileus, and haemodynamic instability.[270]
- Avoid routine administration of laxatives (cathartics) and do not use emetics (e.g. ipecac syrup).[271–273]

Enhanced elimination. Modalities removing a toxin from the body once it has been absorbed include multiple-dose activated charcoal (MDAC), urinary alkalinisation and extracorporeal elimination techniques:

- MDAC, multiple doses of activated charcoal administered over several hours, can increase certain drug elimination.[274,275] Give an initial dose of 50–100 g in adults (25–50 g in children).
- Urinary alkalinisation (urine pH $\geq$ 7.5) involves an IV sodium bicarbonate infusion. It is most commonly performed in patients with salicylate intoxication who do not need dialysis. Consider urine alkalinisation with high urine flow (about 600 mL h^{-1}) in severe poisoning by phenobarbital and herbicides, e.g. 2,4-dichlorophenoxyacetic acid or methylchlorophenoxypropionic acid (mecoprop). Hypokalaemia is the most common complication.[265]

- Haemodialysis removes drugs or metabolites with low molecular weight, low protein binding, small volumes of distribution and high water solubility. In case of hypotension, use continuous veno-venous hemofiltration (CVVH) or continuous veno-venous haemodialysis (CVVHD) alternatively.[257]

Specific poisons

These guidelines address only some of the more common poisons causing cardiac arrest.

Benzodiazepines. Overdose of benzodiazepines can cause loss of consciousness, respiratory depression and hypotension. Flumazenil, a competitive antagonist of benzodiazepines, may be used for reversal of benzodiazepine sedation when there is no history or risk of seizures. Reversal of benzodiazepine intoxication with flumazenil can be associated with significant toxicity (seizure, arrhythmia, hypotension, and withdrawal syndrome) in patients with benzodiazepine dependence or co-ingestion of proconvulsant medications such as tricyclic antidepressants.[276–278] The routine use of flumazenil in the comatose overdose patient is not recommended.There are no specific modifications to the ALS algorithm required for cardiac arrest caused by benzodiazepines.[278–282]

Opioids. Opioid poisoning causes respiratory depression followed by respiratory insufficiency or respiratory arrest. The respiratory effects of opioids are reversed rapidly by the opiate antagonist naloxone.

In severe respiratory depression caused by opioids, there are fewer adverse events when airway opening, oxygen administration and ventilation are carried out before giving naloxone[283–289]; The use of naloxone can prevent the need for intubation. The preferred route for giving naloxone depends on the skills of the rescuer: intravenous (IV), intramuscular (IM), subcutaneous (SC), intraosseous (IO) and intranasal (IN) routes are all suitable.[290,291] The non-IV routes can be quicker because time is saved in not having to establish IV access, which may be difficult in an IV drug abuser. The initial doses of naloxone are 0.4–2 mg IV, IO, IM or SC, and may be repeated every 2–3 min. Additional doses may be needed every 20–60 min. Intranasal dosing is 2 mg IN (1 mg in each nostril) which may be repeated every 5 min. Titrate the dose until the victim is breathing adequately and has protective airway reflexes. Large opioid overdoses may require a total dose of up to 10 mg of naloxone.[283–285,290–300] All patients treated with naloxone must be monitored.

Acute withdrawal from opioids produces a state of sympathetic excess and may cause complications such as pulmonary oedema, ventricular arrhythmias and severe agitation. Use naloxone reversal of opioid intoxication with caution in patients suspected of opioid dependence.

There are no data on the use of any additional therapies beyond standard ALS guidelines in opioid-induced cardiac arrest. In respiratory arrest there is good evidence for the use of naloxone, but not for any other adjuncts or changes in interventions.[284]

Tricyclic antidepressants. This section addresses both tricyclic and related cyclic drugs (e.g. amitriptyline, desipramine, imipramine, nortriptyline, doxepin, and clomipramine). Self-poisoning with tricyclic antidepressants is common and can cause hypotension, seizures, coma and life-threatening arrhythmias. Cardiac toxicity mediated by anticholinergic and Na^+ channel-blocking effects can produce a wide complex tachycardia (VT). Hypotension is exacerbated by alpha-1 receptor blockade. Anticholinergic effects include mydriasis, fever, dry skin, delirium, tachycardia, ileus, and urinary retention. Most life-threatening problems occur within the first 6 h after ingestion.[301–303]

A widening QRS complex (>100 ms) and right axis deviation indicates a greater risk of arrhythmias.[304–306] Give sodium bicarbonate (1–2 mmol kg^{-1}) for the treatment of tricyclic-induced ventricular arrhytmias.[307–312] While no study has investigated the optimal target arterial pH with bicarbonate therapy, a pH of 7.45–7.55 is recommended.[255,257] Administration of bicarbonate may resolve arrhythmias and reverse hypotension even in the absence of acidosis.[312]

Intravenous lipid infusions in experimental models of tricyclic toxicity have suggested benefit but there are few human data.[313,314] Anti-tricyclic antibodies have also been beneficial in experimental models of tricyclic cardiotoxicity.[315–320] One small human study provided evidence of safety but clinical benefit has not been shown.[321]

There are no randomised controlled trials evaluating conventional versus alternative treatments for cardiac arrest caused by tricyclic toxicity. One small case series showed improvement with the use of sodium bicarbonate but the concomitant use of physostigmin prevents the ability to generalise its results.[322]

Cocaine. Sympathetic overstimulation associated with cocaine toxicity can cause agitation, tachycardia, hypertensive crisis, hyperthermia and coronary vasoconstriction causing myocardial ischaemia with angina.

In patients with severe cardiovascular toxicity, alpha blockers (phentolamine),[323] benzodiazepines (lorazepam, diazepam),[324,325] calcium channel blockers (verapamil),[326] morphine,[327] and sublingual nitroglycerine[328,329] may be used as needed to control hypertension, tachycardia, myocardial ischaemia and agitation. The evidence for or against the use of beta-blocker drugs,[330–333] including those beta-blockers with alpha blocking properties (carvedilol and labetolol) is limited.[334–336] The optimal choice of anti-arrhythmic drug for the treatment of cocaine-induced tachyarrhythmias is not known. If cardiac arrest occurs, follow standard resuscitation guidelines.[337]

Local anaesthetics. Systemic toxicity of local anaesthetics involves the central nervous and cardiovascular systems. Severe agitation, loss of consciousness, seizures, bradycardia, asystole or ventricular tachyarrhythmias can all occur. Toxicity typically occurs in the setting of regional anaesthesia, when a bolus of local anaesthetic inadvertently enters an artery or vein (see perioperative cardiac arrest).

Although there are many case reports and case series of patients who were resuscitated after administration of IV lipid emulsion, evidence for its benefit in treating local anaesthetic-induced cardiac arrest is limited. Despite the paucity of data, patients with both cardiovascular collapse and cardiac arrest attributable to local anaesthetic toxicity may benefit from treatment with intravenous 20% lipid emulsion in addition to standard ALS.[338–352] Give an initial intravenous bolus injection of 20% lipid emulsion 1.5 mL kg^{-1} over 1 min followed by an infusion at 15 mL kg^{-1} h^{-1}. Give up to a maximum of two repeat boluses at 5-min intervals and continue until the patient is stable or has received up to a maximum cumulative dose of 12 mL kg^{-1} of lipid emulsion.[259–262,353] Standard cardiac arrests drugs (e.g. adrenaline) should be given according to ALS guidelines, although animal studies provide inconsistent evidence for their role in local anaesthetic toxicity.[349,352,354–356]

Beta-blockers. Beta-blocker toxicity causes bradyarrhythmias and negative inotropic effects that are difficult to treat, and can lead to cardiac arrest.

Evidence for treatment is based on case reports and animal studies. Improvement has been reported with glucagon (50–150 mcg kg^{-1}),[357–370] high-dose insulin and glucose,[371–373] lipid emulsions,[374–377] phosphodiesterase inhibitors,[378,379]

extracorporeal and intra-aortic balloon pump support,[380–382] and calcium salts.[258,383]

Calcium channel blockers. Calcium channel blocker overdose is emerging as a common cause of prescription drug poisoning deaths.[384,385] Overdose of short-acting drugs can rapidly progress to cardiac arrest. Overdose by sustained-release formulations can result in delayed onset of arrhythmias, shock, and sudden cardiac collapse. The treatment for calcium channel blocker poisoning is supported by low-quality evidence.[386]

Give calcium chloride 10% in boluses of 20 mL (or equivalent dose of calcium gluconate every 2–5 min in severe bradycardia or hypotension followed by an infusion if needed.[255,257,258,386,387] While calcium in high doses can overcome some of the adverse effects, it rarely restores normal cardiovascular status. Haemodynamic instability may respond to high doses of insulin (1 unit kg^{-1} followed by an infusion of 0.5–2.0 units kg^{-1} h^{-1}) given with glucose supplementation and electrolyte monitoring in addition to standard treatments including fluids and vasopressors (e.g. dopamine, norepinephrine, vasopressin).[386–398] Extracorporeal life support (ECLS) was associated with improved survival in patients with severe shock or cardiac arrest at the cost of limb ischaemia, thrombosis, and bleeding.[264] Studies on decontamination, 4-aminopyridine, atropine, glucagon, pacemakers, levosimendan, and plasma exchange reported variable results.[386]

Digoxin. Although cases of digoxin poisoning are fewer than those involving calcium channel and beta-blockers, the mortality rate from digoxin is far greater. Other drugs including calcium channel blockers and amiodarone can also cause plasma concentrations of digoxin to rise. Atrioventricular conduction abnormalities and ventricular hyperexcitability due to digoxin toxicity can lead to severe arrhythmias and cardiac arrest.

Specific antidote therapy with digoxin-specific antibody fragments (digoxin-Fab) should be used if there are arrhythmias associated with haemodynamic instability.[257,399–401] Digoxin-Fab therapy may also be effective in poisoning from plants (e.g. oleander) and Chinese herbal medications containing cardiac glycosides.[399,402,403] Digoxin-Fab interfere with digoxin immunoassay measurements and can lead to overestimation of plasma digoxin concentrations. In acute poisoning, give an initial bolus of 2 vials digoxin-Fab (38 mg per vial) and repeat dose if necessary.[401] In a cardiac arrest, consider administration of 2 up to 10 vials IV over 30 min.

Cyanides. Cyanide is generally considered to be a rare cause of acute poisoning; however, cyanide exposure occurs relatively frequently in patients with smoke inhalation from residential or industrial fires. Cyanides are also used in several chemical and industrial processes. Its main toxicity results from inactivation of cytochrome oxidase (at cytochrome a3), thus uncoupling mitochondrial oxidative phosphorylation and inhibiting cellular respiration, even in the presence of adequate oxygen supply. Tissues with the highest oxygen needs (brain and heart) are the most severely affected by acute cyanide poisoning.

Patients with severe cardiovascular toxicity (cardiac arrest, cardiovascular instability, metabolic acidosis, or altered mental status) caused by known or suspected cyanide poisoning should receive cyanide antidote therapy in addition to standard resuscitation, incl. oxygen. Initial therapy should include a cyanide scavenger (either hydroxocobalamin 100 mg kg^{-1} IV or a nitrite – i.e. IV sodium nitrite and/or inhaled amyl nitrite), followed as soon as possible by IV sodium thiosulfate.[404–410] Hydroxocobalamin and nitrites are equally effective but hydroxocobalamin is safer because it does not cause methaemoglobin formation or hypotension.[411–413]

In the case of cardiac arrest caused by cyanide, standard treatment fails to restore spontaneous circulation as long as cellular respiration is blocked. Antidote treatment is needed for reactivation of cytochrome oxidase.

Carbon monoxide. Carbon monoxide poisoning is common. There were about 25,000 carbon monoxide related hospital admissions reported yearly in the United States.[414] Carbon monoxide levels do not correlate with the presence or absence of initial symptoms or with later outcomes.[415] Patients who develop cardiac arrest caused by carbon monoxide rarely survive to hospital discharge, even if ROSC is achieved.[413,416]

Give oxygen as soon as possible. The use of hyperbaric oxygen has been used to treat carbon monoxide exposure in order to reduce the incidence of adverse neurologic outcomes.[417] However, two Cochrane reviews failed to demonstrate convincing benefit from hyperbaric oxygen therapy for patients with carbon monoxide poisoning.[416,418] The role of carbon monoxide in nitric oxide release, reactive oxygen species formation, and its direct action on ion channels may be more significant than its higher affinity for haemoglobin, which is treated by oxygen therapies.[419] There is unproven benefit for transporting critically ill post-arrest patients to a hyperbaric facility and such decision must be considered on a case-by-case basis.[413,416,418,419] Patients who develop myocardial injury caused by carbon monoxide have an increased risk of cardiac and all-cause mortality lasting at least seven years after the event; it is reasonable to recommend cardiology follow-up for these patients.[413,420,421]

B – SPECIAL ENVIRONMENTS

Cardiac arrest in healthcare facilities

Perioperative cardiac arrest

Introduction. Although the safety of routine surgical procedures has increased over recent decades, the greater number of procedures being performed, particularly in more elderly patients and in emergency situations has resulted in a broadly stable incidence of perioperative cardiac arrests over the past decade.

Although the features of perioperative cardiac arrest are often different to those of cardiac arrests occurring in the general hospital population, the principles of treatment are similar. Perioperative cardiac arrest may be caused by the underlying condition being treated, physiological effects of the surgery, anaesthetic drugs and fluids, complications relating to existing co-morbidities, or adverse events.

Epidemiology. The overall incidence of perioperative cardiac arrest ranges from 4.3 to 34.6 per 10,000 procedures.[422–424] This wide range reflects differences in case-mix (some include neonates and/or cardiac surgery) and in the definition of perioperative. The incidence is higher in high-risk groups such as the elderly where it has been reported as 54.4 per 10,000 cases[425] and in patients undergoing emergency surgery where an incidence of 163 per 10,000 cases has been reported.[426] Young age (<2 years old), cardiovascular and respiratory comorbidities, increasing American Society of Anesthesiologists (ASA) physical status classification, preoperative shock, and surgery site have all been identified as risk factors for perioperative cardiac arrest.[426]

The incidence of cardiac arrest attributable primarily to anaesthesia is a relatively small proportion of this overall incidence and in recent studies is estimated to be 1.1–3.26 per 10,000 procedures.[425,427,428] Overall survival from perioperative cardiac arrest is higher than from OHCA, with survival to hospital discharge rates of 30–36.6% being reported recently.[422,424,428]

General versus regional anaesthesia. The incidence of perioperative cardiac arrest during general anaesthesia (GA) is higher than that of regional anaesthesia (RA). The incidence of cardiac arrest for patients receiving general anaesthesia in a study from the Mayo Clinic was higher (almost 3 times higher, at 4.3 per 10,000) than that for those receiving regional anaesthesia or monitored anaesthesia care. The incidence however decreased significantly over a 10-year period.[423]

Causes of cardiac arrest. Overall causes of cardiac arrest have been identified as:

- Hypovolaemia (e.g. bleeding).
- Cardiac-related.
- Other:
  - Drug-induced (e.g. muscle relaxants).
  - Anaesthesia related.
- Airway loss.
- Ventilation failure.
  - Anaphylaxis (drugs, blood products).

The commonest cause of anaesthesia-related cardiac arrest involves airway management.[427,428] Failure of ventilation, medication-related events, complications associated with central venous access, and perioperative myocardial infarction are also common.[423,429] In children, airway obstruction from laryngospasm, hypovolaemia from blood loss and hyperkalemia from transfusion of stored blood are additional causes.[430]

Cardiac arrest caused by bleeding had the highest mortality in non-cardiac surgery, with only 10.3% of these patients surviving to hospital discharge.[423] The primary arrest rhythms during perioperative cardiac arrest recorded in the Mayo Clinic series were asystole in 41.7%, VF in 35.4%, PEA in 14.4% and unknown in 8.5%. Contrary to studies of cardiac arrest in general, the rhythm associated with the best chance of survival to hospital discharge was asystole (43% survival).[423,431]

Management of perioperative cardiac arrest. Patients in the operating room are normally fully monitored and, as such, there should be little or no delay in diagnosing cardiac arrest. High-risk patients will often have invasive blood pressure monitoring, which is invaluable in the event of cardiac arrest. If cardiac arrest is a strong possibility, apply self-adhesive defibrillation electrodes before induction of anaesthesia, ensure adequate venous access and prepare resuscitation drugs and fluids. Use fluid warmers and forced air warmers to limit perioperative hypothermia and monitor the patient's temperature.

In the event of cardiac arrest, follow the ALS algorithm, but with appropriate modifications. Adjust the position and height of the operating table or trolley to optimise delivery of chest compressions. CPR is optimal in the supine position, but is possible in patients who are prone and where immediate turning to a supine position is not possible.[432,433] Risk factors for cardiac arrest in prone patients include cardiac abnormalities in patients undergoing major spinal surgery, hypovolaemia, air embolism, wound irrigation with hydrogen peroxide, occluded venous return.

Identification of causes. In many cases of perioperative cardiac arrest, physiological deterioration is gradual and the cause of the cardiac arrest is known and hence the arrest anticipated. In those where this is not the case, follow the standard ABC algorithm to identify and treat reversible causes. If patients deteriorate, call for senior help immediately. Inform the perioperative team of the deterioration and possible impending cardiac arrest, ensuring that sufficient skilled assistance is present.

C Catastrophic haemorrhage is usually obvious, but may be occult it if involves bleeding into body compartments (abdomen, chest) or into soft tissues in patients with multiple limb fractures. Pelvic and retroperitoneal haemorrhage can also cause rapid hypovolaemia and should be excluded, e.g. by ultrasound if pre-operative haemodynamic instability. In cases where direct surgical intervention is unable to control haemorrhage, early interventional radiography should be considered.

A Loss of the airway is a common cause of perioperative cardiac arrest. Assess the airway carefully before induction of anaesthesia. Prepare all equipment, including suction and an operating table or trolley that can be tipped head-down (Trendelenburg position). Ensure that difficult airway equipment is immediately available, and brief the team on a failed intubation drill if appropriate. Always use waveform capnography. Children are particularly prone to loss of the airway from laryngospasm; ensure an appropriate neuromuscular blocker is available and give before significant hypoxaemia has occurred in order to break the laryngospasm.

B Undiagnosed tension pneumothorax is a readily treatable cause of cardiac arrest. Although usually associated with trauma, consider early in the management of all patients who arrest, particularly those with chronic obstructive pulmonary disease and severe asthma. A sudden increase in airway pressures may indicate a tension pneumothorax or problems with the breathing tubing, but also consider asthma and anaphylaxis.

C Cardiovascular collapse has several causes, but in the context of perioperative cardiac arrest, common causes include hypovolaemia, anaphylaxis, and vagal stimulation. Use of transthoracic echocardiography is a useful tool to exclude cardiac tamponade (if suspected) and to assess myocardial contractility and filling.

Anaphylaxis. The incidence of immune-mediated anaphylaxis during anaesthesia ranges from 1 in 10,000 to 1 in 20,000.[434] Neuromuscular blocking drugs are the commonest cause, being associated with 60% of cases. The associated morbidity and mortality are high, particularly if there are delays in the diagnosis and management. Initial management of anaphylaxis follows the ABC approach and the management principles outlined in the chapter on anaphylaxis. Adrenaline is the most effective drug in anaphylaxis and is given as early as possible. It is appropriate for anaesthetists to give adrenaline by the intravenous route. Repeated doses may be necessary.

If cardiac arrest ensues despite correct treatment for the anaphylaxis (see anaphylaxis), continue resuscitation using the standard ALS algorithm (see adult advanced life support).[168]

Systemic toxicity of local anaesthetics. Cardiac arrest is a rare but well recognised complication of local anaesthetic (LA) overdose, especially following inadvertent intravascular injection. Direct action of the LA on cardiac myocytes causes cardiovascular collapse, usually within 1–5 min of injection, but onset may range from 30 s to as long as 60 min.[435] Significant hypotension, dysrhythmias, and seizures are typical manifestations, but diagnosis may be one of exclusion.[436]

IV lipid therapy has been used as a rescue therapy to treat cardiovascular collapse and cardiac arrest, but its efficacy is debated.[437] In the absence of documented harm, guidelines recommend that 20% lipid emulsion should be available for use wherever patients

receive large doses of LA (e.g. operating rooms, labour wards and the emergency department).[353,438] Stop injecting the LA and call for help. Secure and maintain the airway and, if necessary, intubate. Give 100% oxygen and ensure adequate ventilation (hyperventilation may help by increasing plasma pH in the presence of metabolic acidosis). Control seizures using a benzodiazepine, thiopental or propofol. Give an initial IV bolus injection of 20% lipid emulsion at 1.5 mL kg^{-1} over 1 min and then start an infusion at 15 mL kg^{-1} h^{-1}. If ROSC has not been achieved at 5 min, double the rate of lipid infusion and give a maximum of two additional lipid boluses at 5-min intervals until ROSC has been achieved. Do not exceed a maximum cumulative dose of 12 mL kg^{-1}.[259,260]

Diagnosis of cardiac arrest. Asystole and ventricular fibrillation (VF) will be detected immediately, but the onset of PEA might not be so obvious – loss of the pulse oximeter signal and very low end-tidal CO_2-values will be good clues and should provoke a pulse check. Do not waste time attempting to obtain a non-invasive blood pressure measurement.

Management of cardiac arrest. The management of a cardiac arrest follows the principles of the ALS algorithm. Chest compression in the prone position can be achieved with or without sternal counter-pressure. In one study of prone CPR with sternal counter pressure (provided by a sandbag) versus standard CPR, higher mean arterial pressures were achieved with the prone technique.[439] Consider open cardiac compressions in patients where the thorax is open or the heart can be easily accessed.

Ventricular fibrillation. In the case of VF, call for a defibrillator. If one is not immediately available, apply a precordial thump. If that is unsuccessful, give chest compressions and ventilation until the defibrillator arrives. Look for reversible causes immediately – hypoxaemia and hypovolaemia will be the most common in this setting.

Asystole/extreme bradycardia. Stop any surgical activity likely to be causing excessive vagal activity – if this is the likely cause – give 0.5 mg atropine IV/IO (not 3 mg). Start CPR and immediately look for other reversible causes. Exclude a completely straight line, which suggests an ECG monitoring lead has become detached.

Pulseless electrical activity. Start CPR while looking quickly for reversible causes of PEA. Give fluid unless you are certain that the intravascular volume is adequate. Stop administration of the anaesthetic. While a vasopressor will be required, in these circumstances 1 mg of adrenaline (as directed by the standard ALS guidelines) may be excessive. Give a smaller dose (e.g. 1 mcg kg^{-1}) of adrenaline, or another vasopressor initially; if this fails to restore the cardiac output, increase the dose while continuing to perform chest compressions and ventilation.

Monitoring and feedback during CPR. Unlike OHCAs where monitoring if often limited, patients arresting in the perioperative period can often be monitored with a greater degree of precision.

Monitoring enables assessment of rescuer performance and patient response:

- Rescuer CPR performance.

Feedback sensors (e.g. accelerometers) improve the delivery of effective chest compressions and enable the rescuer to tailor their performance accordingly. Their use should be considered whenever available. Performance feedback can be obtained from invasive and non-invasive patient monitoring and the rescuer should have direct visualisation of monitors displaying these data.

- Patient response.

Monitoring of the patient requires adequate lighting and patient exposure.Non-invasive blood pressure is unlikely to be of assistance until ROSC is achieved, but in patients with invasive arterial monitoring, aim for a diastolic blood pressure >25 mmHg,[440] titrating it to this level (after chest compressions are optimised) by administration of a vasopressor, if necessary. This goal is based on expert consensus derived from experimental and limited clinical data.[441–443]

Waveform capnography is a minimum monitoring standard during anaesthesia and therefore immediately available during a perioperative cardiac arrest. In addition to its use for patients with tracheal intubation where it is particularly valuable to confirm correct tracheal tube placement, it may also be used in patients with supraglottic airway devices (although an air leak may limit quantitative evaluation). An end-tidal carbon dioxide ($ETCO_2$) value <1.4 kPa/10 mmHg suggests a low cardiac output and rescuers may be able to adjust their technique to optimise this variable. An abrupt sustained increase to a normal value (4.7–5.4 kPa/35–40 mmHg) or even higher may be an indicator of ROSC. Optimise CPR to achieve an $ETCO_2$ >2.7 kPa/20 mmHg, while ventilating the lungs at about 10 breaths min^{-1}, with only minimal chest rise).[440]

Team working. Every resuscitation event should have a designated team leader who directs and coordinates all staff and the components of the resuscitation, with a central focus on delivering high-quality CPR. Stop operative surgery unless it is addressing a reversible cause of the cardiac arrest. Patient access and resuscitation tasks may necessitate covering the surgical field and withdrawing the surgical team from the patient. Prioritise team tasks, ensure good quality basic life support (BLS), identify reversible causes and avoid non-priority tasks.[440] If the patient is not responding to resuscitative efforts (i.e. $ETCO_2$ <2.7 kPa/20 mmHg), try to improve the quality of CPR by optimising: (1) compression fraction, (2) compression rate, (3) compression depth, (4) leaning, and (5) by avoiding of excessive ventilation.[440]

Post-resuscitation care. Depending on the circumstances, patients successfully resuscitated after a very brief period of cardiac arrest, e.g. asystole from excessive vagal stimulation may not require anything more than standard post-operative care. All those resuscitated successfully after longer periods of cardiac arrest will require admission to an ICU – unless further active treatment is deemed inappropriate. In most circumstances, anything but immediately life-saving surgery should be abandoned to enable admission to ICU for post-resuscitation care. Patients resuscitated after a prolonged period of cardiac arrest may develop a marked systemic inflammatory response syndrome (SIRS) with the risk of multiple organ failure. They will require optimisation of mean arterial pressure, oxygenation and ventilation. These patients may have sustained a significant cerebral insult. Some may be suitable for targeted temperature management, but this requires careful consideration, given the lack of data on this therapy in the setting of perioperative cardiac arrest. Active bleeding would certainly be a contraindication to induced mild hypothermia but, at the very least, prevent fever in all cases. Avoidance of hyperthermia, from overwarming or a post-cardiac arrest syndrome[444] is important to optimise neurological recovery.

Do not attempt resuscitation decisions. Patients with DNAR decisions presenting for surgery present a dilemma for the anaesthetist. The anaesthetic will induce cardiovascular instability, many of the routine interventions undertaken could be considered as resuscitative, and the chances of surviving a perioperative cardiac arrest are better than those from in-hospital cardiac arrest in general. Consider each case on its individual merits and discuss with the patient and/or relatives. Some patients may wish a DNAR decision to remain valid despite the increased risk of a cardiac arrest and the

presence of potentially reversible causes; others will request that the DNAR decision is suspended temporarily. Discuss and agree on the time at which the DNAR decision is reinstated.[445]

Cardiac arrest following cardiac surgery

Introduction. Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase, with a reported incidence of 0.7–8%.[446–455] It is usually preceded by physiological deterioration,[456] although it can occur suddenly in stable patients.[452] There are usually specific causes of cardiac arrest, such as tamponade, hypovolaemia, myocardial ischaemia, tension pneumothorax, or pacing failure. These are all potentially reversible and if treated promptly cardiac arrest after cardiac surgery has a relatively high survival rate. Key to the successful resuscitation of cardiac arrest in these patients is recognition of the need to perform emergency resternotomy early, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective.

Starting CPR. If VF or asystole is diagnosed, immediately administer external defibrillation or emergency temporary pacing at maximum amplitude. Otherwise start external chest compressions immediately in patients who arrest with monitoring indicating no output. Verify the effectiveness of compressions by looking at the arterial trace, aiming to achieve a systolic blood pressure >60 mmHg [Society of Thoracic Surgeons (STS) Clinical Practice guidelines in preparation – personal communication from Joel Dunning] and a diastolic blood pressure >25 mmHg[440] at a rate of 100–120 min^{-1}. Inability to obtain this goal with external chest compressions indicates that cardiac tamponade or extreme hypovolaemia is likely and emergency resternotomy should be performed.

Consider other reversible causes:

- Hypoxia – check tracheal tube position, ventilate with 100% oxygen.
- Tension pneumothorax – check tracheal position, listen for air entry.
- Pacing failure – check pacing box output and pacing wire integrity. 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).

Defibrillation. There is concern that external chest compressions can cause sternal disruption or cardiac damage.[457–460] In the post-cardiac surgery ICU, a witnessed and monitored VF/pVT 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.[461,462]

Emergency drugs. Use adrenaline very cautiously and titrate to effect (IV doses of up to 100 mcg in adults). Consider amiodarone 300 mg in patients with refractory shockable rhythms (VF/pVT), but do not delay resternotomy. Atropine is not recommended for asystole and temporary or external pacing should be employed.

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/pVT, undertake resternotomy without delay. Emergency resternotomy is also indicated in asystole or PEA, when other treatments have failed, and should be performed within 5 min of the cardiac arrest by anyone with appropriate training.

These guidelines are also appropriate for patients following non-sternotomy cardiac surgery, but surgeons performing these operations should have already made clear their instructions for chest reopening in an arrest.

Special considerations regarding treatment of patients with ventricular assist devices (VADs) are addressed in the section on special patients (see patients with ventricular assist devices).

Cardiac arrest in a cardiac catheterisation laboratory

Cardiac arrest may occur during percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI) or non-STEMI, but it may also be a complication of an angiography such as catheter wedging, air or thrombus embolism in the coronary artery, coronary artery intima dissection from the tip of the angiography catheter or caused by pericardial tamponade from a perforated coronary artery during the procedure. Most complications will result in VF with immediate need for defibrillation. For this reason, patients must be continuously monitored and a defibrillator must be available in the angiography room. Self-adhesive radiolucent defibrillation pads may already be placed at the beginning of the procedure in high-risk patients.

In this special setting with immediate response to monitored VF, defibrillation without preceding chest compressions is recommended. As the patient is early in the electrical phase of a cardiac arrest, in contrast to the guidelines for unmonitored and OHCAs, the result of defibrillation (VF termination and ROSC) can be determined before chest compressions are started. If needed for failed defibrillation or immediately recurring VF, immediate defibrillation may be repeated up to two times.

If VF persists after the initial three shocks or ROSC not immediately established with certainty, chest compressions and ventilations must be initiated without further delay and a cause for the unresolved problem sought with further coronary angiography. It is of extreme importance that chest compressions are not interrupted for angiography. On an angiography table with the image intensifier above the patient, delivering chest compressions with adequate depth and rate is almost impossible and exposes the rescuers to dangerous radiation. Therefore, early transition to the use of a mechanical chest compression device is strongly recommended.[247,463] If the problem is not rapidly resolved, very low quality evidence suggests that the use of extracorporeal life support (ECLS) can be considered as a rescue strategy if the infrastructure is available, and probably to be preferred over intra-aortic balloon pump (IABP).[464] There is no evidence to recommend circulatory support with the Impella pump only during cardiac arrest.

If the cardiac arrest is caused by a non-shockable rhythm, immediate transthoracic echocardiography should identify pericardial tamponade or other conditions.

Cardiac arrest in a dialysis unit

Introduction. Sudden cardiac death is the most common cause of death in haemodialysis patients and is usually preceded by ventricular arrhythmias.[465] Hyperkalemia contributes to 2–5% of deaths amongst haemodialysis patients[466] and accounts for up to 24% of emergency haemodialysis session in haemodialysis patients.[467] The frequency of cardiac arrest is highest on the first session of haemodialysis of the week (i.e. Monday or Tuesday) as fluid and electrolyte disturbances peak after the weekend interval.[468] Primary prevention of cardiac arrest in dialysis patients include the avoidance of low potassium dialysate solutions and proper use of medication, e.g. beta blockers or angiotensin-converting enzyme inhibitors.[465] There is little evidence to guide the treatment of cardiac arrest during haemodialysis, although some special considerations have been suggested.[469]

Initial steps.

- Call the resuscitation team and seek expert help immediately.
- Follow the universal ALS algorithm.
- Assign a trained dialysis nurse to operate the dialysis machine.
- Stop ultrafiltration (i.e. fluid removal) and give a fluid bolus.
- Return the patient's blood volume and disconnect from the dialysis machine.
- Leave dialysis access open and use for drug administration.
- Beware of wet surfaces (i.e. dialysis machines may leak).
- Minimise delay in delivering defibrillation.

Modifications to cardiopulmonary resuscitation.

Defibrillation. A shockable rhythm (VF/pVT) is more common in patients undergoing haemodialysis[465,470,471] than in the general population.[472,473] The safest method to deliver a shock during dialysis requires further study. Most haemodialysis machine manufacturers recommend disconnection from the dialysis equipment prior to defibrillation.[474] Ensure familiarity with local dialysis equipment and check if equipment has defibrillator-proof label in accordance with the International Electrotechnical Committee (IEC) standards. Automated external defibrillators in nurse-led dialysis centres can facilitate early defibrillation by first responders with appropriate training.[475]

Vascular access. Use dialysis access in life-threatening situations and cardiac arrest.[469]

Potentially reversible causes. All of the standard reversible causes (4 Hs and 4 Ts) apply to dialysis patients. Electrolyte disorders, particularly hyperkalaemia (see hypo-/hyperkalaemia and other electrolyte disorders), and fluid overload (e.g. pulmonary oedema) are most common causes.

Post resuscitation care. Dialysis may be required in the early post resuscitation period guided by fluid status and serum biochemistry. Patient transfer to an area with dialysis facilities (i.e. intensive care unit or renal high dependency unit) is essential.

Cardiac arrest in the dental surgery

Introduction. Dental surgery emergencies include a variety of situations ranging from psychosomatic disorders precipitated by fear and anxiety to life-threatening situations requiring immediate life-saving procedures. Cardiac arrest in primary dental practice is rare with an incidence of 0.002–0.011 cases reported per dentist per year.[476–478]

The most frequent medical emergencies include vasovagal (pre-) syncope, orthostatic hypotension, hypertensive crisis, hyperventilation, seizures, moderate allergic reactions, hypoglycaemia, and angina.[476,479] The majority of dentists responded that they would be capable of performing initial treatment of common emergencies, while many felt unable to treat anaphylaxis, myocardial infarction, or cardiac arrest.[476,477]

A cardiac arrest occurring in a dental surgery is an event witnessed by medical professionals who have a duty of care and are required to be competent in the delivery of CPR.

Causes of cardiac arrest. Causes of cardiac arrest usually relate to pre-existing comorbidities or complications of the procedure. The life-threatening emergencies commonly arise from myocardial infarction, grand mal seizures or exacerbation of asthma. Dental procedures may cause loss of airway patency related to the primary pathology or complications of the procedure (e.g. bleeding, secretions, tissue swelling). Choking is rare, with a reported incidence of 0.07–0.09 cases per dentist per year.[476,477] The addition of sedation is a contributory risk in these cases, although provision of dental treatment under both local anaesthesia and sedation has an excellent safety record.[480,481]

Although life-threatening anaphylaxis is rare, it is a documented cause of death during dental procedures. In addition to chlorhexidine mouthwash, other common causes may include penicillin and latex. Anaphylaxis to local anaesthetics is very rare and a reaction to this class of drug is usually due to a direct intravascular injection of adrenaline contained in the solution. True anaphylaxis (all causes) occurs in only 0.004–0.013 cases per dentist per year, compared with coronary symptoms (angina or myocardial infarction) occurring in 0.15–0.18 cases per year.[476,477]

Treatment of cardiac arrest. The following modifications to the initial sequence of actions are recommended if cardiac arrest occurs in a dental chair:

- In case of sudden loss of consciousness, immediately call for help.
- Look into the victim's mouth. Check and remove all solid materials from the oral cavity (e.g. retractor, suction tube, tampons, etc.). Prevention of airway obstruction should preceed positioning the patient on his back.
- Recline the dental chair into a fully horizontal position. Cardiac output can be restored if reduced venous return or vasodilation has caused loss of consciousness, e.g. vasovagal syncope, orthostatic hypotension. In these patients, raising the legs and/or placing the patient in a head-down position may also help.
- Simultaneously open the airway and check breathing (look, listen, feel). If breathing is not normal or absent, assume a cardiac arrest until proven otherwise. Send someone to get an AED if available.
- Some case reports describe successful CPR in a patient left on a dental chair.[482,483] Small simulation studies comparing the effectiveness of CPR on a dental chair and on the floor reported either lower or equivalent CPR quality.[484–487] However, the patient should not be moved from the dental chair because of the risk of injury to the patient and rescuers and the limited space that is likely to be available on the floor next to the patient.[482,483] Ensure that the dental chair is fully reclined into the horizontal position, support its head with a stool to increase stability, and start chest compressions immediately.[482,484]
- If feedback devices are used to monitor CPR quality, those using accelerometers may overestimate depth of compressions if used on a dental chair.[488]
- Follow the standard compression:ventilation ratios for adults and children. Consider the over-the-head technique of CPR if access to either side of the chest is limited.[489–492]
- Maintain the airway and ventilate the patient with a bag-valve-mask device, using the two-hand technique if necessary. Supraglottic airways may be inserted if the operator is skilled in their use, but tracheal intubation is not a recommended intervention required of dental practitioners and should be avoided.
- Switch on the AED and follow the instructions. Deliver the first shock as soon as possible if indicated.
- Continue with CPR until signs of life return, or the patient's handover to the professional resuscitation team (see adult basic life support and automated external defibrillation).[493]

Equipment and training. Follow national guidelines for recommended equipment to treat medical emergencies in a dental practice.[478] Basic resuscitation equipment should be available immediately in all primary care dental premises, including suction, self-inflating bag with face masks, oxygen, and emergency drug kit.[494,495] The role of early defibrillation should be emphasised to increase the availability of AEDs in dental surgeries,[482,496] which is still unsatisfactory, ranging from a reported 0.5–2.6% in Europe[497,498] to 11% in the United States.[499] We recommend that all dental practices delivering clinical care have immediate access to an AED, with all staff trained in its use. Advanced equipment

and special training is needed if analgesia or sedation is used in dental surgeries.[478,500] In patients with pacemakers, ECG monitoring and immediate availability of a defibrillator is recommended if electrical devices are used (e.g. diathermy, electric pulp tester, etc.).[482]

There is rightly a public expectation that dental practitioners and all other dental care professionals should be competent in treating cardiorespiratory arrest. However, only 0.2–0.3% dentists have experience in treating a patient in cardiac arrest,[476,479,501] and their training in CPR varies significantly between countries.[476,477,501–503] Maintaining knowledge and competence to deal with medical emergencies must be an important part of the training of dentists. All dental care professionals should undergo annual practical training in the recognition and management of medical emergencies, and the delivery of CPR, including basic airway management and the use of an AED.[478]

Cardiac arrest in transportation vehicles

In-flight emergencies aboard airplanes

Introduction. Worldwide, 3.2 billion passengers fly on commercial airlines annually. The incidence of in-flight medical emergencies has been reported to be one event per 10,000–40,000 passengers.[504,505] The probability of at least one medical incident reaches 95% after 24 intercontinental flights.[505] Most of the cases involve middle-aged people.[506] Two large studies recently reviewed more than 22,000 in-flight emergencies from five American and two European airlines. The most common medical problems were syncope or presyncope (37.4–53.5%), respiratory symptoms (12.1%), gastrointestinal problems (8.9–9.5%), and cardiac conditions (4.9–7.7%) with some variations across airlines.[504,507] Surgical problems (e.g. deep venous thrombosis, appendicitis, gastrointestinal bleeding) were seen rarely (<0.5%).[504] In-flight incapacitation of the flight crew is very rare, the most common cause being acute myocardial ischaemia.[508]

The in-flight medical emergencies have very limited access to medical care, but the majority can be managed conservatively with fluids, oxygen and other treatment available from first aid kits on board. However, a quarter of these patients subsequently require additional evaluation in a hospital.[507] Immediate diversion of an aircraft is requested in 2.4–7.3% of all incidents, most commonly due to chest pain, suspected stroke, and seizures.[504,507,509,510]

Cardiac arrest on board has an incidence of 1 per 5–10 million passenger flights. An initial shockable rhythm is present in 25–31% patients,[505,511–513] and the in-flight use of an AED can result in 33–50% survival to hospital discharge.[511,513,514] Factors contributing to a high survival rate include a witnessed event, cabin crew trained in BLS and in 73–86% of cases, travelling medical professionals also providing immediate assistance.[504,507,509] However, approximately 1000 lives are lost per year in International Airlines Transport Association (IATA) carriers. Some studies have shown that 41–59% of cardiac arrests on board are unwitnessed, occurring during sleep. There were no survivors if the initial rhythm was asystole or an idioventricular rhythm.[511,513]

Cardiopulmonary resuscitation on the airplane. In case of cardiac arrest, follow the universal algorithm for BLS (see adult basic life support and automated external defibrillation).[493] Immediately request an AED and a first aid kit from cabin crew. Physicians and trained medical providers, e.g. nurses or EMS personnel, should also ask for advanced medical equipment. According to competencies and equipment available, provide the patient with advanced treatment, assuring that there is high quality CPR ongoing, and an AED was deployed appropriately (see adult advanced life support).[168]

Consider the following modifications to CPR:

- Introduce yourself to the cabin crew and state your professional qualifications.
- In case of cardiac arrest, performance of CPR is limited in an aircraft aisle due to space restrictions. Immediately transfer the patient to a suitable location, e.g. galley or exit area. Consider an over-the-head technique of CPR if access precludes conventional CPR.[489–492]
- During CPR, attach oxygen to the facemask or self-inflating bag.
- Request immediate flight diversion to the nearest appropriate airport. In other non-critical medical emergencies, coordinate an optimal course of action with the flight crew. Considerations for flight diversion will depend on the patient's condition and on the need for immediate treatment in a hospital: e.g. acute coronary syndrome, stroke, persistently altered mental status; but also technical and operational factors.
- Ask cabin crew whether medical consultation is provided by the airline, e.g. radiotelephony or satellite communication.[506,510]
- An AED with a monitor can be safely attached to a non-arrested patient for monitoring heart rhythm, e.g. syncope, chest pain, or arrhythmia.[507,512,513]
- Concerns about legal responsibility may arise when travelling physicians are asked for help. Based on ethical duties, every physician is required to offer help within his or her scope of practice, but the legal duty is only applicable for certain countries. However, the so-called Good Samaritan Act and other regulations, depending on the origin of an aircraft, always protect healthcare providers helping on board from possible legal consequences.[504,515]
- Death on board can legally be confirmed only by a physician. If a dead person is found, or CPR has been terminated (see ethics of resuscitation and end-of-life decisions),[243] flight diversion is not recommended.

Education and equipment.

Flight crew training. Both pilots and cabin crew must receive initial and recurrent training on emergency medical event procedures and operation of emergency medical equipment, including AEDs and first aid kits, but local operational procedures may also apply.[516]

Although civil aviation is regulated by a variety of national and international laws, some studies imply that the majority of in-flight emergencies stay unreported or are reported inconsistently.[504,517] Documentation of in-flight emergencies needs standardisation in order to improve cabin crew training and pre-flight assessments of selected groups of passengers.

On-board emergency equipment. The Federal Aviation Administration (FAA) requires every US registered commercial aircraft with a maximum payload capacity of more than 7500 pounds and with at least one flight attendant to carry an AED, intravenous drugs, and advanced emergency equipment,[518] while related regulations in Europe are less precise.[519] On every commercial aircraft registered in Europe, there must be a first-aid kit that all cabin crewmembers are trained to use. Aircraft with at least 30 seats must also carry an advanced medical kit, which can be used by competent personnel, although the contents vary significantly and may be inadequate for all but the most basic of emergencies.[504,517,520] Although most large European airlines carry AEDs, some of them only do so for intercontinental flights, but some do not even provide any equipment for CPR.[517]

Based on the outcome data from survivors of cardiac arrest and in the absence of any alternative treatment for shockable rhythms on board, we strongly recommend mandatory AEDs in all commercial European aircraft, including regional and low-cost carriers.

Healthcare professionals should be aware of the on-board medical equipment and commonly encountered medical

conditions in order to provide appropriate emergency treatment on request.[505] Distribution of supportive information to travelling physicians should be encouraged, e.g. 'Doctor on Board' programme introduced by Lufthansa and Austrian Airlines in 2006.

Cardiac arrest in HEMS and air ambulances

Introduction. Air ambulance services operate either a helicopter emergency medical service (HEMS) or fixed-wing air ambulances that routinely transport critically ill patients directly to specialty centres and perform secondary transfers between hospitals. Cardiac arrest may occur in flight, both in patients being transported from an accident site and also critically ill patients being transported between hospital.[521,522] In a retrospective analysis of 12,140 aeromedical journies, the incidence of cardiac arrest in flight was low (1.1%). Fourty-three percent were medical patients and 57% were patients with traumatic injuries. In the medical cohort, the rate of ROSC was 75%.[523]

The extent of treatment available on board of an air ambulance varies and depends on medical and technical factors, e.g. crew competences and configuration, cabin size and equipment. Ideally, all interventions should be performed before flight so that the need for unplanned treatment during flight is avoided.

Pre-flight preparation. When preparing transport of a critically ill patient, ensure that all necessary monitoring is attached and functioning. Check that IV access is secured and easily accessible and that all necessary drugs and medical equipment are available during flight.

Diagnosis. In monitored patients, asystole and shockable rhythms (VF/pVT) can be immediately identified, but recognition of PEA may be challenging, especially under sedation or general anaesthesia. Unexpected loss of consciousness (in alert patients), change of ECG pattern, and loss of the pulse oximeter signal should provoke a pulse and patient check. A sudden decrease in $ETCO_2$ values in those being ventilated or loss of a waveform in those breathing spontaneously with $ETCO_2$ monitoring are also indicators of cardiac arrest.

Treatment. Cardiac arrest in the air ambulance services should be treated according to the universal ALS algorithm. Start chest compressions and ventilation immediately after confirmation of cardiac arrest, attach monitoring (if not already), and follow universal ALS algorithm.[168] If a shockable rhythm (VF/pVT) is recognised in a monitored patient and defibrillation can be accomplished rapidly, immediately give up to three-stacked shocks before starting chest compressions. In a US study, 33% of patients achieving ROSC following defibrillation did not require any chest compressions.[523]

In smaller helicopters, there may be insufficient room to perform effective resuscitation and an emergency landing may be necessary to allow better patient access.

Mechanical chest compression devices enable delivery of high quality chest compressions in the confined space of an air ambulance and their use should be considered.[248,524] If a cardiac arrest during flight is thought to be a possibility, consider fitting the patient within a mechanical chest compression device during packaging before flight.[50,525]

Cardiac arrest during sports activities

Resuscitation on the field of play

Introduction. The sudden and unexpected collapse, not associated with contact or trauma, of an athlete on the field of play is probably cardiac in origin and requires rapid recognition and effective treatment if the victim is to survive. Sudden cardiac death (SCD) is the most common cause of death of athletes during competition and training. Estimates of the incidence of SCD vary according to the methodology but recently the incidence has been quoted as 1:11,394 in basketball players, 1:21,293 for swimmers and 1:41,695 for cross-county athletes with a wide variation between male and female athletes (incidence expressed as number of athletes per year).[526] Hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC) are the most common causes in under 35 year olds whilst atherosclerotic coronary artery disease accounts for 80% of sudden cardiac arrests in over 35 year olds.[527] Congenital coronary artery abnormalities have been reported in 12–33% of athletes.[528]

Commotio cordis, the disruption of cardiac rhythm by a blow to the precordium, has a quoted incidence of 3%.[529] The striking object must strike the chest within the cardiac silhouette within a 20 ms window of the upstroke of the T-wave.[530] The overall survival rate from commotio cordis is reported to have improved with survival rates of up to 58% reported in recent years.[531]

Whatever the cause the sudden collapse of an athlete there should be an immediate response from the officials or medical team. The standard resuscitation procedures must be followed but with certain additional considerations as described below.

Access. The medical team should gain immediate access to the field of play. It is important that the medical team do observe access rules to the field of play but it would be hoped that the field of play officials will recognise or be alerted to the collapsed athlete and halt play so that it is safe to approach the competitor.

Where there is no medical team, during informal competition or in practice it is the responsibility of the referee, the coach or of the athletes' colleagues to recognise the collapse and to initiate a call for help and resuscitation.

Calling for help. The call for help is essential to providing the collapsed athlete with the best chance of survival. It is essential that sports officials, coaches and sports organisers have a plan for medical collapse or trauma. In its simplest form this could include ensuring the availability of a mobile telephone and knowledge of the site/address of the sport arena (field of play, club house) to provide best access for the ambulance. It would be hoped that more officials and coaches will be trained in BLS and AED usage.

Resuscitation. If the athlete is unresponsive and not breathing normally, commence BLS. If available attach an AED and follow the instructions; if this is SCD then the rhythm will probably be ventricular fibrillation and will respond to defibrillation.

The sports field of play is often an open arena and in major competition may be on view to many thousands of spectators and a television audience. Although treatment must not be compromised moving the collapsed athlete to a quieter and more private site for continued treatment may be considered. Where there is not an immediate response to treatment and there is an organised medical team, this move could be accomplished after three defibrillation attempts on the rationale of providing the highest efficacy of defibrillation in the first three shocks. The move, if decided, should be agreed and may need to be accomplished in stages to allow for near continuous chest compressions. Where there is no medical team or a defibrillator is not immediately available then BLS must continue until advanced care arrives.

If the athlete responds to resuscitation then they must be transported immediately to the nearest cardiac centre for further evaluation and treatment. As there is a possibility of the rhythm reverting this transportation must be under the supervision of a healthcare professional who is equipped and capable of administering resuscitation and further defibrillation.

Prevention. In an effort to predict and prevent SCD, the International Olympic Committee Medical Commission (IOC Medical Commission 2014) and many International Sport Federations have recommended cardiac screening for athletes. However, there is much debate about the effectiveness of the techniques being used and the population that should be screened.[532]

Water rescue and drowning

Introduction

Drowning is a common cause of accidental death.[533] Prompt and effective actions by bystanders, trained rescuers and emergency medical personnel can make the difference between life and death.[534–536] These guidelines provide advice about the initial rescue and resuscitation of victims involved in drowning incidents. They are intended for healthcare professionals and certain groups of lay responders that have a special responsibility in the care of the drowning victim, e.g. lifeguards, lifeboat crews, swimming pool instructors and water rescue teams.

Epidemiology

The World Health Organization (WHO) reports that every hour of every day, more than 40 people lose their lives to drowning; 372,000 deaths each year.[537] The WHO acknowledges that the true number of drownings worldwide is much higher. More than 90% of these deaths occur in low and middle-income countries. The incidence of drowning varies between countries, with eastern Europe having the highest rates in Europe.[533] Although risk groups vary between countries, in general males are much more likely to drown than females. Most accidental drownings are children who are unable to swim. In countries where aquatic leisure in combination with alcohol and drug use is common, young adults are a second group of risk.[538,539] Many countries also report a slight increase of drowning in the age groups above 70 years related to accidents and physical activities close to water. Drowning is commonest in inland waters (e.g. lakes, rivers) and during summer months.[538–540]

Definitions, classifications and reporting

The International Liaison Committee on Resuscitation (ILCOR) defines drowning as a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium. Implicit in this definition is that a liquid/air interface is present at the entrance of the victim's airway, preventing the victim from breathing air. The victim may live or die after this process, but whatever the outcome, he or she has been involved in a drowning incident.[541] Submersion occurs when the face is underwater or covered in water. Asphyxia and cardiac arrest occurs within a matter of minutes of submersion. Immersion, by contrast, is when the head remains above water, in most cases by means of the support of a lifejacket. In most situations of immersion, the victim remains immersed with an open airway and becomes hypothermic, although aspiration of water may occur if water splashes over the face or if the victim becomes unconscious with their face in the water. The difference between submersion and immersion is important in understanding the difference in epidemiology, pathophysiology, clinical course and prognostic parameters between the two drowning processes.

If the casualty is rescued, the process of drowning is interrupted, which is termed a non-fatal drowning. If the person dies at any time as a result of drowning, the term is fatal drowning. Avoid terms such as dry and wet drowning, active and passive drowning, silent drowning, secondary drowning and near-drowning.[541] To improve consistency in information between studies use the Utstein-style registration template for drowning when reporting outcomes from drowning incidents.[542]

Pathophysiology

Detailed summaries of the pathophysiology of drowning have been published.[536,541,543,544] In brief, following submersion, the victim initially breath holds by reflex. During this time the victim frequently swallows water. As breath holding continues, hypoxia and hypercapnia develop. A reflex laryngospasm may temporarily prevent the entrance of water into the lungs. Eventually these reflexes abate and the victim aspirates water. The key feature to note in the pathophysiology of drowning is that bradycardia as a consequence of hypoxia occurs before sustaining a cardiac arrest. Correction of hypoxaemia by ventilation-only resuscitation is critical and in itself may lead to return of spontaneous ventilation or circulation (ROSC) in some cases, probably because the presence of a circulation had not been detected.[545–549]

Drowning chain of survival

The Drowning Chain of Survival describes five critical links for improving survival from drowning (Fig. 4.5).[535] The first two links cover prevention of drowning and recognition of distress.[550,551] This chapter provides guidance on removal from water, initial and post resuscitation care.

Water rescue

Bystander response. Bystanders play a critical role in initial attempts at rescue and resuscitation.[534,548,552–555] At the same time, bystanders who attempt a rescue have died during the rescue attempt, mostly when drowning occurs in surf or fast moving water.[556] Whenever possible, bystanders should attempt to save the drowning victim without entry into the water. Talking to the victim, reaching with a rescue aid (e.g. stick or clothing), or throwing a rope or buoyant rescue aid may be effective if the victim is close to dry land. If entry into the water is essential, take a buoyant rescue aid, flotation device or boat.[535] It is safer to enter the water with two rescuers than alone. Never dive head first in the water when attempting a rescue. You may lose visual contact with the victim and run the risk of a spinal injury.

Trained rescuer response. Trained rescuers are often professionals who work in teams with specialist equipment to assist with search and rescue. Where the rescue takes time, the teams often seek guidance on the likelihood of survival. For this reason, ILCOR reviewed specific prognostic indicators and noted that submersion durations of less than 10 min were associated with a very high chance of favourable outcome; submersion durations longer than 25 min were associated with a low chance of favourable outcomes.[557] Age, emergency medical services (EMS) response time, fresh or salt water, water temperature, and witness status were not useful for predicting survival. Submersion in ice-cold water may prolong the window of survival and justify extended search and rescue activities.[558–560]

In-water resuscitation. Trained individuals may undertake in water ventilation ideally with the support of a buoyant rescue aid.[545,561,562] If a rescuer, in general a surf-lifeguard, finds a non-responding drowning victim in deep open water, the rescuer may start ventilation when trained to do so before moving the victim to dry land or rescue craft. Some victims may respond to this. If not responding, and depending on the local situation, such as sea conditions, distance to shore, availability of rescue boat or rescue helicopter, the rescuer should then decide to bring the victim to shore as quickly as possible without further ventilation while rescue-swimming with the victim or continue on the spot with in-water ventilation until support by crews of a rescue boat or rescue helicopter arrives to take over the resuscitation. One study suggests that the second option has a higher survival rate.[545]

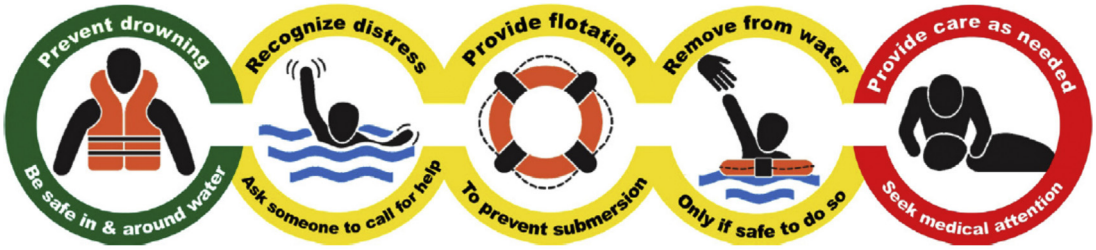

Fig. 4.5. Drowning chain of survival.[535]
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Removal from water. Remove the victim from the water promptly. The chances of a drowning victim sustaining a spinal injury are very low.[563] Spinal precautions are unnecessary unless there is a history of diving in shallow water, or signs of severe injury after water-slide use, waterskiing, kite-surfing, or watercraft racing. If the victim is pulseless and apnoeic, remove them from the water as quickly as possible while attempting to limit neck flexion and extension. Hypovolaemia after prolonged immersion may cause a circum-rescue collapse/arrest. Keep the victim in a horizontal position during and after retrieval from the water.

Initial resuscitation once retrieved from water

Follow the standard BLS sequence, initially by checking for response, opening the airway and checking for signs of life. The drowning victim rescued from the water within a few minutes of submersion is likely to exhibit abnormal (agonal) breathing. Do not confuse this with normal breathing.

Rescue breaths/ventilations. The BLS sequence in drowning (Fig. 4.6) reflects the critical importance of rapid alleviation of hypoxia. Inflation should take about 1 s and be sufficient to see the chest rise. However it often takes more time to insuflate air than under normal conditions due to reduced compliance and high airway resistance. The higher inflation pressure may precipitate inflation of the stomach with regurgitation and also reduce cardiac output. Expert opinion suggests that cricoid pressure applied by trained and skilled personnel in casualties without a secured airway may reduce gastric inflation and enhance ventilation in drowning.

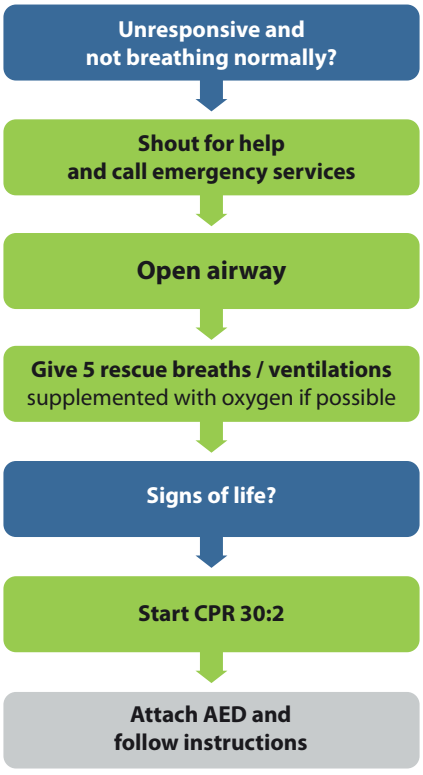


Fig. 4.6. Drowning treatment algorithm for rescuers with a duty to respond.

Chest compressions. If the victim has not responded to initial ventilations, they should be placed on a firm surface before starting chest compressions, as compressions are ineffective in the water.[564,565] Provide CPR in a ratio of 30 compressions to 2 ventilations. Most drowning victims will have sustained cardiac arrest secondary to hypoxia. In these patients, compression-only CPR is likely to be ineffective and should be avoided.

If sufficient rescuers are present, the person performing the aquatic rescue should be relieved of continuing CPR once on land as they are likely to be fatigued, which may impair the quality of CPR.[566,567]

Automated external defibrillation. Defer using an AED until after CPR has commenced. Dry the victim's chest, attach the AED pads and turn the AED on. Deliver shocks according to the AED prompts.

Fluid in the airway. In some situations, massive amounts of foam caused by admixing moving air with water are seen coming out of the mouth of the victim. Do not try and attempt to remove the foam as it will keep coming. Continue rescue breaths/ventilation until an ALS provider arrives and is able to intubate the victim. Regurgitation of stomach contents and swallowed water is common during resuscitation from drowning.[568] If this prevents ventilation completely, turn the victim on their side and remove the regurgitated material using directed suction if possible.

Modifications to advanced life support

Airway and breathing. During the initial assessment of the spontaneously breathing drowning victim, give high-flow oxygen (10–15 L min^{-1}), ideally through an oxygen mask with reservoir bag.[127] For victims who fail to respond to these initial measures, who have a reduced level of consciousness or are in cardiac arrest, consider early tracheal intubation and controlled ventilation by skilled personnel. Reduced pulmonary compliance requiring high inflation pressures may limit the use of a supraglottic airway device.[569] Take care to ensure optimal preoxygenation before attempting tracheal intubation. Pulmonary oedema fluid may pour from the airway and may need continuous suctioning to enable a view of the larynx. After the position of the tracheal tube is confirmed, titrate the inspired oxygen concentration to achieve a SpO_2 of 94–98%.[127] Pulse oximetry can give spurious readings following rescue from drowning.[570] Confirm adequate oxygenation and ventilation with arterial blood gases once available. Set positive end expiratory pressure (PEEP) to at least 5–10 cm H_2O. However, PEEP

levels of 15–20 cm H_2O may be required if the patient is severely hypoxaemic.[571] Decompress the stomach with a gastric tube.

Circulation and defibrillation. Palpation of the pulse as the sole indicator of the presence or absence of cardiac arrest is not always reliable. As soon as possible, use information from monitoring modalities such as the ECG trace, $ETCO_2$ and echocardiography to confirm the diagnosis of cardiac arrest.

If the victim is in cardiac arrest, follow standard ALS protocols. If the victim is hypothermic, modify the approach in accordance with the guidance for treatment of hypothermia (see hypo-/hyperthermia).

After prolonged immersion, most victims will have become hypovolaemic due to the cessation of the hydrostatic pressure of water on the body. Give rapid IV fluid to correct hypovolaemia. This should commence out-of-hospital if transfer time is prolonged.

Discontinuing resuscitation efforts

Making a decision to discontinue resuscitation efforts on a victim of drowning is notoriously difficult. No single factor can accurately predict good or poor survival with certainty. Frequently, decisions made in the field later prove to have been incorrect.[572] Continue resuscitation unless there is clear evidence that such attempts are futile (e.g. massive traumatic injuries, rigour mortis, putrefaction, etc.), or timely evacuation to a medical facility is not possible. Neurologically intact survival has been reported in several victims submerged for longer than 25 min, however these rare case reports almost invariably occur in children submerged in ice-cold water, when immersion hypothermia has preceeded hypoxia or in submersion of car occupants.[558,559,573,574] A retrospective study of 160 children who drowned in the Netherlands found that outcomes were extremely poor if ALS took longer than 30 min to achieve ROSC even if hypothermia was present.[560]

Post resuscitation care

Salt versus fresh water. Small differences in electrolyte disturbance are rarely of any clinical relevance and do not usually require treatment.[575,576]

Lung injury. The predominant pathophysiological process in the lungs is driven by surfactant wash-out and dysfunction, alveolar collapse, atelectasis, and intrapulmonary shunting. The severity of lung injury varies from a mild self-limiting illness to refractory hypoxaemia. Many victims of drowning are at risk of developing acute respiratory distress syndrome (ARDS).[577] Although there are no randomised controlled trials undertaken specifically in this population of patients, it seems reasonable to include strategies such as protective ventilation that have been shown to improve survival in patients with ARDS.[578,579] Extracorporeal membrane oxygenation (ECMO) has been used for those in refractory cardiac arrest, those with refactory hypoxaemia and in selected cases of submersion in ice cold water, although success rates remain low.[580–583]Pneumonia is common after drowning. Prophylactic antibiotics have not been shown to be of benefit[584] but they may be considered after submersion in grossly contaminated water such as sewage. Give broad-spectrum antibiotics if signs of infection develop subsequently.[585–587]

Neurological outcome. Neurological outcome, notably severe permanent neurological damage, is primarily determined by the duration of hypoxia. Attempts have been made to improve neurological outcome following drowning with the use of barbiturates, intracranial pressure (ICP) monitoring, and steroids. None of these interventions has altered outcome.[588]

Wilderness and environmental emergencies

Difficult terrain and remote areas

Geographical and meteorological considerations. Compared to urban areas some terrains will be more difficult to access and are remote from organised medical care. Exposed and steep terrain may render extrication dangerous and challenging. The chances of a good outcome from cardiac arrest may be reduced because of delayed access and prolonged transport. Furthermore, some environments are harsher than urban areas (e.g. cold, windy, wet, very bright due to light-reflection on ice and snow). Human and material resources may be greatly restricted.[589,590]

Compared with the partial pressure of oxygen at sea level (PO_2 about 21 kPa/159 mmHg), the PO_2 at high (>1500 m above sea level), very high (3500–5500 m) and extreme altitude (>5500 m) will be progressively lower, constraining the physical activity of rescuers. There is a physiological limit to acclimatisation (e.g. short term–hyperventilation and increased cardiac output; long-term–haemoglobin increase). The highest permanent settlement is at 5100 m (PO_2 about 11 kPa/84 mmHg). Above 7500 m the risk of lethal acute altitude illness is very high.

There are no epidemiological data on the causes of cardiac arrest at high altitude. However, it is conceivable that primary cardiac arrest is the major (60–70%) cause of sudden cardiac arrest. Thus, public access defibrillator (PAD) programmes in populated areas at altitude seem reasonable. For instance, public access defibrillators (PADs) should be placed in popular ski areas, busy mountain huts and restaurants, at mass-participation events, and in remote but often-visited locations that are not medically covered.[591] In areas where physicians are regularly involved in mountain rescue operations, the provided on-site treatment is more in line with resuscitation guidelines.[592]

Decision making. Continuous monitoring and treatment may be difficult during transport because the patient will be insulated from the harsh environment within a rescue bag, being well wrapped and secured on a stretcher. During transport, CPR may be limited in quality and nearly impossible in some circumstances (e.g. while carrying the patient, during abseiling or winching). In dangerous and difficult terrain where continuous CPR is impossible, delayed and intermittent CPR has been proposed for hypothermic patients.[45] Mechanical resuscitation devices may help to improve CPR quality during difficult extrication and prolonged transport.[50]

Transportation

Effective and safe immobilisation and splinting will reduce morbidity and mortality.[593] Whenever possible, transport the patient with air rescue.[593,594] The organisation of the helicopter emergency medical service (HEMS) affects the outcome.[595–597]

High altitude illness

Given the increasing popularity of travel at altitude, an increasing number of tourists at altitude have cardiovascular and metabolic risk factors for cardiac arrest. The pO_2 falls with increasing altitude and this oxygen deficiency may lead to acute manifestations of mountain sickness.

Persons travelling to an altitude of >3500 m are at risk of developing:

- acute mountain sickness (AMS) with headache, nausea, fatigue and dizziness;
- high altitude pulmonary oedema (HAPO) with severe dyspneoa and cyanosis;
- high altitude cerebral oedema (HACO) with gate disorder, disorientation and confusion.

Risk factors include a fast rate of ascent and a previous history of mountain sickness. If not treated promptly, HAPO and HACO may progress rapidly to loss of consciousness, severe respiratory distress, circulatory instability and cardiac arrest. The most important actions are immediate descent or transport to lower levels of altitude, administration of oxygen (2–6 L min^{-1}, target > 90% SpO_2), treatment in a portable hyperbaric chamber, in cases of HACO administration of dexamethasone 4–8 mg every 8 h, and in cases of HAPO, nifedipine 30 mg every 12 h.

Resuscitation at high altitude does not differ from standard CPR. With the lower pO_2, CPR is more exhausting for the rescuer than at sea level, and the average number of effective chest compressions may decrease within the first minute.[598–600] Use mechanical chest compression devices whenever possible.

Commonly, no physician will be present to give guidance to nurses or paramedics on when to stop CPR. Guidelines have therefore been proposed for these situations.[46]

CPR may be withheld or terminated in a patient with absent vital signs when:

- the risk is unacceptable to the rescuer
- the rescuer is exhausted
- extreme environments prevent CPR
- any of the following apply:
  - decapitation
  - truncal transection
  - whole body incinerated
  - decomposed
  - frozen solid
  - avalanche victim in asystole with obstructed airway and burial time > 60 min (see avalanche burial below).

CPR may be also terminated when all of the following criteria apply:

- unwitnessed loss of vital signs;
- no ROSC during 20 min of CPR;
- no shock advised at any time by AED or only asystole on ECG;
- no hypothermia or other reversible causes warranting extended CPR.

In situations where transport is not possible, and correction of reversible causes is not possible, further resuscitation is futile and CPR should be terminated. These recommendations should be interpreted in the context of local conditions and legislation.

Avalanche burial

Introduction. In Europe and North America together, there are about 150 snow avalanche deaths each year. Most are sports-related and involve skiers, snowboarders and snowmobilers. Fatalities are mainly due to asphyxia, sometimes associated with trauma and hypothermia. Prognostic factors are severity of injury, duration of complete burial, airway patency, core temperature and serum potassium.[601] Completely buried avalanche victims die from asphyxia within 35 min if the airway is obstructed. The average cooling rate is 3 °C h^{-1},[602] ranging from 0.6 °C h^{-1} to 9 °C h^{-1}[603,604]; moderate to severe hypothermia may become important after 60 min of burial if the airway is patent. The highest recorded potassium in an avalanche victim who was successfully resuscitated is 6.4 mmol L^{-1}.[601,605–607] The survival rate of avalanche victims presenting with cardiac arrest ranges from 7% to 17%.[605,606] Survival patterns differ across countries due to terrain, climate and prehospital medical care.[56,608–610]

Decision-making on scene. Avalanche victims are not likely to survive when they are:

- buried > 60 min (or if the initial core temperature is <30 °C) and in cardiac arrest with an obstructed airway on extrication;
- buried and in cardiac arrest on extrication with an initial serum potassium > 8 mmol L^{-1}.

Full resuscitative measures, including extracorporeal rewarming, are indicated for all other avalanche victims without evidence of an unsurvivable injury.

Avalanches occur in areas that are difficult to access by rescuers in a timely manner, and burials frequently involve multiple victims. The decision to initiate full resuscitative measures should be determined by the number of victims and the resources available, and should be informed by the likelihood of survival.[601] As adherence to present guidelines is poor,[611,612] the use of a standardised checklist is recommended.[613]

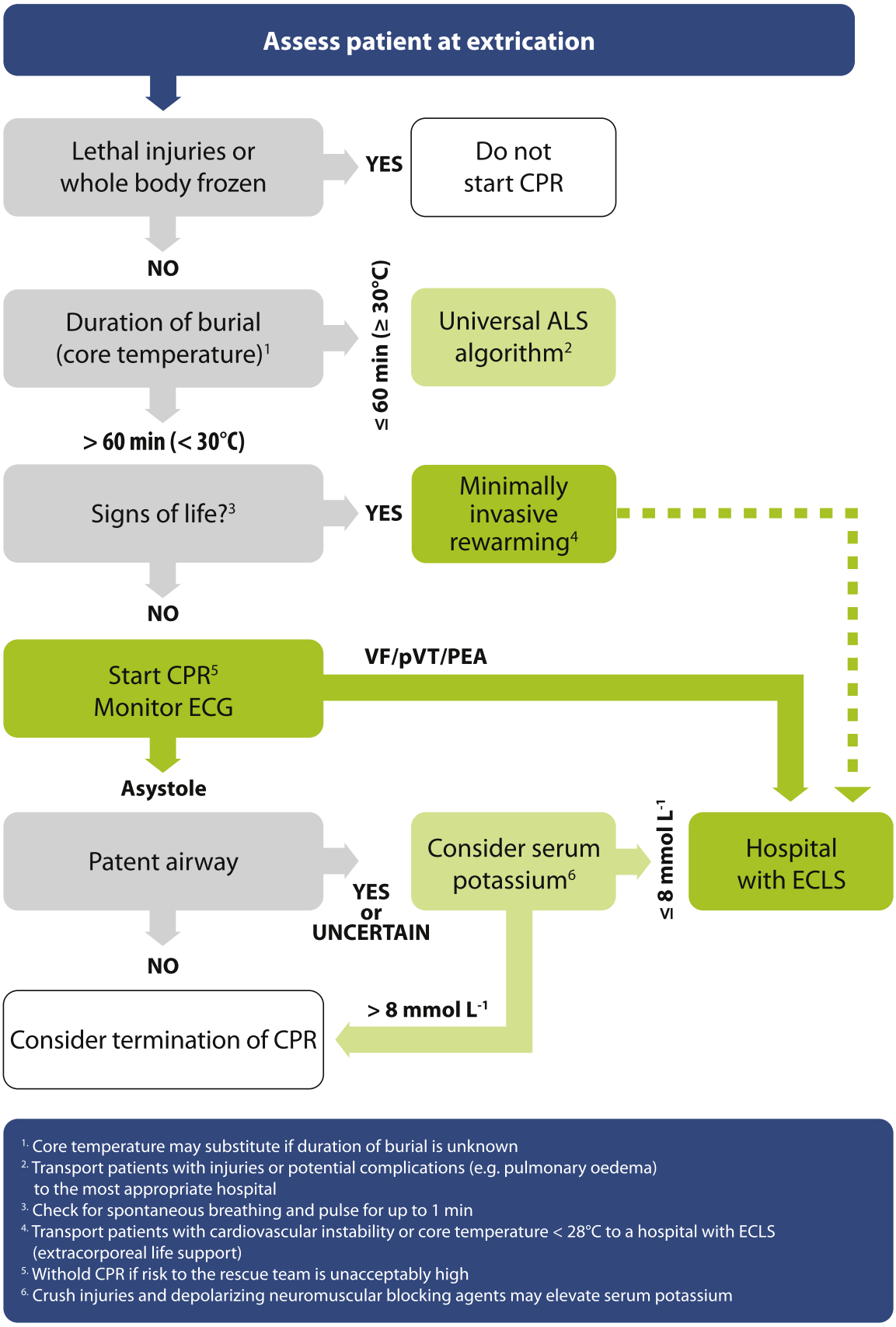
Management of completely buried avalanche victims. The algorithm for the management of buried avalanche victims is shown in Fig. 4.7:

- In all cases, extricate the body gently and use spinal precautions.
- Consider withholding resuscitation at the scene if it increases risk to the rescue team or if the victim is lethally injured or completely frozen.
- Determine the duration of burial. If unknown, core temperature may substitute for decision-making.
- If the duration of burial is ≤60 min (or initial core temperature is ≥30 °C) and cardiac arrest is confirmed, follow standard ALS guidelines (see adult advanced life support).[168] During CPR, measure core temperature, monitor ECG, give oxygen and apply insulation and heat packs to the trunk. Give drugs and fluids only if IV or IO access can be established within a few minutes. Resuscitation may be terminated in a normothermic asystolic patient if ALS is not successful after 20 min, in an absence of reversible cause (see ethics of resuscitation and end-of-life decisions).[243]
- Transport survivors with concern of respiratory (e.g. pulmonary oedema) or other-system critical illness or injury to the most appropriate medical centre. Provide specific trauma care as indicated. The admitting hospital must be capable of advanced active external or core internal rewarming.
- If the duration of burial is > 60 min (or initial core temperature is < 30 °C) and cardiac arrest is confirmed, start CPR and attach monitor. If there is any electrical activity or a patent airway in an asystolic patient, continue CPR. Defibrillation beyond three attempts may be delayed until core temperature ≥30 °C.
- Transport all patients who present with cardiovascular instability (i.e. ventricular arrhythmias, systolic blood pressure < 90 mmHg) or core temperature < 28 °C to an ECLS rewarming centre. Follow regional hypothermia protocols if needed.
- If direct transport to an ECLS rewarming centre is not possible in a timely manner, e.g. by HEMS, check potassium level at the nearest hospital. If potassium exceeds > 8 mmol L^{-1}, consider terminating resuscitation (after excluding crush injuries and considering if depolarizing muscle relaxants were used).

Lightning strike and electrical injuries

Introduction. Electrical injury is a relatively infrequent but potentially devastating multisystem injury with high morbidity and mortality, causing 0.54 deaths per 100,000 people each year. Most electrical injuries occur indoors. In adults, electrical injuries are common in the workplace and are generally associated 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 United States and Canada).[614] Electrocution from lightning strikes is rare, but worldwide it causes 1000 deaths each year.[615]

Electric shock injuries are caused by the direct effects of current on cell membranes and on vascular smooth muscle. The thermal



1. Core temperature may substitute if duration of burial is unknown
2. Transport patients with injuries or potential complications (e.g. pulmonary oedema) to the most appropriate hospital
3. Check for spontaneous breathing and pulse for up to 1 min
4. Transport patients with cardiovascular instability or core temperature < 28°C to a hospital with ECLS (extracorporeal life support)
5. Withold CPR if risk to the rescue team is unacceptably high
6. Crush injuries and depolarizing neuromuscular blocking agents may elevate serum potassium

Fig. 4.7. Avalanche accident algorithm. Management of completely buried victims. (ECLS, extracorporeal life support).

energy associated with high-voltage electrocution will also cause burns. Factors influencing the severity of electrical injury include whether the current is alternating (AC) or direct (DC), voltage, magnitude of energy delivered, resistance to current flow, pathway of current through the patient, and the area and duration of contact. Skin resistance is decreased by moisture, which increases the likelihood of injury. Electric current follows the path of least resistance; conductive neurovascular bundles within limbs are particularly prone to damage. Contact with AC may cause tetanic contraction of skeletal muscle, which may prevent release from the source of electricity. Myocardial or respiratory failure may cause immediate death:

- Respiratory arrest may be caused by paralysis of the central respiratory control system or the respiratory muscles.
- Current may precipitate ventricular fibrillation (VF) if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon).[616] Electrical current may also cause myocardial ischaemia because of coronary artery spasm. Asystole may be primary, or secondary to asphyxia following respiratory arrest.

Current that traverses the myocardium is more likely to be fatal. A transthoracic (hand-to-hand) pathway is more likely to be fatal than a vertical (hand-to-foot) or straddle (foot-to-foot) pathway. There may be extensive tissue destruction along the current pathway.

Associated injuries are common. Blast (hyperbaric) injuries, injuries from being thrown from the point of contact and tetanic contraction causing limb fractures have all been reported.

Lightning strike. Lightning strikes deliver as much as 300 kV over a few milliseconds. Most of the current from a lightning strike passes over the surface of the body in a process called 'external flashover'. Both industrial shocks and lightning strikes cause deep burns at the point of contact. For industrial shocks the points of contact are usually on the upper limbs, hands and wrists, whereas for lightning they are mostly on the head, neck and shoulders. Injury may also occur indirectly through ground current or current splashing from a tree or other object that is hit by lightning.[617] Explosive force may cause blunt trauma.[618] The pattern and severity of injury from a lightning strike varies considerably, even among affected individuals from a single group.[619–621] As with industrial and domestic electric shock, death is caused by cardiac[620–624] or respiratory arrest.[617,625] In those who survive the initial shock, extensive catecholamine release or autonomic stimulation may occur, causing hypertension, tachycardia, non-specific ECG changes (including prolongation of the QT interval and transient T-wave inversion), and myocardial necrosis. Creatine kinase may be released from myocardial and skeletal muscle. Lightning can also cause central and peripheral nerve damage; brain haemorrhage and oedema, and peripheral nerve injury are common. Mortality from lightning injuries is as high as 30%, with up to 70% of survivors sustaining significant morbidity.[626,627]

Diagnosis. The circumstances surrounding the incident are not always known. Unique pattern of skin lesions called feathering or Lichtenberg figure is a pathognomonic symptom that is seen only in patients struck by lightning.[628] Unconscious patients with linear or punctuate burns (feathering) should be treated as victims of lightning strike.[617]

Safety measures. Ensure that any power source is switched off and do not approach the casualty until it is safe. High-voltage (above domestic mains) electricity can arc and conduct through the ground for up to a few metres around the casualty. It is safe to approach and handle casualties after lightning strike, although it would be wise to move to a safer environment, particularly if lightning has been seen within 30 min.[617]

Resuscitation. Patients struck by lightning are most likely to die if they sustain immediate cardiac or respiratory arrest and are not treated rapidly. When multiple victims are struck simultaneously by lightning, rescuers should give highest priority to patients in respiratory or cardiac arrest. Victims with respiratory arrest may require only ventilation to avoid secondary hypoxic cardiac arrest. Resuscitative attempts may have higher success rates in lightning victims than in patients with cardiac arrest from other causes, and efforts may be effective even when the interval before the resuscitative attempt is prolonged.[625] Dilated or non-reactive pupils should never be used as a prognostic sign, particularly in patients suffering a lightning strike.[617]

Start standard BLS and ALS without delay:

- Airway management may be difficult if there are electrical burns around the face and neck. Early tracheal intubation is needed in these cases, as extensive soft-tissue oedema may develop causing airway obstruction. Head and spine trauma can occur after electrocution. Immobilise the spine until evaluation can be performed.
- Muscular paralysis, especially after high voltage, may persist for several hours[627]; 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.[624]
- Consider early surgical intervention in patients with severe thermal injuries.
- Maintain spinal immobilisation if there is a likelihood of head or neck trauma.[629,630]
- Conduct a thorough secondary survey to exclude traumatic injuries caused by tetanic muscular contraction or by the person being thrown.[629,631]
- 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.
- Although rare, consider abdominal visceral injuries caused directly by electrical damage.

There are conflicting reports on the vulnerability of the fetus to electric shock. The clinical spectrum of electrical injury ranges from a transient unpleasant sensation for the mother with no effect on her fetus, to placental abruption, fetal burn or intrauterine fetal death either immediately or a few days later. Several factors, such as the magnitude of the current and the duration of contact, are thought to affect outcome.[632]

Further treatment and prognosis. Immediate resuscitation of young victims in cardiac arrest from electrocution can result in long-term survival. Successful resuscitation has been reported after prolonged life support.

All those who survive electrical injury should be monitored in hospital if they have a history of cardiorespiratory problems or have had:

- loss of consciousness
- cardiac arrest

- electrocardiographic abnormalities
- soft tissue damage and burns.

Severe burns (thermal or electrical), myocardial necrosis, the extent of central nervous system injury, and secondary multisystem organ failure determine the morbidity and long-term prognosis. Bone marrow embolism has also been reported in some cases.[633] There is no specific therapy for electrical injury, and the management is supportive. Prevention remains the best way to minimise the prevalence and severity of electrical injury.

Mass casualty incidents

Introduction

Mass casualty incidents (MCIs), characterised by greater demand for medical care than available resources, are rare events. Among the 19.8 million yearly emergency medical services (EMS) activations in the United States, 0.3% had an MCI code, but incidence of real disasters is much lower.[634] The International Federation of Red Cross and Red Crescent Societies (IFRC) reports about 90 disasters in Europe and 650 events worldwide annually.[635] The MCI or disaster can be caused by variety of chemical, biological, radiological or nuclear (CBRN) incidents, but traumatic incidents (e.g. traffic accidents, acts of crime, or natural and industrial disasters) play a leading role in developed countries.[636] Initial triage of casualties enables identification of patient care priorities. Unlike normal circumstances, CPR is not usually initiated in MCI, in order to avoid delaying potentially effective treatment for salvageable victims. This critical decision depends on available resources in relation to the number of casualties.

Triage and decision-making on scene

Safety.

- Safety at scene is paramount. Those first on scene must identify the actual and potential hazards and appropriate assistance must be requested immediately. The presence of multiple unconscious victims should always alert rescuers to the possibility of a CBRN incident. Unexpected danger may be present at crime scenes, or places polluted by noxious substances e.g. carbon monoxide, industrial cyanides or other chemicals. During sarin attacks in Japan, 10% of 1363 EMS technicians developed poisoning, mostly from primary victims in poorly ventilated ambulances.[637]
- Use adequate protection measures and consider potential risks before approaching casualties. Be aware that wearing some personnel protective equipment may adversely affect performance of treatment interventions and limit the care that can be given in contaminated zones. Simulation studies have shown reduced success rate of advanced airway techniques, prolonged time for securing IV and IO access, and difficulties with drug preparation.[638–640]

Triage.

- Use a triage system to prioritise treatment, e.g. START (Simple Triage and Rapid Transport), *Newport Beach Fire Department, CA, USA*,[641] SALT (Sort-Assess-Lifesaving Interventions-Treat/Transport).[642,643] Advanced prehospital teams involved in the initial scene triage must avoid overtriage. Repeated triage (re-triage) is needed on entering the hospital and responsible personnel at all stages of emergency care must be familiar with the triage system used.
- If the START triage sieve is used, everyone able to walk is directed to clear the scene, and respiratory status of non-walking patients is assessed. If the casualty does not breathe, open the airway using basic manoeuvers (head tilt and chin lift, or jaw thrust). Look, listen and feel for no more than 10 s. A patient who does not begin breathing is triaged as dead. If an unresponsive victim is breathing normally, turn them into the recovery position and label as immediate (highest priority) for treatment. Further assessment of casualties, e.g. respiratory rate, capillary refill time, etc., and depends on individual triage protocols.
- The decision to use an MCI triage sieve, and withhold CPR to those with imminent death (including victims without signs of life), is the responsibility of a medical commander who is usually the most experienced EMS clinician on scene.
- Triage inaccuracy may have fatal consequences in patients with survivable injuries. Healthcare professionals must be regularly trained to use the triage protocols during simulations and live exercises.[644] Modern technologies such as educational video games enhance learning and improve subsequent performance when compared to traditional educational methods, e.g. card-sort exercise.[645] Training allows fast and correct recognition of those requesting life-saving procedures, and reduces the risk of inappropriate care given to futile cases.
- Consider assigning a higher triage risk level to the elderly and to survivors of high-energy trauma in order to reduce preventable deaths. After an aeroplane crash in the Netherlands, 9% of the minor injuries (lowest priority), and 17% of all walking casualties were undertriaged while suffering serious injuries.[646] In the National Trauma Database (NTDB), patients in all triage levels were compared to mortality outcomes. There were 322,162 subjects assigned to the lowest priority triage level of which 2046 died before hospital discharge. Age was the primary predictor of undertriage.[641]
- Perform life-saving interventions in patients triaged as immediate (highest priority) to prevent cardiac arrest: control major haemorrhage, open airway using basic techniques, perform chest decompression for tension pneumothorax, use antidotes, and consider initial rescue breaths in a non-breathing child.[642]
- In children, use of a special triage tape or a paediatric-specific MCI triage system (e.g. JumpSTART, *Team Life Support, Inc., FL, USA*, http://www.jumpstarttriage.com) or a universal SALT system.[647] If it is not available, use any triage system for adults.

C – SPECIAL PATIENTS

Cardiac arrest associated with concomitant diseases

Asthma

Introduction. Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma.[648] The worldwide prevalence of asthma symptoms ranges from 1 to 18% of the population with a high prevalence in some European countries (United Kingdom, Scandinavia and Netherlands) and in Australia.[648,649] In recent years, the prevalence of asthma and its related morbidity and mortality appears to have plateaued and may even have decreased in some countries, especially in children and adolescents.[650–653] The World Health Organization (WHO) has estimated that 15 million disability-adjusted life years (DALYs) are lost annually from asthma, representing 1% of the global disease burden. Annual worldwide deaths from asthma have been estimated at 250,000. The death rate does not appear to be correlated with asthma prevalence.[648] National and international guidance for the management of severe asthma already exists.[654] This guidance focuses on the treatment of patients with near-fatal asthma and subsequent cardiac arrest.

Patients at risk of asthma-related cardiac arrest. The risk of near-fatal asthma attacks is not necessarily related to asthma severity.[655] Patients most at risk include those with:

- a history of near-fatal asthma requiring intubation and mechanical ventilation;[656]
- hospitalisation or emergency care for asthma in the past year;[657]
- low or no use of inhaled corticosteroids;[658]
- increasing use and dependence of beta-2 agonists;[659]
- anxiety, depressive disorders and/or poor compliance with therapy;[660,661]
- food allergy in a patient with asthma.[662]

A national confidential enquiry carried out in the UK in 2014 showed that the majority of asthma-related deaths occurred before admission to hospital.[663] Compared with younger adults, older adults have higher rates of near-fatal asthma-related events and higher comorbidity-adjusted risk of mortality.[664]

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

- severe bronchospasm and mucous plugging leading to asphyxia (this condition causes the vast majority of asthma-related deaths);
- cardiac arrhythmias caused by hypoxia, which is the commonest cause of asthma-related arrhythmia.[665] Arrhythmias can also be caused by stimulant drugs (e.g. beta-adrenergic agonists, aminophylline) or electrolyte abnormalities;
- dynamic hyperinflation, i.e. auto positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure;
- tension pneumothorax (often bilateral).

Diagnosis. Wheezing is a common physical finding, but severity does not correlate with the degree of airway obstruction. The absence of wheezing may indicate critical airway obstruction, whereas increased wheezing may indicate a positive response to bronchodilator therapy. SpO_2 may not reflect progressive alveolar hypoventilation, particularly if oxygen is being given. SpO_2 may initially decrease during therapy because beta-agonists cause both bronchodilation and vasodilation, initially increasing intrapulmonary shunting.

Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis,[101] pneumonia, foreign bodies, pulmonary embolism, and subglottic mass.[666]

The severity of an asthma attack is defined in Table 4.3.

Table 4.3
The severity of asthma (PEF, peak expiratory flow)

Near-fatal asthma	Raised $PaCO_2$ and/or mechanical ventilation with raised inflation pressures	
Life-threatening asthma	Any one of the following in a patient with severe asthma:	
	Clinical signs	Measurements
	Altered conscious level	PEF < 33% best or predicted
	Exhaustion	SpO_2 < 92%
	Arrhythmia	PaO_2 < 8 kPa (60 mmHg)
	Hypotension	'Normal' $PaCO_2$ (4.6–6.0 kPa; 35-45 mmHg)
	Cyanosis	
	Silent chest	
	Poor expiratory effort	
Acute severe asthma	Any one of: - PEF 33–50% best or predicted - respiratory rate ≥ 25 min^{-1} - heart rate ≥ 110 min^{-1} - inability to complete sentences in one breath	

Prevention of cardiac arrest. A patient with severe asthma requires immediate and aggressive medical management to prevent deterioration. Base the assessment and treatment on a systematic ABCDE approach. Patients with SpO_2 < 92% or with features of life-threatening asthma are at risk of hypercapnia and require arterial blood gas measurement. Experienced clinicians should treat these high-risk patients in a critical-care area. The specific drugs and the treatment sequence will vary according to local practice.

Oxygen. Use a concentration of inspired oxygen that will achieve an SpO_2 94–98%.[127] High-flow oxygen by mask is sometimes necessary. Lack of pulse oximetry should not prevent the use of oxygen.

Nebulised beta-2 agonists. Inhaled beta-2 agonists are first line drugs in patients with an acute asthma attack and should be administered as early as possible. Intravenous beta-2 agonists should be reserved for those patients in who inhaled therapy cannot be used reliably. Salbutamol, 5 mg nebulised, is the cornerstone of therapy for acute asthma in most of the world. Repeated doses every 15–20 min are often needed. Severe asthma may necessitate continuous nebulised salbutamol. Nebuliser units that can be driven by high-flow oxygen (at least 6 L min^{-1}) should be available. The hypoventilation associated with severe or near-fatal asthma may prevent effective delivery of nebulised drugs. If a nebuliser is not immediately available, beta-2 agonists can be temporarily administered by repeating activations of a metered dose inhaler via a large volume spacer device.[667,668] Nebulised adrenaline does not provide additional benefit over and above nebulised beta-2 agonists in acute asthma.[669]

Nebulised anticholinergics. Nebulised anticholinergics (ipratropium, 0.5 mg 4–6 hourly) may produce additional bronchodilation in severe asthma or in those who do not respond to beta-agonists.[670,671]

Nebulised magnesium sulphate. Although limited evidence suggests that magnesium sulphate has some bronchodilator effects[672] a review of 16 randomised or pseudo-randomised controlled trials in adults and children with acute asthma showed that inhaled magnesium alone or in combination with inhaled beta(2)-agonists (with or without inhaled ipratropium) was not associated with significant benefit in terms of improved pulmonary function or reduced hospital admissions.[673] Results of small studies in adults with severe exacerbations of asthma showed improvements in pulmonary function with additional inhaled magnesium, however evidence was too limited to come to a definite conclusion. Inhaled magnesium sulphate is currently not recommended for the treatment of acute asthma.

Intravenous magnesium sulphate. Studies of IV magnesium sulphate in acute severe and life-threatening asthma have produced conflicting results.[672,674,675] A systematic review assessing 14 studies (three of which were multicentre trials) including a total of 2313 adult or mostly adult patients treated for acute asthma in the emergency department showed that a single infusion of 1.2 or 2 g IV $MgSO_4$ over 15–30 min significantly reduced hospital admissions compared with placebo (odds ratio [OR] 0.75, 95% confidence interval [CI] 0.60–0.92) and improved lung function.[676] Participants in almost all of the studies had already been given at least oxygen, nebulised short-acting beta-2-agonists and IV corticosteroids in the emergency department. No difference was observed for other

outcomes such as intensive care admissions and length of hospital stay.

Give a single dose of IV magnesium sulphate to patients with acute severe asthma (PEF < 50% best or predicted) who have not had a good initial response to inhaled bronchodilator therapy. The most commonly reported adverse effects of IV magnesium sulphate are flushing, fatigue, nausea, headache and hypotension.

Intravenous corticosteroids. Early use of systemic corticosteroids for acute asthma in the emergency department significantly reduces hospital admission rates, especially for those patients not receiving concomitant corticosteroid therapy.[677] Although there is no difference in clinical effects between oral and IV formulations of corticosteroids,[678] the IV route is preferable because patients with near-fatal asthma may vomit or be unable to swallow.

Intravenous bronchodilators. There is a lack of definitive evidence for or against the use of IV bronchodilators in this setting. Trials have primarily included spontaneously breathing patients with moderate- to life-threatening exacerbations of asthma; evidence in ventilated patients with life-threatening asthma or cardiac arrest is sparse. The use of IV bronchodilators should generally be restricted to patients unresponsive to nebulised therapy or where nebulised/inhaled therapy is not possible (e.g. a patient receiving bag-mask ventilation). A Cochrane review of intravenous beta-2 agonists compared with nebulised beta-2 agonists found no evidence of benefit and some evidence of increased side effects compared with inhaled treatment.[679] Salbutamol may be given as either a slow IV injection (250 mcg IV slowly) or continuous infusion of 3–20 mcg min^{-1}.

Aminophylline. A Cochrane review of intravenous aminophylline found no evidence of benefit and a higher incidence of adverse effects (tachycardia, vomiting) compared with standard care alone.[680,681] Whether aminophylline has a place as an additional therapy after treatment with established medications such as inhaled beta-agonists and systemic corticosteroids remains uncertain. If, after obtaining senior advice, the decision is taken to administer IV aminophylline, give a loading dose of 5 mg kg^{-1} over 20–30 min (unless on maintenance therapy), followed by an infusion of 500–700 mcg kg^{-1} h^{-1}. Maintain serum theophylline concentrations below 20 mcg mL^{-1} to avoid toxicity.

Leukotriene receptor antagonists. There are few data on the use of intravenous leukotriene receptor antagonists.[682] Limited evidence suggests improvement of lung function and a non-significant trend towards reduced hospital admission when the intravenous leukotriene receptor antagonist montelukast was used as a rescue therapy in adults with acute asthma.[683,684] Further studies are required to confirm the usefulness of leukotriene receptor antagonists in this setting.

Intravenous fluids and electrolytes. Severe or near-fatal asthma is associated with dehydration and hypovolaemia, and this will further compromise the circulation in patients with dynamic hyperinflation of the lungs. If there is evidence of hypovolaemia or dehydration, give IV crystalloids. Beta-2 agonists and steroids may induce hypokalaemia, which should be monitored and corrected with electrolyte supplements as required.

Heliox. Heliox is a mixture of helium and oxygen (usually 80:20 or 70:30). A meta-analysis of four clinical trials did not support the use of heliox in the initial treatment of patients with acute asthma.[685]

Intramuscular adrenaline. Sometimes it may be difficult to distinguish severe life-threatening asthma from anaphylaxis. Treat patients presenting with severe 'asthma-like' symptoms, but without pre-existing pulmonary disease (asthma, COPD), as if the cause was anaphylaxis. In these circumstances, administration of adrenaline 0.5 mg IM according to the anaphylaxis guidelines may be appropriate (see anaphylaxis).

Referral to intensive care. An intensive care specialist should assess patients that fail to respond to initial treatment, or develop signs of life-threatening asthma. Intensive care admission after asthma-related cardiac arrest is associated with significantly poorer outcomes compared with those in who a cardiac arrest does not occur.[686]

Consider rapid sequence induction and tracheal intubation if, despite efforts to optimise drug therapy, the patient has:

- a decreasing conscious level, or coma;
- persisting or worsening hypoxaemia;
- deteriorating respiratory acidosis, despite intensive therapy;
- severe agitation, confusion and fighting against the oxygen mask (clinical signs of hypoxaemia);
- progressive exhaustion;
- respiratory or cardiac arrest.

Elevation of the PCO_2 alone does not indicate the need for tracheal intubation.[687] Treat the patient, not the numbers. All patients transferred to intensive care units should be accompanied by a doctor suitably equipped and skilled to intubate.

Non-invasive ventilation. Non-invasive ventilation (NIV) decreases the intubation rate and mortality in COPD[688]; however, its role in patients with severe acute asthma is uncertain. There is insufficient evidence to recommend its routine use in asthma.[689]

Treatment of cardiac arrest.

Basic life support. Give BLS according to standard guidelines. Ventilation will be difficult because of increased airway resistance; try to avoid gastric inflation.

Advanced life support. Modifications to standard ALS guidelines include considering the need for early tracheal intubation. The peak airway pressures recorded during ventilation of patients with severe asthma (mean 67.8 ± 11.1 cmH_2O in 12 patients) are significantly higher than the normal lower oesophageal sphincter pressure (approximately 20 cmH_2O).[690] 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.[691]

Respiratory rates of 8–10 breaths per minute and a tidal volume required for a normal chest rise during CPR should minimise dynamic hyperinflation of the lungs (air 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.[690]

Some case reports have reported ROSC in patients with air trapping when the tracheal tube was disconnected.[692–696] If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest while disconnecting tracheal tube may relieve air trapping. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.

Dynamic hyperinflation increases transthoracic impedance,[697] but modern impedance-compensated biphasic defibrillation waveforms are no less effective in patients with a higher impedance. As with standard ALS defibrillation protocols, consider increasing defibrillation energy if the first shock is unsuccessful and a manual defibrillator is available.

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.[698] If a pneumothorax is suspected, perform needle decompression using a large gauge cannula and being careful to avoid direct puncture of the lung. Any attempt at needle decompression should be followed by insertion of a chest tube. Always consider bilateral pneumothoraces in asthma-related cardiac arrest (see tension pneumothorax).

ECLS can ensure both organ perfusion and gas exchange in cases of otherwise refractory respiratory and circulatory failure. Cases of successful treatment of asthma-related cardiac arrest in adults using ECLS have been reported[699,700]; however, the role of ECLS in cardiac arrest caused by asthma has never been investigated in controlled studies. The use of ECLS requires appropriate skills and equipment that may not be available in all hospitals.

Patients with ventricular assist devices

Introduction. All clinicians caring for patients with ventricular assist devices (VADs) should have received full training in the procedures for equipment failure and the cardiac arrest situation. The management of patients with VADs is more complex, in that a cardiac arrest may be due to mechanical failure and in this situation there may be actions specific to the device that are required. The use of external chest compression in patients with ventricular assist devices has been reviewed.[701] There are isolated case reports of successful external chest compression without damage to the VAD. External chest compression may be particularly useful to decompress a non-functional right ventricle in cardiac arrests and often the right ventricle may be the cause of the loss of output.

Diagnosis of cardiac arrest. Confirming cardiac arrest in these patients may be difficult. A patient with invasive monitoring should be considered to have arrested if the arterial line reads the same as the central venous pressure (CVP) line. In patients without invasive monitoring, if the patient has no signs of life and is not breathing, then they should be considered to have suffered a cardiac arrest. Transthoracic/transoesophageal echocardiography (TTE/TOE), capnography or Doppler flow readings in a major artery may assist in the diagnosis of whether there is meaningful perfusion. These devices also display pump flow and this should be used to assist in a diagnosis of whether there has been a genuine loss of blood flow, or whether there is just a low flow situation with reduced conscious level.

Management of cardiac arrest. Patients with an implantable left ventricular assist devices (LVAD) such as a HeartMate (Thoratec, Pleasanton, CA, USA) or HeartWare (HeartWare, Framingham, MA, USA) device should have the same algorithm followed as the algorithm for arrest after cardiac surgery (see cardiac arrest following cardiac surgery). Check the rhythm; perform defibrillation for shockable rhythms (VF/pVT), start pacing for asystole. In pulseless electrical activity (PEA), turn the pacing off and verify there is no underlying VF, which must be treated by defibrillation. External chest compressions should be performed if immediate resuscitative efforts fail. Importantly, the airway and breathing checks should always be performed.

It is possible for a patient to have asystole or VF, but still have adequate cerebral blood flow due to adequate and continued pump flow. If the patient is conscious and responding then you will have more time in which to resolve this arrhythmia and external chest compressions will not be needed.

Resternotomy should be performed in an established cardiac arrest within 10 days of surgery and after this time, either resternotomy or extracorporeal membrane oxygenation (ECMO) is a reasonable option.

Cardiac arrest associated with neurological disease

Causes of cardiac arrest. Cardiac arrest associated with acute neurological disease is relatively uncommon and can occur with subarachnoid haemorrhage, intracerebral haemorrhage, epileptic seizures, and ischaemic stroke.[702] In addition brain injury associated with trauma can cause cardiac arrest.

Cardiac arrest associated with neurological disease can be due to:

- Loss of consciousness, causing airway obstruction, hypoxaemia and respiratory arrest followed by cardiac arrest. Loss of consciousness is also associated with an increased risk of aspiration of gastric contents into the lungs.
- Respiratory and cardiac depression caused by compression of the brain stem.
- Arrhythmias and myocardial dysfunction associated with acute neurological injury and in particular sub-arachnoid haemorrhage.
- Sudden unexpected death in epilepsy (SUDEP) effects about 1 in every 1000 people with epilepsy.[703]

Neurological symptoms. Patients can have prodromal signs suggesting a neurological cause before cardiac arrest such as headache, seizures, impaired consciousness, and focal signs,[704] but these are often non-specific and can include syncope, shortness of breath and chest pain. Cardiac or respiratory arrest occurs in between 3 and 11% of patents with subarachnoid haemorrhage,[705] and the initial rhythm is usually non-shockable.

Treatment. Preventive measures for cardiac or respiratory arrest should be aimed at treating the underlying cause. Once cardiac arrest occurs, follow standard BLS and ALS guidelines. If ROSC is achieved, address the underlying cause in addition to standard post resuscitation care.

Patients with subarachnoid haemorrhage may have ECG changes that suggest an acute coronary syndrome.[704,706] Certain features such as a young age, female gender, non-shockable initial rhythm and neurological antecedents (e.g. headache, seizures, neurological deficits) are common but non-specific for neurological cause.[707] Individuals with neurological prodromal symptoms who achieve ROSC may be considered for CT brain scan. Whether this is done before or after coronary angiography will depend on clinical judgement regarding the likelihood of a subarachnoid haemorrhage versus acute coronary syndrome.

Outcome. Survival depends on the underlying cause and traditional factors (e.g. witnessed, bystander CPR) associated with survival.[702] Prognosis is poor in those with ROSC after a subarachnoid haemorrhage.[704,706,708] Individuals who achieve ROSC after a primary neurological cause of cardiac arrest will often fulfil neurological criteria for death and should be considered as potential organ donors.[709]

Obesity

Introduction. Worldwide obesity has more than doubled since 1980. In 2014, more than 1.9 billion (39%) adults were overweight, and of these over 600 million (13%) were obese.

The World Health Organization (WHO) uses body mass index (BMI; weight in kg divided by height in m^2) to define obesity in adults as[710–712]:

- overweight (25.0–29.9 $kg\,m^{-2}$);
- obese (30.0–34.9 $kg\,m^{-2}$);
- very obese ($\geq$35.0 $kg\,m^{-2}$).

Many clinical studies have linked BMI to outcomes for a wide variety of cardiovascular and non-cardiovascular conditions.[713–715] Traditional cardiovascular risk factors (hypertension, diabetes, lipid profile, prevalent coronary heart disease, heart failure, and left ventricular hypertrophy) are common in obese patient. Obesity is associated with increased risk of sudden cardiac death.[715] Leading causes of death are dilated cardiomyopathy and severe coronary atherosclerosis.[716]

Modifications to cardiopulmonary resuscitation. No changes to sequence of actions are recommended in resuscitation of obese patients, but delivery of effective CPR may be challenging. Physical and physiological factors related to obesity may adversely affect the delivery of CPR, including patient access and transportation, patient assessment, difficult IV access, airway management, quality of chest compressions, the efficacy of vasoactive drugs, and the efficacy of defibrillation because none of these measures are standardised to a patient's BMI or weight.[710] More rescuers than usual may be required to assist in moving the patient and rescuer fatigue, particularly in relation to the delivery of chest compressions, may necessitate more regular changes of the rescuer than normal.

Chest compressions. As with all cardiac arrests, chest compressions are most effective when performed with the patient lying on a firm surface, but it may be unsafe for the patient and rescuers to attempt to move the patient down onto the floor. However, it is not always necessary in obese patients because the heavier torso sinks into the mattress, leaving less potential for mattress displacement during chest compression.[717]

In order to maintain sufficient depth of chest compresisons (approximately 5 cm but no more than 6 cm), rescuer fatigue may necessitate the need to change rescuers more frequently than the standard 2 min interval. Use of mechanical resuscitation devices is limited by the slope of the anterior chest wall, thoracic dimensions (sternum height up to 303 mm, and maximal width of 449 mm for piston devices (LUCAS); chest circumference up to 130 cm and maximal chest width of 380 mm for devices with a load-distributing band), and patient weight (up to 136 kg) (AutoPulse).

Defibrillation. Optimal defibrillation energy levels in obese patients are unknown. Unlike monophasic defibrillators, modern biphasic defibrillators are impedance-compensated and adjust their output according to the patient's impedance. Two small retrospective studies have demonstrated no apparent weight-based influence on defibrillation efficacy,[718] with a biphasic waveform of 150 J achieving high shock success rates without need for energy escalation.[719] Defibrillation protocols for obese patients should therefore follow those recommended for patients with a normal BMI. Consider higher shock energies for defibrillation if initial defibrillation attempts fail.

Ventilation. Higher inspiration pressure is needed for positive pressure ventilation due to increased intraabdominal pressure.[720] Early tracheal intubation by an experienced provider removes the need for prolonged bag-valve-mask ventilation, and may reduce any risk of aspiration. In all patients with extreme obesity, difficult intubation must be anticipated, with a clear failed intubation drill if necessary.[721] If intubation fails, use of a supraglottic airway device (SAD) with oesophaegeal drainage tube is a suitable option.

Logistical considerations. A patient's BMI should be considered when organising prehospital resuscitation, especially with regard to technical support and number of ambulance crew members.[722] Special response vehicles modified to carry extremely obese patients, equipped with extra-wide interiors, reinforced stretchers and specialised lifting gear, should be used if possible. Weight limits of both stretchers and hospital beds must be checked prior to use.[723] Underestimation of the technical aspects of rescue operations may cause secondary transportation trauma, or even prohibit safe transfer of obese patient to the hospital.[722]

Outcome. The relation between obesity and outcome from cardiac arrest is unclear. One large registry study has shown that survival from cardiac arrests caused by shockable rhythms (VF/pVT) was highest in overweight patients but was significantly lower in those who were very obese.[710] In contrast, survival to discharge of non-shockable rhythms was similar across all BMI groups. Evidence from clinical cohort studies has suggested that overweight and obese patients may actually have a more favourable short-term and long-term prognosis than leaner patients once they are successfully resuscitated from cardiac arrest.[711,724]

Cardiac arrest associated with pregnancy

Introduction

Mortality related to pregnancy is relatively rare in Europe (estimate 16 per 100,000 live births) although there is a large variation between countries.[725] The fetus must always be considered when an adverse cardiovascular event occurs in a pregnant woman. Fetal survival usually depends on maternal survival and initial resuscitation efforts should focus on the pregnant mother. 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.[726,727]

Significant physiological changes occur during pregnancy, e.g. cardiac output, blood volume, minute ventilation and oxygen consumption all increase. Furthermore, the gravid uterus can cause significant compression of iliac and abdominal vessels when the mother is in the supine position, resulting in reduced cardiac output and hypotension.

Causes of cardiac arrest

In developed regions, haemorrhage, embolism (thromboembolic and amniotic fluid), hypertensive disorders of pregnancy, abortion and genital tract sepsis account for most deaths directly associated with pregnancy, and pre-existing medical conditions for those indirectly related to pregnancy.[728] A review of over 2 million pregnancies in the UK showed that maternal deaths (death during pregnancy, childbirth, or within 42 days after delivery) were associated with cardiac disease, neurological conditions, psychiatric conditions, and malignancies.[729] A quarter of pregnant women who died in the UK had sepsis, and 1 in 11 had influenza. Pregnant women can also sustain cardiac arrest from the same causes as women of the same age group.

Prevention of cardiac arrest in pregnancy

In an emergency, use a systematic ABCDE approach. Many cardiovascular problems associated with pregnancy are caused by aorto-caval compression.

Treat a pregnant patient as follows:

- Place the patient in the left lateral position or manually and gently displace the uterus to the left.
- Give oxygen, guided by pulse oximetry to correct any hypoxaemia.
- Give a fluid bolus if there is hypotension or evidence of hypovolaemia.
- Immediately re-evaluate the need for any drugs being given.
- Seek expert help early. Obstetric and neonatal specialists should be involved early in the resuscitation.
- Identify and treat the underlying cause, e.g. rapid recognition and treatment of sepsis, including early intravenous antibiotics.

Modifications to basic life support

From 20 weeks' gestation, the uterus can compress both the inferior vena cava (IVC) and 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 cardiac arrest.[730,731] During cardiac arrest, the compromise in venous return and cardiac output by the gravid uterus limits the effectiveness of chest compressions.

Non-arrest studies show that left lateral tilt improves maternal blood pressure, cardiac output and stroke volume[732–734] and improves fetal oxygenation and heart rate.[735–737] Non cardiac arrest data show that the gravid uterus can be shifted away from the IVC in most cases, by placing the patient in 15° of left lateral decubitus position.[738] The value of relieving aortic or IVC compression during CPR is, however, unknown. Unless the pregnant victim is on a tilting operating table, left lateral tilt is not easy to perform whilst maintaining high-quality chest compressions. A variety of methods to achieve a left lateral tilt have been described including placing the victim on the rescuers knees,[739] pillows or blankets, or the Cardiff wedge[740] although their efficacy in actual cardiac arrests is unknown. Even when a tilting table is used, the angle of tilt is often overestimated.[741] In a manikin study, the ability to provide effective chest compressions decreased as the angle of left lateral tilt increased and at an angle of greater than 30° the manikin tended to roll.[740]

The key steps for BLS in a pregnant patient are:

- Call for expert help early (including an obstetrician and a neonatologist).
- Start BLS according to standard guidelines.
- Ensure high-quality chest compressions with minimal interruptions.
- The hand position for chest compressions may need to be slightly higher on the sternum for patients with advanced pregnancy e.g. third trimester.[726]
- Manually displace the uterus to the left to reduce IVC compression.
- Add left lateral tilt if this is feasible and ensure the chest remains supported on a firm surface (e.g. in the operating room) – the optimal angle of tilt is unknown. Aim for between 15 and 30°. Even a small amount of tilt may be better than no tilt. The angle of tilt used needs to enable high-quality chest compressions and if needed, allow Caesarean delivery of the fetus.
- Start preparing for emergency Caesarean section (see below) – the fetus will need to be delivered if initial resuscitation efforts fail.

Modifications to advanced life support

Defibrillation. For cardiac arrest with a shockable rhythm (VF/pVT) attempt defibrillation as soon as possible. There is no change in transthoracic impedance during pregnancy, suggesting that standard shock energies for defibrillation attempts should be used in pregnant patients.[742] There is no evidence that shocks from a direct current defibrillator have adverse effects on the fetal heart.

Airway management. During pregnancy, there is a greater potential for gastro-oesophageal sphincter insufficiency and risk of pulmonary aspiration of gastric contents.[743,744] Although pregnant patients are at risk of aspiration, oxygenation and ventilation is the priority over aspiration prevention. Early tracheal intubation will however 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.[745] One study documented that the upper airways in the third trimester of pregnancy are narrower compared with their post partum state and to non-pregnant controls.[746] Tracheal intubation may be more difficult in the pregnant patient.[747] Expert help, a failed intubation drill and the use of alternative airway devices may be needed.[748]

Intravascular access. Early intravenous or intraosseous access will enable drug and fluid administration. Aiming for access above the diaphragm may address any theoretical concerns associated with delayed circulation caused by IVC compression if drugs are infused through sites below the IVC.

Reversible causes

Rescuers should attempt to identify common and reversible causes of cardiac arrest in pregnancy during resuscitation (see special causes). The 4 Hs and 4 Ts approach helps identify all the common causes of cardiac arrest in pregnancy. Pregnant patients are also 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 possible causes during cardiac arrest; however, do not delay other treatments and minimise interruptions to chest compressions.

Specific causes of cardiac arrest in pregnancy include the following:

Haemorrhage. Life-threatening haemorrhage can occur both antenatally and postnatally.[728] Postpartum haemorrhage is the commonest single cause of maternal death worldwide and is estimated to cause one maternal death every 7 min.[749] Associations include ectopic pregnancy, placental abruption, placenta praevia, placenta accreta, and uterine rupture.[750] A massive haemorrhage protocol must be used in all units and should be updated in conjunction with the blood bank. Women at high risk of bleeding should be delivered in centres with facilities for blood transfusion, intensive care and other interventions, and plans should be made in advance for their management. Treatment is based on an ABCDE approach. The key step is to stop the bleeding.

Consider the following[751,752]:

- Fluid resuscitation, including use of rapid transfusion system and cell salvage.[753]
- Oxytocin and prostaglandin analogues to correct uterine atony.[754]
- Massaging the uterus.[755]
- Correction of coagulopathy including use of tranexamic acid and/or recombinant activated factor VII.[756–758]
- Uterine balloon tamponade or packing.[759,760]
- Uterine compression sutures.[761]
- Angiography and endovascular embolisation.[762]
- Hysterectomy.[763,764]
- Aortic cross-clamping in catastrophic haemorrhage.[765]

Cardiovascular disease. Myocardial infarction and aneurysm or dissection of the aorta or its branches, and peripartum cardiomyopathy cause most deaths from acquired cardiac disease.[766–768] Patients with known cardiac disease need to be managed in a specialist unit. Pregnant women may develop an acute coronary syndrome, typically in association with risk factors such as obesity, older age, higher parity, smoking, diabetes, pre-existing hypertension and a family history of ischaemic heart disease.[750,769] Pregnant patients can have atypical features such as epigastric pain and vomiting. Percutaneous coronary intervention (PCI) is the reperfusion strategy of choice for ST-elevation myocardial infarction in pregnancy. Thrombolysis should be considered if urgent PCI is unavailable. A review of 200 cases of thrombolysis for massive pulmonary embolism in pregnancy reported a maternal death rate

of 1% and concluded that thrombolytic therapy is reasonably safe in pregnancy.[770]

Increasing numbers of women with congenital heart disease are becoming pregnant.[771] Heart failure and arrhythmias are the commonest problems, especially in those with cyanotic heart disease. Pregnant women with known congenital heart disease should be managed in specialist centres.

Pre-eclampsia and eclampsia. Eclampsia is defined as the development of convulsions and/or unexplained coma during pregnancy or postpartum in patients with signs and symptoms of pre-eclampsia.[772,773] Magnesium sulphate is effective in preventing approximately half of the cases of eclampsia developing in labour or immediately postpartum in women with pre-eclampsia.[774–777] Use magnesium sulphate infusion for the treatment of eclampsia.[778–781]

Pulmonary embolism. The estimated incidence of pulmonary embolism is 1–1.5 per 10,000 pregnancies, with a case fatality of 3.5% (95% CI 1.1–8.0%).[782] Risk factors include obesity, increased age, and immobility. Successful use of fibrinolytics for massive, life-threatening pulmonary embolism in pregnant women has been reported.[770,783–786]

Amniotic fluid embolism. Amniotic fluid embolism (AFE) usually presents around the time of delivery with sudden cardiovascular collapse, breathlessness, cyanosis, arrhythmias, hypotension and haemorrhage associated with disseminated intravascular coagulopathy.[787] Patients may have warning signs preceding collapse including breathlessness, chest pain, feeling cold, lightheadedness, distress, panic, a feeling of pins and needles in the fingers, nausea, and vomiting. The UK Obstetric Surveillance System (UKOSS) identified 120 cases of AFE between 2005 and 2014 with a total and fatal incidence estimated as 1.7 and 0.3 per 100,000, respectively, and association with older maternal age, multiple pregnancy, placenta praevia and induction of labour, instrumental vaginal and Caesarean delivery.[788]

Treatment is supportive, as there is no specific therapy based on an ABCDE approach and correction of coagulopathy. Successful use of extracorporeal life support techniques for women suffering life-threatening amniotic fluid embolism during labour and delivery is reported.[789]

Peri-mortem delivery of the fetus

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. Three observational studies of 154 subjects collectively[790–792] provide very low quality evidence regarding the use of peri-mortem Caesarean section. Based on expert opinion, when initial resuscitation attempts fail, delivery of the fetus may improve the chances of successful resuscitation of the mother and fetus.[793–795] One systematic review documented 38 cases of Caesarean section during CPR, with 34 surviving infants and 13 maternal survivors at discharge, suggesting that Caesarean section may have improved maternal and neonatal outcomes.[796] 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.[793,797–799] This requires that the provider commence the hysterotomy at about 4 min after cardiac arrest. At older gestational ages (30–38 weeks), infant survival is possible even when delivery was after 5 min from the onset of maternal cardiac arrest[796] A case series suggests increased use of Caesarean section during CPR with team training[791]; in this series no deliveries were achieved within 5 min after starting resuscitation. Eight of the twelve women had ROSC after delivery, with two maternal and five newborn survivors. Maternal case fatality rate was 83%. Neonatal case fatality rate was 58%.[791]

Delivery will relieve IVC compression and may improve chances of maternal resuscitation. The Caesarean delivery also enables access to the infant so that newborn resuscitation can begin.

Decision-making for emergency hysterotomy (Caesarean section). The gravid uterus reaches a size that will begin to compromise aorto-caval blood flow at approximately 20 weeks gestation; however, fetal viability begins at approximately 24–25 weeks.[800] Portable ultrasound is available in some emergency departments and may aid in determination of gestational age (in experienced hands) and positioning, provided its use does not delay the decision to perform emergency hysterotomy.[801]

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

Post resuscitation care

Post resuscitation care should follow standard guidelines. Targeted temperature management (TTM) has been used safely and effectively in early pregnancy with fetal heart monitoring and resulted in favourable maternal and fetal outcome after a term delivery.[802] Implantable cardioverter defibrillators (ICDs) have been used in patients during pregnancy.[803]

Preparation for cardiac arrest in pregnancy

ALS in pregnancy requires coordination of maternal resuscitation, Caesarean delivery of the fetus and newborn resuscitation ideally within 5 min.

To achieve this, units likely to deal with cardiac arrest in pregnancy should:

- have plans and equipment in place for resuscitation of both the pregnant woman and newborn
- ensure early involvement of obstetric, anaesthetic and neonatal teams
- ensure regular training in obstetric emergencies.[804,805]

Elderly people

Epidemiology

More than 50% of people resuscitated from OHCAs in the United States are aged 65 years or older.[806] The incidence of cardiac arrests in elderly people is likely to increase as the world population ages. The incidence of cardiac arrest increases with age. In males, the incidence of OHCA at 80 years of age is about seven times greater than at 40 years of age.[807] In females above 70 years of age it is more than 40 times greater than in women below 45 years of age. In an observational study on in-hospital cardiac arrest patients above 65 years of age accounted for 46% of the total hospital admissions in the study period and for 65% of the ward cardiac arrests.[808] In this study, the incidence of arrests was more than twice that in the younger patient population (2.2 versus 1.0 per 1000 patient admissions).

Causes of cardiac arrest

The incidence of both coronary heart disease and chronic heart failure increases with age. As a consequence, elderly people have

an increased incidence of cardiac causes of arrest.[809] However, the proportion of deaths that are sudden (i.e. due to a primary ventricular arrhythmia) decreases with age, due to a parallel increase in the proportion of deaths due to other cardiovascular causes.[810] The incidence of PEA as the first recorded rhythm increases significantly with age[809,811]; with a parallel decrease of the incidence of shockable rhythms (VF/pVT).[812]

Prevention

Deterioration of vital signs leading to cardiac arrest is detected less accurately in elderly patients, compared with younger patients.[813] Clinical signs of acute life-threatening conditions such as sepsis,[814] acute myocardial infarction[815] or heart failure[816] are often blunted or non-specific in elderly patients, resulting in less physiological aberration and a lower Modified Early Warning Score (MEWS) in the 4 h preceding cardiac arrest.[808]

Treatment

Management of periarrest conditions. Ageing is associated with several pathophysiological changes that should be taken into account when managing peri-arrest conditions. Increasing age is associated with autonomic and baroreflex dysfunction and with myocardial stiffening which impairs early diastolic filling.[817] In addition, elderly critically ill patients are often hypovolaemic due to a reduction of both fluid intake and urine-concentrating ability.[818] These changes compromise the cardiovascular response to fluid loss or postural changes and increase the hypotensive effect of sedatives and other vasoactive drugs. Elderly patients are at increased risk of severe hypotension during emergency airway management.[819]

Atrial fibrillation is the most common supraventricular arrhythmia in the elderly. It often causes cardiovascular compromise due to loss of the atrial contribution for diastolic filling, particularly in the elderly who have reduced ventricular compliance. Hypotension and an increased heart rate may reduce coronary perfusion and precipitate cardiac ischaemia, which is more likely in an elderly population with a greater incidence of coronary artery disease.

Older patients are more likely to develop apnea or respiratory depression following the administration of opioid or benzodiazepines.[818] Their lower baseline oxygen tension also increases the risk of developing hypoxia. Advancing age is associated with an increased rate of comorbidities. Elderly patients often take several medications, which may interfere with drugs administered in peri-arrest conditions. The incidence of adverse drug reactions in the elderly is 2–3 times higher than in younger patients.[820]

Management of cardiac arrest. No modifications of standard resuscitation protocols are needed when managing aged patients in cardiac arrest. Rescuers, however, should be aware that the risk of both sternal and rib fractures is higher in elderly.[821–823] The incidence of CPR-related injuries increases with duration of CPR.[823]

Outcome

Older age is associated with an increasingly lower short-term survival rate after cardiac arrest.[824–829] In a large registry of OHCAs, survival to discharge was 8% for those aged 65–79 years, 4% for octogenarians and 2% for nonagenarians.[826] In another study, the adjusted risk for 30-day mortality in elderly resuscitated comatose patients was 1.04 (95% CI 1.03–1.06) per year of age.[812]

Increasing age is also associated with lower long-term survival after resuscitation. In a retrospective cohort study on elderly patients discharged alive after CPR from in-hospital cardiac arrest the risk-adjusted rate of 1-year survival was 63.7%, 58.6%, and 49.7% among patients 65–74, 75–84, and ≥85 years of age, respectively ($P < 0.001$).[827] In another study patients ≥65 years of age discharged alive after resuscitation from VF/pVT cardiac arrest showed a significantly lower long-term survival than an age- and gender-matched controls, while this was not observed in younger resuscitated patients.[830]

In those who do survive, neurological outcome is good in elderly survivors of cardiac arrest, with 95% having a cerebral performance category (CPC) score of 1–2 on discharge from ICU[824] and 72% at hospital discharge.[827]

Decision to resuscitate

Elderly patients with cardiac arrest are significantly less likely to receive resuscitation than younger patients.[831,832] When deciding to resuscitate elderly patients, age alone should not be the only criterion to consider and other more established criteria, i.e. witnessed status, resuscitation times, and first recorded rhythm, are important factors.[833] In addition, we suggest that pre-arrest factors, such as the degree of autonomy, quality of life, mental status and the presence of major comorbidities, should also be considered. Whenever possible, a decision to resuscitate or not, should be discussed in advance with the patient and his/her family (see ethics of resuscitation and end-of-life decisions).[243]

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Conflicts of interest

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European Resuscitation Council and European Society of Intensive Care Medicine Guidelines for Post-resuscitation Care 2015 Section 5 of the European Resuscitation Council Guidelines for Resuscitation 2015☆

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Summary of changes since 2010 guidelines

In 2010, post-resuscitation care was incorporated into the Advanced Life Support section of the European Resuscitation Council (ERC) Guidelines.[1] The ERC and the European Society of Intensive Care Medicine (ESICM) have collaborated to produce these post-resuscitation care guidelines, which recognise the importance of high-quality post-resuscitation care as a vital link in the Chain of Survival.[2] These post-resuscitation care guidelines are being co-published in *Resuscitation* and *Intensive Care Medicine*.

The most important changes in post-resuscitation care since 2010 include:

- There is a greater emphasis on the need for urgent coronary catheterisation and percutaneous coronary intervention (PCI) following out-of-hospital cardiac arrest of likely cardiac cause.
- Targeted temperature management remains important but there is now an option to target a temperature of 36 °C instead of the previously recommended 32–34 °C.
- Prognostication is now undertaken using a multimodal strategy and there is emphasis on allowing sufficient time for neurological recovery and to enable sedatives to be cleared.
- A novel section has been added which addresses rehabilitation after survival from a cardiac arrest. Recommendations include the systematic organisation of follow-up care, which should include screening for potential cognitive and emotional impairments and provision of information.

The international consensus on cardiopulmonary resuscitation science and the guidelines process

The International Liaison Committee on Resuscitation (ILCOR, www.ilcor.org) 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), the 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 most recent International Consensus Conference was held in Dallas in February 2015 and the published conclusions and recommendations from this process form the basis of the ERC Guidelines 2015 and for these ERC-ESICM post-resuscitation care guidelines. During the three years leading up to this conference, 250 evidence reviewers

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from 39 countries reviewed thousands of relevant, peer-reviewed publications to address 169 specific resuscitation questions, each in the standard PICO (Population, Intervention, Comparison, Outcome) format. To assess the quality of the evidence and the strength of the recommendations, ILCOR adopted the GRADE (Grading of Recommendations Assessment, Development and Evaluation) methodology. Each PICO question was reviewed by at least two evidence reviewers who drafted a science statement based on their interpretation of all relevant data on the specific topic and the relevant ILCOR task force added consensus draft treatment recommendations. Final wording of science statements and treatment recommendations was completed after further review by ILCOR member organisations and by the editorial board, and published in *Resuscitation* and *Circulation* as the 2015 Consensus on Science and Treatment Recommendations (CoSTR). These ERC-ESICM guidelines on post-resuscitation care are based on the 2015 CoSTR document and represent consensus among the writing group, which included representatives of the ERC and the ESICM.

Introduction

Successful return of spontaneous circulation (ROSC) is the first step towards the goal of complete recovery from cardiac arrest. The complex pathophysiological processes that occur following whole body ischaemia during cardiac arrest and the subsequent reperfusion response during CPR and following successful resuscitation have been termed the post-cardiac arrest syndrome.[3] Depending on the cause of the arrest, and the severity of the post-cardiac arrest syndrome, many patients will require multiple organ support and the treatment they receive during this post-resuscitation period influences significantly the overall outcome and particularly the quality of neurological recovery.[4–11] The post-resuscitation phase starts at the location where ROSC is achieved but, once stabilised, the patient is transferred to the most appropriate high-care area (e.g., emergency room, cardiac catheterisation laboratory or intensive care unit (ICU)) for continued diagnosis, monitoring and treatment. The post-resuscitation care algorithm (Fig. 1) outlines some of the key interventions required to optimise outcome for these patients.

Some patients do awake rapidly following cardiac arrest – in some reports it is as high as 15–46% of the out-of hospital cardiac arrest patients admitted to hospital.[12–14] Response times, rates of bystander CPR, times to defibrillation and the duration of CPR impact on these numbers.[14] Although we have no data, it is reasonable to recommend that if there is any doubt about the patient's neurological function, the patient's trachea should be intubated and treatment to optimise haemodynamic, respiratory and metabolic variables, together with targeted temperature management started, following the local standardised treatment plan.

Of those comatose patients admitted to ICUs after cardiac arrest, as many as 40–50% survive to be discharged from hospital depending on the cause of arrest, system and quality of care.[7,10,13–20] Of the patients who survive to hospital discharge, the vast majority have a good neurological outcome although many with subtle cognitive impairment.[21–24]

Post-cardiac arrest syndrome

The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and the persistent precipitating pathology.[3,25,26] 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 approximately two thirds after out-of hospital cardiac arrest and approximately 25% after in-hospital cardiac arrest.[27–30] Cardiovascular failure accounts for most deaths in the first three days, while brain injury accounts for most of the later deaths.[27,30,31] Withdrawal of life sustaining therapy (WLST) is the most frequent cause of death (approximately 50%) in patients with a prognosticated bad outcome,[14,30] emphasising the importance of the prognostication plan (see below). Post-cardiac arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypotension, hypercarbia, hypoxaemia, hyperoxaemia, pyrexia, hypoglycaemia, hyperglycaemia and seizures. Significant myocardial dysfunction is common after cardiac arrest but typically starts to recover by 2–3 days, although full recovery may take significantly longer.[32–34] The whole body ischaemia/reperfusion of cardiac arrest activates immune and coagulation pathways contributing to multiple organ failure and increasing the risk of infection.[35–41] Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion, vasodilation, endothelial injury and abnormalities of the microcirculation.[42–48]

Airway and breathing

Control of oxygenation

Patients who have had a brief period of cardiac arrest responding immediately to appropriate treatment may achieve an immediate return of normal cerebral function. These patients do not require tracheal intubation and ventilation but should be given with oxygen via a facemask if their arterial blood oxygen saturation is less than 94%. 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 early after ROSC causes oxidative stress and harms post-ischaemic neurones.[49–53] One animal study showed that adjusting the fractional inspired concentration (FiO_2) to produce an arterial oxygen saturation of 94–96% in the first hour after ROSC (controlled reoxygenation) achieved better neurological outcomes than achieved with the delivery of 100% oxygen.[54] One clinical registry study that included more than 6000 patients supports the animal data and shows post-resuscitation hyperoxaemia in the first 24 h is associated with worse outcome, compared with both normoxaemia and hypoxaemia.[55] A further analysis by the same group showed that the association between hyperoxia and outcome was dose-dependent and that there was not a single threshold for harm.[56] An observational study that included only those patients treated with mild induced hypothermia also showed an association between hyperoxia and poor outcome.[57] In contrast, an observational study of over 12,000 post-cardiac arrest patients showed that after adjustment for the inspired oxygenation concentration and other relevant covariates (including sickness severity), hyperoxia was no longer associated with mortality.[58] A meta-analysis of 14 observational studies showed significant heterogeneity across studies.[59]

The animal studies showing a relationship between hyperoxia and worse neurological outcome after cardiac arrest have generally evaluated the effect of hyperoxia in the first hour after ROSC. There are significant practical challenges with the titration of inspired oxygen concentration immediately after ROSC, particularly in the out-of hospital setting. The only prospective clinical study to

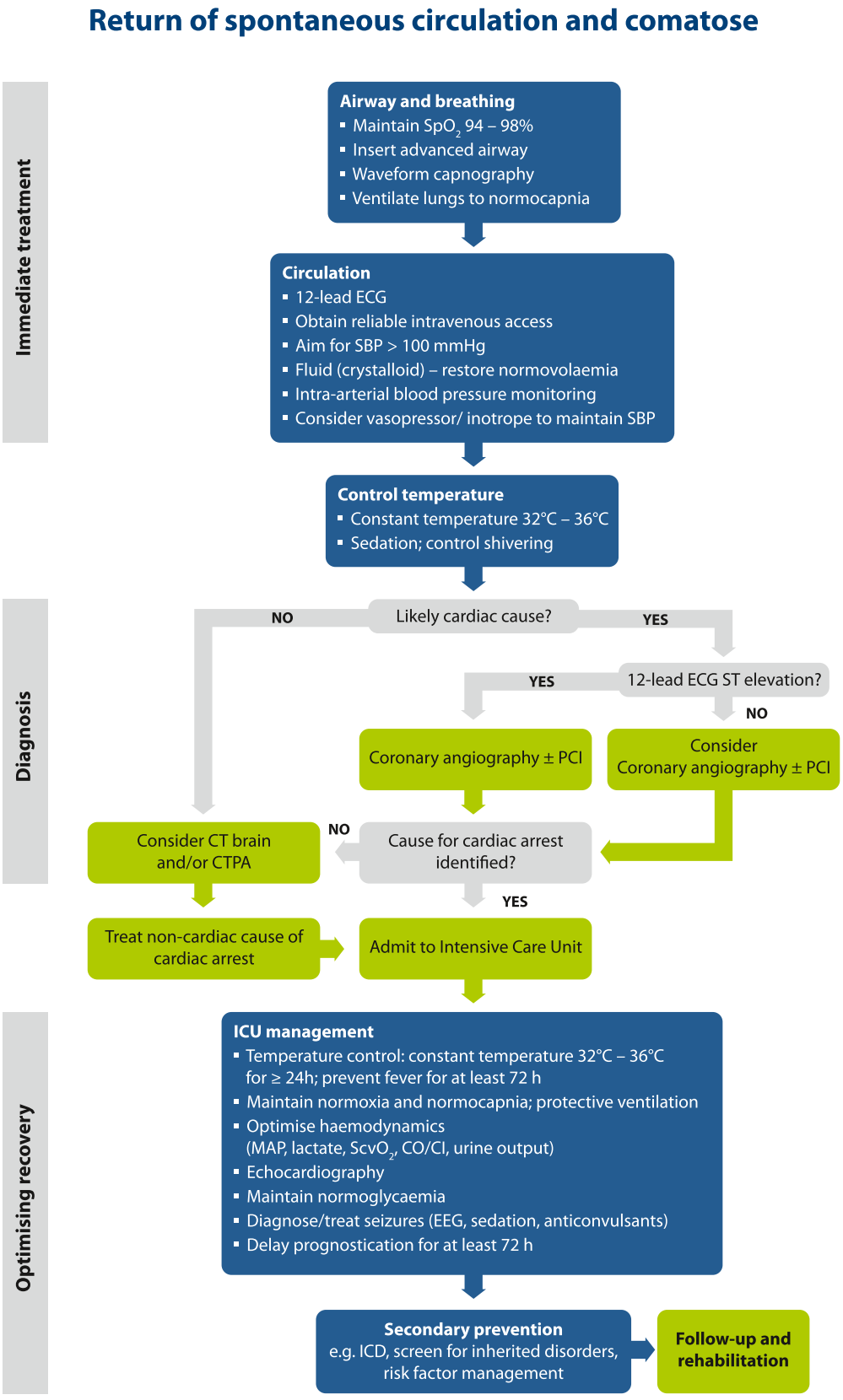

Fig. 5.1. Post-resuscitation care algorithm. SBP: systolic blood pressure; PCI: percutaneous coronary intervention; CTPA: computed tomography pulmonary angiogram; ICU: intensive care unit; MAP: mean arterial pressure; $ScvO_2$: central venous oxygenation; CO/CI: cardiac output/cardiac index; EEG: electroencephalography; ICD: implanted cardioverter defibrillator.

compare oxygen titrated to a target range (in this case 90–94% oxygen saturation) versus giving 100% oxygen after out of hospital cardiac arrest was stopped after enrolling just 19 patients because it proved very difficult to obtain reliable arterial blood oxygen saturation values using pulse oximetry.[60] A recent study of air versus supplemental oxygen in ST-elevation myocardial infarction showed that supplemental oxygen therapy increased myocardial injury, recurrent myocardial infarction and major cardiac arrhythmia and was associated with larger infarct size at 6 months.[61]

Given the evidence of harm after myocardial infarction and the possibility of increased neurological injury after cardiac arrest, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%. Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration.

Control of ventilation

Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. Ensure the tracheal tube is positioned correctly, well above the carina. Hypocarbia causes cerebral vasoconstriction and a decreased cerebral blood flow.[62] After cardiac arrest, hypocapnia induced by hyperventilation causes cerebral ischaemia.[63–67] Observational studies using cardiac arrest registries document an association between hypocapnia and poor neurological outcome.[68,69] Two observation studies have documented an association with mild hypercapnia and better neurological outcome among post-cardiac arrest patients in the ICU.[69,70] Until prospective data are available, it is reasonable to adjust ventilation to achieve normocarbia and to monitor this using the end-tidal CO_2 and arterial blood gas values. Lowering the body temperature decreases the metabolism and may increase the risk of hypocapnia during the temperature intervention.[71]

Although protective lung ventilation strategies have not been studied specifically in post-cardiac arrest patients, given that these patients develop a marked inflammatory response, it seems rational to apply protective lung ventilation: tidal volume 6–8 ml kg^{-1} ideal body weight and positive end expiratory pressure 4–8 cm H_2O.[48,72]

Insert a gastric tube to decompress the stomach; gastric distension caused by mouth-to-mouth or bag-mask ventilation will splint the diaphragm and impair ventilation. Give adequate doses of sedative, which will reduce oxygen consumption. A sedation protocol is highly recommended. Bolus doses of a neuromuscular blocking drug may be required, particularly if using targeted temperature management (TTM) (see below). Limited evidence shows that short-term infusion ($\leq$48 h) of short-acting neuromuscular blocking drugs given to reduce patient-ventilator dysynchrony and risk of barotrauma in ARDS patients is not associated with an increased risk of ICU-acquired weakness and may improve outcome in these patients.[73] There are some data suggesting that continuous neuromuscular blockade is associated with decreased mortality in post-cardiac arrest patients[74]; however, infusions of neuromuscular blocking drugs interfere with clinical examination and may mask seizures. Continuous electroencephalography (EEG) is recommended to detect seizures in these patients, especially when neuromuscular blockade is used.[75] Obtain a chest radiograph to check the position of the tracheal tube, gastric tube and central venous lines, assess for pulmonary oedema, and detect complications from CPR such as a pneumothorax associated with rib fractures.[76,77]

Circulation

Coronary reperfusion

Acute coronary syndrome (ACS) is a frequent cause of out-of-hospital cardiac arrest (OHCA): in a recent meta-analysis, the prevalence of an acute coronary artery lesion ranged from 59% to 71% in OHCA patients without an obvious non-cardiac aetiology.[78] Since the publication of a pioneering study in 1997,[79] many observational studies have shown that emergent cardiac catheterisation laboratory evaluation, including early percutaneous coronary intervention (PCI), is feasible in patients with ROSC after cardiac arrest.[80,81] The invasive management (i.e., early coronary angiography followed by immediate PCI if deemed necessary) of these patients, particularly those having prolonged resuscitation and nonspecific ECG changes, has been controversial because of the lack of specific evidence and significant implications on use of resources (including transfer of patients to PCI centres).

Percutaneous coronary intervention following ROSC with ST-elevation

In patients with ST segment elevation (STE) or left bundle branch block (LBBB) on the post-ROSC electrocardiogram (ECG) more than 80% will have an acute coronary lesion.[82] There are no randomised studies but given that many observational studies reported increased survival and neurologically favourable outcome, it is highly probable that early invasive management is beneficial in STE patients.[83] Based on available data, emergent cardiac catheterisation laboratory evaluation (and immediate PCI if required) should be performed in adult patients with ROSC after OHCA of suspected cardiac origin with STE on the ECG. This recommendation is based on low quality of evidence from selected populations. Observational studies also indicate that optimal outcomes after OHCA are achieved with a combination of TTM and PCI, which can be included in a standardised post-cardiac arrest protocol as part of an overall strategy to improve neurologically intact survival.[81,84,85]

Percutaneous coronary intervention following ROSC without ST-elevation

In contrast to the usual presentation of ACS in non-cardiac arrest patients, the standard tools to assess coronary ischaemia in cardiac arrest patients are less accurate. The sensitivity and specificity of the usual clinical data, ECG and biomarkers to predict an acute coronary artery occlusion as the cause of OHCA are unclear.[86–89] Several large observational series showed that absence of STE may also be associated with ACS in patients with ROSC following OHCA.[90–93] In these non-STE patients, there are conflicting data from observational studies on the potential benefit of emergent cardiac catheterisation laboratory evaluation.[92,94,95] A recent consensus statement from the European Association for Percutaneous Cardiovascular Interventions (EAPCI) has emphasised that in OHCA patients, cardiac catheterisation should be performed immediately in the presence of ST-elevation and considered as soon as possible (less than 2 h) in other patients in the absence of an obvious non-coronary cause, particularly if they are haemodynamically unstable.[96] Currently, this approach in patients without STE remains controversial and is not accepted by all experts. However, it is reasonable to discuss and consider emergent cardiac catheterisation laboratory evaluation after ROSC in patients with the highest risk of a coronary cause for their cardiac arrest. Factors such as patient age, duration of CPR, haemodynamic instability, presenting cardiac rhythm, neurological status upon hospital arrival, and perceived likelihood of cardiac aetiology can influence

the decision to undertake the intervention in the acute phase or to delay it until later on in the hospital stay.

Indications and timing of computed tomography (CT) scanning

Cardiac causes of OHCA have been extensively studied in the last few decades; conversely, little is known on non-cardiac causes. Early identification of a respiratory or neurological cause would enable transfer of the patient to a specialised ICU for optimal care. Improved knowledge of prognosis also enables discussion about the appropriateness of specific therapies, including TTM. Early identification of a respiratory or neurological cause can be achieved by performing a brain and chest CT-scan at hospital admission, before or after coronary angiography. In the absence of signs or symptoms suggesting a neurological or respiratory cause (e.g., headache, seizures or neurological deficits for neurological causes, shortness of breath or documented hypoxia in patients suffering from a known and worsening respiratory disease) or if there is clinical or ECG evidence of myocardial ischaemia, coronary angiography is undertaken first, followed by CT scan in the absence of causative lesions. Several case series showed that this strategy enables diagnosis of non-cardiac causes of arrest in a substantial proportion of patients.[97,98] In those with cardiac arrest associated with trauma or haemorrhage a whole body CT scan may be indicated.[99,100]

Haemodynamic management

Post-resuscitation myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias.[32,101] Perform early echocardiography in all patients in order to detect and quantify the degree of myocardial dysfunction.[33,102] Post-resuscitation myocardial dysfunction often requires inotropic support, at least transiently. Based on experimental data, dobutamine is the most established treatment in this setting,[103,104] but the systematic inflammatory response that occurs frequently in post-cardiac arrest patients may also cause vasoplegia and severe vasodilation.[32] Thus, noradrenaline, with or without dobutamine, and fluid is usually the most effective treatment. Infusion of relatively large volumes of fluid is tolerated remarkably well by patients with post-cardiac arrest syndrome.[7,8,32] If treatment with fluid resuscitation, inotropes and vasoactive drugs is insufficient to support the circulation, consider insertion of a mechanical circulatory assistance device (e.g., IMPELLA, Abiomed, USA).[7,105]

Treatment may be guided by blood pressure, heart rate, urine output, rate of plasma lactate clearance, and central venous oxygen saturation. Serial echocardiography may also be used, especially in haemodynamically unstable patients. In the ICU an arterial line for continuous blood pressure monitoring is essential. Cardiac output monitoring may help to guide treatment in haemodynamically unstable patients but there is no evidence that its use affects outcome. Some centres still advocate use of an intra aortic balloon pump (IABP) in patients with cardiogenic shock, although the IABP-SHOCK II Trial failed to show that use of the IABP improved 30-day mortality in patients with myocardial infarction and cardiogenic shock.[106,107]

Similarly to the early goal-directed therapy that is recommended in the treatment of sepsis,[108] although challenged by several recent studies,[109–111] a bundle of therapies, including a specific blood pressure target, has been proposed as a treatment strategy after cardiac arrest.[8] However its influence on clinical outcome is not firmly established and optimal targets for mean arterial pressure and/or systolic arterial pressure remain unknown.[7,8,112–114] One observational study of 151 post-cardiac arrest patients identified an association between a time-weighted average mean arterial pressure (measured every 15 min) of greater than 70 mmHg and good neurological outcome.[113] A recent study showed an inverse relationship between mean arterial pressure and mortality.[101] However, whether the use of vasoactive drugs to achieve such a blood pressure target achieves better neurological outcomes remains unknown. 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] These targets may vary depending on individual physiology and co-morbid status. Importantly, hypothermia may increase urine output[115] and impair lactate clearance.[101]

Tachycardia was associated with bad outcome in one retrospective study.[116] During mild induced hypothermia the normal physiological response is bradycardia. In animal models this has been shown to reduce the diastolic dysfunction that usually is present early after cardiac arrest.[117] Bradycardia was previously considered to be a side effect, especially below a rate of $40\ min^{-1}$; however, recent retrospective studies have shown that bradycardia is associated with a good outcome.[118,119] As long as blood pressure, lactate, SvO_2 and urine output are sufficient, a bradycardia of $\leq 40\ min^{-1}$ may be left untreated. Importantly, oxygen requirements during mild induced hypothermia are reduced.

Relative adrenal insufficiency occurs frequently after successful resuscitation from cardiac arrest and it appears to be associated with a poor prognosis when accompanied by post-resuscitation shock.[120,121] Two randomised controlled trials involving 368 patients with IHCA showed improved ROSC with the use of methylprednisolone and vasopressin in addition to adrenaline, compared with the use of placebo and adrenaline alone: combined RR 1.34 (95% CI 1.21–1.43).[122,123] No studies have assessed the effect of adding steroids alone to standard treatment for IHCA. These studies come from a single group of investigators and the population studied had very rapid advanced life support, a high incidence of asystolic cardiac arrest, and low baseline survival compared with other IHCA studies. Further confirmatory studies are awaited but, pending further data, do not give steroids routinely after IHCA. There is no clinical evidence for the routine use of steroids after OHCA.

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

Implantable cardioverter defibrillators

Insertion of an implantable cardioverter defibrillator (ICD) should be considered in ischaemic patients with significant left ventricular dysfunction, who have been resuscitated from a ventricular arrhythmia that occurred later than 24–48 h after a primary coronary event.[124–126] ICDs may also reduce mortality in cardiac arrest survivors at risk of sudden death from structural heart diseases or inherited cardiomyopathies.[127,128] In all cases, a specialised electrophysiological evaluation should be performed before discharge for placement of an ICD for secondary prevention of sudden cardiac death.

Disability (optimising neurological recovery)

Cerebral perfusion

Animal studies show that immediately after ROSC there is a short period of multifocal cerebral no-reflow followed by transient

global cerebral hyperaemia lasting 15–30 min.[129–131] This is followed by up to 24 h of cerebral hypoperfusion while the cerebral metabolic rate of oxygen gradually recovers. After asphyxial cardiac arrest, brain oedema may occur transiently after ROSC but it is rarely associated with clinically relevant increases in intracranial pressure.[132,133] In many patients, autoregulation of cerebral blood flow is impaired (absent or right-shifted) for some time after cardiac arrest, which means that cerebral perfusion varies with cerebral perfusion pressure instead of being linked to neuronal activity.[134,135] In a study that used near-infrared spectroscopy to measure regional cerebral oxygenation, autoregulation was disturbed in 35% of post-cardiac arrest patients and the majority of these had been hypertensive before their cardiac arrest[136]; this tends to support the recommendation made in the 2010 ERC Guidelines: after ROSC, maintain mean arterial pressure near the patient's normal level.[1] However, there is a significant gap in the knowledge about how temperature impacts the optimal blood pressure.

Sedation

Although it has been common practice to sedate and ventilate patients for at least 24 h after ROSC, there are no high-level data to support a defined period of ventilation, sedation and neuromuscular blockade after cardiac arrest. Patients need to be sedated adequately during treatment with TTM, and the duration of sedation and ventilation is therefore influenced by this treatment. A meta-analysis of drugs used for sedation during mild induced hypothermia showed considerable variability among 68 ICUs in a variety of countries.[137] There are no data to indicate whether or not the choice of sedation influences outcome, but a combination of opioids and hypnotics is usually used. Short-acting drugs (e.g., propofol, alfentanil, remifentanil) will enable more reliable and earlier neurological assessment and prognostication (see Section 7).[138] Volatile anaesthetics have been used to sedate post cardiac arrest patients[139] but although there are some animal data suggesting myocardial and neurological benefits,[140] there are no clinical data showing an advantage with this strategy. Adequate sedation will reduce oxygen consumption. During hypothermia, optimal sedation can reduce or prevent shivering, which enables the target temperature to be achieved more rapidly. Use of published sedation scales for monitoring these patients (e.g., the Richmond or Ramsay Scales) may be helpful.[141,142]

Control of seizures

Seizures are common after cardiac arrest and occur in approximately one-third of patients who remain comatose after ROSC. Myoclonus is most common and occurs in 18–25%, the remainder having focal or generalised tonic–clonic seizures or a combination of seizure types.[31,143–145] Clinical seizures, including myoclonus may or may not be of epileptic origin. Other motor manifestations could be mistaken for seizures[146] and there are several types of myoclonus[147] the majority being non-epileptic. Use intermittent electroencephalography (EEG) to detect epileptic activity in patients with clinical seizure manifestations. Consider continuous EEG to monitor patients with a diagnosed status epilepticus and effects of treatment.

In comatose cardiac arrest patients, EEG commonly detects epileptiform activity. Unequivocal seizure activity according to strict EEG-terminology[148] is less common but post-anoxic status epilepticus was detected in 23–31% of patients using continuous EEG-monitoring and more inclusive EEG-criteria.[75,149,150] Patients with electrographic status epilepticus may or may not have clinically detectable seizure manifestations that may be masked by sedation. Whether systematic detection and treatment of electrographic epileptic activity improves patient outcome is not known.

Seizures may increase the cerebral metabolic rate[151] and have the potential to exacerbate brain injury caused by cardiac arrest: treat with sodium valproate, levetiracetam, phenytoin, benzodiazepines, propofol, or a barbiturate. Myoclonus can be particularly difficult to treat; phenytoin is often ineffective. Propofol is effective to suppress post-anoxic myoclonus.[152] Clonazepam, sodium valproate and levetiracetam are antimyoclonic drugs that may be effective in post-anoxic myoclonus.[147] After the first event, start maintenance therapy once potential precipitating causes (e.g., intracranial haemorrhage, electrolyte imbalance) are excluded.

The use of prophylactic anticonvulsant drugs after cardiac arrest in adults has been insufficiently studied.[153,154] Routine seizure prophylaxis in post-cardiac arrest patients is not recommended because of the risk of adverse effects and the poor response to antiepileptic agents among patients with clinical and electrographic seizures.

Myoclonus and electrographic seizure activity, including status epilepticus, are related to a poor prognosis but individual patients may survive with good outcome (see Section 7).[145,155] Prolonged observation may be necessary after treatment of seizures with sedatives, which will decrease the reliability of a clinical examination.[156]

Glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome.[13,15,20,157–163] Although one randomised controlled trial in a cardiac surgical intensive care unit showed that tight control of blood glucose (4.4–6.1 mmol l^{-1} or 80–110 mg dl^{-1}) using insulin reduced hospital mortality in critically ill adults,[164] a second study by the same group in medical ICU patients showed no mortality benefit from tight glucose control.[165] In one randomised trial of patients resuscitated from OHCA with ventricular fibrillation, strict glucose control (72–108 mg dl^{-1}, 4–6 mmol l^{-1}) gave no survival benefit compared with moderate glucose control (108–144 mg dl^{-1}, 6–8 mmol l^{-1}) and there were more episodes of hypoglycaemia in the strict glucose control group.[166] A large randomised trial of intensive glucose control (81 mg dl^{-1} – 108 mg dl^{-1}, 4.5–6.0 mmol l^{-1}) versus conventional glucose control (180 mg dl^{-1}, 10 mmol l^{-1} or less) in general ICU patients reported increased 90-day mortality in patients treated with intensive glucose control.[167,168] Severe hypoglycaemia is associated with increased mortality in critically ill patients,[169] and comatose patients are at particular risk from unrecognised hypoglycaemia. Irrespective of the target range, variability in glucose values is associated with mortality.[170] Compared with normothermia, mild induced hypothermia is associated with higher blood glucose values, increased blood glucose variability and greater insulin requirements.[171] Increased blood glucose variability is associated with increased mortality and unfavourable neurological outcome after cardiac arrest.[157,171]

Based on the available data, following ROSC maintain the blood glucose at $\leq$10 mmol l^{-1} (180 mg dl^{-1}) and avoid hypoglycaemia.[172] Do not implement strict glucose control in adult patients with ROSC after cardiac arrest because it increases the risk of hypoglycaemia.

Temperature control

Treatment of hyperpyrexia

A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest.[13,173–176] Several studies document an association between post-cardiac arrest pyrexia and poor

outcomes.[13,173,175–178] The development of hyperthermia after a period of mild induced hypothermia (rebound hyperthermia) is associated with increased mortality and worse neurological outcome.[179–182] There are no randomised controlled trials evaluating the effect of treatment of pyrexia (defined as ≥37.6 °C) compared to no temperature control in patients after cardiac arrest and the elevated temperature may only be an effect of a more severely injured brain. Although the effect of elevated temperature on outcome is not proven, it seems reasonable to treat hyperthermia occurring after cardiac arrest with antipyretics and to consider active cooling in unconscious patients.

Targeted temperature management

Animal and human data indicate that mild induced hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia.[183,184] Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen ($CMRO_2$) by about 6% for each 1 °C reduction in core temperature and this may reduce the release of excitatory amino acids and free radicals.[183,185] 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. However, in the temperature range 33–36 °C, there is no difference in the inflammatory cytokine response in adult patients according to a recent study.[186]

All studies of post-cardiac arrest mild induced hypothermia have included only patients in coma. One randomised trial and a pseudo-randomised trial demonstrated improved neurological outcome at hospital discharge or at 6 months in comatose patients after out-of-hospital VF cardiac arrest.[187,188] Cooling was initiated within minutes to hours after ROSC and a temperature range of 32–34 °C was maintained for 12–24 h.

Three cohort studies including a total of 1034 patients, have compared mild induced hypothermia (32–34 °C) to no temperature management in OHCA and found no difference in neurological outcome (adjusted pooled odds ratio (OR), 0.90 [95% CI 0.45–1.82].[189–191] One additional retrospective registry study of 1830 patients documented an increase in poor neurological outcome among those with nonshockable OHCA treated with mild induced hypothermia (adjusted OR 1.44 [95% CI 1.039–2.006]).[192]

There are numerous before and after studies on the implementation of temperature control after in hospital cardiac arrest but these data are extremely difficult to interpret because of other changes in post cardiac arrest care that occurred simultaneously. One retrospective cohort study of 8316 in-hospital cardiac arrest (IHCA) patients of any initial rhythm showed no difference in survival to hospital discharge among those who were treated with mild induced hypothermia compared with no active temperature management (OR 0.9, 95% CI 0.65–1.23) but relatively few patients were treated with mild induced hypothermia.[193]

In the Targeted Temperature Management (TTM) trial, 950 all-rhythm OHCA patients were randomised to 36 h of temperature control (comprising 28 h at the target temperature followed by slow rewarm) at either 33 °C or 36 °C.[31] Strict protocols were followed for assessing prognosis and for withdrawal of life-sustaining treatment (WLST). There was no difference in the primary outcome – all cause mortality, and neurological outcome at 6 months was also similar (hazard ratio (HR) for mortality at end of trial 1.06, 95% CI 0.89–1.28; relative risk (RR) for death or poor neurological outcome at 6 months 1.02, 95% CI 0.88–1.16). Detailed neurological outcome at 6 months was also similar.[22,24] Importantly, patients in both arms of this trial had their temperature well controlled so that fever was prevented in both groups. TTM at 33 °C was associated with decreased heart rate, elevated lactate, the need for increased vasopressor support, and a higher extended cardiovascular SOFA score compared with TTM at 36 °C.[101,194] Bradycardia during mild induced hypothermia may be beneficial – it is associated with good neurological outcome among comatose survivors of OHCA, presumably because autonomic function is preserved.[118,119]

The optimal duration for mild induced hypothermia and TTM is unknown although it is currently most commonly used for 24 h. Previous trials treated patients with 12–28 h of targeted temperature management.[31,187,188] Two observational trials found no difference in mortality or poor neurological outcome with 24 h compared with 72 h of hypothermia.[195,196] The TTM trial provided strict normothermia (<37.5 °C) after hypothermia until 72 h after ROSC.[31]

The term targeted temperature management or temperature control is now preferred over the previous term therapeutic hypothermia. The Advanced Life Support Task Force of the International Liaison Committee on Resuscitation made several treatment recommendations on targeted temperature management[128] and these are reflected in these ERC guidelines:

- Maintain a constant, target temperature between 32 °C and 36 °C for those patients in whom temperature control is used (strong recommendation, moderate-quality evidence).
- Whether certain subpopulations of cardiac arrest patients may benefit from lower (32–34 °C) or higher (36 °C) temperatures remains unknown, and further research may help elucidate this.
- TTM is recommended for adults after OHCA with an initial shockable rhythm who remain unresponsive after ROSC (strong recommendation, low-quality evidence).
- TTM is suggested for adults after OHCA with an initial nonshockable rhythm who remain unresponsive after ROSC (weak recommendation, very low-quality evidence).
- TTM is suggested for adults after IHCA with any initial rhythm who remain unresponsive after ROSC (weak recommendation, very low-quality evidence).
- If targeted temperature management is used, it is suggested that the duration is at least 24 h (as undertaken in the two largest previous RCTs[31,187]) (weak recommendation, very low-quality evidence).

It is clear that the optimal target temperature after cardiac arrest is not known and that more high-quality large trials are needed.[197]

When to control temperature? Whichever target temperature is selected, active temperature control is required to achieve and maintain the temperature in this range. Prior recommendations suggest that cooling should be initiated as soon as possible after ROSC, but this recommendation was based only on preclinical data and rational conjecture.[198] Animal data indicate that earlier cooling after ROSC produces better outcomes.[199,200] Observational studies are confounded by the fact that there is an association between patients who cool faster spontaneously and worse neurological outcome.[201–203] It is hypothesised that those with the most severe neurological injury are more prone to losing their ability to control body temperature.

Five randomised controlled trials used cold intravenous fluids after ROSC to induce hypothermia,[204–207] one trial used cold intravenous fluid during resuscitation,[208] and one trial used intra-arrest intranasal cooling.[209] The volume of cold fluid ranged from 20 to 30 ml kg^{-1} and up to 2 l, although some patients did not receive the full amount before arrival at hospital. All seven trials suffered from the unavoidable lack of blinding of the clinical team, and three also failed to blind the outcomes assessors. These trials showed no overall difference in mortality for patients treated with prehospital cooling (RR, 0.98; 95% CI 0.92–1.04) compared with

those who did not receive prehospital cooling. No individual trial found an effect on either poor neurological outcome or mortality.

Four RCTs provided low quality evidence for an increased risk of re-arrest among subjects who received prehospital induced hypothermia (RR, 1.22; 95% CI 1.01–1.46),[204,205,207] although this result was driven by data from the largest trial.[207] Three trials reported no pulmonary oedema in any group, two small pilot trials found no difference in the incidence of pulmonary oedema between groups,[204,208] and one trial showed an increase in pulmonary oedema in patients who received prehospital cooling (RR, 1.34; 95% CI 1.15–1.57).[207]

Based on this evidence, prehospital cooling using a rapid infusion of large volumes of cold intravenous fluid immediately after ROSC is not recommended. It may still be reasonable to infuse cold intravenous fluid where patients are well monitored and a lower target temperature (e.g., 33 °C) is the goal. Early cooling strategies, other than rapid infusion of large volumes of cold intravenous fluid, and cooling during cardiopulmonary resuscitation in the prehospital setting have not been studied adequately. Whether certain patient populations (e.g., patients for whom transport time to a hospital is longer than average) might benefit from early cooling strategies remains unknown.

How to control temperature? The practical application of TTM is divided into three phases: induction, maintenance and rewarming.[210] External and/or internal cooling techniques can be used to initiate and maintain TTM. If a target temperature of 36 °C is chosen, for the many post cardiac arrest patients who arrive in hospital with a temperature less than 36 °C, a practical approach is to let them rewarm spontaneously and to activate a TTM-device when they have reached 36 °C. The maintenance phase at 36 °C is the same as for other target temperatures; shivering, for example, does not differ between patients treated at 33 °C and 36 °C.[31] When using a target of 36 °C, the rewarming phase will be shorter.

If a lower target temperature, e.g., 33 °C is chosen, an infusion of 30 ml kg^{-1} of 4 °C saline or Hartmann's solution will decrease core temperature by approximately 1.0–1.5 °C.[206,207,211] However, in one prehospital randomised controlled trial this intervention was associated with increased pulmonary oedema (diagnosed on the initial chest radiograph) and an increased rate of re-arrest during transport to hospital.[207]

Methods of inducing and/or maintaining TTM include:

- Simple ice packs and/or wet towels are inexpensive; however, these methods may be more time consuming for nursing staff, may result in greater temperature fluctuations, and do not enable controlled rewarming.[11,19,188,212–219] Ice cold fluids alone cannot be used to maintain hypothermia,[220] but even the addition of simple ice packs may control the temperature adequately.[218]
- Cooling blankets or pads.[221–227]
- Water or air circulating blankets.[7,8,10,182,226,228–234]
- Water circulating gel-coated pads.[7,224,226,233,235–238]
- Transnasal evaporative cooling[209] – this technique enables cooling before ROSC and is undergoing further investigation in a large multicentre randomised controlled trial.[239]
- Intravascular heat exchanger, placed usually in the femoral or subclavian veins.[7,8,215,216,226,228,232,240–245]
- Extracorporeal circulation (e.g., cardiopulmonary bypass, ECMO).[246,247]

In most cases, it is easy to cool patients initially after ROSC because the temperature normally decreases within this first hour.[13,176] Admission temperature after OHCA is usually between 35 °C and 36 °C and in a recent large trial the median temperature was 35.3 °C.[31] If a target temperature of 36 °C is chosen allow a slow passive rewarm to 36 °C. If a target temperature of 33 °C is chosen, initial cooling is facilitated by neuromuscular blockade and sedation, which will prevent shivering.[248] Magnesium sulphate, a naturally occurring NMDA receptor antagonist, that reduces the shivering threshold slightly, can also be given to reduce the shivering threshold.[210,249]

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.[250] The temperature is typically monitored from a thermistor placed in the bladder and/or oesophagus.[210,251,252] As yet, there are no data indicating that any specific cooling technique increases survival when compared with any other cooling technique; however, internal devices enable more precise temperature control compared with external techniques.[226,250]

Plasma electrolyte concentrations, effective intravascular volume and metabolic rate can change rapidly during rewarming, as they do during cooling. Rebound hyperthermia is associated with worse neurological outcome.[179,180] Thus, rewarming should be achieved slowly: the optimal rate is not known, but the consensus is currently about 0.25–0.5 °C of rewarming per hour.[228] Choosing a strategy of 36 °C will reduce this risk.[31]

Physiological effects and side effects of hypothermia. The well-recognised physiological effects of hypothermia need to be managed carefully[210]:

- Shivering will increase metabolic and heat production, thus reducing cooling rates – strategies to reduce shivering are discussed above. The occurrence of shivering in cardiac arrest survivors who undergo mild induced hypothermia is associated with a good neurological outcome[253,254]; it is a sign of a normal physiological response. Occurrence of shivering was similar at a target temperature of 33 °C and 36 °C.[31] A sedation protocol is required.
- Mild induced hypothermia increases systemic vascular resistance and causes arrhythmias (usually bradycardia).[241] Importantly, the bradycardia caused by mild induced hypothermia may be beneficial (similar to the effect achieved by beta-blockers); it reduces diastolic dysfunction[117] and its occurrence has been associated with good neurological outcome.[118,119]
- Mild induced hypothermia causes a diuresis and electrolyte abnormalities such as hypophosphataemia, hypokalaemia, hypomagnesaemia and hypocalcaemia.[31,210,255]
- Hypothermia decreases insulin sensitivity and insulin secretion, and causes hyperglycaemia,[188] which will need treatment with insulin (see glucose control).
- Mild induced hypothermia impairs coagulation and may increase bleeding, although this effect seems to be negligible[256] and has not been confirmed in clinical studies.[7,31,187] In one registry study, an increased rate of minor bleeding occurred with the combination of coronary angiography and mild induced hypothermia, but this combination of interventions was the also the best predictor of good outcome.[20]
- Hypothermia can impair the immune system and increase infection rates.[210,217,222] Mild induced hypothermia is associated with an increased incidence of pneumonia[257,258]; however, this seems to have no impact on outcome. Although prophylactic antibiotic treatment has not been studied prospectively, in an observational study, use of prophylactic antibiotics was associated with a reduced incidence of pneumonia.[259] In another observational study of 138 patients admitted to ICU after OHCA, early use of antibiotics was associated with improved survival.[260]

- The serum amylase concentration is commonly increased during hypothermia but the significance of this unclear.
- The clearance of sedative drugs and neuromuscular blockers is reduced by up to 30% at a core temperature of 34 °C.[261] Clearance of sedative and other drugs will be closer to normal at a temperature closer to 37.0 °C.

Contraindications to targeted temperature management. Generally recognised contraindications to TTM at 33 °C, but which are not applied universally, include: severe systemic infection and pre-existing medical coagulopathy (fibrinolytic therapy is not a contraindication to mild induced hypothermia). Two observational studies documented a positive inotropic effect from mild induced hypothermia in patients in cardiogenic shock,[262,263] but in the TTM study there was no difference in mortality among patients with mild shock on admission who were treated with a target temperature of 33 °C compared with 36 °C.[194] Animal data also indicate improved contractile function with mild induced hypothermia probably because of increased Ca^{2+} sensititvity.[264]

Other therapies

Neuroprotective drugs (Coenzyme Q10,[223] thiopental,[153] glucocorticoids,[123,265] nimodipine,[266,267] lidoflazine[268] or diazepam[154]) used alone, or as an adjunct to mild induced hypothermia, have not been shown to increase neurologically intact survival when included in the post arrest treatment of cardiac arrest. The combination of xenon and mild induced hypothermia has been studied in a feasibility trial and is undergoing further clinical evaluation.[269]

Prognostication

This section has been adapted from the Advisory Statement on Neurological Prognostication in comatose survivors of cardiac arrest,[270] written by members of the ERC ALS Working Group and of the Trauma and Emergency Medicine (TEM) Section of the European Society of Intensive Care Medicine (ESICM), in anticipation of the 2015 Guidelines.

Hypoxic-ischaemic brain injury is common after resuscitation from cardiac arrest.[271] Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury; this has been shown both before[28] and after[27,30,31] the implementation of target temperature management (TTM) for post-resuscitation care. Most of these deaths are due to active withdrawal of life sustaining treatment (WLST) based on prognostication of a poor neurological outcome.[27,30] For this reason, when dealing with patients who are comatose after resuscitation from cardiac arrest minimising the risk of a falsely pessimistic prediction is essential. Ideally, when predicting a poor outcome the false positive rate (FPR) should be zero with the narrowest possible confidence interval (CI). However, most prognostication studies include so few patients that even if the FPR is 0%, the upper limit of the 95% CI is often high.[272,273] Moreover, many studies are confounded by self-fulfilling prophecy, which is a bias occurring when the treating physicians are not blinded to the results of the outcome predictor and use it to make a decision on WLST.[272,274] Finally, both TTM itself and sedatives or neuromuscular blocking drugs used to maintain it may potentially interfere with prognostication indices, especially those based on clinical examination.[156]

Clinical examination

Bilateral absence of pupillary light reflex at 72 h from ROSC predicts poor outcome with close to 0% FPR, both in TTM-treated and in non-TTM-treated patients (FPR 1 [0–3] and 0 [0–8], respectively)[156,275–284] and a relatively low sensitivity (19% and 18%, respectively). Similar performance has been documented for bilaterally absent corneal reflex.[272,273]

In non-TTM-treated patients[276,285] an absent or extensor motor response to pain at 72 h from ROSC has a high (74 [68–79]%) sensitivity for prediction of poor outcome, but the FPR is also high (27 [12–48]%). Similar results were observed in TTM-treated patients.[156,277–280,282–284,286–288] Nevertheless, the high sensitivity of this sign may enable it to be used to identify the population with poor neurological status needing prognostication. Like the corneal reflex, the motor response can be suppressed by sedatives or neuromuscular blocking drugs.[156] When interference from residual sedation or paralysis is suspected, prolonging observation of these clinical signs beyond 72 h from ROSC is recommended, in order to minimise the risk of obtaining false positive results.

Myoclonus is a clinical phenomenon consisting of sudden, brief, involuntary jerks caused by muscular contractions or inhibitions. A prolonged period of continuous and generalised myoclonic jerks is commonly described as status myoclonus. Although there is no definitive consensus on the duration or frequency of myoclonic jerks required to qualify as status myoclonus, in prognostication studies in comatose survivors of cardiac arrest the minimum reported duration is 30 min. The names and definitions used for status myoclonus vary among those studies.

While the presence of myoclonic jerks in comatose survivors of cardiac arrest is not consistently associated with poor outcome (FPR 9%),[145,272] a status myoclonus starting within 48 h from ROSC was consistently associated with a poor outcome (FPR 0 [0–5]%; sensitivity 8%) in prognostication studies made in non-TTM-treated patients,[276,289,290] and is also highly predictive (FPR 0% [0–4]; sensitivity 16%) in TTM-treated patients.[144,156,291] However, several case reports of good neurological recovery despite an early-onset, prolonged and generalised myoclonus have been published. In some of these cases myoclonus persisted after awakening and evolved into a chronic action myoclonus (Lance–Adams syndrome).[292–297] In others it disappeared with recovery of consciousness.[298,299] The exact time when recovery of consciousness occurred in these cases may have been masked by the myoclonus itself and by ongoing sedation. Patients with post-arrest status myoclonus should be evaluated off sedation whenever possible; in those patients, EEG recording can be useful to identify EEG signs of awareness and reactivity and to reveal a coexistent epileptiform activity.

While predictors of poor outcome based on clinical examination are inexpensive and easy to use, they cannot be concealed from the treating team and therefore their results may potentially influence clinical management and cause a self-fulfilling prophecy. Clinical studies are needed to evaluate the reproducibility of clinical signs used to predict outcome in comatose post-arrest patients.

Electrophysiology

Short-latency somatosensory evoked potentials (SSEPs)

In non-TTM-treated post-arrest comatose patients, bilateral absence of the N20 SSEP wave predicts death or vegetative state (CPC 4–5) with 0 [0–3]% FPR as early as 24 h from ROSC,[276,300,301] and it remains predictive during the following 48 h with a consistent sensitivity (45–46%).[276,300,302–304] Among a total of 287 patients with absent N20 SSEP wave at ≤72 h from ROSC, there was only one false positive result (positive predictive value 99.7 [98–100]%).[305]

In TTM-treated patients, bilateral absence of the N20 SSEP wave is also very accurate in predicting poor outcome both during mild induced hypothermia[278,279,301,306] (FPR 2 [0–4]%) and after rewarming[277,278,286,288,304] (FPR 1 [0–3]%). The few cases of false reports observed in large patient cohorts were due mainly

to artefacts.[279,284] SSEP recording requires appropriate skills and experience, and utmost care should be taken to avoid electrical interference from muscle artefacts or from the ICU environment. Interobserver agreement for SSEPs in anoxic–ischaemic coma is moderate to good but is influenced by noise.[307,308]

In most prognostication studies bilateral absence of N20 SSEP has been used as a criterion for deciding on withdrawal of life-sustaining treatment (WLST), with a consequent risk of self-fulfilling prophecy.[272] SSEP results are more likely to influence physicians' and families' WLST decisions than those of clinical examination or EEG.[309]

Electroencephalography

Absence of EEG reactivity. In TTM-treated patients, absence of EEG background reactivity predicts poor outcome with 2 [1–7]% FPR[288,310,311] during TH and with 0 [0–3]% FPR[286,288,310] after rewarming at 48–72 h from ROSC. However, in one prognostication study in posthypoxic myoclonus three patients with no EEG reactivity after TTM had a good outcome.[144] Most of the prognostication studies on absent EEG reactivity after cardiac arrest are from the same group of investigators. Limitations of EEG reactivity include lack of standardisation as concerns the stimulation modality and modest interrater agreement.[312]

Status epilepticus. In TTM-treated patients, the presence of status epilepticus (SE), i.e., a prolonged epileptiform activity, during TH or immediately after rewarming[150,291,313] is almost invariably – but not always – followed by poor outcome (FPR from 0% to 6%), especially in presence of an unreactive[150,314] or discontinuous EEG background.[75] All studies on SE included only a few patients. Definitions of SE were inconsistent among those studies.

Burst-suppression. Burst-suppression has recently been defined as more than 50% of the EEG record consisting of periods of EEG voltage <10 μV, with alternating bursts.[148] However, most of prognostication studies do not comply with this definition.

In comatose survivors of cardiac arrest, either TTM-treated or non-TH-treated, burst-suppression is usually a transient finding. During the first 24–48 h after ROSC[305] in non-TTM-treated patients or during hypothermia in TTM-treated patients[288,306,315] burst-suppression may be compatible with neurological recovery while at ≥72 h from ROSC[75,276,316] a persisting burst-suppression pattern is consistently associated with poor outcome. Limited data suggest that specific patterns like a pattern of identical bursts[317] or association with status epilepticus[75] have very high specificity for prediction of poor outcome.

Apart from its prognostic significance, recording of EEG – either continuous or intermittent – in comatose survivors of cardiac arrest both during TH and after rewarming is helpful to assess the level of consciousness – which may be masked by prolonged sedation, neuromuscular dysfunction or myoclonus – and to detect and treat non-convulsive seizures[318] which may occur in about one quarter of comatose survivors of cardiac arrest.[75,149,291]

Biomarkers

NSE and S-100B are protein biomarkers that are released following injury to neurons and glial cells, respectively. Their blood values after cardiac arrest are likely to correlate with the extent of anoxic–ischaemic neurological injury and, therefore, with the severity of neurological outcome. S-100B is less well documented than is NSE.[319] Advantages of biomarkers over both EEG and clinical examination include quantitative results and likely independence from the effects of sedatives. Their main limitation as prognosticators is that it is difficult to find a consistent threshold for identifying patients destined to a poor outcome with a high degree of certainty. In fact, serum concentrations of biomarkers are per se continuous variables, which limits their applicability for predicting a dichotomous outcome, especially when a threshold for 0% FPR is desirable.

Neuron-specific enolase (NSE)

In non-TTM-treated patients the NSE threshold for prediction of poor outcome with 0% FPR at days 24–72 from ROSC was 33 mcg l^{-1} or less in some studies.[276,320,321] However, in other studies this threshold was 47.6 mcg l^{-1} at 24 h, 65.0 mcg l^{-1} at 48 h and 90.9 mcg l^{-1} at 72 h.[302]

In TTM-treated patients the threshold for 0% FPR varied between 49.6 mcg l^{-1} and 151.4 mcg l^{-1} at 24 h,[313,322–326] between 25 mcg l^{-1} and 151.5 mcg l^{-1} at 48 h,[279,313,322–329] and between 57.2 mcg l^{-1} and 78.9 mcg l^{-1} at 72 h.[321,324,327]

The main reasons for the observed variability in NSE thresholds include the use of heterogeneous measurement techniques (variation between different analysers),[330–332] the presence of extra-neuronal sources of biomarkers (haemolysis and neuroendocrine tumours),[333] and the incomplete knowledge of the kinetics of its blood concentrations in the first few days after ROSC. Limited evidence suggests that the discriminative value of NSE levels at 48–72 h is higher than at 24 h.[323,325,334] Increasing NSE levels over time may have an additional value in predicting poor outcome.[323,324,334] In a secondary analysis of the TTM trial, NSE values were measured at 24, 48 and 72 h in 686 patients; an increase in NSE values between any two points was associated with a poor outcome.[335]

Imaging

Brain CT

The main CT finding of global anoxic–ischaemic cerebral insult following cardiac arrest is cerebral oedema,[133] which appears as a reduction in the depth of cerebral sulci (sulcal effacement) and an attenuation of the grey matter/white matter (GM/WM) interface, due to a decreased density of the GM, which has been quantitatively measured as the ratio (GWR) between the GM and the WM densities. The GWR threshold for prediction of poor outcome with 0% FPR in prognostication studies ranged between 1.10 and 1.22.[281,325,336] The methods for GWR calculation were inconsistent among studies.

MRI

MRI changes after global anoxic–ischaemic brain injury due to cardiac arrest appear as a hyperintensity in cortical areas or basal ganglia on diffusion weighted imaging (DWI) sequences. In two small studies,[337,338] the presence of large multilobar changes on DWI or FLAIR MRI sequences performed within five days from ROSC was consistently associated with poor outcome while focal or small volume lesions were not.[329]

Apparent diffusion coefficient (ADC) is a quantitative measure of ischaemic DWI changes. ADC values between 700 and 800×10^{-6} mm^2 s^{-1} are considered to be normal.[339] Brain ADC measurements used for prognostication include whole-brain ADC,[340] the proportion of brain volume with low ADC[341] and the lowest ADC value in specific brain areas, such as the cortical occipital area and the putamen.[322,342] The ADC thresholds associated with 0% FPR vary among studies. These methods depend partly on subjective human decision in identifying the region of interest to be studied and in the interpretation of results, although automated analysis has recently been proposed.[343]

Advantages of MRI over brain CT include a better spatial definition and a high sensitivity for identifying ischaemic brain injury; however, its use can be problematic in the most clinically unstable patients.[339] MRI can reveal extensive changes when results of other predictors such as SSEP or ocular reflexes are normal.[329,339]

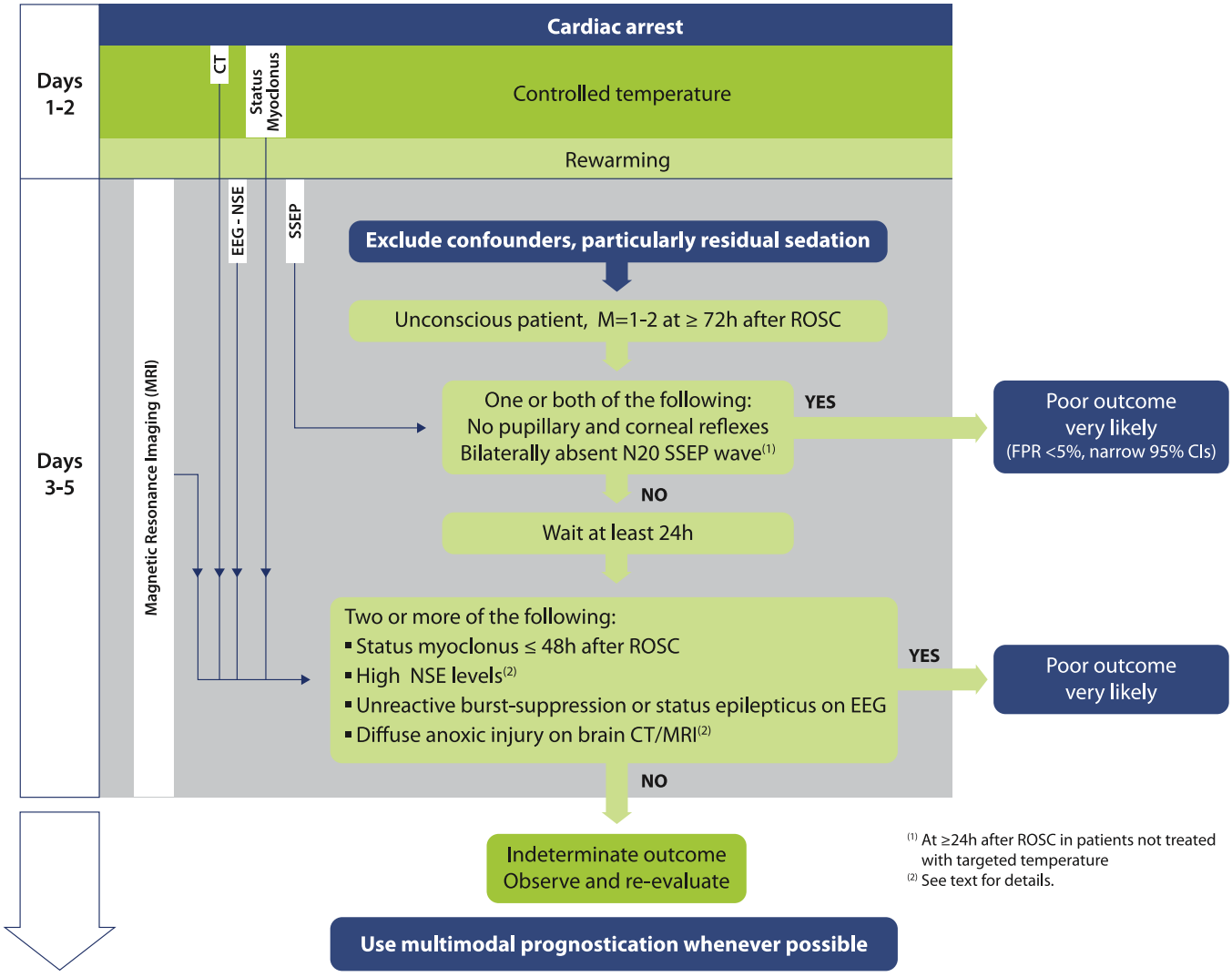

Fig. 5.2. Prognostication strategy algorithm. EEG: electroencephalography; NSE: neuron-specific enolase; SSEP: somatosensory evoked potentials; ROSC: return of spontaneous circulation; FPR: false positive rate; CI: confidence interval.

All studies on prognostication after cardiac arrest using imaging have a small sample size with a consequent low precision, and a very low quality of evidence. Most of those studies are retrospective, and brain CT or MRI had been requested at the discretion of the treating physician, which may have caused a selection bias and overestimated their performance.

Suggested prognostication strategy

A careful clinical neurological examination remains the foundation for prognostication of the comatose patient after cardiac arrest.[344] Perform a thorough clinical examination daily to detect signs of neurological recovery such as purposeful movements or to identify a clinical picture suggesting that brain death has occurred.

The process of brain recovery following global post-anoxic injury is completed within 72 h from arrest in most patients.[290,345] However, in patients who have received sedatives ≤12 h before the 72 h post ROSC neurological assessment, the reliability of clinical examination may be reduced.[156] Before decisive assessment is performed, major confounders must be excluded[346,347]; apart from sedation and neuromuscular blockade, these include hypothermia, severe hypotension, hypoglycaemia, and metabolic and respiratory derangements. Suspend sedatives and neuromuscular blocking drugs for long enough to avoid interference with clinical examination. Short-acting drugs are preferred whenever possible. When residual sedation/paralysis is suspected, consider using antidotes to reverse the effects of these drugs.

The prognostication strategy algorithm (Fig. 2) is applicable to all patients who remain comatose with an absent or extensor motor response to pain at ≥72 h from ROSC. Results of earlier prognostic tests are also considered at this time point.

Evaluate the most robust predictors first. These predictors have the highest specificity and precision (FPR <5% with 95% CIs <5% in patients treated with controlled temperature) and have been documented in >5 studies from at least three different groups of investigators. They include bilaterally absent pupillary reflexes at ≥72 h from ROSC and bilaterally absent SSEP N20 wave after rewarming (this last sign can be evaluated at ≥24 h from ROSC in patients who have not been treated with controlled temperature). Based on expert opinion, we suggest combining the absence of pupillary reflexes with those of corneal reflexes for predicting poor outcome at this time point. Ocular reflexes and SSEPs maintain their predictive value irrespective of target temperature.[283,284]

If none of the signs above is present to predict a poor outcome, a group of less accurate predictors can be evaluated, but the degree of confidence in their prediction will be lower. These have FPR <5% but wider 95% CIs than the previous predictors, and/or their definition/

threshold is inconsistent in prognostication studies. These predictors include the presence of early status myoclonus (within 48 h from ROSC), high values of serum NSE at 48–72 h after ROSC, an unreactive malignant EEG pattern (burst-suppression, status epilepticus) after rewarming, the presence of a marked reduction of the GM/WM ratio or sulcal effacement on brain CT within 24 h after ROSC or the presence of diffuse ischaemic changes on brain MRI at 2–5 days after ROSC. Based on expert opinion, we suggest waiting at least 24 h after the first prognostication assessment and confirming unconsciousness with a Glasgow motor score of 1–2 before using this second set of predictors. We also suggest combining at least *two* of these predictors for prognostication.

No specific NSE threshold for prediction of poor outcome with 0% FPR can be recommended at present. Ideally, every hospital laboratory assessing NSE should create its own normal values and cut-off levels based on the test kit used. Sampling at multiple timepoints is recommended to detect trends in NSE levels and to reduce the risk of false positive results.[335] Care should be taken to avoid haemolysis when sampling NSE.

Although the most robust predictors showed no false positives in most studies, none of them singularly predicts poor outcome with absolute certainty when the relevant comprehensive evidence is considered. Moreover, those predictors have often been used for WLST decisions, with the risk of a self-fulfilling prophecy. For this reason, we recommend that prognostication should be multimodal whenever possible, even in presence of one of these predictors. Apart from increasing safety, limited evidence also suggests that multimodal prognostication increases sensitivity.[286,311,325,348]

When prolonged sedation and/or paralysis is necessary, for example, because of the need to treat severe respiratory insufficiency, we recommend postponing prognostication until a reliable clinical examination can be performed. Biomarkers, SSEP and imaging studies may play a role in this context, since they are insensitive to drug interference.

When dealing with an uncertain outcome, clinicians should consider prolonged observation. Absence of clinical improvement over time suggests a worse outcome. Although awakening has been described as late as 25 days after arrest,[291,298,349] most survivors will recover consciousness within one week.[31,329,350–352] In a recent observational study,[351] 94% of patients awoke within 4.5 days from rewarming and the remaining 6% awoke within ten days. Even those awakening late, can still have a good neurological outcome.[351]

Rehabilitation

Although neurological outcome is considered to be good for the majority of cardiac arrest survivors, cognitive and emotional problems and fatigue are common.[23,24,279,353–356] Long-term cognitive impairments are present in half of survivors.[22,357,358] Memory is most frequently affected, followed by problems in attention and executive functioning (planning and organisation).[23,359] The cognitive impairments can be severe, but are mostly mild.[22] In one study, of 796 OHCA survivors who had been employed before their cardiac arrest, 76.6% returned to work.[360] Mild cognitive problems are often not recognised by health care professionals and cannot be detected with standard outcome scales such as the Cerebral Performance Categories (CPC) or the Mini-Mental State Examination (MMSE).[24,361] Emotional problems, including depression, anxiety and posttraumatic stress are also common.[362,363] Depression is present in 14–45% of the survivors, anxiety in 13–61% and symptoms of posttraumatic stress occur in 19–27%.[355] Fatigue is also a complaint that is often reported after cardiac arrest. Even several years after a cardiac arrest, 56% of the survivors suffer severe fatigue.[356]

It is not only the patients who experience problems; their partners and caregivers can feel highly burdened and often have emotional problems, including symptoms of posttraumatic stress.[356,364] After hospital discharge both survivors and caregivers frequently experience a lack of information on important topics including physical and emotional challenges, implantable cardioverter defibrillators (ICD), regaining daily activities, partner relationships and dealing with health care providers.[365] A systematic review on coronary heart disease patients also showed the importance of active information supply and patient education.[366]

Both cognitive and emotional problems have significant impact and can affect a patient's daily functioning, return to work and quality of life.[356,367,368] Therefore, follow-up care after hospital discharge is necessary. Although the evidence on the rehabilitation phase appears scarce, three randomised controlled trials have shown that the outcome after cardiac arrest can be improved.[369–371] First, an eleven-session nursing intervention reduced cardiovascular mortality and depressive symptoms. It did so by focussing on physiological relaxation, self-management, coping strategies and health education.[369] Another nursing intervention was found to improve physical symptoms, anxiety, self-confidence and disease knowledge.[370,371] This intervention consisted of eight telephone sessions, a 24/7 nurse pager system and an information booklet and was directed at improving self-efficacy, outcome efficacy expectations and enhancing self-management behavioural skills.[372] A third intervention called 'Stand still. . ., and move on', improved overall emotional state, anxiety and quality of life, and also resulted in a faster return to work.[373] This intervention aimed to screen early for cognitive and emotional problems, to provide information and support, to promote self-management and to refer to specialised care, if needed.[374,375] It generally consisted of only one or two consultations with a specialised nurse and included supply of a special information booklet.

The organisation of follow-up after cardiac arrest varies widely between hospitals and countries in Europe. Follow-up care should be organised systematically and can be provided by a physician or specialised nurse. It includes at least the following aspects:

Screening for cognitive impairments. There is currently no gold standard on how to perform such screening. A good first step would be to ask the patient and a relative or caregiver about cognitive complaints (for example problems with memory, attention, planning). If feasible, administer a structured interview or checklist, such as the Checklist Cognition and Emotion,[376] or a short cognitive screening instrument, such as the Montreal Cognitive Assessment (MoCA) (freely available in many languages at http://www.mocatest.org). In cases where there are signs of cognitive impairments, refer to a neuropsychologist for neuropsychological assessment or to a specialist in rehabilitation medicine for a rehabilitation programme.[377]

Screening for emotional problems. Ask whether the patient experiences any emotional problems, such as symptoms of depression, anxiety or posttraumatic stress. General measures that can be used include the Hospital Anxiety and Depression Scale (HADS) and the Impact of Event Scale.[378,379] In case of emotional problems refer to a psychologist or psychiatrist for further examination and treatment.[355]

Provision of information. Give active information on the potential non-cardiac consequences of a cardiac arrest including cognitive impairment, emotional problems and fatigue. Other topics that can be addressed include heart disease, ICDs, regaining daily activities, partner relationships and sexuality, dealing with health care providers and caregiver strain.[365] It is best to combine written information with the possibility for personal consultation. An example of an information booklet is available (in Dutch and English).[373,374]

Organ donation

Organ donation should be considered in those who have achieved ROSC and who fulfil criteria for death using neurological criteria.[380] In those comatose patients in whom a decision is made to withdraw life-sustaining therapy, organ donation should be considered after circulatory death occurs. Organ donation can also be considered in individuals where CPR is not successful in achieving ROSC. All decisions concerning organ donation must follow local legal and ethical requirements, as these vary in different settings.

Non-randomised studies have shown that graft survival at one year is similar from donors who have had CPR compared with donors who have not had CPR: adult hearts (3230 organs[381–387]), adult lungs (1031 organs[383,385,388]), adult kidneys (5000 organs[381,383]), adult livers (2911 organs[381,383]), and adult intestines (25 organs[383]).

Non-randomised studies have also shown that graft survival at one year was similar when organs recovered from donors with ongoing CPR were compared to other types of donors for adult kidneys (199 organs[389–391]) or adult livers (60 organs[390,392,393]).

Solid organs have been successfully transplanted after circulatory death. This group of patients offers an opportunity to increase the organ donor pool. Organ retrieval from donation after circulatory death (DCD) donors is classified as controlled or uncontrolled.[394,395] Controlled donation occurs after planned withdrawal of treatment following non-survivable injuries and illnesses. Uncontrolled donation describes donation from patients with unsuccessful CPR in whom a decision has been made that CPR should be stopped. Once death has been diagnosed, the assessment of which includes a pre-defined period of observation to ensure a spontaneous circulation does not return,[396] organ preservation and retrieval takes place. Aspects or uncontrolled organ donation are complex and controversial as some of the same techniques used during CPR to attempt to achieve ROSC are also used for organ preservation after death has been confirmed, e.g., mechanical chest compression and extracorporeal circulation. Locally agreed protocols must therefore be followed.

Screening for inherited disorders

Many sudden death victims have silent structural heart disease, most often coronary artery disease, but also primary arrhythmia syndromes, cardiomyopathies, familial hypercholesterolaemia and premature ischaemic heart disease. Screening for inherited disorders is crucial for primary prevention in relatives as it may enable preventive antiarrhythmic treatment and medical follow-up.[397–399] This screening should be performed using clinical examination, electrophysiology and cardiac imaging. In selected cases, genetic mutations associated with inherited cardiac diseases should also be searched.[400]

Cardiac arrest centres

There is wide variability in survival among hospitals caring for patients after resuscitation from cardiac arrest.[9,13,16,17,401–403] Many studies have reported an association between survival to hospital discharge and transport to a cardiac arrest centre but there is inconsistency in the hospital factors that are most related to patient outcome.[4,5,9,17,401,404–416] There is also inconsistency in the services that together define a cardiac arrest centre. Most experts agree that such a centre must have a cardiac catheterisation laboratory that is immediately accessible 24/7 and the facility to provide targeted temperature management. The availability of a neurology service that can provide neuroelectrophysiological monitoring (electroencephalography (EEG)) and investigations (e.g., EEG and somatosensory evoked potentials (SSEPs)) is also essential.

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[17]; however, differences in case mix could account for these differences. An observational study showed that unadjusted survival to discharge was greater in hospitals that received ≥40 cardiac arrest patients/year compared with those that received <40 per year, but this difference disappeared after adjustment for patient factors.[417]

Several studies with historic control groups have shown improved survival after implementation of a comprehensive package of post-resuscitation care that includes mild induced hypothermia and percutaneous coronary intervention.[7,10,11,418] There is also evidence of improved survival after out-of-hospital cardiac arrest in large hospitals with cardiac catheter facilities compared with smaller hospitals with no cardiac catheter facilities.[9] In a study of 3981 patients arriving with a sustained pulse at one of 151 hospitals, the Resuscitation Outcome Consortium (ROC) investigators have shown that early coronary intervention and mild induced hypothermia were associated with a favourable outcome.[84] These interventions were more frequent in hospitals that treated higher number of OHCA patients per year.

Several studies of OHCA arrest failed to demonstrate any effect of transport interval from the scene to the receiving hospital on survival to hospital discharge if ROSC was achieved at the scene and transport intervals were short (3–11 min).[406,412,413] This implies that it may be safe to bypass local hospitals and transport the post-cardiac arrest patient to a regional cardiac arrest centre. There is indirect evidence that regional cardiac resuscitation systems of care improve outcome after ST elevation myocardial infarction (STEMI).[407,419–442]

The implication from all these data is that specialist cardiac arrest centres and systems of care may be effective.[443–446] Despite the lack of high quality data to support implementation of cardiac arrest centres, it seems likely that regionalisation of post-cardiac arrest care will be adopted in most countries.

Conflicts of interest

Jerry P. Nolan	Editor-in-Chief Resuscitation
Alain Cariou	Speakers honorarium BARD-France
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European Resuscitation Council Guidelines for Resuscitation 2015 Section 6. Paediatric life support

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Introduction

These guidelines on paediatric life support are based on three main principles: (1) the incidence of critical illness, particularly cardiopulmonary arrest, and injury in children is much lower than in adults; (2) the illnesses and pathophysiological responses of paediatric patients often differ from those seen in adults; (3) many paediatric emergencies are managed primarily by providers who are not paediatric specialists and who have limited paediatric emergency medical experience. Therefore, guidelines on paediatric life support must incorporate the best available scientific evidence but must also be simple and feasible. Finally, international guidelines need to acknowledge the variation in national and local emergency medical infrastructures and allow flexibility when necessary.

The process

The European Resuscitation Council (ERC) published guidelines for paediatric life support (PLS) in 1994, 1998, 2000, 2005 and 2010.[1–5] The latter three were based on the paediatric work of the International Consensus on Science published by the International Liaison Committee on Resuscitation (ILCOR).[6–10] This process was repeated in 2014/2015, and the resulting Consensus on Science with Treatment Recommendations (CoSTR) was published simultaneously in Resuscitation, Circulation and Pediatrics using the GRADE process.[11–13] The PLS Writing Group of the ERC has developed the ERC PLS Guidelines based on the 2015 CoSTR and supporting scientific literature. The guidelines for resuscitation of Babies at Birth are covered in the ERC GL2015 Babies at Birth.[14] Information pertaining to children are also found in the ERC GL2015 First Aid,[15] the ERC GL2015 chapter on Education[16] and in the GL2015 chapter on the Ethics of Resuscitation and End-of-Life Decisions.[17]

Summary of changes since 2010 Guidelines

Guideline changes have been made in response to convincing new scientific evidence and, by using clinical, organisational and educational findings, they have been adapted to promote their use and ease for teaching.

The 2015 ILCOR process was informed by librarians who helped paediatric experts in performing in-depth systematic searches on 21 different key questions relating to paediatric resuscitation. Relevant adult literature was also considered and, in a few cases, extrapolated to the paediatric questions when they overlapped with other Task Forces, or when there were insufficient paediatric data. In rare circumstances, appropriate animal studies were incorporated into reviews of the literature. However, these data were considered only when higher levels of evidence were not available. The topic areas that the paediatric COSTR questions dealt with related to: pre-cardiac arrest care, basic life support care, advanced life support during cardiac arrest and post-resuscitation care.

As in previous ILCOR deliberations, there remains a paucity of good-quality evidence on paediatric resuscitation with many gaps in knowledge about paediatric resuscitation having been identified in this round of the CoSTR process.

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These ERC GL2015 have included the recommendations from the ILCOR CoSTR 2015, updating the scientific base in addition to these recommendations and accompanied by points of clarification on matters about which there have been questions since 2010.[12,13]

This section of the ERC GL 2015 on Paediatric Life Support includes:

- Basic life support.
- Management of foreign bodies in the airway.
- Prevention of cardiac arrest.
- Advanced life support during cardiac arrest.
- Post resuscitation care.

New topics in the ERC GL2015 include those from CoSTR recommendations as well as the deliberations of the PLS Writing Group of the ERC.

These include:

In BLS

- The duration of delivering a breath is about 1 s, to coincide with adult practice.
- For chest compressions, the lower sternum should be depressed by at least one third the anterior–posterior diameter of the chest, or by 4 cm for the infant and 5 cm for the child.

In managing the seriously ill child

- If there are no signs of septic shock, then children with a febrile illness should receive fluid with caution and reassessment following its administration. In some forms of septic shock, restricting fluids with isotonic crystalloid may be better than the liberal use of fluids.
- For cardioversion of a supraventricular tachycardia (SVT), the initial dose has been revised to 1 J kg^{-1}.

In the paediatric cardiac arrest algorithm

- Many of the features are now common with adult practice.

In post resuscitation care

- Preventing fever in children who have return of spontaneous circulation (ROSC) from an out-of-hospital setting.
- Targeted temperature management of children post ROSC should comprise treatment with either normothermia or mild hypothermia.
- There is no single predictor for when to stop resuscitation.

Terminology

In the following text the masculine includes the feminine and child refers to both infants and children unless noted otherwise. The term newly born refers to a neonate immediately after delivery. A neonate is an infant within 4 weeks of being born. An infant is a child under one year of age (but does not include newly borns) and the term child refers to children between 1 year and onset of puberty. From puberty children are referred to as adolescents for whom the adult guidelines apply. Furthermore, it is necessary to differentiate between infants and older children, as there are some important differences with respect to diagnostic and interventional techniques between these two groups. The onset of puberty, which is the physiological end of childhood, is the most logical landmark for the upper age limit for use of paediatric guidance. If rescuers believe the victim to be a child they should use the paediatric guidelines. If a misjudgement is made and the victim turns out to be a young adult, little harm will accrue, as studies of aetiology have shown that the paediatric pattern of cardiac arrest continues into early adulthood.[18]

The terms paediatrician and paediatric nurse are used in this text as a generic term to represent clinicians who routinely manage ill or injured children, and could apply to others trained in the delivery of paediatric care, such as emergency department clinicians, or Paediatric Intensive Care Unit (PICU) specialists/paediatric anaesthetists.

Healthcare professionals are those people who look after patients and should have a higher level of training than lay people. This term relates particularly to the delivery of basic life support.

Paediatric basic life support

From the ILCOR CoSTR statement on the sequence for manoeuvres in BLS, there was found to be equipoise between the CAB sequence (compression for circulation, airway and breathing) and the ABC sequence (airway, breathing and compression for circulation).[19–21] Given that the ABC sequence has become an established and well recognised method for the delivery of CPR to children in Europe, the ERC PLS Writing Group determined that the use of this sequence should continue, particularly as the previous guidelines have led to its instruction to many hundreds of thousands of healthcare providers and lay people. This position will continue to be reviewed on the basis of any new knowledge that may be forthcoming.

Sequence of actions in BLS

Bystander CPR is associated with a better neurological outcome in adults and children.[22–26]

Rescuers who have been taught adult BLS or the chest compression-only sequence and have no specific knowledge of paediatric resuscitation may use this, as the outcome is worse if they do nothing. However, it is better to provide rescue breaths as part of the resuscitation sequence when applied to children as the asphyxial nature of most paediatric cardiac arrests necessitates ventilation as part of effective CPR.[25,26]

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 one minute of CPR before they go for help (see adult BLS guidelines).

BLS 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 professionals) (Fig. 6.1).

Although the following sequence describes expired air ventilation, health professionals with a responsibility for treating children will usually have access to, and training in the use of bag mask ventilation systems (BMV), and these should be used to provide rescue breaths.

1. Ensure the safety of rescuer and child.
2. Check the child's responsiveness.

- Stimulate the child and ask loudly: Are you all right?

3A. If the child responds by answering, crying or moving:

- Leave the child in the position in which you find him (provided he is not in further danger).
- Check his condition and call for help.
- Reassess him regularly.

Fig. 6.1. Paediatric basic life support algorithm.

3B. If the child does not respond:

- Shout for help.
- Turn the child carefully 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. This is especially important in infants.
- If you still have difficulty in opening the airway, try a jaw thrust: place the first two fingers of each hand behind each side of the child's mandible and push the jaw forward.

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

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

Fig. 6.2. Mouth to mouth and nose ventilation–infant.

- *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). If there is a history of trauma, cervical spine injury should be considered.
- Send or go for help—call the emergency services.
- Check for continued breathing.

5B. If breathing is not normal or absent:

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

Rescue breaths for an infant (Fig. 6.2)

- Ensure a neutral position of the head as an infant's head is usually flexed when supine, this may require some extension (a rolled towel/blanket under the upper part of the body may help to maintain the position) 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 for about 1 s, sufficient to make the chest visibly rise.
- Maintain head position and chin lift, take your mouth away from the victim and watch for his chest to fall as air comes out.
- Take another breath and repeat this sequence five times.

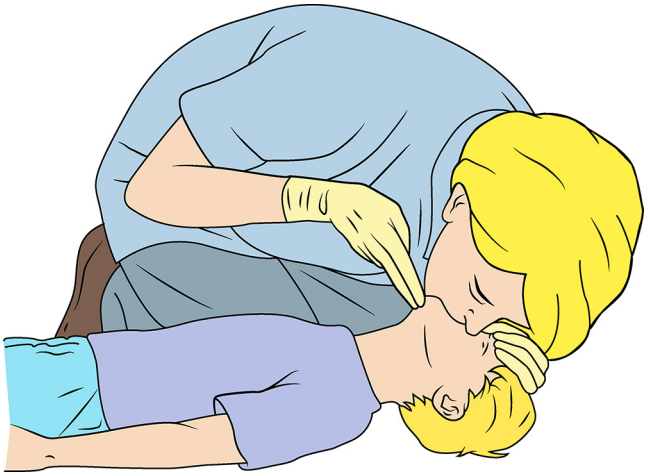

Fig. 6.3. Mouth to mouth ventilation–child.

Rescue breaths for a child over 1 year of age (Fig. 6.3):

- 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 for about 1 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.
- 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.
- Reposition the head. 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 (gasps or infrequent, irregular breaths are abnormal). If you check the pulse, ensure that you take no more than 10 s. Pulse check is unreliable and therefore the complete picture of how the patient appears must guide whether BLS is required, i.e. if there are no signs of life, start BLS.[27,28]

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 his side (into the recovery position, with caution if there is a history of trauma) if he remains unconscious.
- Re-assess the child frequently.

7B. If there are no signs of life

- Start chest compressions.

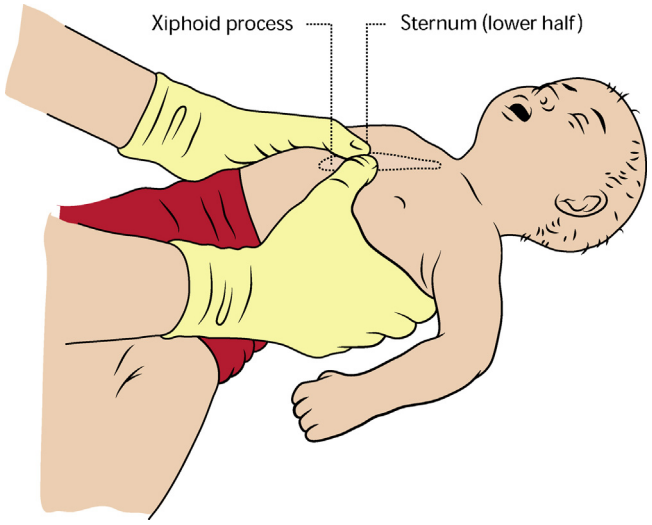

Fig. 6.4. Chest compression—infant.

- Combine rescue breathing and chest compressions at a ratio of 15 compressions to 2 ventilations.

Chest compressions

For all children, compress the lower half of the sternum. The compression should be sufficient to depress the sternum by at least one third of the anterior–posterior diameter of the chest. Release the pressure completely and repeat at a rate 100–120 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.

Chest compression in infants (Fig. 6.4)

The lone rescuer compresses the sternum with the tips of two fingers. If there are two or more rescuers, use the encircling technique. Place both thumbs flat side by side on the lower half of the sternum (as above) with the tips pointing towards the infant's head. Spread both hands with the fingers together to encircle the lower part of the infant's rib cage. The fingers should support the infant's back. For both methods, depress the lower sternum by at least one third the anterior–posterior dimension of the infant's chest or by 4 cm.[29]

Chest compression in children over 1 year of age (Figs. 6.5 and 6.6)

To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Place the heel of one hand on the sternum one finger's breadth above this. Lift the fingers to ensure that pressure is not applied onto the child's ribs. Position yourself above the victim's chest and, with your arm straight, compress the sternum to at least one third of the anterior–posterior dimension of the chest or by 5 cm.[29,30]

In larger children or for small rescuers, this is achieved most easily by using both hands, with the rescuer's fingers interlocked.

Do not interrupt resuscitation until

- The child shows signs of life (starts to wake up, to move, opens eyes and to breathe normally).
- More healthcare workers arrive and can either assist or take over.
- You become exhausted.

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

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.

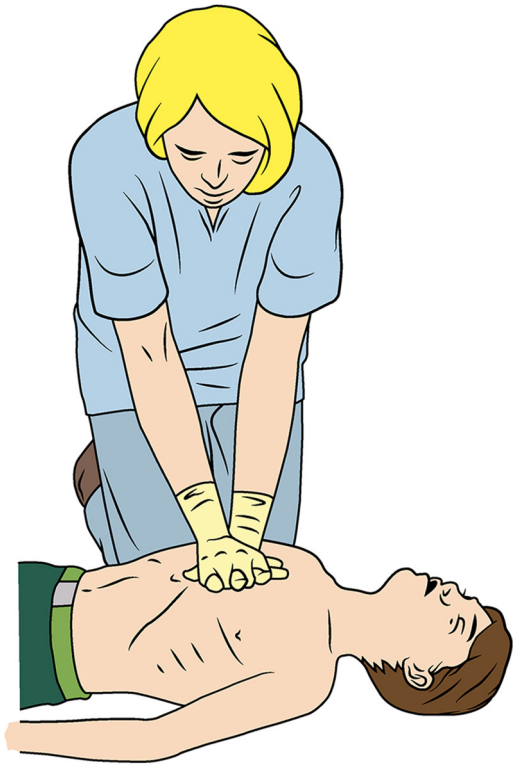

Fig. 6.6. Chest compression with two hands—child.

- If only one rescuer is present, undertake resuscitation for about 1 min or 5 cycles of CPR before going for assistance. To minimise interruption in CPR, it may be possible to carry an infant or small child whilst summoning help.
- If you are on your own, witness a child suddenly collapse and you suspect a primary cardiac arrest, call for help first and then start CPR as the child will likely need urgent defibrillation. This is an uncommon situation.

AED and BLS

Continue with CPR until the AED arrives. Attach the AED and follow the instructions. For 1–8 year old, use attenuated pads if available, as explained in the chapter on Basic Life Support and Automated External Defibrillation.[31]

Recovery position

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

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

There are important principles to be followed.

- Place the child in as near true lateral position as possible, with his mouth dependent, which should enable the free drainage of fluid.
- The position should be stable. In an infant, this may require a small pillow or a rolled-up blanket to be placed along his back to maintain the position, so preventing the infant from rolling into either the supine or prone position
- Avoid any pressure on the child's chest that may impair breathing.
- It should be possible to turn the child onto his side and back again to the recovery position easily and safely, taking into consideration the possibility of cervical spine injury by in-line cervical stabilisation techniques.
- Regularly change side to avoid pressure points (i.e. every 30 min).
- The adult recovery position is suitable for use in children.

Foreign body airway obstruction (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.[32] 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 (Fig. 6.7).

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

Recognition of foreign body airway obstruction

When a foreign body enters the airway the child reacts immediately by coughing in an attempt to expel it. A spontaneous cough is likely to be more effective and safer than any manoeuvre a rescuer might perform. However, if coughing is absent or ineffective

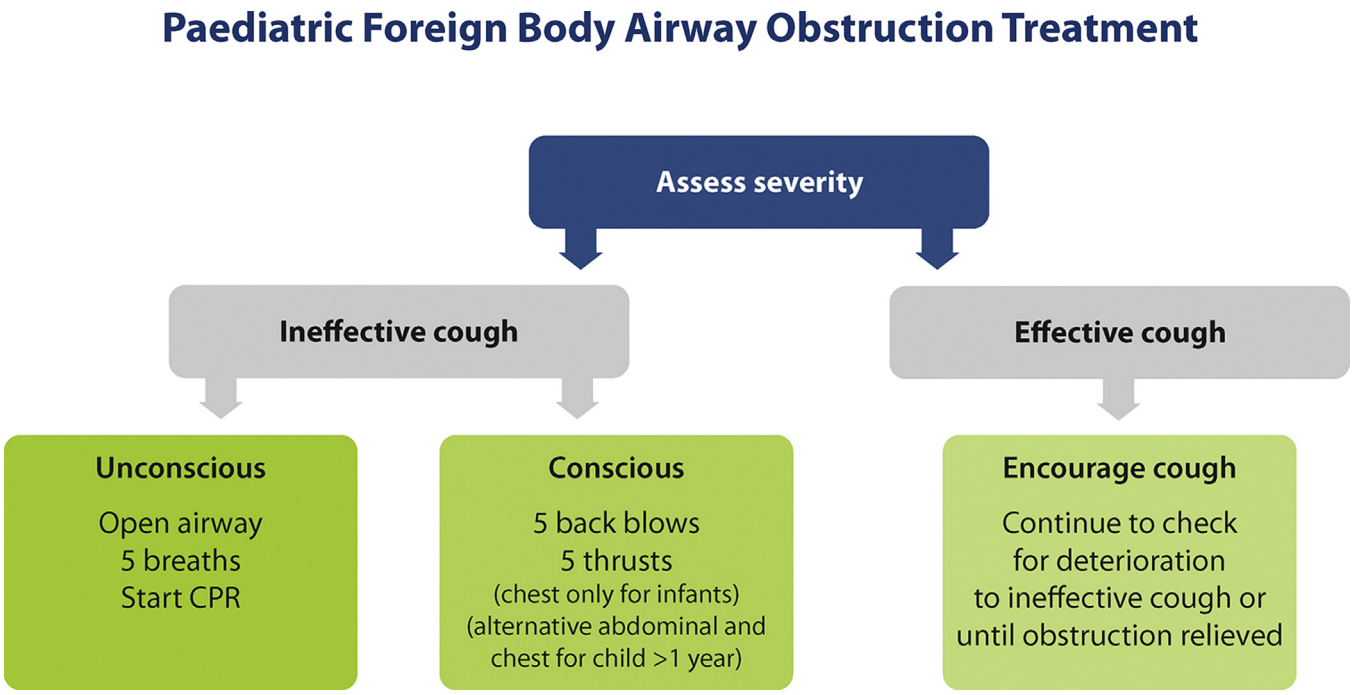

Fig. 6.7. Paediatric foreign body airway obstruction algorithm.

and the object completely obstructs the airway, the child will rapidly become asphyxiated. Active interventions to relieve FBAO are therefore required only when coughing becomes ineffective, but they then need to be commenced rapidly and confidently. The majority of choking events in infants and children occur during play or eating episodes, when a carer is usually present; thus, the events are frequently witnessed and interventions are usually initiated when the child is conscious.

Foreign body airway obstruction is characterised by the sudden onset of respiratory distress associated with coughing, gagging or stridor (Table 6.1). Similar signs and symptoms may be associated with other causes of airway obstruction such as laryngitis or epiglottitis; these conditions are managed differently to that of FBAO. Suspect FBAO if the onset was very sudden and there are no other signs of illness; there may be clues to alert the rescuer, e.g. a history of eating or playing with small items immediately before the onset of symptoms.

Relief of FBAO (Fig. 6.7)

Safety and summoning assistance. The principle of do no harm should be applied i.e. if the child is able to breath and cough, even with difficulty, encourage these spontaneous efforts. Do not intervene at this point as this may move the foreign body and worsen the problem, e.g. by causing full airway obstruction.

If the child is coughing effectively, no manoeuvre is necessary. Encourage the child to cough and continue monitoring the child's condition.

If the child's coughing is (or is becoming) ineffective, *shout for help* immediately and determine the child's conscious level.

Conscious child with FBAO. If the child is still conscious but has absent or ineffective coughing, give back blows.

If back blows do not relieve the FBAO, give chest thrusts to infants or abdominal thrusts to children. These manoeuvres create an artificial cough, increasing intrathoracic pressure and dislodging the foreign body.

Back blows for infants

- Support the infant in a head downward, prone position, to enable gravity to assist removal of the foreign body.
- A seated or kneeling rescuer should be able to support the infant safely across their lap.
- Support the infant's head by placing the thumb of one hand, at the angle of the lower jaw, and one or two fingers from the same hand, at the same point on the other side of the jaw.
- Do not compress the soft tissues under the infant's jaw, as this will worsen the airway obstruction.
- Deliver up to five sharp back blows with the heel of one hand in the middle of the back between the shoulder blades.
- The aim is to relieve the obstruction with each blow rather than to give all five.

Back blows for children over 1 year

- Back blows are more effective if the child is positioned head down.
- A small child may be placed across the rescuer's lap as with the infant.

Table 6.1
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	

- If this is not possible, support the child in a forward leaning position and deliver the back blows from behind.

If back blows fail to dislodge the object, and the child is still conscious, use chest thrusts for infants or abdominal thrusts for children. Do not use abdominal thrusts (Heimlich manoeuvre) in infants.

Chest thrusts for infants

- Turn the infant into a head downward supine position. This is achieved safely by placing your free arm along the infant's back and encircling the occiput with the hand.
- Support the infant down your arm, which is placed down (or across) your thigh.
- Identify the landmark for chest compressions (on the lower half of the sternum, approximately a finger's breadth above the xiphisternum).
- Give five chest thrusts; these are similar to chest compressions but sharper and delivered at a slower rate.

Abdominal thrusts for children over 1 year

- Stand or kneel behind the child; place your arms under the child's arms and encircle his torso.
- Clench your fist and place it between the umbilicus and the xiphisternum.
- Grasp this hand with the other hand and pull sharply inwards and upwards.
- Repeat up to five times.
- Ensure that pressure is not applied to the xiphoid process or the lower rib cage—this may cause abdominal trauma.

Following the chest or abdominal thrusts, reassess the child. If the object has not been expelled and the victim is still conscious, continue the sequence of back blows and chest (for infant) or abdominal (for children) thrusts. Call out, or send, for help if it is still not available. Do not leave the child at this stage.

If the object is expelled successfully, assess the child's clinical condition. It is possible that part of the object may remain in the respiratory tract and cause complications. If there is any doubt, seek medical assistance. Abdominal thrusts may cause internal injuries and all victims treated with abdominal thrusts should be examined by a doctor.[4]

Unconscious child with FBAO If the child with FBAO is, or becomes, unconscious, place him on a firm, flat surface. Call out, or send, for help if it is still not available. Do not leave the child at this stage; proceed as follows:

Airway opening Open the mouth and look for any obvious object. If one is seen, make an attempt to remove it with a single finger sweep. Do not attempt blind or repeated finger sweeps—these could push the object deeper into the pharynx and cause injury.

Rescue breaths Open the airway using a head tilt/chin lift and attempt five rescue breaths. Assess the effectiveness of each breath: if a breath does not make the chest rise, reposition the head before making the next attempt.

Chest compressions and CPR

- Attempt five rescue breaths and if there is no response (moving, coughing, spontaneous breaths) proceed to chest compressions without further assessment of the circulation.
- Follow the sequence for single rescuer CPR (step 7B above) for approximately a minute or 5 cycles of 15 compressions to 2 ventilations before summoning the EMS (if this has not already been done by someone else).
- When the airway is opened for attempted delivery of rescue breath, check if the foreign body can be seen in the mouth.
- If an object is seen and can be reached, attempt to remove it with a single finger sweep.
- If it appears the obstruction has been relieved, open and check the airway as above; deliver rescue breaths if the child is not breathing.
- If the child regains consciousness and exhibits spontaneous effective breathing, place him in a safe position on his side (recovery position) and monitor breathing and the level of consciousness whilst awaiting the arrival of the EMS.

Paediatric advanced life support

Assessment of the seriously ill or injured child—The 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.[22,33–42] So-called asphyxial arrests or respiratory arrests are also more common in young adulthood (e.g. trauma, drowning and poisoning).[25,43–56]

Without treatment, the ill/injured child's initial physiological responses involve compensatory mechanisms. This means the affected system tries to adapt to the underlying physiological disturbance. So, for a circulatory problem, the initial physiological response will be in the circulatory system, and if there is a respiratory problem, then respiratory changes may take place. As things worsen, the other systems may become involved as part of the compensatory process. However, the child may continue to deteriorate, leading to decompensated respiratory or circulatory failure. Further physiological deterioration to cardiopulmonary failure may occur with the then inevitable progression to cardiopulmonary arrest. As the outcome from cardiopulmonary arrest in children is poor, identifying the preceding stages of circulatory or respiratory failure is a priority as effective early intervention in these stages may be lifesaving.

The order of assessment and intervention for any seriously ill child follows the ABCDE principles.

- A indicates airway.
- B indicates breathing.
- C indicates circulation.
- D indicates disability.
- E indicates exposure.

The topics of D (disability i.e. neurological status) and E (exposure with any subsequent conditions that may be found e.g. non-blanching rashes) are beyond the remit of these guidelines but are taught in paediatric life support courses.

Interventions are made at each step of the assessment as abnormalities are identified. The next step of the assessment is not started until the preceding abnormality has been managed and corrected if possible.

The role of the team leader is to co-ordinate care and to anticipate problems in the sequence. Each team member must be aware of the ABC principles.[57] Should deterioration occur, reassessment based on ABCDE is strongly recommended, starting at A again.

Summoning a paediatric rapid response team or medical emergency team may reduce the risk of respiratory and/or cardiac arrest in hospitalised children outside the intensive care setting but the evidence is limited on this point as the literature tends not to separate out the team response alone from the other systems in place to

identify early deterioration.[58–69] This team should ideally include at least one physician experienced in acute paediatric care and a paediatric nurse (see the definitions in the terminology section above for the clinicians involved), and be called to evaluate a potentially critically ill child not already in a paediatric intensive care unit (PICU) or paediatric emergency department (ED).[70,71]

The ERC PLS writing group recognised that there is national and regional variation in countries as to the compositions of such a team but it is clear that processes to detect the early deterioration are key in reducing the morbidity and mortality of seriously ill and injured children. These processes with subsequent intervention by attending nurses and doctors have a higher priority for implementation than there solely being a rapid response or medical emergency team.[29,72–74]

Specific scores can be used (e.g. the paediatric early warning score, PEWS),[70,75–96] but there is no evidence that these improve the decision making process, or the clinical outcome.[29,71]

Diagnosing respiratory failure: Assessment of A and B

Assessment of a potentially critically ill child starts with the assessment of airway (A) and breathing (B).

Respiratory failure can be defined as the body's inability to maintain adequate blood levels of oxygen and carbon dioxide. Physiological compensatory mechanisms may be seen, such as an increase in respiratory rate and heart rate, and increased work of breathing, but these signs are not always present.

The signs of respiratory failure, as features of those physiological responses, may include:

- Respiratory rate outside the normal range for the child's age—either too fast or too slow.[97]
- Initially increased work of breathing, which may progress to inadequate/decreased work of breathing as the child tires or compensatory mechanisms fail.
- Additional noises such as stridor, wheeze, crackles, grunting, or the loss of breath sounds.
- Decreased tidal volume marked by shallow breathing, decreased chest expansion or decreased air entry at auscultation.
- Hypoxaemia (without/with supplemental oxygen) generally identified by cyanosis but it is often detectable prior to this by pulse oximetry.

There are uncommon conditions that can be associated with respiratory failure in which there is an inability of the body to raise these physiological compensatory signs. These are mostly due to abnormal neurological conditions (e.g. intoxication or coma) or muscular conditions (e.g. myopathy) where owing to muscle weakness, the child may not have the capacity to increase the work of breathing. A history or the presence of any features of these conditions is important to take into account when assessing the patient.

There may be associated signs in other organ systems. Even though the primary problem is respiratory, other organ systems will be involved to try to ameliorate the overall physiological disturbance.

These are detectable in step C of the assessment and include:

- Increasing tachycardia (compensatory mechanism to increase tissue oxygen delivery).
- Pallor.
- Bradycardia (an ominous indicator of the loss of compensatory mechanisms).
- Alteration in the level of consciousness (a sign that compensatory mechanisms are failing) owing to poor perfusion of the brain.

Diagnosing circulatory failure: Assessment of C

Circulatory failure is characterised by a mismatch between the metabolic demand by the tissues, and the delivery of oxygen and nutrients by the circulation.[97,98] Physiological compensatory mechanisms lead to changes in heart rate, in the systemic vascular resistance, and in tissue and organ perfusion. In some conditions, there may be vasodilation as part of the body's response to illness, e.g. toxic shock syndrome.

Signs of circulatory failure might include:

- Increased heart rate (bradycardia is an ominous sign of physiological decompensation).[97]
- Decreased systemic blood pressure.
- Decreased peripheral perfusion (prolonged capillary refill time, decreased skin temperature, pale or mottled skin)—signs of increased vascular resistance.
- Bounding pulses, vasodilation with widespread erythema may be seen in conditions with decreased vascular resistance.
- Weak or absent peripheral pulses.
- Decreased intravascular volume.
- Decreased urine output.

The transition from a compensatory state to decompensation may occur in an unpredictable way. Therefore, the child should be monitored, to detect and correct any deterioration in their physiological parameters promptly.

Other systems may be affected, for example:

- The respiratory rate may be increased initially, as an attempt to improve oxygen delivery, later becoming slower; this is usually accompanied by decompensated circulatory failure.
- The level of consciousness may decrease owing to poor cerebral perfusion.
- Poor cardiac functioning can lead to other signs, such as pulmonary oedema, enlarged liver, raised jugular veins.
- Poor tissue perfusion, metabolic acidosis and increased/increasing blood lactate levels may become progressively worse without correction.

Diagnosing cardiopulmonary arrest

Signs of cardiopulmonary arrest include:

- Unresponsiveness to pain (coma).
- Apnoea or gasping respiratory pattern.
- Absent circulation.
- Pallor or deep cyanosis.

Palpation of a pulse is not reliable as the sole determinant of the need for chest compressions.[27,99–101] In the absence of signs of life, rescuers (lay and professional) should begin CPR unless they are certain that they can feel a central pulse within 10 s (infants—brachial or femoral artery; children—carotid or femoral artery). If there is any doubt, start CPR.[99,102–104] If personnel skilled in echocardiography are available, this investigation may help to detect cardiac activity and potentially treatable causes for the arrest.[100] However, echocardiography must not interfere with or delay the performance of chest compressions.

Management of respiratory and circulatory failure

In children, there are many causes of respiratory and circulatory failure and they may develop gradually or suddenly. Both may be initially compensated but will normally decompensate without adequate treatment. Untreated decompensated respiratory or circulatory failure will lead to cardiopulmonary arrest. Hence, the

aim of paediatric life support is the early and effective intervention in children with respiratory and circulatory failure to prevent progression to full arrest.[105–110]

Airway and breathing

- Open the airway.
- Optimise ventilation.
- Ensure adequate oxygenation, start with 100% oxygen.
- Establish respiratory monitoring (first line – pulse oximetry/peripheral oxygen saturation – SpO_2).
- Achieving adequate ventilation and oxygenation—this may require the use of airway adjuncts ± bag-mask ventilation (BMV), the use of an LMA or other supraglottic airway, securing a definitive airway by tracheal intubation and positive pressure ventilation.
- For intubated children, it is standard practice that their end tidal carbon dioxide levels are monitored. End tidal carbon dioxide monitoring can be used in non-intubated critically ill patients.
- Very rarely, a surgical airway may be required.

Circulation

- Establish cardiac monitoring (first line—pulse oximetry/SpO_2, electrocardiography (ECG) and non-invasive blood pressure (NIBP)).
- Secure intravascular access. This may be achieved by peripheral intravenous (IV) or by intraosseous (IO) route. 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) to treat circulatory failure due to hypovolaemia, e.g. from fluid loss or maldistribution, as seen in septic shock and anaphylaxis.
- Consider carefully the use of fluid bolus in primary cardiac functioning disorders, e.g. myocarditis, cardiomyopathy.
- Do not give a fluid bolus in severe febrile illness when circulatory failure is absent.[29,111–113]
- Isotonic crystalloids are recommended as initial resuscitation fluid in infants and children with any type of shock, including septic shock.[29,114–119]
- Assess and re-assess the child repeatedly, beginning each time with the airway before proceeding to breathing and then the circulation. Blood gas and lactate measurement may be helpful.
- 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 treatment of respiratory and/or circulatory failure.[120,121] Whilst the evidence for the use of these techniques is of low quality, the general principles of monitoring and assessing the impact of any interventions and those responses are key in managing seriously ill children.

Airway

Open the airway by using basic life support techniques. Oropharyngeal and nasopharyngeal airways adjuncts can help maintain the airway. An oropharyngeal airway may be helpful in the unconscious child, in whom there is no gag reflex. Use the appropriate size (as measured from the incisors to the angle of the mandible) to avoid pushing the tongue backward during insertion, as this may further obstruct the airway. The soft palate may be damaged by forceful insertion of the oropharyngeal airway—avoid this by inserting the oropharyngeal airway with care. Do not use force if the child resists.

The nasopharyngeal airway is usually tolerated better in the conscious or semi-conscious child (who has an effective gag reflex), but should not be used if there is a basal skull fracture or a coagulopathy. The correct insertion depth should be sized from the nostrils to the angle of the mandible and must be re-assessed after insertion. These simple airway adjuncts do not protect the airway from aspiration of secretions, blood or stomach contents.

Supraglottic airways devices (SADs) (including LMA)

Although BVM ventilation remains the recommended first line method for achieving airway control and ventilation in children, the SADs represent a range of acceptable airway devices that may assist providers trained in their use.[122,123] SADs may be particularly helpful in airway obstruction caused by supraglottic airway abnormalities, or if BVM ventilation is difficult or not possible.[124,125] SADs do not totally protect the airway from aspiration of secretions, blood or stomach contents, and therefore close observation is required.[126,127]

Tracheal intubation

Tracheal intubation is the most secure and effective way to establish and maintain the airway, prevent gastric distension, protect the lungs against pulmonary aspiration, enable optimal control of the airway pressure and provide positive end expiratory pressure (PEEP). The oral route for tracheal intubation is preferable during resuscitation. Oral intubation is quicker and simpler, and is associated with fewer complications than nasal intubation. In the conscious child, the judicious use of anaesthetics, sedatives and neuromuscular blocking drugs is essential to avoid multiple intubation attempts or intubation failure.[128–137] Only skilled and experienced practitioners should perform intubation.

The anatomy of a child's airway differs significantly from that of an adult, and tube sizes and insertion depth vary considerably with age; hence, intubation of a child requires special training and ongoing experience. Clinical examination and capnography should be used to ensure that the tracheal tube remains secured and vital signs should be monitored.[136] It is also essential to anticipate potential cardiorespiratory problems and to plan an alternative airway management technique in case the trachea cannot be intubated.

There is currently no evidence-based recommendation defining the setting-, patient- and operator-related criteria for pre-hospital tracheal intubation of children. Pre-hospital tracheal intubation of children may be considered if the airway and/or breathing is seriously compromised or threatened. The mode and duration of transport (e.g., air transport) may play a role in the decision to secure the airway before transport.

Anyone intending to intubate must be adequately skilled in advanced paediatric airway management including pre-oxygenation and the use of drugs to facilitate tracheal intubation.[138]

Intubation during cardiopulmonary arrest. The child who is in cardiopulmonary arrest does not require sedation or analgesia to be intubated. As previously stated, intubation of the seriously ill/injured child should be undertaken by an experienced and trained practitioner.

Tracheal tube sizes. Table 6.2 shows which tracheal tube internal diameters (ID) should be used for different ages.[139–144] 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 indicated by resuscitation tapes.[145,146]

Cuffed versus uncuffed tracheal tubes. Uncuffed tracheal tubes have been used traditionally in children up to 8 years of age but cuffed tubes may offer advantages in certain circumstances e.g. in facial burns,[147] when lung compliance is poor, airway resistance is high or if there is a large air leak from the glottis.[139,148,149] The use of

Table 6.2
General recommendation for cuffed and uncuffed tracheal tube sizes (internal diameter in mm).

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

cuffed tubes also makes it more likely that the correct tube size will be chosen on the first attempt.[139,140,147] The correctly sized cuffed tracheal tube is as safe as an uncuffed tube for infants and children (not for neonates) provided attention is paid to its placement, size and cuff inflation pressure.[148–150] 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.[150]

Confirmation of correct tracheal tube placement. Displaced, misplaced or obstructed tubes occur frequently in the intubated child and are associated with an increased risk of death.[151,152] No single technique is 100% reliable for distinguishing oesophageal from tracheal intubation.[153–155]

Assessment of the correct tracheal tube position is made by:

- Laryngoscopic observation of the tube passing through the vocal cords.
- Detection of end-tidal CO_2 (preferably by capno*graphy* or by capno*metry* or colorimetry) 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!).
- Improvement of heart rate towards the age-expected value (or remaining within the normal range) (delayed sign!).

If the child is in cardiopulmonary arrest and exhaled CO_2 is not detected despite adequate chest compressions, or if there is any doubt as to the tube position, confirm the placement of the tracheal tube by direct laryngoscopy. After correct placement and confirmation, secure the tracheal tube and reassess its position. Maintain the child's head in the neutral position. Flexion of the head drives the tube further into the trachea whereas extension may pull it out of the airway.[156] Confirm the position of the tracheal tube at the mid-trachea by chest X-ray; the tracheal tube tip should be at the level of the 2nd or 3rd thoracic vertebra.

DOPES is a useful acronym for the causes of sudden deterioration in an intubated child. It is also helpful in the case of a child who requires intubation and thereafter fails to improve following being intubated. When the cause is found, steps should be taken to remedy the situation.

Displacement of the tracheal tube (in the oesophagus, pharynx or endobronchially). **O**bstruction of the tracheal tube, or of the heat and moisture exchanger (HME) or the respirator pipes. **P**neumothorax and other **p**ulmonary disorders (bronchospasm, oedema, pulmonary hypertension, etc.). **E**quipment failure (source of gas, bag-mask, ventilator, etc.). **S**tomach (gastric distension may alter diaphragm mechanics).

Breathing

Oxygenation

Give oxygen at the highest concentration (i.e. 100%) during initial resuscitation.

Studies in newly borns suggest advantages of using room air during resuscitation.[14] In infants and older children, however, there is no evidence of benefit for using air instead of oxygen so use 100% oxygen for the initial resuscitation. Once the child is stabilised and/or there is ROSC, titrate the fraction of inspired oxygen (FiO_2) to achieve normoxaemia, or at least (if arterial blood gas is not available), maintain SpO_2 in the range of 94–98%.[157,158] In smoke inhalation (carbon monoxide poisoning) and severe anaemia, however, high FiO_2 should be maintained until the underlying disorder is ameliorated as in these circumstances, dissolved oxygen in the blood plays an important role in oxygen transport to tissues.

Ventilation

Healthcare providers commonly provide excessive ventilation during CPR and this may be harmful. Hyperventilation causes increased intrathoracic pressure, decreased cerebral and coronary perfusion, and there is some evidence of poorer survival rates in animals although other evidence suggests that survival rates are not worse.[159–166] A simple guide to deliver an appropriate tidal volume is to achieve normal chest wall rise. Use a ratio of 15 chest compressions to 2 ventilations and a compression rate of 100–120 min^{-1}.

Inadvertent hyperventilation during CPR occurs frequently, especially when the trachea is intubated and ventilations are given continuously along with asynchronous chest compressions.

Once the airway is protected by tracheal intubation, continue positive pressure ventilation at 10 breaths min^{-1} without interrupting the chest compressions. Take care to ensure that lung inflation is adequate during chest compressions. Once ROSC has been achieved, provide normal ventilation (rate/volume) based on the child's age, and by monitoring end-tidal CO_2 and blood gas values, to achieve a normal arterial carbon dioxide tension ($PaCO_2$) and arterial oxygen levels. Both hypocarbia and hypercarbia are associated with poor outcomes following cardiac arrest.[167] This means that the child with ROSC should usually be ventilated at 12–24 breaths min^{-1}, according to their age normal values.

In a few children the normal values for carbon dioxide and oxygenation levels may be different to that of the rest of the paediatric population; take care to restore the carbon dioxide and oxygen values to that child's normal levels, e.g. in children with chronic lung disease or congenital heart conditions.

Bag mask ventilation (BMV). Bag mask ventilation (BMV) is effective and safe for a child requiring assisted ventilation for a short period, i.e., in the pre-hospital setting or in an emergency department.[168,169] Assess the effectiveness of BMV by observing adequate chest rise, monitoring heart rate and auscultating for breath sounds, and measuring SpO_2. Any healthcare provider with a responsibility for treating children must be able to deliver BMV effectively.

Monitoring of breathing and ventilation

1.1.1.1. End-tidal CO_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 a child.[170–173] 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 $ETCO_2$ may reflect low or absent pulmonary blood flow.[174–177]

In this circumstance, the tube placement should be checked by direct laryngoscopy and the chest auscultated for the sounds of air entry into the lungs.

Capnography may also provide information on the effectiveness of chest compressions and can give an early indication of ROSC.[178,179] Care must be taken when interpreting $ETCO_2$ values especially after the administration of adrenaline or other vasoconstrictor drugs when there may be a transient decrease in values[180–184] or after the use of sodium bicarbonate causing a transient increase.[185] Although an $ETCO_2$ higher than 2 kPa (15 mmHg) may be an indicator of adequate resuscitation, current evidence does not support the use of a threshold $ETCO_2$ value as an indicator for the quality of CPR or for the discontinuation of resuscitation.[29]

Peripheral pulse oximetry, SpO_2. Clinical evaluation to determine the degree of oxygenation in a child is unreliable; therefore, monitor the child's peripheral oxygen saturation continuously by pulse oximetry. Pulse oximetry can be unreliable under certain conditions, e.g. if the child is in circulatory failure, in cardiopulmonary arrest or has poor peripheral perfusion. In some circumstances the SpO_2 reading may not give a true assessment of the total amount of oxygen in the blood as it only measures the relative amount of oxygen bound to haemoglobin. Hence, in anaemia, methaemoglobinaemia or in carbon monoxide poisoning, SpO_2 values must be interpreted with caution.

Although pulse oximetry is relatively simple, it is a poor guide to tracheal tube displacement and must not be relied upon. Capnography detects tracheal tube dislodgement more rapidly than pulse oximetry and is the monitoring system of choice.[186]

Circulation

Vascular access

Vascular access is essential to enable drugs and fluids to be given, and blood samples obtained. Venous access can be difficult to establish during resuscitation of an infant or child. In critically ill children, whenever venous access is not readily attainable, intraosseous access should be considered early, especially if the child is in cardiac arrest or decompensated circulatory failure.[187–193] In any case, in critically ill children, if attempts at establishing intravenous (IV) access are unsuccessful after one minute, insert an intra-osseous (IO) needle instead.[190,194]

IO access. IO access is a rapid, safe, and effective route to give drugs, fluids and blood products.[195–205] The onset of action and time to achieve adequate plasma drug concentrations are similar to that achieved via the central venous route.[206–209] Bone marrow samples can be used to cross match for blood type or group for chemical analysis[210–212] and for blood gas measurement (the values may be comparable to central venous blood gases if no drug has been injected in the cavity).[206,209,211,213–215] However, these bone marrow samples can damage auto-analysers and should be used preferably in a cartridge analyser.[216] After taking blood samples, flush each given drug with a bolus of normal saline to ensure dispersal beyond the marrow cavity, and to achieve faster distribution to the central circulation. Inject large boluses of fluid using manual pressure or a pressure bag.[217] Maintain IO access until definitive IV access has been established.[107,192,203,218,219]

Intravenous access and other routes. Peripheral IV access provides plasma concentrations of drugs and clinical responses equivalent to central or IO access.[220–222] The intramuscular route is preferred for the administration of adrenaline in anaphylaxis.[223,224] Other routes are useful for different circumstances e.g. intranasal, buccal etc. but are beyond the remit of these guidelines.[225] Central venous lines provide more secure long-term access but, compared with IO or peripheral IV access, offer no advantages during resuscitation.[190,191,221,226,227] The tracheal route for the administration of drugs is no longer recommended.[228,229]

Fluids and drugs

When a child shows signs of circulatory failure caused by hypovolaemia, controlled volume administration is indicated.[230] For children with febrile illness and not showing signs of circulatory failure, adopt a cautious approach to fluid therapy with frequent reassessment of the child.[29,111–113] Isotonic crystalloids are recommended as the initial resuscitation fluid for infants and children with any type of circulatory failure.[231,232] If there are signs that the systemic perfusion is inadequate, give a bolus of 20 ml kg^{-1} of an isotonic crystalloid even if the systemic blood pressure is normal. Following each bolus, re-assess the child's clinical state, using the ABCDE system of assessment, to decide whether a further bolus or other treatment is required (and how much and how fast). In some children, early inotropic or vasopressor support may be needed.[108,233] In addition, owing to decreased/decreasing consciousness or progressive respiratory failure, some patients will need intubation and mechanical ventilation, so be prepared in case this occurs.

There is growing evidence to prefer the use of balanced crystalloids as these induce less hyperchloraemic acidosis.[234–237]

In life-threatening hypovolaemic shock, as may be seen in rapid blood loss in trauma, limiting the use of crystalloids in favour of a regime of massive blood transfusion may be required. There are varying regimes of combining plasma, platelets and other blood products in delivering massive blood transfusion,[238,239] so the regime used should be according to local protocols. Similarly, in other types of shock, when multiple boluses of crystalloids are given, timely blood products should be considered to treat dilutional effects. Avoid glucose containing solutions unless there is hypoglycaemia.[240–244] Monitor blood glucose levels and avoid hypoglycaemia; infants and small children are particularly prone to hypoglycaemia.[245]

Adenosine

Adenosine is an endogenous nucleotide that causes a brief atrioventricular (AV) block and impairs accessory bundle re-entry at the level of the AV node. Adenosine is recommended for the treatment of supraventricular tachycardia (SVT).[246] It has a short half-life (10 s); give it intravenously via upper limb or central veins to minimise the time taken to reach the heart. It causes asystole, which is usually short lived, hence adenosine must be given under ECG monitoring. Give adenosine rapidly, followed by a flush of 5 ml of normal saline.[247] Adenosine must be used with caution in asthmatics, second or third degree AV block, long QT syndromes and in cardiac transplant recipients.

Adrenaline (epinephrine)

Adrenaline is an endogenous catecholamine with potent α, β_1 and β_2 adrenergic actions. It plays a central role in the cardiac arrest treatment algorithms for non-shockable and shockable rhythms. Adrenaline induces vasoconstriction, increases diastolic pressure and thereby improves coronary artery perfusion pressure, enhances myocardial contractility, stimulates spontaneous contractions, and increases the amplitude and frequency of

ventricular fibrillation (VF), so increasing the likelihood of successful defibrillation.

For cardiopulmonary resuscitation, the recommended IV/IO dose of adrenaline in children for the first and for subsequent doses is 10 micrograms kg^{-1}. The maximum single dose is 1 mg. If needed, give further doses of adrenaline every 3–5 min, i.e. every 2 cycles.

The use of single higher doses of adrenaline (above 10 micrograms kg^{-1}) is not recommended because it does not improve survival or neurological outcome after cardiopulmonary arrest.[248–252]

Once spontaneous circulation is restored, a continuous infusion of adrenaline may be required. Its haemodynamic effects are dose-related; there is also considerable variability in response between children; therefore, titrate the infusion dose to the desired effect. High infusion rates may cause excessive vasoconstriction, so compromising extremity, mesenteric, and renal blood flow. High-dose adrenaline can cause severe hypertension and tachyarrhythmias.[253] To avoid tissue damage it is essential to give adrenaline through a secure intravascular line (IV or IO). Adrenaline (and other catecholamines) is inactivated by alkaline solutions and should never be mixed with sodium bicarbonate.[254]

Amiodarone for shock-resistant paediatric VF/pulseless VT

Amiodarone can be used to treat paediatric shock-resistant VF/pulseless VT (pVT). Amiodarone is a non-competitive inhibitor of adrenergic receptors: it depresses conduction in myocardial tissue and therefore slows AV conduction, and prolongs the QT interval and the refractory period. Amiodarone can be given as part of the cardiac arrest algorithm in managing refractory VF/pVT. It is given after the third shock as a 5 mg kg^{-1} bolus (and can be repeated following the fifth shock). When treating other cardiac rhythm disturbances, amiodarone must be injected slowly (over 10–20 min) with systemic blood pressure and ECG monitoring to avoid causing hypotension.[255] This side effect is less common with the aqueous solution.[256] Other rare but significant adverse effects are bradycardia and polymorphic VT.[257]

Lidocaine has been suggested by COSTR as an alternative but most practitioners will have followed the guidance that has stated amiodarone is the drug of choice. The European Resuscitation Council advises that the clinician should use the drug with which they are familiar and for which they have knowledge of expected and unexpected listed side effects.

Lidocaine is a commonly used local anaesthetic as well as being a Class-1b antiarrhythmic drug. Lidocaine is an alternative to amiodarone in defibrillation-resistant VF/pulseless VT in children.[29,258–260] It can be used with a loading dose of 1 mg kg^{-1} (maximum dose 100 mg/dose) followed by continuous infusion at 20–50 micrograms kg^{-1} min^{-1}. Toxicity can occur if there is underlying renal or hepatic disease.

Atropine

Atropine accelerates sinus and atrial pacemakers by blocking the parasympathetic response. The commonly used dose is 20 micrograms kg^{-1}. It may also increase AV conduction. Small doses (<100 micrograms) may cause paradoxical bradycardia.[261] In bradycardia with poor perfusion unresponsive to ventilation and oxygenation, the first line drug is adrenaline, not atropine. Atropine is recommended only for bradycardia caused by increased vagal tone or cholinergic drug toxicity.[262–264] Its role in emergency intubation for the child is still unclear as there are no reported long-term benefits following ROSC.[29,265,266]

Calcium

Calcium is essential for myocardial function,[267] but the routine use of calcium does not improve the outcome from cardiopulmonary arrest.[268–272] Calcium is indicated in the presence of hypocalcaemia, calcium channel blocker overdose, hypermagnesaemia and hyperkalaemia.[46,272–274] Calcium supplementation may be required when massive blood transfusion is given, e.g. as in response to blood loss in trauma, or when any other large fluid volumes are given; the calcium levels must be monitored and replacement given to maintain normal blood levels.[238]

Glucose

Data from neonates, children and adults indicate that both hyper- and hypo- glycaemia are associated with poor outcome after cardiopulmonary arrest,[275,276] but it is uncertain if this is causative or merely an association.[241,276–278] Check blood or plasma glucose concentration and monitor closely in any ill or injured child, including after cardiac arrest. Do not give glucose-containing fluids during CPR unless hypoglycaemia is present.[245] Avoid hyper- and hypoglycaemia following ROSC.[279] In adults strict glucose control does not increase survival when compared with moderate glucose control[280,281] and it increases the risk of hypoglycaemia in neonates, children and adults.[282,283]

Magnesium

There is no evidence for giving magnesium routinely during cardiopulmonary arrest.[284,285] Magnesium treatment is indicated in the child with documented hypomagnesaemia or with torsade de pointes VT, (50 mg kg^{-1}) regardless of the cause.[286]

Sodium bicarbonate

There is no clear evidence for giving sodium bicarbonate routinely during cardiopulmonary arrest.[287–290] After effective ventilation and chest compressions have been achieved and adrenaline given, sodium bicarbonate may be considered for the child with prolonged cardiopulmonary arrest and/or severe metabolic acidosis. Sodium bicarbonate may also be considered in case of haemodynamic instability and co-existing hyperkalaemia, or in the management of tricyclic antidepressant drug overdose. Excessive quantities of sodium bicarbonate may impair tissue oxygen delivery and cause hypokalaemia, hypernatraemia, hyperosmolality and cerebral acidosis.

Procainamide

Procainamide slows intra-atrial conduction and prolongs the QRS and QT intervals. It can be used in supraventricular tachycardia (SVT)[291,292] or VT[293] resistant to other medications in the haemodynamically stable child. However, paediatric data are sparse and procainamide should be used cautiously.[294–297] Procainamide is a potent vasodilator and can cause hypotension: infuse it slowly with careful monitoring.[255,294]

Vasopressin—Terlipressin

Vasopressin is an endogenous hormone that acts at specific receptors, mediating systemic vasoconstriction (via V_1 receptor) and the reabsorption of water in the renal tubule (by the V_2 receptor).[298] There is currently insufficient evidence to support or refute the use of vasopressin or terlipressin as an alternative to, or in combination with, adrenaline in any cardiac arrest rhythm in adults or children.[299–306] These drugs may be considered in cardiac arrest refractory to adrenaline.

Some studies have reported that terlipressin (a long-acting analogue of vasopressin with comparable effects) improves haemodynamics in children with refractory, vasodilatory septic shock, but its impact on survival is less clear.[307–309] Two paediatric case series suggested that terlipressin may be effective in refractory cardiac arrest.[303,310]

Defibrillators

Defibrillators are either automated or manually operated, and may be capable of delivering either monophasic or biphasic shocks. Manual defibrillators capable of delivering the full energy requirements from neonates upwards must be available within hospitals and in other healthcare facilities caring for children at risk of cardiopulmonary arrest. Automated external defibrillators (AEDs) are pre-set for all variables including the energy dose.

Pad/Paddle size for defibrillation

Select the largest possible available paddles to provide good contact with the chest wall. The ideal size is unknown but there should be good separation between the pads.[311,312]

Recommended sizes are:

- 4.5 cm diameter for infants and children weighing <10 kg.
- 8–12 cm diameter for children weighing >10 kg (older than one year).

To decrease skin and thoracic impedance, an electrically conducting interface is required between the skin and the paddles. Preformed gel pads or self-adhesive defibrillation electrodes are effective and are recommended for maximal delivery of the energy. Self-adhesive pads facilitate continuous good quality CPR. Do not use saline-soaked gauze/pads, alcohol-soaked gauze/pads or ultrasound gel.

Position of the paddles

Apply the paddles firmly to the bare chest in the antero-lateral position, one paddle placed below the right clavicle and the other in the left axilla (Fig. 6.8). If the paddles are too large and there is a danger of charge arcing across the paddles, one should be placed on the upper back, below the left scapula and the other on the front, to the left of the sternum. This is known as the antero-posterior position and is also acceptable.

Optimal paddle force

To decrease transthoracic impedance during defibrillation, apply a force of 3 kg for children weighing <10 kg and 5 kg for larger children.[313,314] In practice, this means that the paddles should be applied firmly.

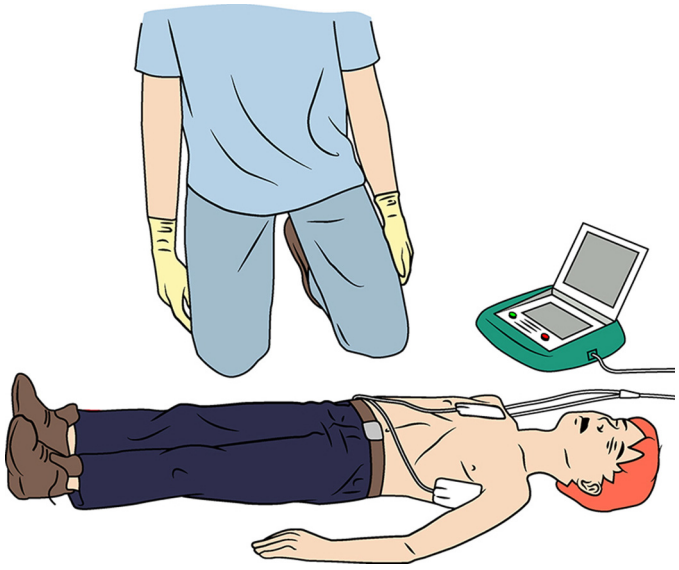

Fig. 6.8. Paddle positions for defibrillation—child.

Energy dose in children

The ideal energy dose for safe and effective defibrillation is unknown. Biphasic shocks are at least as effective and produce less post-shock myocardial dysfunction than monophasic shocks.[315] Animal models show better results with paediatric doses of 3–4 J kg^{-1} than with lower doses,[316] or adult doses,[317] but there are no data to support a different strategy to the current one of an initial dose of 2–4 J kg^{-1}. In Europe, for the sake of simplicity, we continue to recommend 4 J kg^{-1} for initial and subsequent defibrillation. Doses higher than 4 J kg^{-1} (as much as 9 J kg^{-1}) have defibrillated children effectively with negligible side effects.[318,319] When using a manual defibrillator, use 4 J kg^{-1} (preferably biphasic but monophasic waveform is also acceptable) for the first and subsequent shocks.

If no manual defibrillator is available, use an AED that can recognise paediatric shockable rhythms.[320–322] The AED should be equipped with a dose attenuator that decreases the delivered energy to a lower dose more suitable for children aged 1–8 years (50–75 J).[317,323] If such an AED in not available, use a standard adult AED and the pre-set adult energy levels. For children older than 8 years, use a standard AED with standard paddles. Experience with the use of AEDs (preferably with dose attenuator) in children younger than 1 year is limited; its use is acceptable if no other option is available.

Advanced management of cardiopulmonary arrest (Fig. 6.9)

A, B and C: Commence and continue with basic life support.

A and B	Oxygenate and ventilate with BMV • Provide positive pressure ventilation with a high concentration of inspired oxygen (100%) • Establish cardiac monitoring • Avoid rescuer fatigue by frequently changing the rescuer performing chest compressions
C	Assess cardiac rhythm and signs of life (+ check for a central pulse for no more than 10 s)

Non shockable—asystole, pulseless electrical activity (PEA)

- Give adrenaline IV or IO (10 micrograms kg^{-1}) and repeat every 3–5 min (every 2nd cycle) (Fig. 6.10).
- Identify and treat any reversible causes (4Hs & 4Ts).

Reversible causes of cardiac arrest

The reversible causes of cardiac arrest can be considered quickly by recalling the 4Hs and 4Ts:

- Hypoxia
- Hypovolaemia
- Hyper/hypokalaemia, metabolic
- Hypothermia
- Thrombosis (coronary or pulmonary)
- Tension pneumothorax
- Tamponade (cardiac)
- Toxic/therapeutic disturbances

Shockable—VF/pulseless VT

Attempt defibrillation immediately (4 J kg^{-1}) (Fig. 6.11):

- Charge the defibrillator while another rescuer continues chest compressions
- Once the defibrillator is charged, pause the chest compressions and ensure that all rescuers are clear of the patient. Minimise the delay between stopping chest compressions and delivery of the

Paediatric Advanced Life Support

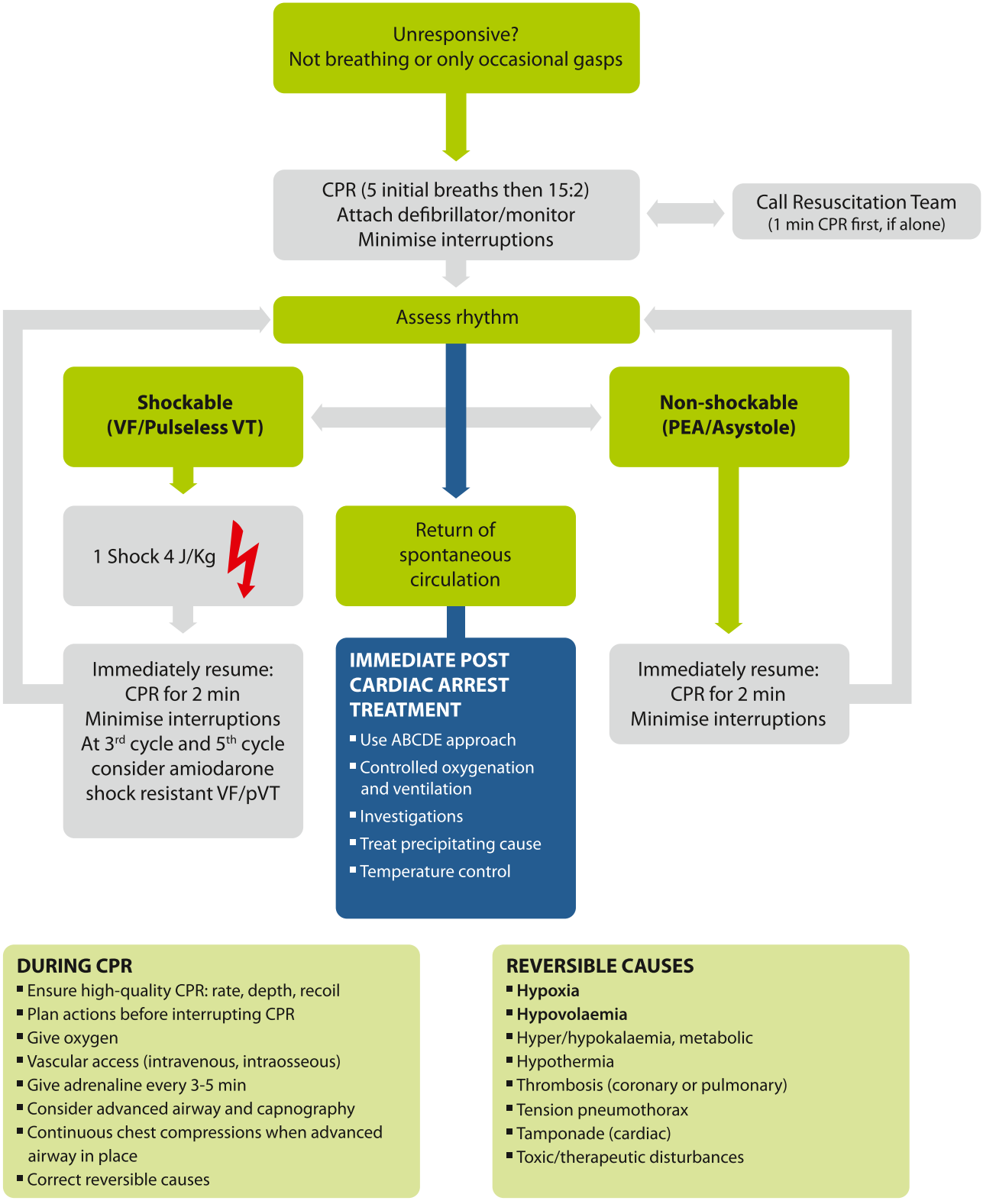

Fig. 6.9. Paediatric advanced life support algorithm.

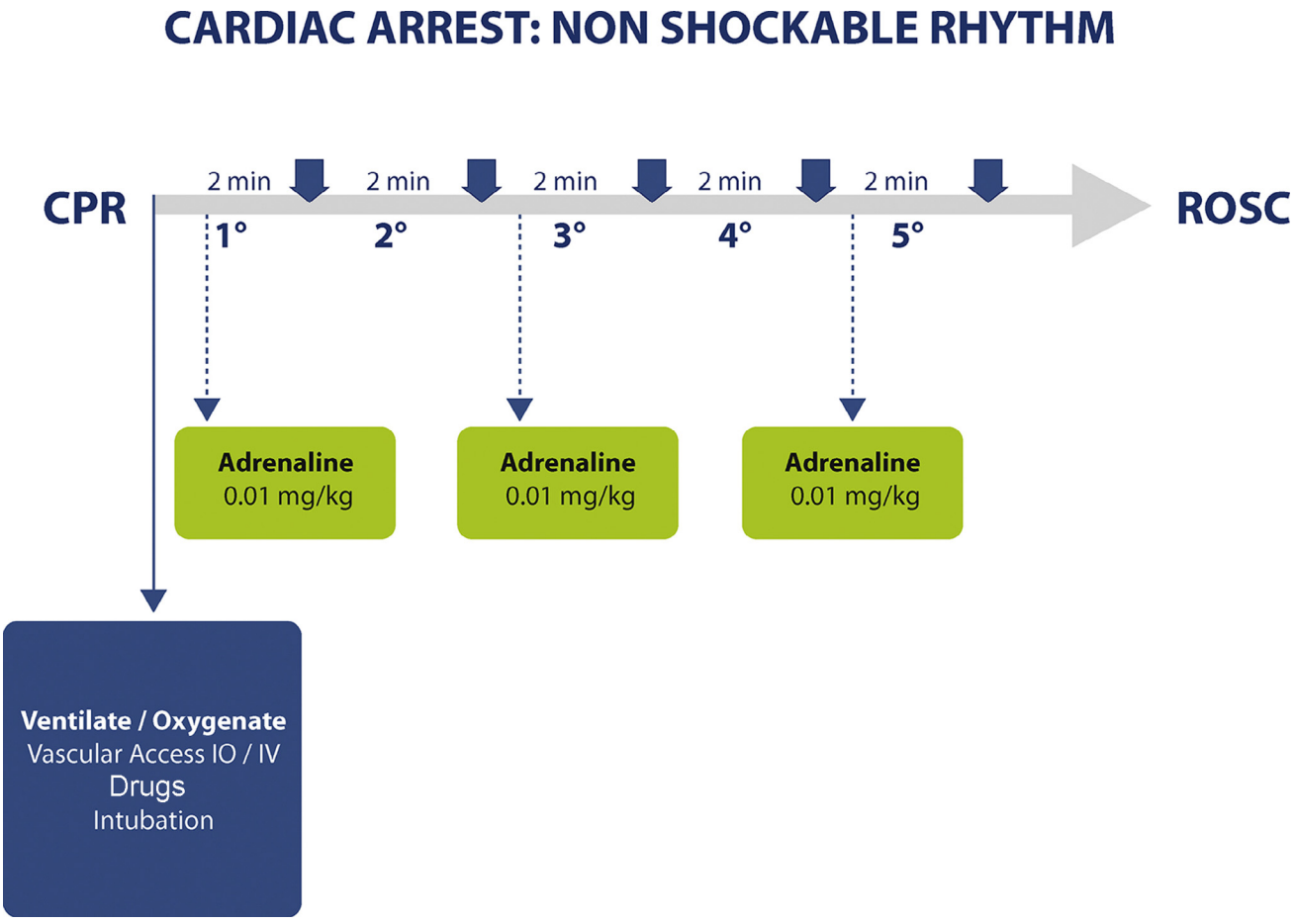

Fig. 6.10. Paediatric algorithm for non-shockable rhythm.

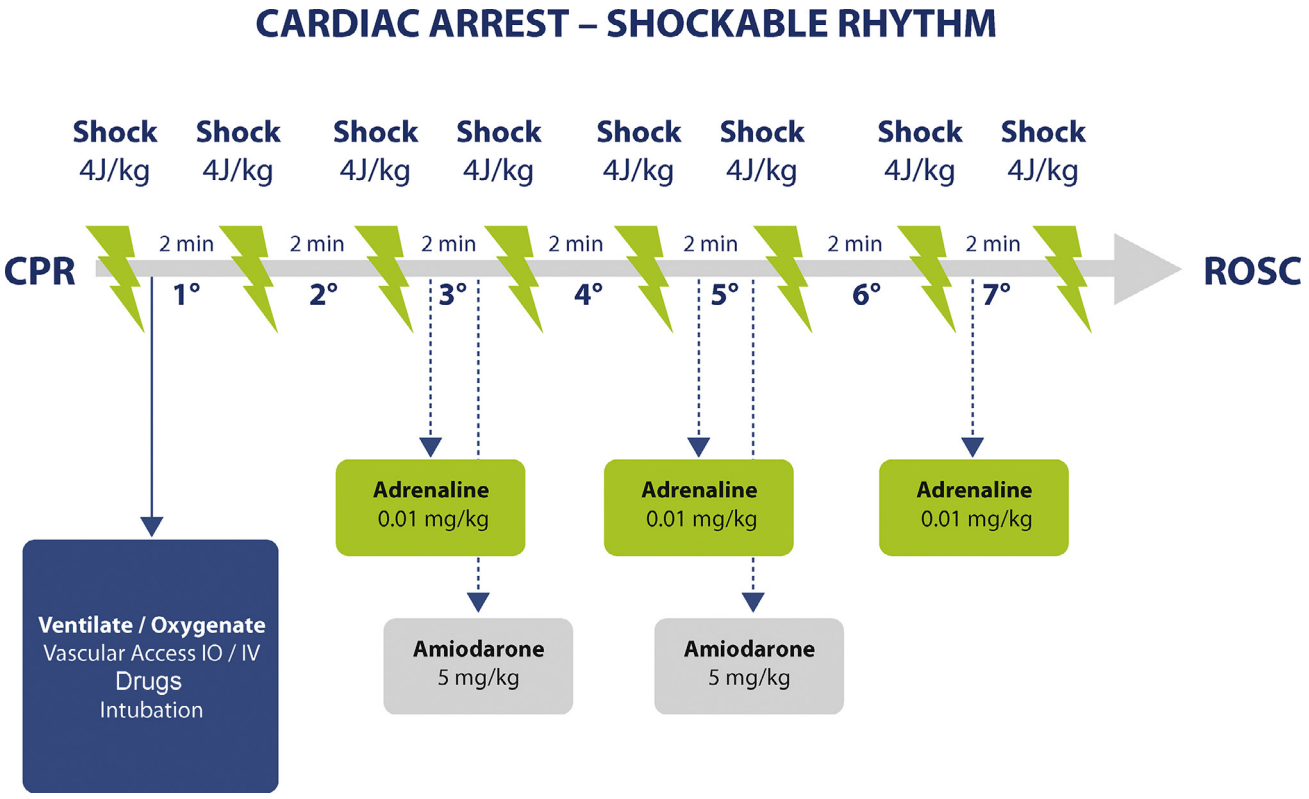

Fig. 6.11. Paediatric algorithm for shockable rhythm.

shock—even 5–10 s delay will reduce the chances of the shock being successful.
- 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/pVT.
- Give CPR for 2 min as soon as possible without reassessing the rhythm.
- Pause briefly to assess the rhythm; if still in VF/pVT give a third shock at 4 J kg^{-1}
- Give adrenaline 10 micrograms 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}[324] if still in VF/pVT after the fifth shock.

Lidocaine may be used as an alternative to amiodarone.

If the child remains in VF/pVT, 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 hypoxia and hypovolaemia have the highest prevalence in critically ill or injured children, and that electrolyte disturbances and toxicity are common causes of arrhythmia.

If defibrillation has been successful but VF/pVT recurs, resume CPR, give amiodarone or lidocaine and defibrillate again at the energy level that was effective previously.

Cardiac monitoring

Position the cardiac monitor leads or self-adhesive pads soon as possible to enable differentiation between a shockable and a non-shockable cardiac rhythm. Defibrillation paddles can be used to determine a rhythm if monitor leads or self-adhesive pads are not immediately available. Invasive monitoring of systemic blood pressure may help to improve the effectiveness of chest compression if present but it must never delay the provision or hamper the quality of basic or advanced resuscitation.

Non-shockable rhythms are pulseless electrical activity (PEA), bradycardia (<60 min^{-1} with no signs of circulation) and asystole. PEA and bradycardia often have wide QRS complexes.

Shockable rhythms are pVT and VF. These rhythms are more likely after sudden collapse in children with heart disease or in adolescents.

Non-shockable rhythms

Most cardiopulmonary arrests in children and adolescents are of respiratory origin.[325–327] A period of immediate CPR is therefore mandatory in this age group before searching for an AED or manual defibrillator, as its immediate availability will not improve the outcome of a respiratory arrest. The most common ECG patterns in infants, children and adolescents with cardiopulmonary arrest are asystole and PEA. PEA is characterised by electrical activity on the ECG, and absent pulses. It commonly follows a period of hypoxia or myocardial ischaemia, but occasionally can have a reversible cause (i.e., one of the 4Hs and 4Ts) that led to a sudden impairment of cardiac output.

Shockable rhythms

Primary VF occurs in 3.8% to 19% of cardiopulmonary arrests in children, the incidence of VF/pVT increases as the age increases.[48–56,328] The primary determinant of survival from VF/pVT cardiopulmonary arrest is the time to defibrillation. Prehospital defibrillation within the first 3 min of witnessed adult VF arrest results in >50% survival. However, the success of defibrillation decreases dramatically the longer the time until defibrillation: for every minute delay in defibrillation (without any CPR), survival decreases by 7–10%. Secondary VF is present at some point in up to 27% of in-hospital resuscitation events. It has a much poorer prognosis than primary VF.[329]

Drugs in shockable rhythms

Adrenaline (adrenaline). Adrenaline is given every 3–5 min, every 2 cycles by the IV or IO route.

Amiodarone or lidocaine. Either drug can be given in defibrillation-resistant VF/pVT.

Extracorporeal life support. Extracorporeal life support should be considered for children with cardiac arrest refractory to conventional CPR with a potentially reversible cause, if the arrest occurs where expertise, resources and sustainable systems are available to rapidly initiate extracorporeal life support (ECLS).

Arrhythmias

Unstable arrhythmias

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

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

Bradycardia

Bradycardia is caused commonly by hypoxia, acidosis and/or severe hypotension; it may progress to cardiopulmonary arrest. Give 100% oxygen, and positive pressure ventilation if required, to any child presenting with bradyarrhythmia and circulatory failure.

If a child with decompensated circulatory failure has a heart rate <60 beats min^{-1}, and they do not respond rapidly to ventilation with oxygen, start chest compressions and give adrenaline.

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.[330]

Tachycardia

Narrow complex tachycardia. If SVT is the likely rhythm, vagal manoeuvres (Valsalva or diving reflex) may be used in haemodynamically stable children. They can also be used in haemodynamically unstable children, but only if they do not delay chemical or electrical cardioversion.[331]

Adenosine is usually effective in converting SVT into sinus rhythm. It is given by rapid, intravenous injection as close as practicable to the heart (see above), and followed immediately by a bolus of normal saline. If the child has signs of decompensated shock with depressed conscious level, omit vagal manoeuvres and adenosine and attempt electrical cardioversion immediately.

Electrical cardioversion (synchronised with R wave) is also indicated when vascular access is not available, or when adenosine has failed to convert the rhythm. The first energy dose for electrical cardioversion of SVT is $1\,J\,kg^{-1}$ and the second dose is $2\,J\,kg^{-1}$. If unsuccessful, give amiodarone or procainamide under guidance from a paediatric cardiologist or intensivist before the third attempt. Verapamil may be considered as an alternative therapy in older children but should not be routinely used in infants.

Amiodarone has been shown to be effective in the treatment of SVT in several paediatric studies.[324,332–339] However, since most studies of amiodarone use in narrow complex tachycardias have been for junctional ectopic tachycardia in postoperative children, the applicability of its use in all cases of SVT may be limited. If the child is haemodynamically stable, early consultation with an expert is recommended before giving amiodarone. An expert should also be consulted about alternative treatment strategies because the evidence to support other drugs in the treatment of SVT is limited and inconclusive.[340,341] If amiodarone is used in this circumstance, avoid rapid administration because hypotension is common.

Wide complex tachycardia. In children, wide-QRS complex tachycardia is uncommon and more likely to be supraventricular than ventricular in origin.[342] Nevertheless, in haemodynamically unstable children, it must be considered to be VT until proven otherwise. Ventricular tachycardia occurs most often in the child with underlying heart disease (e.g., after cardiac surgery, cardiomyopathy, myocarditis, electrolyte disorders, prolonged QT interval, central intracardiac catheter).

Synchronised cardioversion is the treatment of choice for unstable VT with signs of life. Consider anti-arrhythmic therapy if a second cardioversion attempt is unsuccessful or if VT recurs.

Amiodarone has been shown to be effective in treating paediatric arrhythmias,[343] although cardiovascular side effects are common.[324,332,334,339,344]

Stable arrhythmias

Whilst maintaining the child's airway, breathing and circulation, contact an expert before initiating therapy. Depending on the child's clinical history, presentation and ECG diagnosis, a child with stable, wide-QRS complex tachycardia may be treated for SVT and be given vagal manoeuvres or adenosine.

Special circumstances

Life support for blunt or penetrating trauma

Cardiac arrest from major (blunt or penetrating) trauma is associated with a very high mortality.[345–352] The 4Ts and 4Hs should be considered as potentially reversible causes. There is little evidence to support any additional specific interventions that are different from the routine management of cardiac arrest; however, the use of resuscitative thoracotomy may be considered in children with penetrating injuries.[353–359]

Extracorporeal membrane oxygenation (ECMO)

For infants and children with a cardiac diagnosis and an in-hospital arrest ECMO should be considered as a useful rescue strategy if sufficient expertise and resources are available. There is insufficient evidence to suggest for or against the use of ECMO in non-cardiac arrest or for children with myocarditis or cardiomyopathy who are not in arrest.[29]

Pulmonary hypertension

There is an increased risk of cardiac arrest in children with pulmonary hypertension.[360,361] 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.[362] Resuscitation is most likely to be successful in patients with a reversible cause who are treated with intravenous epoprostenol or inhaled nitric oxide.[363] 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.[364–368] Right ventricular support devices may improve survival.[369–373]

Post-resuscitation care

After prolonged, complete, whole-body hypoxia-ischaemia ROSC has been described as an unnatural pathophysiological state, created by successful CPR.[374] Post-cardiac arrest care must be a multidisciplinary activity and include all the treatments needed for complete neurological recovery. The main goals are to reverse brain injury and myocardial dysfunction, and to treat the systemic ischaemia/reperfusion response and any persistent precipitating pathology.

Myocardial dysfunction

Myocardial dysfunction is common after cardiopulmonary resuscitation.[374–378] Parenteral fluids and vasoactive drugs (adrenaline, dobutamine, dopamine and noradrenaline) may improve the child's post-arrest haemodynamic status and should be titrated to maintain a systolic blood pressure of at least >5th centile for age.[29,379–390]

Although the measurement of blood pressure has limitations in determining perfusion of vital organs, it is a practical and valued measurement of haemodynamic status. Alternative perfusion endpoints (such as serum lactate levels, measures of cardiac output, mean blood pressure) can be targeted but the evidence for each of them individually is still equivocal. Ideally, they should be considered as a part of a global 'gestalt' observation. The optimal strategy to avoid hypotension i.e. the relative use of parenteral fluids versus inotropes and/or vasopressors in children post ROSC following cardiac arrest currently remains unclear. The need to use agents to maintain a normal blood pressure is a poor prognostic factor.[390]

Finally, subgroups of children might respond differently to components of the above interventions, such as cardiac patients or trauma patients who may be particularly sensitive to preload status and changes in afterload. Any interventions must be monitored and adapted according to the child's physiological responses. Reassessment of the child is key in improving their outcome.

Oxygenation and ventilation goals

Aim for a normal PaO_2 range (normoxaemia) post-ROSC once a patient is stabilised.[167,391–393] Balance the titration of oxygen delivery against the risk of inadvertent hypoxaemia.[29] Further challenges for paediatrics include identifying what the appropriate targets should be for specific patient subpopulations (e.g. infants and children with cyanotic heart disease).

There is insufficient paediatric evidence to suggest a specific $PaCO_2$ target, however, $PaCO_2$ should be measured post-ROSC and adjusted according to patient characteristics and needs.[29,167,394,395]

Adult data do not suggest any added benefit of either hypocapnia or hypercapnia; hypocapnia has even been associated with worse outcome. It is sensible to aim in general for normocapnia, although this decision might be in part influenced by context and disease. For instance, it is unclear if a strategy of permissive mild hypercapnia could be beneficial in ventilated children with respiratory failure.

Temperature control and management post ROSC

Mild hypothermia has an acceptable safety profile in adults[396,397] and neonates.[398–403] Recently the THAPCA out of hospital study showed that both hypothermia (32–34 °C) and controlled normothermia (36–37.5 °C) could be used in children.[404] The study did not show a significant difference for the primary outcome (neurologic status at one year) with either approach. The study was, however, underpowered to show a significant difference for survival, for which the lower 95% confidence interval approached 1. Furthermore, hyperthermia occurred frequently in the post-arrest period; hyperthermia is potentially harmful and should be avoided. After ROSC, a strict control of the temperature must be maintained to avoid hyperthermia (>37.5 °C) and severe hypothermia (<32 °C).[29]

Glucose control

Both hyper- and hypoglycaemia may impair outcome of critically ill adults and children and should be avoided,[405–407] but tight glucose control may also be harmful.[408] Although there is insufficient evidence to support or refute a specific glucose management strategy in children with ROSC after cardiac arrest, it is appropriate to monitor blood glucose and to avoid hypoglycaemia and hyperglycaemia.[280,281,374]

Prognosis of cardiopulmonary arrest

Although several factors are associated with outcome after cardiopulmonary arrest and resuscitation there are no simple guidelines to determine when resuscitative efforts become futile.[29,394,409–414]

The relevant considerations in the decision to continue the resuscitation include the duration of CPR, cause of arrest, pre-existing medical conditions, age, site of arrest, whether the arrest was witnessed,[36,415] the duration of untreated cardiopulmonary arrest ('no flow' time), the presence of a shockable rhythm as the first or subsequent rhythm, and associated special circumstances (e.g., icy water drowning[416,417] exposure to toxic drugs). The role of the EEG as a prognostic factor is still unclear. Problems with the literature in this area to identify individual factors are that the studies have largely not been designed in this context and therefore there may be bias as to its use in determining poor or good outcomes. Guidance on the termination of resuscitation attempts is discussed in the chapter on Ethics in Resuscitation and End-of-Life Decisions.[17]

Parental presence

In some Western societies, the majority of parents want to be present during the resuscitation of their child.[418–440] Parental presence has neither been perceived as disruptive nor stressful for the staff.[418,420,436,441] Parents witnessing their child's resuscitation believe their presence to be beneficial to the child.[418–420,427,438,442,443] Allowing parents to be at the side of their child helps them to gain a realistic view of the attempted resuscitation and the child's death. Furthermore, they may have the opportunity to say goodbye to their child. Families who are present at their child's death show better adjustment and undergo a better grieving process.[419–421,438,439,443,444]

Parental presence in the resuscitation room may help healthcare providers maintain their professional behaviour, whilst helping them to see the child as a human being and a family member.[435,440] However, in out-of-hospital resuscitation, some EMS providers may feel anxious owing to the presence of relatives or are concerned that relatives may interfere with their resuscitation efforts.[445] Evidence about parental presence during resuscitation comes from selected countries and can probably not be generalised to all of Europe, where there may be different socio-cultural and ethical considerations.[446,447]

Family presence guidelines

When parents are in the resuscitation room, a member of the resuscitation team should be allocated to them and explain the process in an empathetic manner, ensuring that they do not interfere with or distract the resuscitation process. If the presence of the relatives is impeding the progress of the resuscitation, they should be sensitively asked to leave. When appropriate, physical contact with the child should be allowed and, wherever possible, the parents should be allowed to be with their dying child at the final moment.[435,448–451] The number of relatives present should be at the discretion of the resuscitation team leader.

The leader of the resuscitation team, not the parents, will decide when to stop the resuscitation; this should be expressed with sensitivity and understanding. After the event, the team should be debriefed, to enable any concerns to be expressed and for the team to reflect on their clinical practice in a supportive environment.

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Conflict of interest statement

The authors declare no conflict of interest.

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European Resuscitation Council Guidelines for Resuscitation 2015 Section 7. Resuscitation and support of transition of babies at birth

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Introduction

The following guidelines for resuscitation at birth have been developed during the process that culminated in the 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations (CoSTR, 2015).[1,2] They are an extension of the guidelines already published by the ERC[3] and take into account recommendations made by other national and international organisations and previously evaluated evidence.[4]

Summary of changes since 2010 guidelines

The following are the main changes that have been made to the guidelines for resuscitation at birth in 2015:

- **Support of transition**: Recognising the unique situation of the baby at birth, who rarely requires 'resuscitation' but sometimes needs medical help during the process of postnatal transition. The term 'support of transition' has been introduced to better distinguish between interventions that are needed to restore vital organ functions (resuscitation) or to support transition.
- **Cord clamping**: For uncompromised babies, a delay in cord clamping of at least 1 min from the complete delivery of the infant, is now recommended for term and preterm babies. As yet there is insufficient evidence to recommend an appropriate time for clamping the cord in babies who require resuscitation at birth.
- **Temperature**: The temperature of newly born non-asphyxiated infants should be maintained between 36.5 °C and 37.5 °C after birth. The importance of achieving this has been highlighted and reinforced because of the strong association with mortality and morbidity. The admission temperature should be recorded as a predictor of outcomes as well as a quality indicator.
- **Maintenance of temperature**: At <32 weeks gestation, a combination of interventions may be required to maintain the temperature between 36.5 °C and 37.5 °C after delivery through admission and stabilisation. These may include warmed humidified respiratory gases, increased room temperature plus plastic wrapping of body and head, plus thermal mattress or a thermal mattress alone, all of which have been effective in reducing hypothermia.
- **Optimal assessment of heart rate**: It is suggested in babies requiring resuscitation that the ECG can be used to provide a rapid and accurate estimation of heart rate.
- **Meconium**: Tracheal intubation should not be routine in the presence of meconium and should only be performed for suspected tracheal obstruction. The emphasis should be on initiating ventilation within the first minute of life in non-breathing or ineffectively breathing infants and this should not be delayed.
- **Air/Oxygen**: Ventilatory support of term infants should start with air. For preterm infants, either air or a low concentration of oxygen (up to 30%) should be used initially. If, despite effective ventilation, oxygenation (ideally guided by oximetry) remains unacceptable, use of a higher concentration of oxygen should be considered.
- **Continuous Positive Airways Pressure (CPAP)**: Initial respiratory support of spontaneously breathing preterm infants with respiratory distress may be provided by CPAP rather than intubation.

The guidelines that follow do not define the only way that resuscitation at birth should be achieved; they merely represent a widely accepted view of how resuscitation at birth can be carried out both safely and effectively (Fig. 7.1).

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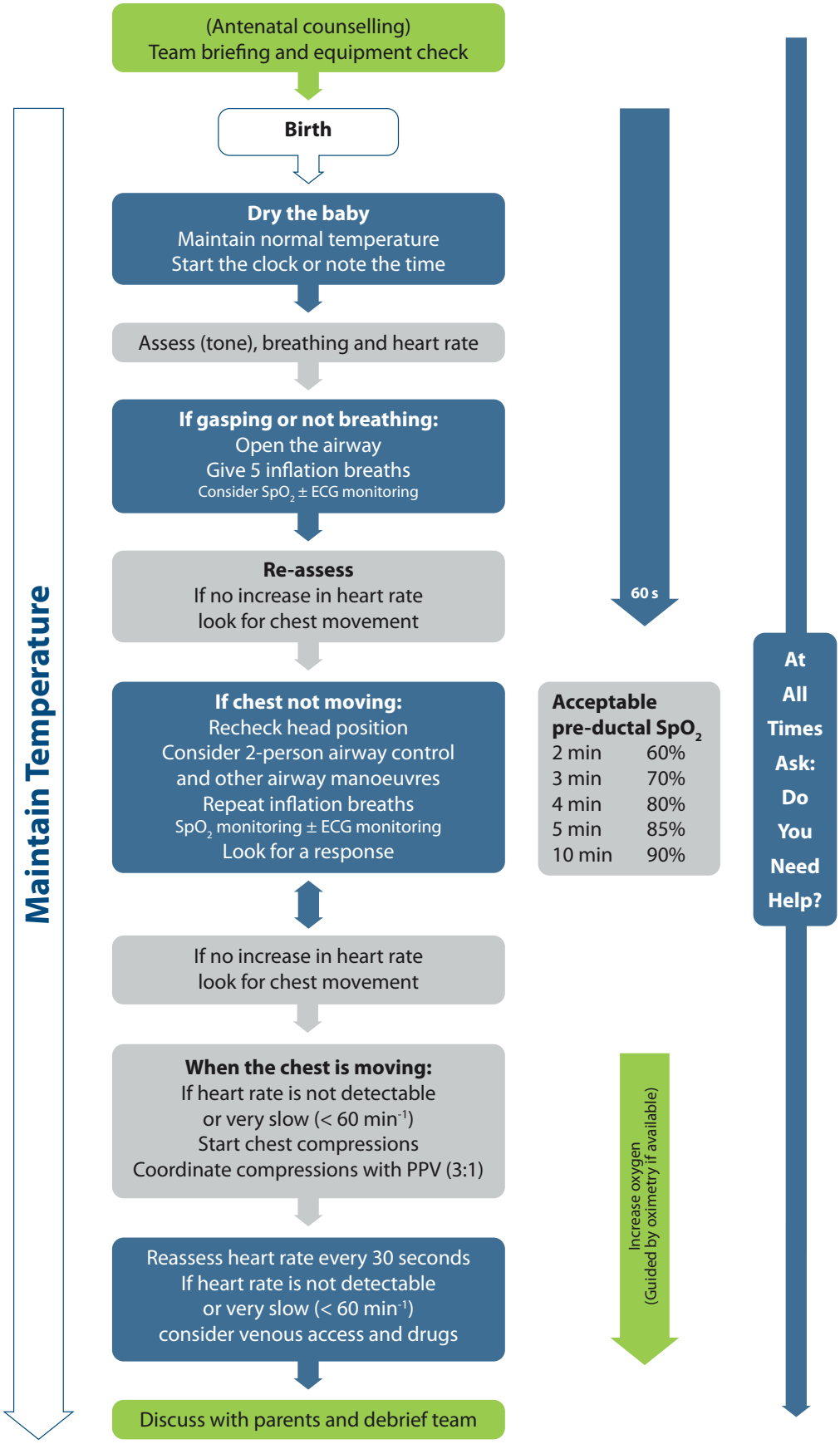

Fig. 7.1. Newborn life support algorithm. SpO_2: transcutaneous pulse oximetry, ECG: electrocardiograph, PPV: positive pressure ventilation.

Preparation

The fetal-to-neonatal transition, which occurs at the time of birth, requires anatomic and physiological adjustments to achieve the conversion from placental gas exchange with intra-uterine lungs filled with fluid, to pulmonary respiration with aerated lungs. The absorption of lung fluid, the aeration of the lungs, the initiation of air breathing, and cessation of the placental circulation bring about this transition.

A minority of infants require resuscitation at birth, but a few more have problems with this perinatal transition, which, if no support is given, might subsequently result in a need for resuscitation. Of those needing any help, the overwhelming majority will require only assisted lung aeration. A tiny minority may need a brief period of chest compressions in addition to lung aeration. In a retrospective study, approximately 85% of babies born at term initiated spontaneous respirations within 10 to 30 s of birth; an additional 10% responded during drying and stimulation, approximately 3% initiated respirations following positive pressure ventilation, 2% were intubated to support respiratory function and 0.1% received chest compressions and/or adrenaline.[5–7] However, of 97,648 babies born in Sweden in one year, only 10 per 1000 (1%) babies of 2.5 kg or more appeared to need any resuscitation at delivery.[8] Most of those, 8 per 1000, responded to mask inflation of the lungs 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 or help with transition at delivery. Of these, 90% responded to mask ventilation alone while the remaining 10% appeared not to respond to mask inflation and therefore were intubated at birth. There was almost no need for cardiac compressions.

Resuscitation or support of transition 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, maternal infection and multiple pregnancies.[9] Furthermore, caesarean delivery is associated with an increased risk of problems with respiratory transition at birth requiring medical interventions especially for deliveries before 39 weeks gestation.[10–13] However, elective caesarean delivery at term does not increase the risk of needing newborn resuscitation in the absence of other risk factors.[14–17]

Although it is sometimes possible to predict the need for resuscitation or stabilisation before a baby is born, this is not always the case. Any newborn may potentially develop problems during birth, therefore, personnel trained in newborn life support should be easily available for every delivery. In deliveries with a known increased risk of problems, specially trained personnel should be present with at least one person experienced in tracheal intubation. Should there be any need for intervention, the care of the baby should be their sole responsibility. Local guidelines indicating who should attend deliveries should be developed, based on current practice and clinical audit. Each institution should have a protocol in place for rapidly mobilising a team with competent resuscitation skills for any birth. Whenever there is sufficient time, the team attending the delivery should be briefed before delivery and clear role assignment should be defined. It is also important to prepare the family in cases where it is likely that resuscitation might be required.

A structured educational programme, teaching the standards and skills required for resuscitation of the newborn is therefore essential for any institution or clinical area in which deliveries may occur. Continued experiential learning and practice is necessary to maintain skills.

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 assessment, stabilisation or 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 take place in a warm, well-lit, draught free area with a flat resuscitation surface placed below a radiant heater (if in hospital), with other resuscitation equipment immediately available. All equipment must be regularly checked and tested.

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 and subsequent ventilation of an appropriate size for the newborn, warm dry towels and blankets, a sterile instrument for cutting and clamping the umbilical cord and clean gloves for the attendant and assistants. Unexpected deliveries outside hospital are most likely to involve emergency services that should plan for such events.

Timing of clamping the umbilical cord

Cine-radiographic studies of babies taking their first breath at delivery showed that those whose cords were clamped prior to this had an immediate decrease in the size of the heart during the subsequent three or four cardiac cycles. The heart then increased in size to almost the same size as the fetal heart. The initial decrease in size could be interpreted as the significantly increased pulmonary blood flow following the decrease in pulmonary vascular resistance upon lung aeration. The subsequent increase in size would, as a consequence, be caused by the blood returning to the heart from the lung.[18] Brady et al drew attention to the occurrence of a bradycardia apparently induced by clamping the cord before the first breath and noted that this did not occur in babies where clamping occurred after breathing was established.[19] Experimental evidence from similarly treated lambs suggest the same holds true for premature newborn.[20]

Studies of delayed clamping have shown an improvement in iron status and a number of other haematological indices over the next 3–6 months and a reduced need for transfusion in preterm infants.[21,22] They have also suggested greater use of phototherapy for jaundice in the delayed group but this was not found in a randomised controlled trial.[21]

A systematic review on delayed cord clamping and cord milking in preterm infants found improved stability in the immediate postnatal period, including higher mean blood pressure and haemoglobin on admission, compared to controls.[23] There were also fewer blood transfusions in the ensuing weeks.[23] Some studies have suggested a reduced incidence of intraventricular haemorrhage and periventricular leukomalacia[22,24,25] as well as of late-onset sepsis.[24]

No human studies have yet addressed the effect of delaying cord clamping on babies apparently needing resuscitation at birth because such babies have been excluded from previous studies.

Delaying umbilical cord clamping for at least 1 min is recommended for newborn infants not requiring resuscitation. A similar delay should be applied to preterm babies not requiring immediate resuscitation after birth. Until more evidence is available, infants who are not breathing or crying may require the umbilical cord to be clamped, so that resuscitation measures can commence promptly. Umbilical cord milking may prove an alternative in these infants although there is currently not enough evidence available to recommended this as a routine measure.[1,2] Umbilical cord milking produces improved short term haematological outcomes, admission temperature and urine output when compared to delayed cord clamping (>30 s) in babies born by caesarean section, although these differences were not observed in infants born vaginally.[26]

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.[27] Exposure of the newborn to cold stress will lower arterial oxygen tension[28] and increase metabolic acidosis.[29] The association between hypothermia and mortality has been known for more than a century,[30] and the admission temperature of newborn non-asphyxiated infants is a strong predictor of mortality at all gestations and in all settings.[31–65] Preterm infants are especially vulnerable and hypothermia is also associated with serious morbidities such as intraventricular haemorrhage[35,42,55,66–69] need for respiratory support[31,35,37,66,70–74] hypoglycaemia[31,49,60,74–79] and in some studies late onset sepsis.[49]

The temperature of newly born non-asphyxiated infants should be maintained between 36.5 °C and 37.5 °C after birth. For each 1 °C decrease in admission temperature below this range there is an associated increase in mortality by 28%.[1,2,49] The admission temperature should be recorded as a predictor of outcomes as well as a quality indicator.

Prevent heat loss:

- Protect the baby from draughts.[80] Make certain windows closed and air-conditioning appropriately programmed.[52]
- Dry the term baby immediately after delivery. Cover the head and body of the baby, apart from the face, with a warm and dry towel to prevent further heat loss. Alternatively, place the baby skin to skin with mother and cover both with a towel.
- Keep the delivery room warm at 23–25 °C.[1,2,48,80] For babies less than 28 weeks gestation the delivery room temperature should be >25 °C.[27,48,79,81]
- If the baby needs support in transition or resuscitation then place the baby on a warm surface under a preheated radiant warmer.
- All babies less than 32 weeks gestation should have the head and body of the baby (apart from the face) covered with polyethylene wrapping, without drying the baby beforehand, and also placed under a radiant heater.[73,77,82,83]
- In addition, babies <32 weeks gestation, may require a combination of further interventions to maintain the temperature between 36.5 °C and 37.5 °C after delivery through admission and stabilisation. These may include warmed humidified respiratory gases,[84,85] increased room temperature plus cap plus thermal mattress [70,72,86,87] or thermal mattress alone,[88–92] which have all been effective in reducing hypothermia.
- Babies born unexpectedly outside a normal delivery environment may benefit from placement in a food grade plastic bag after drying and then swaddling.[93,94] Alternatively, well newborns >30 weeks gestation may be dried and nursed with skin to skin contact or kangaroo mother care to maintain their temperature whilst they are transferred.[95–101] They should be covered and protected from draughts.

Whilst maintenance of a baby's temperature is important, this should be monitored in order to avoid hyperthermia (>38.0 °C). Infants born to febrile mothers have a higher incidence of perinatal respiratory depression, neonatal seizures, early mortality and cerebral palsy.[102,103] Animal studies indicate that hyperthermia during or following ischaemia is associated with a progression of cerebral injury.[104,105]

Initial assessment

The Apgar score was not designed to be assembled and ascribed in order to then identify babies in need of resuscitation.[106,107] However, individual components of the score, namely respiratory rate, heart rate and tone, if assessed rapidly, can identify babies needing resuscitation, (and Virginia Apgar herself found that heart rate was the most important predictor of immediate outcome).[106] 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

Immediately after birth the heart rate is assessed to evaluate the condition of the baby and subsequently is the most sensitive indicator of a successful response to interventions. Heart rate is initially most rapidly and accurately assessed by listening to the apex beat with a stethoscope[108] or by using an electrocardiograph.[109–112] Feeling the pulse in the base of the umbilical cord is often effective but can be misleading because cord pulsation is only reliable if found to be more than 100 beats per minute (bpm)[108] and clinical assessment may underestimate the heart rate.[108,109,113] For babies requiring resuscitation and/or continued respiratory support, a modern pulse oximeter can give an accurate heart rate.[111] Several studies have demonstrated that ECG is faster than pulse oximetry and more reliable, especially in the first 2 min after birth;[110–115] however, the use of ECG does not replace the need to use pulse oximetry to assess the newborn baby's oxygenation.

Colour

Colour is a poor means of judging oxygenation,[116] 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 hypoxaemia. 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 preductal 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}.**

There is no need for immediate clamping of the cord. 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. It remains important to ensure the baby's temperature is maintained.

(2)	**Breathing inadequately or apnoeic.** **Normal or reduced tone.** **Heart rate less than 100 min^{-1}.**

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

(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.

Preterm babies may be breathing and showing signs of respiratory distress in which case they should be supported initially with CPAP.

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

Newborn life support

Commence newborn life support if initial assessment shows that the baby has failed to establish adequate regular normal breathing, or has a heart rate of less than 100 min^{-1} (Fig. 7.1). Opening the airway and aerating the lungs is usually all that is necessary. Furthermore, more complex interventions will be futile unless these two first steps have been successfully completed.

Airway

Place the baby on his or her back with the head in a neutral position (Fig. 7.2). A 2 cm thickness of the blanket or towel placed under the baby's shoulder may be helpful in maintaining proper head position. In floppy babies application of jaw thrust or the use of an appropriately sized oropharyngeal airway may be essential in opening the airway.

The supine position for airway management is traditional but side-lying has also been used for assessment and routine delivery room management of term newborns but not for resuscitation.[117]

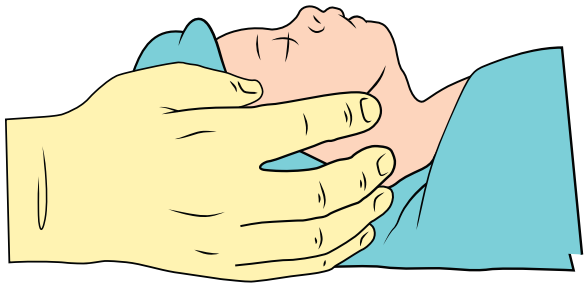

Fig. 7.2. Newborn with head in neutral position.

There is no need to remove lung fluid from the oropharynx routinely.[118] Suction is needed only if the airway is obstructed. Obstruction may be caused by particulate meconium but can also be caused by blood clots, thick tenacious mucus or vernix even in deliveries where meconium staining is not present. However, aggressive pharyngeal suction can delay the onset of spontaneous breathing and cause laryngeal spasm and vagal bradycardia.[119–121]

Meconium

For over 30 years it was hoped that clearing meconium from the airway of babies at birth would reduce the incidence and severity of meconium aspiration syndrome (MAS). However, studies supporting this view were based on a comparison of suctioning on the outcome of a group of babies with the outcome of historical controls.[122,123] Furthermore other studies failed to find any evidence of benefit from this practice.[124,125]

Lightly meconium stained liquor is common and does not, in general, give rise to much difficulty with transition. The much less common finding of very thick meconium stained liquor at birth is an indicator of perinatal distress and should alert to the potential need for resuscitation. Two multi-centre randomised controlled trials showed that routine elective intubation and tracheal suctioning of these infants, if vigorous at birth, did not reduce MAS [126] and that suctioning the nose and mouth of such babies on the perineum and before delivery of the shoulders (intrapartum suctioning) was ineffective.[127] Hence intrapartum suctioning and routine intubation and suctioning of vigorous infants born through meconium stained liquor are not recommended. A small RCT has recently demonstrated no difference in the incidence of MAS between patients receiving tracheal intubation followed by suctioning and those not intubated.[128]

The presence of thick, viscous meconium in a non-vigorous baby is the only indication for initially considering visualising the oropharynx and suctioning material, which might obstruct the airway. Tracheal intubation should not be routine in the presence of meconium and should only be performed for suspected tracheal obstruction.[128–132] The emphasis should be on initiating ventilation within the first minute of life in non-breathing or ineffectively breathing infants and this should not be delayed. If suctioning is attempted use a 12–14 FG suction catheter, or a paediatric Yankauer sucker, connected to a suction source not exceeding −150 mmHg.[133] The routine administration of surfactant or bronchial lavage with either saline or surfactant is not recommended.[134,135]

Initial breaths and assisted ventilation

After initial steps at birth, if breathing efforts are absent or inadequate, lung aeration is the priority and must not be delayed (Fig. 7.3). In term babies, respiratory support should start with air.[136] The primary measure of adequate initial lung inflation is a

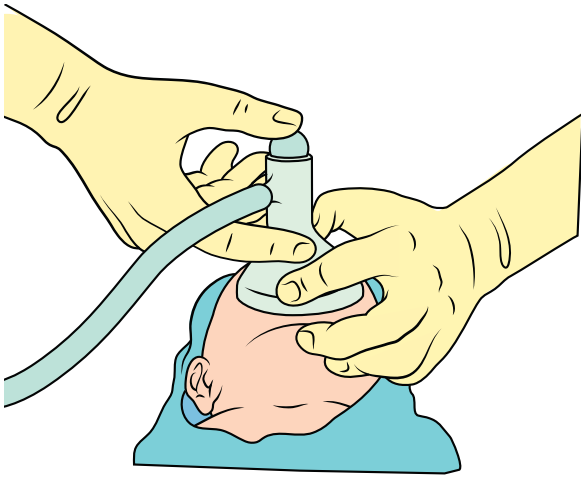

Fig. 7.3. Mask ventilation of newborn.

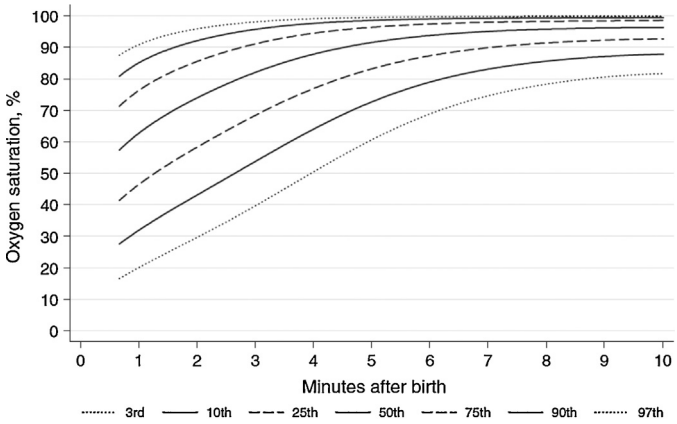

Fig. 7.4. Oxygen saturations (3rd, 10th, 25th, 50th, 75th, 90th, and 97th SpO_2 percentiles) in healthy infants at birth without medical intervention. Reproduced with permission from. [157]

prompt improvement in heart rate. If the heart rate is not improving assess the chest wall movement. In term infants, spontaneous or assisted initial inflations create a functional residual capacity (FRC).[137–141] The optimum pressure, inflation time and flow required to establish an effective FRC has not been determined.

For the first five positive pressure inflations maintain the initial inflation pressure for 2–3 s. This will usually help lung expansion.[137,142] The pressure required to aerate the fluid filled lungs of newborn babies requiring resuscitation is 15–30 cm H_2O (1.5–2.9 kPa) with a mean of 20 cm H_2O.[137,141,142] For term babies use an inflation pressure of 30 cm H_2O and 20–25 cm H_2O in preterm babies.[143,144]

Efficacy of the intervention can be estimated by a prompt increase in heart rate or observing the chest rise. If this is not obtained it is likely that repositioning of the airway or mask will be required and, rarely, higher inspiratory pressures may be needed. Most babies needing respiratory support at birth will respond with a rapid increase in heart rate within 30 s of lung inflation. If the heart rate increases but the baby is not breathing adequately, ventilate at a rate of about 30 breaths min^{-1} allowing approximately 1 s for each inflation, until there is adequate spontaneous breathing.

Adequate passive ventilation is usually indicated by either a rapidly increasing heart rate or a heart rate that is maintained faster than 100 beats min^{-1}. If the baby does not respond in this way the most likely cause is inadequate airway control or inadequate ventilation. Look for passive chest movement in time with inflation efforts; if these are present then lung aeration has been achieved. If these are absent then airway control and lung aeration has not been confirmed. Mask leak, inappropriate airway position and airway obstruction, are all possible reasons, which may need correction.[145–149] In this case, consider repositioning the mask to correct for leakage and/or reposition the baby's head to correct for airway obstruction.[145] Alternatively using a two person approach to mask ventilation reduces mask leak in term and preterm infants.[146,147] Without adequate lung aeration, chest compressions will be ineffective; therefore, confirm lung aeration and ventilation 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.

Sustained inflations (SI) > 5 s

Several animal studies have suggested that a longer SI may be beneficial for establishing functional residual capacity at birth during transition from a fluid-filled to air-filled lung.[150,151] Review of the literature in 2015 disclosed three RCTs[152–154] and two cohort studies,[144,155] which demonstrated that initial SI reduced the need for mechanical ventilation. However, no benefit was found for reduction of mortality, bronchopulmonary dysplasia, or air leak. One cohort study[144] suggested that the need for intubation was less following SI. It was the consensus of the COSTR reviewers that there was inadequate study of the safety, details of the most appropriate length and pressure of inflation, and long-term effects, to suggest routine application of SI of greater than 5 s duration to the transitioning newborn.[1,2] Sustained inflations >5 s should only be considered in individual clinical circumstances or in a research setting.

Air/Oxygen

Term babies. In term infants receiving respiratory support at birth with positive pressure ventilation (PPV), it is best to begin with air (21%) as opposed to 100% oxygen. If, despite effective ventilation, there is no increase in heart rate or oxygenation (guided by oximetry wherever possible) remains unacceptable, use a higher concentration of oxygen to achieve an adequate preductal oxygen saturation.[156,157] High concentrations of oxygen are associated with an increased mortality and delay in time of onset of spontaneous breathing,[158] therefore, if increased oxygen concentrations are used they should be weaned as soon as possible.[136,159]

Preterm babies. Resuscitation of preterm infants less than 35 weeks gestation at birth should be initiated in air or low concentration oxygen (21–30%).[1,2,136,160] The administered oxygen concentration should be titrated to achieve acceptable pre-ductal oxygen saturations approximating to the 25th percentile in healthy term babies immediately after birth (Fig. 7.4).[156,157]

In a meta-analysis of seven randomized trials comparing initiation of resuscitation with high (>65%) or low (21–30%) oxygen concentrations, the high concentration was not associated with any improvement in survival,[159,161–166] bronchopulmonary dysplasia,[159,162,164–166] intraventricular haemorrhage[159,162,165,166] or retinopathy of prematurity.[159,162,166] There was an increase in markers of oxidative stress.[159]

Pulse oximetry. Modern pulse oximetery, using neonatal probes, provides reliable readings of heart rate and transcutaneous oxygen saturation within 1–2 min of birth (Fig. 7.4).[167,168] A reliable

pre-ductal reading can be obtained from >90% of normal term births, approximately 80% of those born preterm, and 80-90% of those apparently requiring resuscitation, within 2 min of birth.[167] Uncompromised babies born at term at sea level have SpO_2 ~60% during labour,[169] which increases to >90% by 10 min.[156] The 25th percentile is approximately 40% at birth and increases to ~80% at 10 min.[157] Values are lower in those born by Caesarean delivery,[170] those born at altitude[171] and those managed with delayed cord clamping.[172] Those born preterm may take longer to reach >90%.[157]

Pulse oximetry should be used to avoid excessive use of oxygen as well as to direct its judicious use (Figs. 7.1 and 7.4). Transcutaneous oxygen saturations above the acceptable levels should prompt weaning of any supplemental oxygen.

Positive end expiratory pressure

All term and preterm babies who remain apnoeic despite initial steps must receive positive pressure ventilation after initial lung inflation. It is suggested that positive end expiratory pressure (PEEP) of ~5 cm H_2O should be administered to preterm newborn babies receiving PPV.[173]

Animal studies show that preterm lungs are easily damaged by large-volume inflations immediately after birth[174] and suggest that maintaining a PEEP immediately after birth may protect against lung damage[175,176] although some evidence suggests no benefit.[177] PEEP also improves lung aeration, compliance and gas exchange.[178–180] Two human newborn RCTs demonstrated no improvement in mortality, need for resuscitation or bronchopulmonary dysplasia they were underpowered for these outcomes.[181,182] However, one of the trials suggested that PEEP reduced the amount of supplementary oxygen required.[182]

Assisted ventilation devices

Effective ventilation can be achieved with a flow-inflating, a self-inflating bag or with a T-piece mechanical device designed to regulate pressure.[181–185] The blow-off valves of self-inflating bags are flow-dependent and pressures generated may exceed the value specified by the manufacturer if compressed vigorously.[186,187] Target inflation pressures, tidal volumes and long inspiratory times are achieved more consistently in mechanical models when using T-piece devices than when using bags,[187–190] although the clinical implications are not clear. More training is required to provide an appropriate pressure using flow-inflating bags compared with self-inflating bags.[191] A self-inflating bag, a flow-inflating bag or a T-piece mechanical device, all designed to regulate pressure or limit pressure applied to the airway can be used to ventilate a newborn. However, self-inflating bags are the only devices, which can be used in the absence of compressed gas but cannot deliver continuous positive airway pressure (CPAP) and may not be able to achieve PEEP even with a PEEP valve in place[189,192–195]

Respiratory function monitors measuring inspiratory pressures and tidal volumes [196] and exhaled carbon dioxide monitors to assess ventilation [197,198] have been used but there is no evidence that they affect outcomes. Neither additional benefit above clinical assessment alone, nor risks attributed to their use have so far been identified. The use of exhaled CO_2 detectors to assess ventilation with other interfaces (e.g., nasal airways, laryngeal masks) during PPV in the delivery room has not been reported.

Face mask versus nasal prong

A reported problem of using the facemask for newborn ventilation is mask leak caused by a failure of the seal between the mask and the face.[145–148] To avoid this some institutions are using nasopharyngeal prongs to deliver respiratory support. Two randomised trials in preterm infants have compared the efficacy and did not find any difference between the methods.[199,200]

Table 1
Oral tracheal tube lengths by gestation.

Gestation (weeks)	ETT at lips (cm)
23–24	5·5
25–26	6·0
27–29	6·5
30–32	7·0
33–34	7·5
35–37	8·0
38–40	8·5
41–43	9·0

Laryngeal mask airway

The laryngeal mask airway can be used in resuscitation of the newborn, particularly if facemask ventilation is unsuccessful or tracheal intubation is unsuccessful or not feasible. The LMA may be considered as an alternative to a facemask for positive pressure ventilation among newborns weighing more than 2000 g or delivered ≥34 weeks gestation.[201] One recent unblinded RCT demonstrated that following training with one type of LMA, its use was associated with less tracheal intubation and neonatal unit admission in comparison to those receiving ventilation via a facemask.[201] There is limited evidence, however, to evaluate its use for newborns weighing <2000 gram or delivered <34 weeks gestation. The laryngeal mask airway may be considered as an alternative to tracheal intubation as a secondary airway for resuscitation among newborns weighing more than 2000 g or delivered ≥34 weeks gestation.[201–206] The LMA is recommended during resuscitation of term and preterm newborns ≥34 weeks gestation when tracheal intubation is unsuccessful or not feasible. The laryngeal mask airway has not been evaluated in the setting of meconium stained fluid, during chest compressions, or for the administration of emergency intra-tracheal medications.

Tracheal tube placement

Tracheal intubation may be considered at several points during neonatal resuscitation:

- When suctioning the lower airways to remove a presumed tracheal blockage.
- When, after correction of mask technique and/or the baby's head position, bag-mask ventilation is ineffective or prolonged.
- When chest compressions are performed.
- Special circumstances (e.g., congenital diaphragmatic hernia or to give tracheal surfactant).

The use and timing of tracheal intubation will depend on the skill and experience of the available resuscitators. Appropriate tube lengths based on gestation are shown in Table 1.[207] It should be recognised that vocal cord guides, as marked on tracheal tubes by different manufacturers to aid correct placement, vary considerably.[208]

Tracheal tube placement must be assessed visually during intubation, and positioning confirmed. Following tracheal intubation and intermittent positive-pressure, a prompt increase in heart rate is a good indication that the tube is in the tracheobronchial tree. [209] Exhaled CO_2 detection is effective for confirmation of tracheal tube placement in infants, including VLBW infants[210–213] and neonatal studies suggest that it confirms tracheal intubation in neonates with a cardiac output more rapidly and more accurately than clinical assessment alone.[212–214] Failure to detect exhaled CO_2 strongly suggests oesophageal intubation[210,212] but false negative readings have been reported during cardiac arrest [210] and in VLBW infants

despite models suggesting efficacy.[215] However, neonatal studies have excluded infants in need of extensive resuscitation. False positives may occur with colorimetric devices contaminated with adrenaline (epinephrine), surfactant and atropine.[198]

Poor or absent pulmonary blood flow or tracheal obstruction may prevent detection of exhaled CO_2 despite correct tracheal tube placement. Tracheal tube placement is identified correctly in nearly all patients who are not in cardiac arrest[211]; however, in critically ill infants with poor cardiac output, inability to detect exhaled CO_2 despite correct placement may lead to unnecessary extubation. Other clinical indicators of correct tracheal tube placement include evaluation of condensed humidified gas during exhalation and presence or absence of chest movement, but these have not been evaluated systematically in newborn babies.

Detection of exhaled carbon dioxide in addition to clinical assessment is recommended as the most reliable method to confirm tracheal placement in neonates with spontaneous circulation.[3,4]

CPAP

Initial respiratory support of all spontaneously breathing preterm infants with respiratory distress may be provided by CPAP, rather than intubation. Three RCTs enrolling 2358 infants born at <30 weeks gestation demonstrated that CPAP is beneficial when compared to initial tracheal ventilation and PPV in reducing the rate of intubation and duration of mechanical ventilation without any short term disadvantages.[216–218] There are few data to guide the appropriate use of CPAP in term infants at birth and further clinical studies are required.[219,220]

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. As ventilation is the most effective and important intervention in newborn resuscitation, and may be compromised by compressions, it is vital to ensure that effective ventilation is occurring before commencing chest compressions.

The most effective technique for providing chest compressions is with two thumbs over the lower third of the sternum with the fingers encircling the torso and supporting the back (Fig. 7.5).[221–224] This technique generates higher blood pressures and coronary artery perfusion with less fatigue than the previously used two-finger technique.[222–234] In a manikin study overlapping the thumbs on the sternum was more effective than positioning them adjacent but more likely to cause fatigue.[235] 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.[225,236–240] Use a 3:1 compression to ventilation ratio, aiming to achieve approximately 120 events per minute, i.e. approximately 90 compressions and 30 ventilations.[241–246] There are theoretical advantages to allowing a relaxation phase that is very slightly longer than the compression phase.[247] However, the quality of the compressions and breaths are probably more important than the rate. Compressions and ventilations should be coordinated to avoid simultaneous delivery.[248] A 3:1 compression to ventilation ratio is used for resuscitation at birth where compromise of gas exchange is nearly always the primary cause of cardiovascular collapse, but rescuers may consider using higher ratios (e.g., 15:2) if the arrest is believed to be of cardiac origin.

When resuscitation of a newborn baby has reached the stage of chest compressions, the steps of trying to achieve return of spontaneous circulation using effective ventilation with low

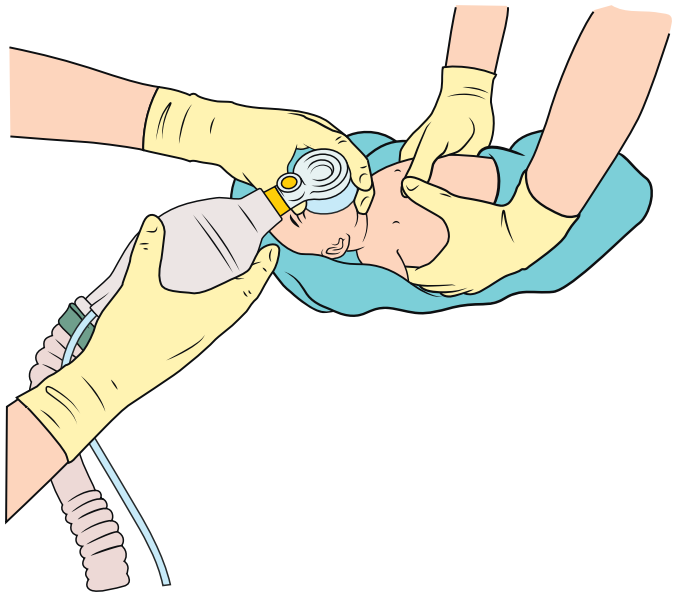

Fig. 7.5. Ventilation and chest compression of newborn.

concentration oxygen should have been attempted. Thus, it would appear sensible to try increasing the supplementary oxygen concentration towards 100%. There are no human studies to support this and animal studies demonstrate no advantage to 100% oxygen during CPR.[249–255]

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}. Exhaled carbon dioxide monitoring and pulse oximetry have been reported to be useful in determining the return of spontaneous circulation[256–260]; however, current evidence does not support the use of any single feedback device in a clinical setting.[1,2]

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 a centrally positioned umbilical venous catheter (Fig. 7.6).

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

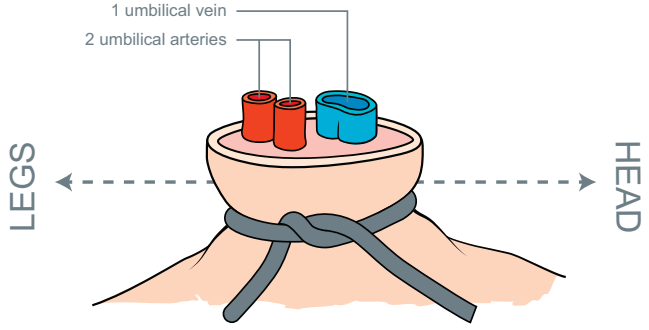

Fig. 7.6. Newborn umbilical cord showing the arteries and veins.

adrenaline is used, an initial dose 10 micrograms kg^{-1} (0.1 ml kg^{-1} of 1:10,000 adrenaline) should be administered intravenously as soon as possible[1,2,4] with subsequent intravenous doses of 10–30 micrograms kg^{-1} (0.1–0.3 ml kg^{-1} of 1:10,000 adrenaline) if required.

The tracheal route is not recommended but if it is used, it is highly likely that doses of 50–100 micrograms kg^{-1} will be required.[3,7,136,261–265] Neither the safety nor the efficacy of these higher tracheal doses has been studied. Do not give these high doses intravenously.

Bicarbonate

If effective spontaneous cardiac output is not restored despite adequate ventilation and adequate chest compressions, reversing intracardiac acidosis may improve myocardial function and achieve a spontaneous circulation. There are insufficient data to recommend routine use of bicarbonate in resuscitation of the newly born. The hyperosmolarity and carbon dioxide-generating properties of sodium bicarbonate may impair myocardial and cerebral function. Use of sodium bicarbonate is not recommended 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.[266] 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. When resuscitating preterm infants volume is rarely needed and has been associated with intraventricular and pulmonary haemorrhages when large volumes are infused rapidly.

Withholding or discontinuing resuscitation

Mortality and morbidity for newborns varies according to region and to availability of resources.[267] Social science studies indicate that parents desire a larger role in decisions to resuscitate and to continue life support in severely compromised babies.[268] Opinions vary amongst providers, parents and societies about the balance of benefits and disadvantages of using aggressive therapies in such babies.[269,270] Local survival and outcome data are important in appropriate counselling of parents. A recent study suggests that the institutional approach at the border of viability affects the subsequent results in surviving infants.[271]

Discontinuing resuscitation

Local and national committees will define recommendations for stopping resuscitation. If the heart rate of a newly born baby is not detectable and remains undetectable for 10 min, it may be appropriate to consider stopping resuscitation. The decision to continue resuscitation efforts when the heart rate has been undetectable for longer than 10 min is often complex and may be influenced by issues such as the presumed aetiology, the gestation of the baby, the potential reversibility of the situation, the availability of therapeutic hypothermia and the parents' previous expressed feelings about acceptable risk of morbidity.[267,272–276] The decision should be individualised. 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.

Withholding resuscitation

It is possible to identify conditions associated with high mortality and poor outcome, where withholding resuscitation may be considered reasonable, particularly when there has been the opportunity for discussion with parents.[38,272,277–282] There is no evidence to support the prospective use of any particular delivery room prognostic score presently described, over gestational age assessment alone, in preterm infants <25 weeks gestation.

A consistent and coordinated approach to individual cases by the obstetric and neonatal teams and the parents is an important goal.[283] Withholding resuscitation and discontinuation of life-sustaining treatment during or following resuscitation are considered by many to be ethically equivalent and clinicians should not be hesitant to withdraw support when the possibility of functional survival is highly unlikely. The following guidelines must be interpreted according to current regional outcomes.

- Where gestation, birth weight, and/or congenital anomalies are associated with almost certain early death, and unacceptably high morbidity is likely among the rare survivors, resuscitation is not indicated.[38,277,284] Examples from the published literature include: extreme prematurity (gestational age less than 23 weeks and/or birth weight less than 400 g), and anomalies such as anencephaly and confirmed Trisomy 13 or 18.
- Resuscitation is nearly always indicated in conditions associated with a high survival rate and acceptable morbidity. This will generally include babies with gestational age of 25 weeks or above (unless there is evidence of fetal compromise such as intrauterine infection or hypoxia-ischaemia) and those with most congenital malformations.
- In conditions associated with uncertain prognosis, where there is borderline survival and a relatively high rate of morbidity, and where the anticipated burden to the child is high, parental desires regarding resuscitation should be supported.[283]
- When withdrawing or withholding resuscitation, care should be focused on the comfort and dignity of the baby and family.

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 the routine local plan and, if possible, hand the baby to the mother at the earliest opportunity. If resuscitation is required inform the parents of the procedures undertaken and why they were required.

European guidelines are supportive of family presence during cardiopulmonary resuscitation.[285] In recent years healthcare professionals are increasingly offering family members the opportunity to remain present during CPR and this is more likely if resuscitation takes place within the delivery room. Parents' wishes to be present during resuscitation should be supported where possible.[286]

The members of the resuscitation team and family members, without coercion or pressure, make the decision about who should be present during resuscitation jointly. It is recommended to provide a healthcare professional whose sole responsibility is to care for the family member. Whilst this may not always be possible it should not mean the exclusion of the family member from the resuscitation. Finally, there should be an opportunity for the immediate relative to reflect, ask questions about details of the resuscitation and be informed about the support services available.[286]

Decisions to discontinue resuscitation should ideally involve senior paediatric staff. Whenever possible, the decision to attempt resuscitation of an extremely preterm baby should be taken in close consultation with the parents and senior paediatric and obstetric staff. Where a difficulty has been foreseen, for example in the case of severe congenital malformation, discuss the options and prognosis with the parents, midwives, obstetricians and birth attendants before delivery.[283] Record carefully all discussions and decisions in the mother's notes prior to delivery and in the baby's records after birth.

Post-resuscitation care

Babies who have required resuscitation may later deteriorate. Once adequate ventilation and circulation are established, the infant should be maintained in or transferred to an environment in which close monitoring and anticipatory care can be provided.

Glucose

Hypoglycaemia was associated with adverse neurological outcome in a neonatal animal model of asphyxia and resuscitation.[287] Newborn animals that were hypoglycaemic at the time of an anoxic or hypoxic-ischemic insult had larger areas of cerebral infarction and/or decreased survival compared to controls.[288,289] One clinical study demonstrated an association between hypoglycaemia and poor neurological outcome following perinatal asphyxia.[290] In adults, children and extremely low-birth-weight infants receiving intensive care, hyperglycaemia has been associated with a worse outcome.[288–292] However, in paediatric patients, hyperglycaemia after hypoxia-ischaemia does not appear to be harmful,[293] which confirms data from animal studies[294] some of which suggest it may be protective.[295] However, the range of blood glucose concentration that is associated with the least brain injury following asphyxia and resuscitation cannot be defined based on available evidence. Infants who require significant resuscitation should be monitored and treated to maintain glucose in the normal range.

Induced hypothermia

Newly born infants born at term or near-term with evolving moderate to severe hypoxic - ischemic encephalopathy should, where possible, be offered therapeutic hypothermia.[296–301] Whole body cooling and selective head cooling are both appropriate strategies. Cooling should be initiated and conducted under clearly defined protocols with treatment in neonatal intensive care facilities and with the capabilities for multidisciplinary care. Treatment should be consistent with the protocols used in the randomized clinical trials (i.e. commence within 6 h of birth, continue for 72 h of birth and re-warm over at least 4 h). Animal data would strongly suggest that the effectiveness of cooling is related to early intervention. There is no evidence in human newborns that cooling is effective if started more than 6 h after birth. Commencing cooling treatment >6 h after birth is at the discretion of the treating team and should only be on an individualised basis. Carefully monitor for known adverse effects of cooling such as thrombocytopenia and hypotension. All treated infants should be followed longitudinally.

Prognostic tools

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).[106] Although widely used in clinical practice, for research purposes and as a prognostic tool,[302] its applicability has been questioned due to large inter- and intra-observer variations. These are partly explained by a lack of agreement on how to score infants receiving medical interventions or being born preterm. Therefore a development of the score was recommended as follows: all parameters are scored according to the conditions regardless of the interventions needed to achieve the condition and considering whether being appropriate for gestational age. In addition, the interventions needed to achieve the condition have to be scored as well. This Combined-Apgar has been shown to predict outcome in preterm and term infants better than the conventional score.[303,304]

Briefing/debriefing

Prior to resuscitation it is important to discuss the responsibilities of each member of the team. After the management in the delivery room a team debrief of the event using positive and constructive critique techniques should be conducted and personal bereavement counselling offered to those with a particular need. Studies of the effect of briefings or debriefings following resuscitation have generally shown improved subsequent performance.[305–310] However, many of these have been following simulation training. A method that seems to further improve the management in the delivery room is videotaping and subsequent analysis of the videos.[311] A structured analysis of perinatal management with feedback has been shown to improve outcomes, reducing the incidence of intraventricular haemorrhage in preterm infants.[312]

Regardless of the outcome, witnessing the resuscitation of their baby may be distressing for parents. Every opportunity should be taken to prepare parents for the possibility of a resuscitative effort when it is anticipated and to keep them informed as much as possible during and certainly after the resuscitation. Whenever possible, information should be given by a senior clinician. Early contact between parents and their baby is important.

Conflicts of interest

Jonathan Wyllie	No conflict of interest reported
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European Resuscitation Council Guidelines for Resuscitation 2015 Section 8. Initial management of acute coronary syndromes

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Summary of main changes since 2010 guidelines

The following is a summary of the most important views and changes in new recommendations for the diagnosis and treatment of acute coronary syndromes (ACS) since the last ERC guidelines in 2010.

Diagnostic interventions in ACS

- Pre-hospital recording of a 12-lead electrocardiogram (ECG) is recommended in patients with suspected ST segment elevation acute myocardial infarction (STEMI). For those with STEMI this expedites prehospital and in-hospital reperfusion and reduces mortality for both those planned for primary percutaneous coronary intervention (PPCI) and those who receive fibrinolytic therapy.
- Non-physician ECG STEMI interpretation with or without the aid of computer interpretation is suggested if adequate diagnostic performance can be maintained through carefully monitored quality assurance programs.
- Pre-hospital STEMI activation of the catheterisation laboratory may not only reduce treatment delays but may also reduce patient mortality.
- The use of negative high-sensitivity cardiac troponins (hs-cTn) during initial patient evaluation cannot be used as a standalone measure to exclude an ACS, but in patients with very low risk scores may justify early discharge.

Therapeutic interventions in ACS

- Adenosine diphosphate (ADP) receptor antagonists (clopidogrel, ticagrelor, or prasugrel-with specific restriction), may be given either pre-hospital or in the ED for STEMI patients planned for primary PCI.
- Unfractionated heparin (UFH) can be administered either in the pre-hospital or in-hospital setting in patients with STEMI and a planned primary PCI approach.
- Pre-hospital enoxaparin may be used as an alternative to pre-hospital UFH for STEMI.
- Patients with acute chest pain with presumed ACS do not need supplemental oxygen unless they present with signs of hypoxia, dyspnoea, or heart failure.

Reperfusion decisions in STEMI

- Reperfusion decisions have been reviewed in a variety of possible local situations.
- When fibrinolysis is the planned treatment strategy, we recommend using pre-hospital fibrinolysis in comparison to in-hospital fibrinolysis for STEMI where transport times are >30 min and pre-hospital personnel are well trained.
- In geographic regions where PCI facilities exist and are available, direct triage and transport for PCI is preferred to pre-hospital fibrinolysis for STEMI.
- Patients presenting with STEMI in the emergency department (ED) of a non-PCI capable hospital should be transported immediately to a PCI centre provided that treatment delays for PPCI are less than 120 min (60 to 90 min for early presenters and those with extended infarctions), otherwise patients should receive fibrinolysis and be transported to a PCI centre.
- Patients who receive fibrinolytic therapy in the emergency department of a non-PCI centre should be transported if possible for early routine angiography (within 3 to 24 h from fibrinolytic therapy) rather than be transported only if indicated by the presence of ischemia.
- PCI in less than 3 h following administration of fibrinolytics is not recommended and can be performed only in case of failed fibrinolysis.

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Hospital reperfusion decisions after return of spontaneous circulation (ROSC)

- We recommend emergency cardiac catheterisation lab evaluation (and immediate PCI if required), in a manner similar to patients with STEMI without cardiac arrest, in selected adult patients with ROSC after out-of-hospital cardiac arrest (OHCA) of suspected cardiac origin with ST-elevation on ECG.
- In patients who are comatose and with ROSC after OHCA of suspected cardiac origin without ST-elevation on ECG It is reasonable to consider an emergency cardiac catheterisation lab evaluation in patients with the highest risk of coronary cause cardiac arrest.

Introduction

The incidence of acute ST-elevation myocardial infarction (AMI) is decreasing in many European countries;[1] however, the incidence of non-STEMI acute coronary syndrome (non-STEMI ACS) is increasing.[2] 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.[3] Thus, the best way of improving survival from an ischaemic attack is reducing the delay from symptom onset to first medical contact and targeted treatment started in the early out-of hospital phase.

The term acute coronary syndrome (ACS) encompasses three different entities of the acute manifestation of coronary heart disease (Fig. 8.1): ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction 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.[4] Electrocardiographic (ECG) characteristics (absence or presence of ST elevation) differentiate STEMI from non-STEMI ACS. The latter may present with ST segment depression, nonspecific ST segment wave abnormalities, or even a normal ECG. In the absence of ST elevation, an increase in the plasma concentration of cardiac biomarkers, particularly troponin T or I as the most specific markers of myocardial cell necrosis, indicates non-STEMI.

Acute coronary syndromes are the commonest cause of malignant arrhythmias leading to sudden cardiac death. The therapeutic goals are to treat acute life-threatening conditions, such as ventricular fibrillation (VF) or extreme bradycardia, and to preserve left ventricular function and prevent heart failure by minimising the extent of myocardial damage. The current guidelines address the first hours after onset of symptoms. Out-of-hospital treatment and initial therapy in the emergency department (ED) may vary according to local capabilities, resources and regulations. The data supporting out-of-hospital treatment are often extrapolated from studies of initial treatment after hospital admission; there are few high-quality out-of-hospital studies. The European Society of Cardiology and the American College of Cardiology/American Heart Association have published comprehensive guidelines for the diagnosis and treatment of ACS with and without ST elevation. The current recommendations are in line with these guidelines.[5,6]

Diagnosis and risk stratification in acute coronary syndromes

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. None of these signs and symptoms of ACS can be used alone for the diagnosis of ACS. A reduction in chest pain after nitroglycerin administration can be misleading and is not recommended as a diagnostic manoeuvre.[7] Symptoms may be more intense and last longer in patients with STEMI but are not reliable for discriminating between STEMI and non-STEMI-ACS.[5,8–10]

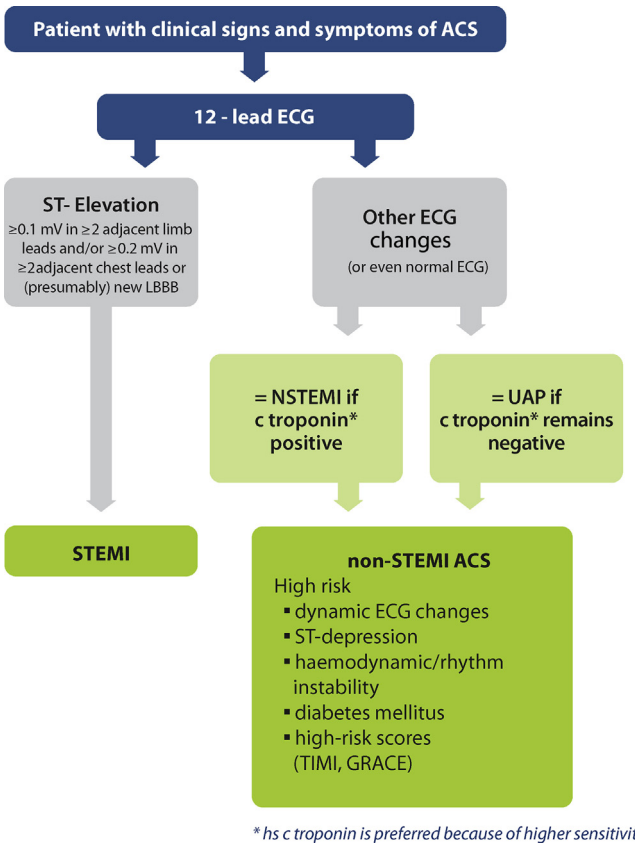

Fig. 8.1. Definitions of acute coronary syndromes (ACS); ECG, electrocardiogram; LBBB, left bundle branch block; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; c troponin, cardiac troponin; UAP, unstable angina pectoris; TIMI, thrombolysis in acute myocardial infarction; GRACE, global registry of acute coronary events.

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

The clinical recognition of ACS is a challenge emphasising that training of emergency providers including EMS dispatchers, doctors and non-doctors depending on the type of EMS system is essential. Clinical pathway protocols are strongly recommended and must be available for emergency teams working in the pre hospital setting and the emergency department (ED).

12-lead ECG

A 12-lead ECG is the key investigation for assessment of an ACS. In the case of STEMI, it indicates the need for immediate reperfusion therapy (i.e. primary percutaneous coronary intervention (PCI) or pre-hospital fibrinolysis). When an ACS is suspected, printout of a 12-lead-ECG should be acquired and interpreted as soon as possible after first patient contact, to facilitate earlier diagnosis and triage.[6,8,10] STEMI is typically diagnosed when ST-segment

elevation, measured at the J point, fulfills specific voltage criteria in the absence of left ventricular (LV) hypertrophy or left bundle branch block (LBBB).[5] In patients with clinical suspicion of ongoing myocardial ischaemia with new or presumed new LBBB, consider prompt reperfusion therapy, preferably using primary PCI (PPCI). Ventricular pacing may also mask the presence of an evolving MI and may require urgent angiography to confirm diagnosis and initiate therapy.

Right precordial leads should be recorded in all patients with inferior STEMI in order to detect right ventricular MI. Isolated ST-depression ≥0.05 mV in leads V1 through V3 represents STEMI in the inferobasal portion of the heart which may be confirmed by ST segment elevation in posterior leads (V7–V9). Pre-hospital or ED ECG yields useful diagnostic information when interpreted by trained health care providers.

Recording of a 12-lead ECG out-of-hospital enables advanced notification to the receiving facility and expedites treatment decisions after hospital arrival. In many studies, using pre-hospital 12-lead ECG, the time from hospital admission to initiating reperfusion therapy is reduced by 10 to 60 min. This is associated with shorter times to reperfusion and improved patient survival in both patients with PCI and those undergoing fibrinolysis.[11–19]

Trained EMS personnel (emergency physicians, paramedics and nurses) can identify STEMI, defined by ST elevation of ≥0.1 mV elevation in at least two adjacent limb leads or >0.2 mV in two adjacent precordial leads, with a high specificity and sensitivity comparable to diagnostic accuracy in the hospital.[20,21] It is thus reasonable that paramedics and nurses be trained to diagnose STEMI without direct medical consultation, as long as there is strict concurrent provision of quality assurance.

If interpretation of the pre-hospital ECG is not available on-site, computer interpretation[22,23] or field transmission of the ECG is reasonable.[14,22–29] Recording and transmission of diagnostic quality ECGs to the hospital usually takes less than 5 min. When used for the evaluation of patients with suspected ACS, computer interpretation of the ECG may increase the specificity of diagnosis of STEMI, especially for clinicians inexperienced in reading ECGs. The benefit of computer interpretation however, is dependent on the accuracy of the ECG report. Incorrect reports may mislead inexperienced ECG readers. Thus computer-assisted ECG interpretation should not replace, but may be used as an adjunct to, interpretation by an experienced clinician.

Biomarkers, rules for early discharge and chest pain observation protocols

In the absence of ST elevation on the ECG, the presence of a suggestive history and elevated concentrations of biomarkers (troponins, CK and CKMB) characterise non-STEMI and distinguish it from STEMI and unstable angina, respectively. Measurement of a cardiac-specific troponin is used routinely because of its higher sensitivity and specificity. Elevated concentrations of troponin are particularly helpful in identifying patients at increased risk of adverse outcome.[30,31]

In order to use the measured biomarker optimally, clinicians should be familiar with the sensitivity, precision and institutional norms of the assay, and also the release kinetics and clearance. Highly sensitive (ultrasensitive) cardiac troponin assays have been developed. They can increase sensitivity and accelerate diagnosis of MI in patients with symptoms suspicious of cardiac ischaemia.[32]

Cardiac biomarker testing should be part of the initial evaluation of all patients presenting to the ED with symptoms suggestive of cardiac ischaemia. However, the delay in release of biomarkers from damaged myocardium prevents their use in diagnosing myocardial infarction in the first hours after the onset of symptoms. For patients who present within 6 h of symptom onset, and have an initial negative cardiac troponin, biomarkers should be measured again between 2 and 3 and up to 6 h later for hs-cTn (12 h with regular troponin). The majority of patients with possible ACS do not have an ACS but the identification of those with ACS is challenging. The recently reported rate of patients with a 'missed' diagnosis of ACS in the ED is up to 3.5% with significant morbidity and mortality.[33–35]

With the implementation of high-sensitivity (hs)-assays, many hs-cTn based pathways for rapid decision-making have been tested which resulted in a proliferation of proposed diagnostic algorithms in the ED including ECG, troponin, and TIMI risk score. Data from large observational multicentre studies showed an excellent performance of 2 h rule-out protocols that combine hs-cTn values with clinical information, but also for a 1 h rule-out and rule-in protocol exclusively based on hs-cTnT values.[36–39]

It is not recommended to use high sensitivity cardiac troponins as a stand-alone measure at 0 and 2 h to exclude the diagnosis of ACS, defined as <1% 30-day major adverse cardiac events (MACE).[40] Negative hs-cTnI measured at 0 and 2 h may be used together with low risk stratification (TIMI score of 0 or 1) to exclude the diagnosis of ACS. Also negative cTnI or cTnT measured at 0 and 3–6 h together may be used in conjunction with very low risk stratification (Vancouver score of 0 or North American CP score of 0 and age <50) to exclude the diagnosis of ACS.

There is no evidence to support the use of troponin point-of-care testing (POCT) in isolation as a primary test in the pre-hospital setting to evaluate patients with symptoms suspicious of cardiac ischaemia.[32] In the ED, use of point-of-care troponin assays may help to shorten time to treatment and length of ED stay.[41] Until further randomised control trials are performed, other serum assays should not be considered first-line steps in the diagnosis and management of patients presenting with ACS symptoms.[42–44]

Risk assessment scores and clinical prediction algorithms using clinical history, physical examination, ECG, and cardiac troponins have been developed to help identify patients with ACS at increased risk of adverse outcome(s). Both accurate discrimination and calibration are needed from a risk prediction equation. Clinicians need to know which ACS patients are at highest risk so they can be prioritised for earlier and more aggressive treatment. But they also need to know what the absolute risk is, so patients can be advised about the risk and benefits of various treatment options, and to support them in making rational cost-benefit decisions. Global Registry of Acute Coronary Events (GRACE) and Thrombolysis In Myocardial Infarction (TIMI) risk score are the most commonly used. In a recent meta-analysis, TIMI and GRACE risk scores were the only ones validated in multiple clinical setting, with GRACE showing a better performance with an area under the curve (AUC) around 0.85.[45]

The GRACE score identified a sizable low-risk cohort potentially safe for early ED discharge with outpatient assessment with high sensitivity and negative predictive value; however the complexity of this tool may limit its utility.[46,47] It could be difficult to use these scores in the context of a pre-hospital setting where biological parameters (biomarkers and creatinine) are not available. This is probably the reason why little attention has been focused on the pre-hospital aspects of care in non-STEMI despite its commonality and dominant role as the major overall contributor to mortality from myocardial infarction. Whether implementation of a pre-hospital regional program of early-risk stratification, initiation of evidence-based care, and a timely invasive strategy delivered to patients with non-STEMI at moderate to high risk would enhance outcomes still needs investigation.[48] The new version of the GRACE risk score (GRACE 2.0) uses non-linear functions and seems to be more accurate than the original version. It is now validated over the longer term (to 1 and 3 years) and with substitutions possible for creatinine values and Killip class GRACE 2.0 will enable risk stratification at the patient presentation wherever the management care will start.[49]

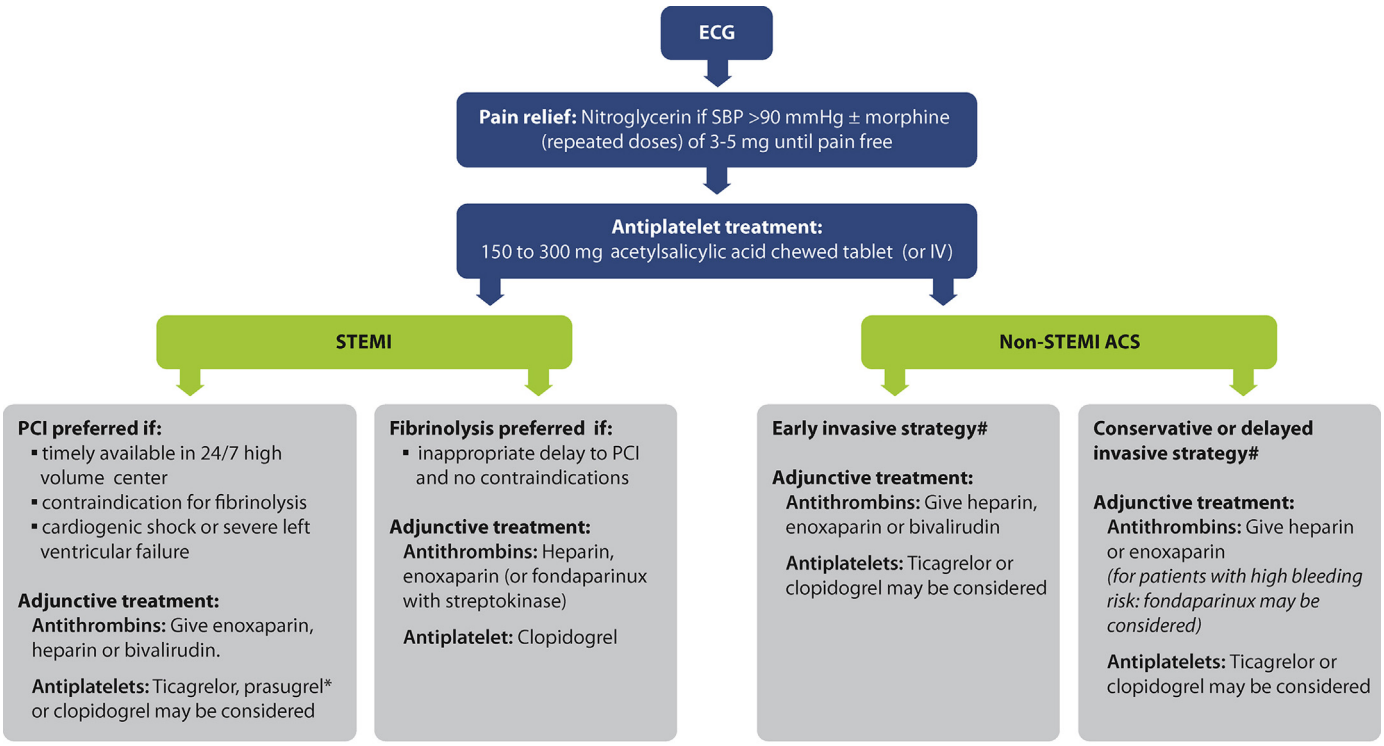

Fig. 8.2. Treatment algorithm for acute coronary syndromes; ECG, electrocardiogram; SBP, systolic blood pressure; STEMI, ST-elevation myocardial infarction; Non-STEMI-ACS, non-ST-elevation acute coronary syndrome; PCI, percutaneous coronary intervention.

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

Chest pain observation protocols are rapid systems for assessment of patients with suspected ACS. They should generally include a history and physical examination, followed by a period of observation, during which serial electrocardiography and cardiac marker measurements are performed. At some point after AMI is excluded, the evaluation of the patient should be complemented by either a non-invasive evaluation for anatomical coronary disease or provocative testing for inducible myocardial ischaemia. These protocols may be used to improve accuracy in identifying patients requiring inpatient admission or further diagnostic testing while maintaining patient safety, reducing length of stay and reducing costs.[50]

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. The potential therapeutic and diagnostic yield of provocative testing may take on an increasingly central role in determining the usefulness of provocative testing for low-risk and moderate-risk patients with chest pain evaluated in accelerated diagnostic protocols. Multicentre studies are needed to demonstrate the impact of chest pain (observation) units in the use of provocative testing.[51] They may be recommended as a means to reduce length of stay, hospital admissions and healthcare costs, improve diagnostic accuracy and improve quality of life.[52] There is no direct evidence demonstrating that chest pain units or observation protocols reduce adverse cardiovascular outcomes, particularly mortality, for patients presenting with possible ACS.

Imaging techniques

Effective screening of patients with suspected ACS, but with negative ECG and negative cardiac biomarkers, remains challenging. Non invasive imaging techniques (CT angiography,[53] cardiac magnetic resonance, myocardial perfusion imaging,[54] and echocardiography[55]) have been evaluated as means of screening these low-risk patients and identifying subgroups that can be discharged home safely.[31,56–58] Furthermore, differential diagnoses such as aortic dissection, pulmonary embolism, aortic stenosis, hypertrophic cardiomyopathy, pericardial effusion, or pneumothorax may be identified. Therefore, echocardiography should be routinely available in ED, and used in all patients with suspected ACS. Studies are needed to evaluate the impact of echocardiography in the pre-hospital setting. Although there are no large multicentre trials, existing evidence indicates that these diagnostic modalities enable early and accurate diagnosis with a reduction in length of stay and costs without increasing cardiac events. Both the exposure to radiation and iodinated contrast should be considered when using myocardial perfusion imaging and multi-detector computer tomography coronary angiography (MDCTCA).

MDCTCA has been recently proposed in the management acute chest pain in the ED. It is accurate compared with invasive coronary angiography, enabling differential diagnosis, and it is feasible and practical in the ED.[53,59–63] MDCTCA has a high ability to rule out obstructive coronary artery disease (CAD).[64,65] Early use of cardiac MDCTCA in patients presenting to the ED with chest pain and a low to intermediate risk of ACS quickly identifies a group of particularly low-risk patients (<1% risk of adverse events within 30 days) and enables safe and expedited discharge. By preventing unnecessary admissions and prolonged lengths of stay, a strategy based on early cardiac MDCTCA has been shown to be efficient.[66–68] In a significant

number of low-risk ACS patients, MDCTCA detects severe coronary lesions and enables dedicated further diagnostic and therapeutic intervention. In a recent meta-analysis, MDCTCA demonstrated a high sensitivity and a low negative likelihood ratio of 0.06, and was effective in ruling out the presence of ACS in low to intermediate risk patients presenting to the ED with acute chest pain.[69] But the inability of anatomical findings to prove the presence of ischaemia, the cancer risk induced by radiation exposure and potential overuse still raise concerns about the relevance of this strategy.

Treatment of acute coronary syndromes—Symptoms

Nitrates

Glyceryl trinitrate is an effective treatment for ischaemic chest pain and has beneficial haemodynamic effects, such as dilation of the venous capacitance vessels, dilation of the coronary arteries and, to a minor extent, the peripheral arteries. Glyceryl trinitrate may be considered if the systolic blood pressure (SBP) is above 90 mmHg and the patient has ongoing ischaemic chest pain (Fig. 8.2). Glyceryl trinitrate can also be useful in the treatment of acute pulmonary congestion. Do not use nitrates in patients with hypotension (SBP $\leq$ 90 mmHg), 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. Do not use nitrates if 5′-phosphodiesterase inhibitors have been used recently (<48 h).[5,9,70,71]

Give glyceryl trinitrate 0.4 mg sublingual or equivalent every 5 min up to 3 doses as SBP allows. Begin IV dosing at 10 10 micrograms min^{-1} in persistent pain or pulmonary edema; titrate to desired BP effect.

Analgesia

Morphine is the analgesic of choice for nitrate-refractory pain and also has calming effects on the patient making sedatives unnecessary in most cases. Since morphine is a dilator of venous capacitance vessels, it may have additional benefit in patients with pulmonary congestion. Give morphine in initial doses of 3–5 mg intravenously and repeat every few min until the patient is pain-free. Caution is needed in presence of lethargy, hypotension, bradycardia or known hypersensitivity.[5,9,71] Avoid non-steroidal anti-inflammatory drugs (NSAIDs) for analgesia because they have pro-thrombotic effects.[72]

Oxygen

Evidence is accumulating about the questionable role of supplemental oxygen in cardiac arrest, after ROSC and in acute coronary syndromes. Patients with acute chest pain with presumed ACS do not need supplemental oxygen unless they present with signs of hypoxia, dyspnoea or heart failure. There is increasing evidence suggesting that hyperoxia may be harmful in patients with uncomplicated myocardial infarction.[73–76]

In ACS complicated with cardiac arrest, hypoxia develops rapidly. Ischaemic brain injury is a major determinant for neurologically intact survival. Therefore during CPR adequate oxygenation is essential. After ROSC, avoid both hypoxia and hyperoxia (see post resuscitation care).[77] Use 100% inspired oxygen until the arterial oxygen saturation can be measured reliably. As soon as the arterial blood oxygen saturation can be measured reliably, titrate the inspired oxygen concentration to achieve arterial blood oxygen saturation in the range of 94–98%, or 88–92% in chronic obstructive pulmonary disease.[5,71]

Treatment of acute coronary syndromes—Cause

Inhibitors of platelet aggregation

Platelet activation and aggregation following atherosclerotic plaque rupture are central pathophysiologic mechanisms of acute coronary syndromes and antiplatelet therapy is a pivotal treatment of ACS whether with or without ST segment elevation, with or without reperfusion and with or without revascularisation.

Acetylsalicylic acid (ASA)

Large randomised controlled trials indicate decreased mortality when ASA (75–325 mg) is given to hospitalised patients with ACS independent of the reperfusion or revascularisation strategy. A few studies have suggested reduced mortality if ASA is given earlier.[78–80] Therefore, give an oral loading dose of ASA (150 to 300 mg of a non-enteric coated formulation) or 150 mg of an IV preparation as soon as possible to all patients with suspected ACS unless the patient has a known true allergy to ASA or has active bleeding. ASA may be given by the first healthcare provider, bystander or by dispatcher assistance according to local protocols.

ADP receptor inhibitors

The inhibition of the platelet ADP receptor by the thienopyridines clopidogrel and prasugrel (irreversible inhibition) and the cyclo-pentyl-triazolo-pyrimidine ticagrelor (reversible inhibition) leads to further inhibition of platelet aggregation in addition to that produced by ASA. In contrast to clopidogrel, the effect of prasugrel and ticagrelor are largely independent of a genetically determined variability of drug metabolism and activation. Therefore prasugrel and ticagrelor (reversible) lead to a more reliable, faster and stronger inhibition of platelet aggregation.

A large randomised study comparing a loading dose of 300 mg clopidogrel followed by 75 mg daily with prasugrel (loading dose 60 mg, followed by 10 mg daily) in patients with ACS (see specific remarks on non-STEMI-ACS below) planned for PCI resulted in fewer MACE with prasugrel; the bleeding rate was higher, however. Bleeding risk was increased markedly in patients weighing less than 60 kg and those older than 75 years.[81] A significantly increased intracranial bleeding rate was observed in patients with a history of transient ischaemic attack (TIA) and/or stroke in association with prasugrel. In another study, ticagrelor (loading dose 180 mg, followed by 90 mg twice daily) proved to be superior to clopidogrel (loading dose 300–600 mg followed by 75 mg daily) with respect to mortality and MACE in the general setting of ACS but also associated with higher risks of bleeding.[82]

ADP-receptor inhibitors in non-STEMI-ACS.

Clopidogrel. If given in addition to heparin and ASA in high-risk non-STEMI-ACS patients, clopidogrel improves outcome.[83] 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. There is no large-scale study investigating pre-treatment with clopidogrel, compared with peri-interventional application – either with a 300 mg or 600 mg loading dose.

Prasugrel. Prasugrel (60 mg loading dose) may be given to patients with high-risk non-STEMI-ACS and planned PCI only after angiography, provided that coronary stenoses are suitable for PCI. Contraindications (history of TIA/stroke) and the benefit – risk balance in patients with high bleeding risk (weight < 60 kg, age > 75 years) should be considered. A RCT, comparing pre versus post-angiography treatment by prasugrel in non-STEMI-ACS showed that pre-treatment was associated with higher risk of major bleeding without reducing thrombotic events excluding prasugrel from

any possible pre-treatment strategy in non-STEMI-ACS whether initiated in or out of hospital before coronary anatomy is known.[84]

Ticagrelor. According to the latest ESC guidelines,[6] ticagrelor (180 mg loading dose) should be given in addition to ASA in all patients with moderate to high-risk non-STEMI-ACS whether an invasive strategy is planned or not. In patients with non-STEMI-ACS planned for a conservative approach, give ticagrelor or clopidogrel as soon as the diagnosis is confirmed. There is insufficient evidence to recommend for or against pre-treatment with these agents when PCI is the initial strategy.

ADP-receptor inhibitors in STEMI.

Clopidogrel. Pre-hospital versus in-hospital administration of clopidogrel has been assessed in two small studies which have shown its safety but no evident clinical benefit.[85,86] However, a meta-analysis of pre-PCI versus post-PCI (and not pre-hospital versus in-hospital) administration of clopidogrel in STEMI subgroup of patients has shown a significant benefit in terms of mortality reduction and MI rates without a bleeding excess risk in association with pre-treatment.[87] Although there is no large study on the use of clopidogrel for pre-PCI 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 may be considered for patients presenting with STEMI and planned PCI.

Two large randomised trials studied clopidogrel compared with placebo in patients with STEMI treated conservatively or with fibrinolysis.[88–90] One study included patients up to 75 years, treated with fibrinolysis, ASA, an anti-thrombin and a loading dose of 300 mg clopidogrel.[88] Treatment with clopidogrel resulted in fewer occluded culprit coronary arteries at angiography and fewer re-infarctions, without an increased bleeding risk. The other study investigated STEMI patients without age limits to be treated conservatively or with fibrinolysis. In this trial, clopidogrel (no loading, 75 mg daily) compared with placebo resulted in fewer deaths and a reduction of the combined endpoint of death and stroke.[89] Therefore patients with STEMI treated with fibrinolysis should be treated with clopidogrel (300 mg loading dose up to an age of 75 years and 75 mg without loading dose if >75 years of age) in addition to ASA and an antithrombin.

Prasugrel. Prasugrel with a loading dose of 60 mg up to 24 h before, at the time of, or even after, PCI may be given in addition to ASA and an antithrombin to patients presenting with STEMI with planned PCI.[91] Contraindications (history of TIA/stroke), and relation of bleeding risk versus benefit in patients with a body weight < 60 kg or aged > 75 years should be taken into account. There is no data on prehospital treatment with prasugrel and no data on prasugrel if used in the context of fibrinolysis.

Ticagrelor. Ticagrelor may be given with a loading dose of 180 mg to patients presenting with STEMI and planned PCI. The benefit of pre-hospital versus in-catheterisation laboratory administration of ticagrelor (180 mg loading dose) was assessed in a RCT including 1862 STEMI patients presenting within 6 h after symptom onset and planned for primary PCI. The study showed no benefit in terms of angiographic coronary artery flow or ST segment elevation resolution (primary end-point) or major clinical endpoints. The study also showed that pre-hospital administration of ticagrelor was associated with a reduced rate of definite acute stent thrombosis (OR 0.19, 95% CI 0.04–0.86) without excess in the risk of bleeding.[92] However, this end-point was not pre-specified and the finding should be considered as hypothesis generating only. There are no data on ticagrelor when used in the context of fibrinolysis.

The relative benefit of pre-hospital administering an ADP antagonist routinely in patients planned for PCI for STEMI may be marginal and offset by additional harms that should be evaluated in larger RCTs that include additional patient-oriented outcomes. However, the use of ADP antagonists in patients transferred for primary PCI may be considered after cautious evaluation of the risk-benefit balance for each patient.

Glycoprotein (Gp) IIB/IIIA inhibitors

Glycoprotein (Gp) IIB/IIIA receptor activation 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. Older studies from the pre-stent era mostly support the use of this class of drugs.[93] Recent studies mostly document neutral or worse outcomes[94] with the exception of the recently published ON-TIME-2 trial comparing pre-hospital systematic versus in-hospital provisional administration of tirofiban associated with secondary PCI and showing a benefit of pre-treatment with Gp IIB/IIIA receptor blockers on the primary thrombotic endpoint with no relevant excess in bleeding risk.[95] Also a recent meta-analysis studying the results of 7 randomised trials including 722 patients comparing early versus late administration of abciximab in the setting of primary PCI for STEMI showed a benefit of early strategy on coronary artery patency translating to a benefit in terms of mortality.[96] However, in almost all supporting, neutral or opposing studies, bleeding occurred in more patients treated with Gp IIB/IIIA receptor blockers. There are insufficient data to support routine pre-treatment with Gp IIB/IIIA receptor blockers in patients with STEMI or non-STEMI-ACS. It is not recommended to give Gp IIB/IIIA receptor blockers before coronary anatomy is known. For high-risk patients with non-STEMI-ACS, in-hospital pre-treatment with eptifibatide or tirofiban may be acceptable whereas abciximab may be given only in the context of PCI. Taking into account the increased bleeding risk with GpIIB/IIIA receptor blockers when used with heparins, alternative treatment strategies with the use of ADP antagonists should be considered.[97]

Antithrombins

Unfractionated heparin (UFH) is an indirect inhibitor of thrombin, which in combination with ASA is used as an adjunct with fibrinolytic therapy or PPCI and is an important part of treatment of unstable angina and STEMI. Limitations of unfractionated heparin include its unpredictable anticoagulant effect in individual patients, the need to give it intravenously and the need to monitor aPTT. Moreover, heparin can induce thrombocytopaenia. Since publication of the 2010 ERC guidelines on ACS, several randomised trials have been performed testing alternative antithrombins versus UFH for the treatment of patients with ACS.[98–100] These alternatives are characterised by a more specific factor Xa activity (low molecular weight heparins [LMWH], fondaparinux) or are direct thrombin inhibitors (bivalirudin). With the latter antithrombins, the coagulation system does not need be monitored and the risk of thrombocytopenia is reduced. There are no studies evaluating prehospital versus delayed in-hospital administration of anti-thrombins other than UFH. Rivaroxaban, apixaban and other oral direct thrombin antagonists may have an indication after stabilisation in specific patient groups but not in the initial treatment of ACS.[101]

Antithrombins in non-STEMI-ACS

Parenteral anticoagulation, in addition to anti-platelet drugs, is recommended at the time of diagnosis because it effectively reduces the rate of MACE in patients with non-STEMI-ACS. Even if there is a rationale in use of early treatment to avoid MACE, there is no scientific proof of superiority of pre-hospital over in-hospital initiation of anti-thrombin therapy.

Compared with UFH (70–100 IU kg^{-1} IV), enoxaparin (30 mg IV followed by 1 mg kg^{-1} every 12 h) 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.[102,103] Although enoxaparin causes more minor bleeding than UFH, the incidence of serious bleeding is not increased. Additional Activated Clotting Time (ACT)-activated IV boluses of UFH may be considered following initial UFH treatment.

Bleeding worsens the prognosis of patients with ACS.[104] Fondaparinux (2.5 mg sc daily) and bivalirudin (0.1 mg kg^{-1} iv followed by a 0.25 mg kg^{-1} infusion) cause less bleeding than UFH.[105–107] Fondaparinux is recommended as having the most favourable efficacy—safety profile regardless of the management strategy. Since catheter thrombi were observed in patients undergoing PCI additional UFH during PCI is necessary.[105]

Enoxaparin or UFH are recommended when fondaparinux is not available. In the trials on patients presenting with non-STEMI-ACS, UFH, fondaparinux, enoxaparin and bivalirudin were given only after hospital admission; it therefore may be invalid to extrapolate the results to the pre-hospital or ED setting.

Because of the reduced risk of bleeding, fondaparinux may be the preferred anticoagulant. Because enoxaparin and fondaparinux may accumulate in patients with renal impairment, dosing must be adjusted. For patients with a planned invasive approach, bivalirudin and enoxaparin are reasonable alternatives to UFH. The bleeding risk may be increased by switching between UFH and enoxaparin.[108] Consider stopping anticoagulation after PCI unless otherwise indicated.

Antithrombins in STEMI

Antithrombins for patients to be treated with fibrinolysis.

Enoxaparin-UFH. It is reasonable to give UFH for patients treated with pre-hospital fibrinolysis for STEMI.

Several randomised studies of patients with STEMI undergoing fibrinolysis, however, have shown that additional treatment with enoxaparin instead of UFH resulted in 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).[109] Reduced doses of enoxaparin in elderly and low weight patients maintained the improved outcome while reducing the bleeding rate.[110]

Dosing of enoxaparin: in patients <75 years, give an initial bolus of 30 mg IV followed by 1 mg kg^{-1} SC every 12 h (first SC dose shortly after the IV bolus). Treat patients ≥75 years with 0.75 mg kg^{-1} SC every 12 h without an initial IV dose. Patients with known impaired renal function (creatinine clearance <30 ml^{-1} min^{-1}) may be given 1 mg kg^{-1} enoxaparin SC once daily or may be treated with UFH. There are insufficient data to recommend other LMWH.

Fondaparinux. Several studies show superiority or neutral outcome when fondaparinux was compared with UFH as an adjunct for fibrinolysis in STEMI patients.[105] Fondaparinux (initially 2.5 mg SC followed by 2.5 mg SC daily) may be considered specifically with non-fibrin-specific fibrinolytics (i.e. streptokinase) in patients with a plasma creatinine concentration <3 mg dl^{-1} (250 micromol l^{-1}). In case of planned PPCI, enoxaparin or UFH are preferred.

Bivalirudin. There are insufficient data to recommend bivalirudin instead of UFH or enoxaparin in STEMI patients to be treated with fibrinolysis. Since switching the anticoagulants may increase bleeding risk, the initial agent should be maintained, with the exception of fondaparinux, where additional UFH is necessary if an additional invasive procedure is planned.[108]

Anti-thrombins for STEMI patients to be treated with primary PCI (PPCI). An injectable anticoagulant must be used in primary PCI for STEMI. After publication of the ERC 2010 Guidelines, studies have been performed comparing different antithrombin treatments with pre-hospital initiation for patients with STEMI and planned PPCI.[98,99,111] With exemption of UFH (F), however, there is still a paucity of studies comparing the efficacy of pre-hospital with in-hospital initiation of treatment with the same anticoagulant i.e. the role of earlier start of therapy. Therefore treatment recommendations for these settings have to be extrapolated mainly from in-hospital investigations without proof of advantage of pre-hospital initiation of therapy, until more specific study results are available.

UFH. In one observational study pre-hospital injection of 500 mg aspirin together with >5000 IU UFH led to a significantly higher rate of TIMI flow 2 and 3 and TIMI flow 3 at initial angiography.[112] There was, however, no improvement in infarct size or 30 day mortality.

Enoxaparin. In one larger randomised study enoxaparin was compared with UFH in planned PPCI for STEMI. In 71% of patients anticoagulants were started in the ambulance.[99] The study revealed no difference in the primary combined endpoint of death, procedure failure or major bleeding, but reductions in several secondary combined endpoints such as death, recurrent ACS and urgent revascularisation. Several registries and smaller studies also documented favourable or neutral outcome when enoxaparin was compared with UFH for PPCI (with broad use of thienopyridines and/or Gp IIB/IIIA receptor blockers).[113] Therefore, enoxaparin is a safe and effective alternative to UFH and may be preferred over UFH also in the pre-hospital setting. There are insufficient data to recommend any LMWH other than enoxaparin for PPCI in STEMI. Switching from UFH to enoxaparin or vice versa may lead to an increased bleeding risk and therefore should be avoided.[108] Dose adjustment of enoxaparin is necessary for patients with renal impairment.

Bivalirudin. Two large randomised studies documented less bleeding and a reduction in short and long term mortality when bivalirudin was compared with UFH plus Gp IIB/IIIA receptor blockers in patients with STEMI and planned PCI.[114,115] Several other studies and case series showed also better or neutral results and less bleeding when bivalirudin was compared with UFH. Feasibility and safety of pre-hospital administration as well as reduced bleeding rates were shown in non-randomised studies when compared with historical controls.[100,111] In newer trials testing bivalirudin against UFH with less extensive addition of GpIIb/IIIa receptor blockers, or a modified antiplatelet strategy differences in major bleedings were only minor, whereas results in ischaemic endpoints were neutral or even inferior with bivalirudin.[116,117] A further study tested prehospital initiation of bivalirudin vs. UFH plus optional GpIIB/IIIa receptor antagonists, which was given in 69% of patients. Bleedings but not death were significantly reduced with bivalirudin; the rate of stent thromboses within the first 24 h after PCI was higher with bivalirudin as in other studies.[98,118] Weighing reduced bleeding rates against higher rates of stent thrombosis bivalirudin may still be considered as an alternative to UFH in PPCI for STEMI.

Fondaparinux. When compared with UFH, fondaparinux resulted in similar clinical outcomes but less bleeding when used in the context of PPCI;[105] however, thrombus formation on catheters required treatment with additional UFH. Therefore fondaparinux is not recommended for planned PPCI of STEMI.

Reperfusion strategy in patients presenting with STEMI

Reperfusion therapy in patients with STEMI is the most important advance in the treatment of myocardial infarction in the last 30 years. For patients presenting with STEMI within 12 h of symptom onset, reperfusion should be initiated as soon as possible using the most appropriate available strategy.[119–122] Reperfusion may be achieved with fibrinolysis, with PPCI, or a combination of both. Efficacy of reperfusion therapy is profoundly dependent on the time

Table 8.1
Contraindications for fibrinolysis

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

According to the guidelines of the European Society of Cardiology

interval from symptom onset to reperfusion. Fibrinolysis is effective specifically in the first 2 to 3 h after symptom onset; PPCI is less time sensitive.

Fibrinolysis

A meta-analysis of 3 RCTs including 531 patients showed benefit of prehospital versus inhospital fibrinolysis in terms of survival to hospital discharge without evidence of additional harm in terms of major or intracranial bleeding.[123–125] An effective and safe system for out-of-hospital fibrinolytic therapy requires adequate facilities for the diagnosis and treatment of STEMI and its complications. Fibrinolytic therapy can be given safely by trained paramedics, nurses or physicians using an established protocol, comprehensive training programs, and quality assurance programs with medical oversight.[126] Ideally, there should be a capability of communicating with experienced hospital doctors. (e.g. emergency physicians or cardiologists). The real advantage of prehospital fibrinolysis is where there are long transport times, i.e. >30–60 min. The RCTs that showed benefit with pre-hospital fibrinolysis were conducted in healthcare settings with a mean difference in time between pre-hospital treatment and in hospital treatment of 33–52 min. Additionally, transport times to hospital were a mean of 38–60 min. As the transport time shortens, any expected advantage is lost. Thus, giving fibrinolytics out-of-hospital to patients with STEMI or signs and symptoms of an ACS with presumed new LBBB is beneficial. The efficacy is greatest early after onset of symptoms. Patients with symptoms of ACS and ECG evidence of STEMI (or presumably new LBBB or true posterior infarction) presenting directly to the ED should be given fibrinolytic therapy as soon as possible unless there is timely access to PPCI.

Risks of fibrinolytic therapy

Healthcare professionals who give fibrinolytic therapy must be aware of its contraindications (Table 8.1) and risks. Patients with large AMIs (e.g. indicated by extensive ECG changes) are likely to gain most from fibrinolytic therapy. Benefits of fibrinolytic therapy are less impressive in inferior wall infarctions than in anterior infarctions. Older patients have an absolute higher risk of death, but the absolute benefit of fibrinolytic therapy is similar to that of younger patients. Patients over 75 years have an increased risk of intracranial bleeding from fibrinolysis; thus, the absolute benefit of fibrinolysis is reduced by this complication. The risk of intracranial bleeding is increased in patients with a systolic blood pressure of over 180 mmHg; this degree of hypertension is a relative contraindication to fibrinolytic therapy. The risk of intracranial bleeding is also dependent on the chosen fibrinolytic, antithrombin and antiplatelet therapy. An alternative, while using enoxaparin, is halving the dose for tenecteplase in patients >75 years, which reduces the rate of intracranial bleeding without loss of efficacy.[127,128]

Primary percutaneous intervention

Coronary angioplasty with or without stent placement has become the first-line treatment for patients with STEMI. PPCI performed with a limited delay to first balloon inflation after first medical contact, at a high-volume centre, by an experienced operator who maintains an appropriate expert status, is the preferred treatment as it improves morbidity and mortality as compared with immediate fibrinolysis.[129]

Fibrinolysis vs primary PCI

Primary PCI has been limited by access to catheter laboratory facilities, appropriately skilled clinicians and delay to first balloon inflation. Fibrinolysis therapy is a widely available reperfusion strategy. Both treatment strategies are well established and have been the subject of large randomised multicentre trials over the last decades. Time from onset of symptoms and PPCI related delay (diagnosis to balloon interval minus the diagnosis to needle interval) are key in selecting the most appropriate revascularisation strategy.

Fibrinolytic therapy is most effective in patients presenting within 2–3 h from onset of ischaemic symptoms. It compares favourably with PPCI when started within 2 h from symptom onset and is combined with rescue or delayed PCI.[40,130,131] In the randomised studies comparing PPCI with fibrinolytic therapy, the typical delay from decision to the beginning of treatment with either PPCI or fibrinolytic therapy was less than 60 min. In registries that reflect standard practice more realistically the acceptable PPCI-related delay (i.e. the diagnosis to balloon interval minus the diagnosis to needle interval) to maintain the superiority of PPCI over fibrinolysis varied considerably between 45 and >180 min depending on the patients' conditions (i.e. age, localisation of infarction, and duration of symptoms). In STEMI registries, system delays to PCI may exceed 120 min in as many as 58% of patients with STEMI.[132] Thus continuous monitoring of system performance is needed to assure optimal performance and outcomes for patients with STEMI.

In early presenters, patients of younger age and large anterior infarctions, PPCI related delays of 60 min may be unacceptable while in late presenters (>3 h from the onset of symptoms) PPCI related delays of up to 120 min may be acceptable.[133]

The presence of certain comorbidities such as previous CABG, diabetes and renal failure are additional factors to be considered for the selection of the most appropriate therapy.[134]

Time delay to PPCI may be significantly shortened by improving the systems of care[135,136]:

- A pre-hospital ECG should be acquired as soon as possible and interpreted for the diagnosis of STEMI. This can reduce mortality in both patients planned for PPCI and fibrinolytic therapy.
- STEMI recognition may be accomplished by ECG transmission or onsite interpretation by physicians, or highly trained nurses or paramedics, with or without the aid of computer ECG interpretation.
- When PPCI is the planned strategy, pre-hospital activation of catheterisation laboratory for PPCI will contribute to a mortality benefit.[40]

Additional elements for an effective system of care include:

- Requiring the catheterisation laboratory to be ready within 20 min available 24/7.
- Providing real-time data feedback on the real time course from symptom onset to PCI.

For those patients with a contraindication to fibrinolysis, PCI should still be pursued despite the delay, rather than not providing reperfusion therapy at all. For those STEMI patients presenting in shock, primary PCI (or coronary artery bypass surgery) is the preferred reperfusion treatment. Fibrinolysis should only be considered if there is a substantial delay to PCI.

Triage and inter-facility transfer for primary PCI

The majority of patients with an ongoing STEMI will be first diagnosed either in the pre-hospital environment or in the setting of the ED of a non-PCI capable hospital. Therefore decisions have to be made as for the most appropriate strategy for revascularisation.

In the pre-hospital setting there is evidence suggesting that although pre-hospital fibrinolysis is not inferior to immediate transfer for primary PPCI in terms of mortality, it is associated with increased risk for intracranial haemorrhage. When PCI can be performed within a time limit of 60–90 min, then direct triage and transport for PCI is preferred to pre-hospital fibrinolysis.[40,127,137–139]

When the patient with STEMI first appears in the emergency department of a non PCI hospital, data from 8 RCTs[140–147] enrolling 3119 patients indicate that immediate transfer for PPCI is superior to local fibrinolytic therapy and transfer only for rescue PCI in terms of mortality, reinfarction and stroke without evidence for additional harm. Therefore, for adult patients presenting with STEMI in the ED of a non-PCI capable hospital emergent transfer without fibrinolysis to a PCI centre should be considered provided that PPCI can be performed within acceptable time delays.

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.[133] 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 and maintain coronary blood flow and myocardial perfusion. There are several ways in which the two therapies can be combined. There is some lack of uniformity in the nomenclature used to describe PCI in these regimens. Facilitated PCI is used to describe PCI performed immediately after fibrinolysis, a pharmaco-invasive strategy refers to PCI performed routinely 3 to 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 to 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.

Routine immediate angiography post fibrinolytic therapy is associated with increased intracranial haemorrhage (ICH) and major bleeding without offering any benefit in terms of mortality or reinfarction.[148–152]

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.[153]

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

Analysis of data form seven RCTs[138,146,154–158] enrolling 2355 patients show benefit in terms of less reinfarctions in immediate routine transfer for angiography at 3–6 h (or up to 24 h) in first 24 h after ED fibrinolysis versus only transfer for rescue PCI after in hospital fibrinolysis (OR 0.57; 95% CI 0.38–0.85). There was no evidence of benefit in terms of short term and 1 year mortality or for additional harm in terms of major haemorrhage or intracranial bleeding.

Data from two RCTs[138,159] and one non RCT[160] indicate no benefit from transfer to immediate PCI in comparison to fibrinolytic therapy followed by routine transfer for PCI 3 to 24 h later. Therefore in case PPCI cannot be achieved in due time, onsite fibrinolysis and transfer for angiography 3 to 24 h later is a reasonable alternative.

Special situations

Cardiogenic shock

Acute coronary syndrome (ACS) is the most common cause of cardiogenic shock, mainly through a large zone of myocardial ischaemia or a mechanical complication of myocardial infarction. Although uncommon, the short-term mortality of cardiogenic shock is up to 40%[161] contrasting with a good quality of life in patients discharged alive. An early invasive strategy (i.e. primary PCI, PCI early after fibrinolysis) is indicated for those patients who are suitable for revascularisation.[162] Observational studies suggest that this strategy could be also beneficial in elderly patients (over 75 years). Even if commonly used in clinical practice, there is no evidence supporting the use of IABP in cardiogenic shock.[161]

Suspect right ventricular infarction in patients with inferior infarction, clinical shock and clear lung fields. ST segment elevation ≥0.1 mV in lead V4R is a useful indicator of right ventricular infarction. These patients have an in-hospital mortality of up to 30% and many benefit greatly from reperfusion therapy. Avoid nitrates and other vasodilators, and treat hypotension with intravenous fluids.

Reperfusion after successful CPR

As it is often accompanied by an acute coronary artery occlusion or by a high degree stenosis, acute coronary syndrome (ACS) is a frequent cause of out-of-hospital cardiac arrest (OHCA) In a recent meta-analysis, the prevalence of acute coronary artery lesion ranged from 59% to 71% in OHCA patients without an obvious non-cardiac cause.[163] Since the publication of the pioneering study,[164] many observational studies have shown that emergent cardiac catheter laboratory evaluation, including early percutaneous coronary intervention (PCI), is feasible in patients with return of spontaneous circulation (ROSC) after cardiac arrest.[165] The invasive management (i.e. early coronary angiography (CAG) followed by immediate PCI if deemed necessary) of this patient group, particularly patients after prolonged resuscitation and having nonspecific ECG changes, has been controversial due to the lack of specific evidence and significant implications on resource utilization (including transfer of patients to PCI centres).

PCI following ROSC with ST-elevation. The highest prevalence of acute coronary lesion is observed in patients with ST segment elevation (STE) or left bundle branch block (LBBB) on post-ROSC electrocardiogram (ECG). There is no randomised study but as many observational studies reported a benefit regarding survival and neurological outcome, it is highly probable that this early invasive

management is a strategy associated with a clinically relevant benefit in patients with ST segment elevation. A recent meta-analysis indicates that early angiography is associated with reduction of hospital mortality [OR 0.35 (0.31 to 0.41)] and increased neurologically favourable survival [OR 2.54 (2.17 to 2.99)].[40] However patients that underwent early angiography were highly selected populations with higher prevalence of male gender, VF, witnessed arrest, therapeutic hypothermia and more intense LV support. Diabetes mellitus, renal and heart failure were less prevalent in these patients.

Based on the available data, emergent cardiac catheterisation lab evaluation (and immediate PCI if required) should be performed in selected adult patients with ROSC after OHCA of suspected cardiac origin with ST segment elevation on ECG.[166]

Observational studies also indicate that optimal outcomes after OHCA are achieved with a combination of targeted temperature management and PCI, which can be combined in a standardised post–cardiac-arrest protocol as part of an overall strategy to improve neurologically intact survival in this patient group.

PCI following ROSC without ST-elevation. In contrast to the usual presentation of ACS in non cardiac arrest patients, recommended tools to assess coronary ischaemia are less accurate in this setting. Both sensitivity and specificity of clinical data, ECG and biomarkers to predict an acute coronary artery occlusion as the cause of OHCA are debatable.[167] In particular several large observational series showed that absence of STE may also be associated with ACS in patients with ROSC following OHCA.[168] In these non-STE patients, data are conflicting regarding the potential benefit of an emergent cardiac catheterisation lab evaluation, all coming from observational studies,[169,170] or subgroup analysis.[171] It is reasonable to discuss an emergent cardiac catheterisation lab evaluation after ROSC in patients with the highest risk of coronary cause of CA. A variety of factors such as patient age, duration of CPR, haemodynamic instability, presenting cardiac rhythm, neurologic status upon hospital arrival, and perceived likelihood of cardiac aetiology can influence the decision to undertake the intervention. A recent consensus statement from the European Association for Percutaneous Cardiovascular Interventions (EAPCI) has emphasised that in OHCA patients, cardiac catheterisation should be performed immediately in the presence of ST-elevation and considered as soon as possible (less than two hours) in other patients in the absence of an obvious non-coronary cause, particularly if they are haemodynamically unstable.[172] In patients who present in a non-PCI centre transfer for angiography and PPCI if indicated should be considered on an individual basis, weighing the expected benefits from early angiography against the risks from patient transport.

Preventive interventions

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

Beta-blockers

Several studies, undertaken mainly in the pre-reperfusion era, indicate a decreased mortality, incidence of reinfarction and cardiac rupture as well as a lower incidence of ventricular fibrillation and supraventricular arrhythmia in patients treated early with a beta-blocker.[173] Beta-blocker studies are very heterogeneous with respect to time of start of treatment. There is paucity of data on administration in the pre-hospital or ED settings. Moreover, studies indicate an increased risk of cardiogenic shock with IV beta-blockers in patients with STEMI, even if the rate of severe tachyarrhythmia is reduced by beta-blockade.[174] There is no evidence to support routine intravenous beta-blockers in the pre-hospital or initial ED settings. Early IV use of beta-blockers is contraindicated in patients with clinical signs of hypotension or congestive heart failure. It may be indicated in special situations such as severe hypertension or tachyarrhythmias in the absence of contraindications. It is reasonable to start oral beta-blockers at low doses only after the patient is stabilised.

Other anti-arrhythmics

Apart from beta-blockers, there is no evidence to support the use of anti-arrhythmic prophylaxis after ACS. Ventricular fibrillation (VF) accounts for most of the early deaths from ACS; the incidence of VF is highest in the first hours after onset of symptoms. This explains why numerous studies have been performed with the aim of demonstrating the prophylactic effect of antiarrhythmic therapy.[175] The effects of antiarrhythmic drugs (lidocaine, magnesium, disopyramide, mexiletine, verapamil, sotalol, tocainamide) given prophylactically to patients with ACS have been studied. Prophylaxis with lidocaine reduces the incidence of VF but may increase mortality.[176] Routine treatment with magnesium in patients with AMI does not reduce mortality. Arrhythmia prophylaxis using disopyramide, mexiletine, verapamil, or other anti-arrhythmics given within the first hours of an ACS does not improve mortality. Therefore prophylactic anti-arrhythmics are not recommended.

Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers

Oral ACE inhibitors reduce mortality when given to patients with AMI with or without early reperfusion therapy. The beneficial effects are most pronounced in patients presenting with anterior infarction, pulmonary congestion or left ventricular ejection fraction <40%. Do not give ACE inhibitors if the systolic blood pressure is less than 100 mmHg on admission or if there is a known contraindication to these drugs. A trend towards higher mortality has been documented if an intravenous ACE inhibitor is started within the first 24 h after onset of symptoms. This therapy is safe, well tolerated and associated with a small but significant reduction in 30-day mortality.[177] Therefore, give an oral ACE inhibitor within 24 h after symptom onset in patients with AMI regardless of whether early reperfusion therapy is planned, particularly in those patients with anterior infarction, pulmonary congestion or a left ventricular ejection fraction below 40%. Do not give intravenous ACE inhibitors within 24 h of onset of symptoms.[178,179] Give an angiotensin receptor blocker (ARB) to patients intolerant of ACE inhibitors.[180]

Lipid-lowering therapy

Statins reduce the incidence of major adverse cardiovascular events when given early within the first days after onset an ACS.[181,182] Consider starting statin therapy in all patients within 24 h of onset of symptoms of ACS unless contraindicated. If patients are already receiving statin therapy, do not stop it.[183]

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European Resuscitation Council Guidelines for Resuscitation 2015 Section 9. First aid

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Introduction

In 2005, the American Heart Association (AHA) together with the American Red Cross (ARC) formed the National First Aid Science Advisory Board to evaluate the science associated with the practice of First Aid and published the 2005 AHA and ARC Guidelines for First Aid. This advisory board was subsequently expanded to include representatives from several international first aid organizations to become the International First Aid Science Advisory Board (IFASAB). IFASAB evaluated the scientific literature of first aid and published the treatment recommendations for 2010 in association with the International Liaison Committee on Resuscitation (ILCOR) resuscitation recommendations.[1,2]

It was not until 2012 that ILCOR convened a full international First Aid Task Force with representation from all constituent International Councils together with the ARC. The ERC contributed directly to the Task Force as individual members, question owners and by providing expert evidence reviewers. By the ILCOR Consensus Conference in early 2015 the Task Force had completed comprehensive reviews of twenty-two questions using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) methodology in combination with ILCOR's Scientific Evidence Evaluation and Review system (SEERS). Seventeen of these questions were derived from the 2010 AHA and ARC consensus document, the remaining five questions were new topics selected by the First Aid Task Force and based on current medical requirements. All 22 questions were devised in a Population Intervention Comparison Outcome (PICO) format and librarians assisted in developing the search strategies so that the science could be reassessed at regular intervals throughout the process and into the future using the same search criteria.

In publishing these guidelines based on the 2015 Consensus on Science and Treatment Recommendations the ERC recognises that this is not a comprehensive review of all First Aid topics. The twenty-two questions reviewed in this section provide important evidence-based support for current first aid practice or changes from current practice. It is hoped that the search strategies that have been developed will be used to access newly published research. The Task Force will continue by re-examining the remaining 30 topics reviewed in 2010 and develop new questions based on current and evolving medical practice.

GRADE and First Aid

GRADE is a standardised and transparent process for the evaluation of scientific data. For the 2015 Consensus on Science ILCOR combined GRADE with the development of PICO search strings and its own SEERS system. The process contained over 50 planned steps and involved the selection of a PICO question, the development of an appropriate search string for interrogating the science databases, the analysis of the searched publications to select those relevant to the PICOs, the analysis of the individually selected papers for risk of bias and quality indicators across selected outcomes, analysis of the results of the science, and insertion of

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this information into Summary of Findings tables and then into a GRADE evidence profile. For each PICO question, two evidence reviewers carried out study selection and risk of bias assessment independently. A draft recommendation was formulated, involving a balance between the quality of the evidence identified, benefits and harms. The final results were presented in a standardised format to the ILCOR First Aid Task Force and discussed. The resulting treatment recommendations were presented to ILCOR at the 2015 Consensus on Science conference and the final recommendations formulated.[3]

Certain aspects of First Aid have little or no published data to support their practice and much has been built on expert consensus opinion, tradition and common sense. The GRADE process underlined the lack of true science behind many of the current practices and in some cases the Task Force was unable to make a treatment recommendation founded on evidence-based science. For each treatment recommendation the Task Force added a 'values and preferences' statement as a description of limitations or qualifiers for the treatment recommendations and the 'knowledge gaps' to guide future investigation and research.

In writing these guidelines the Writing Group were conscious that the consensus on science had led to a treatment recommendation that required qualification in terms of safe clinical practice. The Writing Group have added these additional clinical recommendations as expert consensual opinion and labelled them as Good Practice Points to differentiate them from guidelines derived directly from scientific review.

The 2015 definition of First Aid

First Aid is defined as the *helping behaviours* and *initial care* provided for an acute illness or injury. First Aid can be initiated by anyone in any situation. A First Aid Provider is defined as someone trained in First Aid who should:

- recognise, assess and prioritise the need for first aid;
- provide care using appropriate competencies;
- recognise limitations and seek additional care when needed.

The goals of First Aid are to preserve life, alleviate suffering, prevent further illness or injury, and promote recovery.

This 2015 definition for First Aid, as created by the ILCOR First Aid Task Force, addresses the need to recognise injury and illness, the requirement to develop a specific skill base and the need for first aid providers to simultaneously provide immediate care and to activate Emergency Medical Services or other medical care as required. First aid assessments and interventions should be medically sound and based on scientific evidence-based medicine or, in the absence of such evidence, on expert medical consensus. The scope of first aid is not purely scientific, as both training and regulatory requirements will influence it. Because the scope of first aid varies between countries, states and provinces, the guidelines contained herein may need to be refined according to circumstances, need, and regulatory constraints. Dispatcher assisted First Aid was not evaluated in the Guideline process 2015 and has not been included in these guidelines.

Summary of the 2015 First Aid Guidelines

First Aid for medical emergencies

Positioning of a breathing but unresponsive victim

Position individuals who are unresponsive but breathing normally into a lateral, side-lying recovery position as opposed to leaving them supine (lying on back). In certain situations such as resuscitation related agonal respirations or trauma, it may not be appropriate to move the individual into a recovery position.

Optimal position for a shock victim

Place individuals with shock into the supine (lying on back) position. Where there is no evidence of trauma use passive leg raising to provide a further transient (<7 min) improvement in vital signs; the clinical significance of this transient improvement is uncertain.

Oxygen administration for first aid

There are no direct indications for the use of supplemental oxygen by first aid providers.

Bronchodilator administration

Assist individuals with asthma who are experiencing difficulty in breathing with their bronchodilator administration. First aid providers must be trained in the various methods of administering a bronchodilator.

Stroke recognition

Use a stroke assessment system to decrease the time to recognition and definitive treatment for individuals with suspected acute stroke. First Aid providers must be trained in the use of FAST (Face, Arm, Speech Tool) or CPSS (Cincinnati Pre-hospital Stroke Scale) to assist in the early recognition of stroke.

Aspirin administration for chest pain

In the pre-hospital environment, administer 150–300 mg chewable aspirin early to adults with chest pain due to suspected myocardial infarction (ACS/AMI). There is a relatively low risk of complications particularly anaphylaxis and serious bleeding. Do not administer aspirin to adults with chest pain of unclear aetiology.

Second dose of adrenaline for anaphylaxis

Administer a second intramuscular dose of adrenaline to individuals in the pre-hospital environment with anaphylaxis that has not been relieved within 5 to 15 min by an initial intramuscular auto-injector dose of adrenaline. A second intramuscular dose of adrenaline may also be required if symptoms re-occur.

Hypoglycaemia treatment

Treat conscious patients with symptomatic hypoglycaemia with glucose tablets equating to glucose 15–20 g. If glucose tablets are not available, use other dietary forms of sugar.

Exertion-related dehydration and rehydration therapy

Use 3–8% oral carbohydrate–electrolyte (CE) beverages for rehydration of individuals with simple exercise-induced dehydration. Alternative acceptable beverages for rehydration include water, 12% CE solution, coconut water, 2% milk, or tea with or without carbohydrate electrolyte solution added.

Eye injury from chemical exposure

For an eye injury due to exposure to a chemical substance, take immediate action by irrigating the eye using continuous, large volumes of clean water. Refer the individual for emergency healthcare professional review.

First Aid for trauma emergencies

Control of bleeding

Apply direct pressure, with or without a dressing, to control external bleeding where possible. Do not try to control major external bleeding by the use of proximal pressure points or elevation of an extremity. However it may be beneficial to apply localised cold

therapy, with or without pressure, for minor or closed extremity bleeding.

Haemostatic dressings

Use a haemostatic dressing when direct pressure cannot control severe external bleeding or the wound is in a position where direct pressure is not possible. Training is required to ensure the safe and effective application of these dressings.

Use of a tourniquet

Use a tourniquet when direct wound pressure cannot control severe external bleeding in a limb. Training is required to ensure the safe and effective application of a tourniquet.

Straightening an angulated fracture

Do not straighten an angulated long bone fracture.

Protect the injured limb by splinting the fracture. Realignment of fractures should only be undertaken by those specifically trained to perform this procedure.

First aid treatment for an open chest wound

Leave an open chest wound exposed to freely communicate with the external environment without applying a dressing, or cover the wound with a non-occlusive dressing if necessary. Control localised bleeding with direct pressure.

Spinal motion restriction

The routine application of a cervical collar by a first aid provider is not recommended. In suspected cervical spine injury, manually support the head in position limiting angular movement until experienced healthcare provision is available.

Recognition of concussion

Although a concussion scoring system would greatly assist first aid providers in the recognition of concussion, there is no simple validated scoring system in use in current practice. An individual with a suspected concussion should be evaluated by a healthcare professional.

Cooling of burns

Actively cool thermal burns as soon as possible for a minimum of 10 min duration using water.

Burn dressings

Subsequent to cooling, burns should be dressed with a loose sterile dressing.

Dental avulsion

If a tooth cannot be immediately re-implanted, store it in Hank's Balanced Salt Solution. If this is not available use propolis, egg white, coconut water, ricetral, whole milk, saline or Phosphate Buffered Saline (in order of preference) and refer the individual to a dentist as soon as possible.

Education

First aid education programmes, public health campaigns and formal first aid training are recommended in order to improve prevention, recognition and management of injury and illness.

First Aid for medical emergencies

Positioning of the breathing but unresponsive victim

The priority management of a breathing but unresponsive victim, including one whose circulation has been successfully restored following cardiac arrest, is the maintenance of an open airway. Victims with agonal breathing should not be placed in the recovery position. The ERC 2015 Guidelines for Basic Life Support include the use of a recovery position aimed at achieving this.[4]

Although the available evidence is weak, the use a recovery position places a high value on the importance of decreasing the risk of aspiration or the need for more advanced airway management. Given the absence of high quality evidence, the recovery position is recommended due to the lack of demonstrated associated risk.

A number of different side-lying recovery positions have been compared (left lateral versus right lateral versus prone positions,[5] ERC versus Resuscitation Council (UK) positions,[6] and AHA versus ERC versus Rautek versus Morrison, Mirakhur and Craig (MMC) positions.[7] The quality of evidence is low, but overall no significant differences between the positions have been identified.

In certain situations such as trauma, it may not be appropriate to move the individual into a recovery position. The HAINES position has been reported to reduce the likelihood of causing cervical spinal injury compared with the side-lying positions.[8] The evidence for this is of very low quality with little if any difference between the positions being demonstrated.[9]

2015 First Aid Guideline

Position individuals who are unresponsive but breathing normally into a lateral, side-lying recovery position as opposed to leaving them supine (lying on back). In certain situations such as resuscitation related agonal respirations or trauma, it may not be appropriate to move the individual into a recovery position.

Overall, there is little evidence to suggest an optimal recovery position, but the ERC recommends the following sequence of actions:

- kneel beside the victim and make sure that both legs are straight,
- place the arm nearest to you out at right angles to the body, elbow bent with the hand palm uppermost;
- bring the far arm across the chest, and hold the back of the hand against the victim's cheek nearest to you;
- with your other hand, grasp the far leg just above the knee and pull it up, keeping the foot on the ground;
- keeping the hand pressed against the cheek, pull on the far leg to roll the victim towards you onto his or her side;
- adjust the upper leg so that both hip and knee are bent at right angles;
- tilt the head back to make sure the airway remains open;
- adjust the hand under the cheek if necessary, to keep the head tilted and facing downwards to allow liquid material to drain from the mouth;
- check breathing regularly.

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

Optimal position for shock victim

Shock is a condition in which there is failure of the peripheral circulation. It may be caused by sudden loss of body fluids (such as in bleeding), serious injury, myocardial infarction (heart attack), pulmonary embolism, and other similar conditions. While the primary treatment is usually directed at the cause of shock, support of the circulation is important. Although the evidence is weak, there is potential clinical benefit of improved vital signs and cardiac function by placing individuals with shock into the supine (lying on back) position, rather than by moving a victim with shock into an alternative position.

The use of passive leg raising (PLR) may provide a transient (<7 min) improvement in heart rate, mean arterial pressure, cardiac index, or stroke volume;[10–12] for those with no evidence of trauma. The clinical significance of this transient improvement is uncertain. The optimal degree of elevation has not been determined, with studies of PLR ranging between 30 and 60 degrees elevation. No study however, has reported adverse effects due to PLR.

These recommendations place an increased value on the potential, but uncertain, clinical benefit of improved vital signs and cardiac function, by positioning a victim with shock in the supine position (with or without PLR), over the risk of moving the victim.

The Trendelenburg position (legs raised–head down) was excluded from evaluation in this review and is not recommended due to the inability or impracticality of first aid providers placing a victim into this position in an out-of-hospital setting.

2015 First Aid Guideline

Place individuals with shock into the supine (lying on back) position. Where there is no evidence of trauma use passive leg raising to provide a further transient (<7 min) improvement in vital signs but the clinical significance of this transient improvement is uncertain.

Oxygen administration for first aid

Oxygen is probably one of the most commonly used drugs in medicine. Administration of oxygen in the pre-hospital environment has traditionally been considered crucial in the care of patients with an acute illness or injury, aiming at treating or preventing hypoxaemia. However, there is no evidence for or against the routine administration of supplemental oxygen by first aid providers.[13–16] Further, supplemental oxygen might have potential adverse effects that complicate the disease course or even worsen clinical outcomes and therefore its usefulness is not universally proved. If used, supplemental oxygen should only be administered by first aid providers who have been properly trained in its use and if they can monitor its effects.

2015 First Aid Guideline

There are no direct indications for the use of supplemental oxygen by first aid providers:

Bronchodilator administration

Asthma is a common chronic disease affecting millions of people worldwide, while its incidence continues to increase, especially in urban and industrialised areas. Bronchodilators are integral to asthma management and work by relaxing the bronchial smooth muscles, thereby improving respiratory function and reducing respiratory distress. The administration of a bronchodilator decreased the time to resolution of symptoms in children and reduced the time for the subjective improvement of dyspnoea in young adult asthma sufferers.[17,18] Bronchodilator administration can be achieved via different methods ranging from assisting the individual with their bronchodilator to administering a bronchodilator as part of an organised response team with medical oversight.

Individuals with asthma who experience breathing difficulties may be severely incapacitated and not be able to administer a bronchodilator themselves due to the severity of the attack or due to poor inhalation technique. Although first aid providers cannot routinely be expected to make a diagnosis of asthma, they may be able to aid an individual experiencing difficulty in breathing due to asthma by helping them to sit upright, and then assisting the patient with the administration of a prescribed bronchodilator.

Administration of bronchodilators or use of inhalers requires competency in bronchoconstriction recognition and nebuliser use and first aid providers should be trained in these methods.[19–21] National organisations must assure the quality of training in their local setting. If the patient has no bronchodilator or the bronchodilator is having no effect, activate EMS and continue to observe and assist the patient until help arrives.

2015 First Aid Guideline

Assist individuals with asthma who are experiencing difficulty in breathing with their bronchodilator administration. First aid providers must be trained in the various methods of administering a bronchodilator.

Stroke recognition

Stroke is a non-traumatic, focal vascular-induced injury of the central nervous system and typically results in permanent damage in the form of cerebral infarction, intracerebral haemorrhage and/or subarachnoid haemorrhage.[22] Every year, 15 million people worldwide suffer a stroke, nearly six million die and another five million are left permanently disabled. Stroke is the second leading cause of death above the age of 60 years and the second leading cause of disability (loss of vision, speech or partial or complete paralysis).[23]

Early admission to a stroke centre and early treatment greatly improves stroke outcome and highlights the need for first aid providers to quickly recognize stroke symptoms.[24,25] The stroke management goal is to administer definitive treatment early in the course of the stroke and to benefit from the best therapies, e.g. receiving clot-busting treatment within the first hours of the onset of stroke symptoms or in the case of intra-cerebral haemorrhage, a surgical intervention.[26] There is good evidence that the use of a stroke-screening tool improves the time to definitive treatment.[27–30]

First aid providers should be trained to utilize a simple stroke assessment tool such as the Face, Arm, Speech, Test scale (FAST)[31–35] or the Cincinnati Prehospital Stroke Scale (CPSS)[31,36,37] to identify individuals with suspected acute stroke. The specificity of stroke recognition can be improved by using a stroke assessment tool that includes the measurement of blood glucose such as the Los Angeles Prehospital Stroke Scale (LAPSS),[28,31,36,38–40] the Ontario Prehospital Stroke Scale (OPSS),[41] Recognition of Stroke in the Emergency Room (ROSIER)[32,34,35,42,43] or the Kurashiki Prehospital Stroke Scale (KPSS)[44] but it is recognised that blood glucose measurement may not be routinely available to first aid providers.

2015 First Aid Guideline

Use a stroke assessment system to decrease the time to recognition and definitive treatment for individuals with suspected acute stroke. First aid providers must be trained in the use of FAST (Face, Arm, Speech Tool) or CPSS (Cincinnati Prehospital Stroke Scale) to assist in the early recognition of stroke.

Aspirin administration for chest pain

The pathogenesis of acute coronary syndromes (ACS) including acute myocardial infarction (AMI) is most frequently a ruptured plaque in a coronary artery. As the plaque contents leak into the artery, platelets clump around them and coronary thrombosis occurs completely or partially occluding the lumen of the artery, leading to myocardial ischemia and possible infarction.

The use of aspirin as an antithrombotic agent to potentially reduce mortality and morbidity in ACS/AMI is considered beneficial even when compared with the low risk of complications, particularly anaphylaxis and serious bleeding (requiring transfusion).[45–49] The early administration of aspirin in the pre-hospital environment,

within the first few hours of the onset of symptoms, also reduces cardiovascular mortality,[50,51] supporting the recommendation that first aid providers should administer aspirin to those individuals with chest pain from suspected myocardial infarction.

All patients with chest pain due to suspected myocardial infarction should seek immediate healthcare professional advice and be transferred to hospital for definitive medical care. First aid providers should call for help and administer a single oral dose of 150–300 mg chewable or soluble aspirin whilst waiting for healthcare professional assistance to arrive.[52] This early administration of aspirin should never delay the transfer of the patient to a hospital for definitive care.

Aspirin should not be administered to patients who have a known allergy or contraindications to aspirin.

It is recognised that a first aid provider might have difficulty in identifying chest pain of cardiac origin and the pre-hospital administration of aspirin by first aid providers to adults with chest pain of unclear aetiology is not recommended. If there is any doubt call for the advice and assistance of a healthcare professional.

2015 First Aid Guidelines

In the pre-hospital environment, administer 150–300 mg chewable aspirin early to adults with chest pain due to suspected myocardial infarction (ACS/AMI). There is a relatively low risk of complications particularly anaphylaxis and serious bleeding. Do not administer aspirin to adults with chest pain of unclear aetiology.

Second dose of adrenaline for anaphylaxis

Anaphylaxis is a potentially fatal, allergic reaction that requires immediate recognition and intervention. It is a rapid multi-organ system reaction, affecting the cutaneous, respiratory, cardiovascular, and gastrointestinal systems, usually characterised by swelling, breathing difficulty, shock and even death. Adrenaline reverses the pathophysiological manifestations of anaphylaxis and remains the most important drug, especially if it is given within the first few minutes of a severe allergic reaction.[53–55] Although adrenaline should be administered as soon as the diagnosis is suspected, the majority of patients die due to lack of adrenaline or delays in its administration.[54,56]

In the pre-hospital setting, adrenaline is administered via prefilled auto-injectors, which contain one dose of 300 mcg of adrenaline for intra-muscular self-administration or assisted by a trained first aid provider. If symptoms are not relieved within 5–15 min of the initial dose or reoccur, a second dose of intramuscular adrenaline is recommended.[57–66]

No absolute contraindications to the use of adrenaline for anaphylaxis have been identified.[54,67,68] Adverse effects have previously been reported in the literature when adrenaline is administered at an incorrect dose or via inappropriate routes such as the intravenous route. Use of auto-injectors by first aid providers should minimize the opportunity for mis-dosing or administration of adrenaline by the intravenous route.

2015 First Aid Guideline

Administer a second intramuscular dose of adrenaline to individuals in the pre-hospital environment with anaphylaxis that has not been relieved within 5 to 15 min by an initial intramuscular auto-injector dose of adrenaline. A second intramuscular dose of adrenaline may also be required if symptoms re-occur.

Hypoglycaemia treatment

Diabetes is a chronic disease that occurs either when the pancreas does not produce enough insulin, a hormone that regulates blood sugar, or when the body cannot effectively use the insulin it produces.

Diabetes is frequently complicated by serious events such as heart attack and stroke but significant or extreme alterations of blood sugar level (hyper- and hypoglycaemia) can present as a medical emergency. Hypoglycaemia is usually a sudden and life-threatening event with the typical symptoms of hunger, headache, agitation, tremor, sweating, psychotic behaviour (frequently resembling drunkenness) and loss of consciousness. It is most important that these symptoms are recognised as hypoglycaemia as the victim requires rapid first aid treatment.

Glucose tablets equating to glucose 15–20 g should be used by first aid providers, before dietary forms of sugar for treating symptomatic hypoglycaemia, in conscious patients who are able to follow commands and swallow. Glucose tablets, however, may not always be immediately available and various alternative forms of dietary sugars such as Skittles™, Mentos™, sugar cubes, jellybeans and orange juice can be used to treat symptomatic hypoglycaemia.[69–71] Glucose gels and paste are not directly equivalent to oral glucose tablets in terms of dosing and absorption.

If the patient is unconscious or unable to swallow then oral treatment should be withheld due to the risk of aspiration, and the emergency medical services should be called.

2015 First Aid Guideline

Treat conscious patients with symptomatic hypoglycaemia with glucose tablets equating to glucose 15–20 g. If glucose tablets are not available, use other dietary forms of sugar.

Exertion-related dehydration and rehydration therapy

First aid providers are often called upon to assist at "hydration stations" for sporting events such as bicycle races or foot races. Failure to hydrate adequately before, during and following exercise contributes to exercise-associated dehydration. When vigorous exercise takes place during periods of high ambient temperatures, dehydration may be associated with heat cramps, heat exhaustion or heat stroke.

Water is commonly used for rehydration following exercise, but newer commercial "sports drinks" are often promoted for this purpose. Furthermore, alternative beverages (tea or coconut water) have recently been promoted as acceptable for oral rehydration and some athletes may have a cultural preference for these beverages. Solutions made from oral rehydration salt packets and homemade solutions are more commonly used for rehydration following gastrointestinal losses and are not as practical for use following exercise-associated dehydration.

3–8% oral carbohydrate–electrolyte (CE) beverages were found to be superior to water and are therefore recommended for rehydration of individuals with simple exercise-induced dehydration.[72–80] It is recognised that water may be the simplest and most readily available rehydrating solution and that palatability and gastro-intestinal tolerance may be a factor that limits rehydration with fluids other than water. Other alternative acceptable beverages for rehydration include 12% CE solution,[72] coconut water,[73,79,80] 2% milk,[77] or tea with or without carbohydrate electrolyte solution added.[74,81]

It is known that thirst is not an accurate guide for rehydration, and the volume of oral fluids ingested typically must at least equal the volume fluid lost. The exact amount of liquid required for adequate rehydration may not be determinable in the first aid setting.

Oral hydration may not be appropriate for individuals with severe dehydration associated with hypotension, hyperpyrexia or mental status changes. Such individuals should receive care by an

advanced medical provider capable of administering intravenous fluids (Good Practice Point).

2015 First Aid Guideline

Use 3–8% oral carbohydrate–electrolyte (CE) beverages for rehydration of individuals with simple exercise-induced dehydration. Alternative acceptable beverages for rehydration include water, 12% CE solution, coconut water, 2% milk, or tea with or without carbohydrate electrolyte solution added.

Eye injury from chemical exposure

Accidental exposure of the eye to chemical substances is a common problem in both the household and industrial setting and it is often difficult to identify precisely what chemical has entered the eye. Alkali injury to the cornea has been shown to cause severe corneal injury and risk of blindness. Irrigation with large volumes of water was more effective at improving corneal pH as compared to using low volumes or saline irrigation.[82]

Trying to identify the chemical substance may delay treatment and it is recommended that the first aid provider should flush the eye with continuous large volumes of clean water immediately after the injury has been sustained and to refer the patient for emergency healthcare review.

Where there is a known high risk of eye contamination by particular chemicals, specific antidotes should be readily available.

2015 First Aid Guideline

In case of eye injury due to exposure to a chemical substance, take immediate action. Put on disposable gloves. Irrigate the eye using continuous, large volumes of clean water. Take care that the rinsing water does not come into contact with the other eye (Good Practice Point). Call 112 and the Poison Control Centre. Wash your hands after giving first aid. Refer the individual for emergency healthcare professional review (Good Practice Point).

First aid for trauma emergencies

Control of bleeding

There is a paucity of literature comparing different bleeding control strategies commonly employed by first aiders. The best control of bleeding is to apply direct pressure to the wound where possible. Localised cold therapy, with or without pressure, may be beneficial in haemostasis for minor or closed bleeding in extremities although this is based on in-hospital evidence.[83,84] There is no published evidence for the effective use of proximal pressure points to control bleeding.

Where bleeding cannot be controlled by direct pressure it may be possible to control bleeding by the use of a haemostatic dressing or a tourniquet (see below).

2015 First Aid Guideline

Apply direct pressure, with or without a dressing, to control external bleeding where possible. Do not try to control major external bleeding by the use of proximal pressure points or elevation of an extremity. However it may be beneficial to apply localised cold therapy, with or without pressure, for minor or closed extremity bleeding.

Haemostatic dressings

Haemostatic dressings are commonly used to control bleeding in the surgical and military settings especially when the wound is in a non-compressible area such as the neck, abdomen, or groin. Early generation haemostatic agents were powder or granules that were poured directly into the wound. Some of these were associated with exothermic reactions that could exacerbate tissue injury. Major improvements have been made in the composition, texture, and active constituent materials of haemostatic dressings.[85–89] In human studies there was a reported improvement in haemostasis associated with a low complication rate of 3% from the use of haemostatic dressings and a decrease in mortality.[90–93]

2015 First Aid Guideline

Use a haemostatic dressing when direct pressure cannot control severe external bleeding or the wound is in a position where direct pressure is not possible. Training is required to ensure the safe and effective application of these dressings.

Use of a tourniquet

Haemorrhage from vascular injured extremities may result in life-threatening exsanguination and is one of the leading causes of preventable death on the battlefield and in the civilian setting.[94,95] Initial management of severe external limb bleeding is direct pressure but this may not be possible and even a tight compression bandage directly over the wound may not completely control major arterial bleeding.

Tourniquets have been used in military settings for severe external limb bleeding for many years.[96,97] The application of a tourniquet has resulted in a decrease in mortality,[96,98–106] haemostasis being achieved with an associated incidence of complications of 6% and 4.3%.[96,97,99,100,103,105–109]

2015 First Aid Guideline

Use a tourniquet when direct wound pressure cannot control severe external bleeding in a limb. Training is required to ensure the safe and effective application of a tourniquet.

Straightening an angulated fracture

Fractures, dislocations, sprains and strains are extremity injuries commonly cared for by first aid providers. Long bone fractures, particularly of the leg or forearm, may be angulated on presentation. Severe angulation may limit the ability to properly splint the extremity or move the injured individual.

First aid for fractures begins with manual stabilisation of the fracture, followed by splinting in the position found. Splinting, to include the joint above and the joint below the break, protects the injury from further movement and thus prevents or reduces pain and the potential for converting a closed fracture to an open fracture.

Although there are no published studies that show a benefit to stabilising or splinting a fractured extremity, common sense and expert opinion support the use of a splint to immobilize the injured extremity for the purpose of preventing further harm and reducing pain. Splinting of an extremity injury by first aid providers should be “in the position found”, with as little movement as possible to apply the splint. In some cases, an extremity fracture will present with severe angulation, making application of a splint and transportation extremely difficult or impossible. In these cases, the first aid provider may defer to a provider with specific training to perform minimal realignment to facilitate splinting and transportation to a hospital.

2015 First Aid Guideline

Do not straighten an angulated long bone fracture (Good Practice Point).

Protect the injured limb by splinting the fracture to reduce movement, limit pain, reduce the chance for further injury and to facilitate safe and prompt transport. Realignment of fractures

should only be undertaken by those specifically trained to perform this procedure.

First aid treatment for an open chest wound

The correct management of an open chest wound is critical, as the inadvertent sealing of these wounds by the incorrect use of occlusive dressings or device or the application of a dressing that becomes occlusive may result in the potential life-threatening complication of a tension pneumothorax.[110] A decrease in the incidence of respiratory arrest and improvements in oxygen saturation, tidal volume, respiratory rate and mean arterial pressure has been shown using a non-occlusive device in an animal model.[111] It is important that an open chest wound, especially with associated underlying lung damage, is not occluded and that the inside of the chest is in open communication with the external environment.

2015 First Aid Guideline

Leave an open chest wound exposed to freely communicate with the external environment without applying a dressing, or cover the wound with a non-occlusive dressing if necessary. The use of occlusive devices or dressings can be associated with the potentially life-threatening complication of a tension pneumothorax. Control localised bleeding with direct pressure.

Cervical spinal motion restriction

Definitions

- Spinal immobilisation is defined as the process of immobilising the spine using a combination of devices (e.g. backboard and cervical collar) intended to restrict spinal motion.
- Cervical spinal motion restriction is defined as the reduction or limitation of cervical spine movement using mechanical cervical devices including cervical collars and sandbags with tape.
- Spinal stabilisation is defined as physical maintenance of the spine in a neutral position prior to applying spinal motion restriction devices.

In suspected cervical spine injury it has been routine to apply cervical collars to the neck, in order to avoid further injury from spinal movement. However, this intervention has been based on consensus and opinion rather than on scientific evidence.[112,113] Furthermore, clinically significant adverse effects, such as raised intracranial pressure, have been shown to occur following the application of a cervical collar.[114–118]

2015 First Aid Guideline

The routine application of a cervical collar by a first aid provider is not recommended.

In suspected cervical spine injury, manually support the head in a position limiting angular movement until experienced healthcare providers are available (Good Practice Point).

Recognition of concussion

Minor head injuries without loss of consciousness are common in adults and children. The first aid providers may find it difficult to recognise concussion (minor traumatic brain injury (TBI)) due to the complexity of the symptoms and signs, and this can lead to a delay in providing proper concussion management and post-concussion advice and treatment.

In sport, the use of a sport concussion assessment tool (SCAT3) is widely advocated and used.[119] This tool is advocated for use by healthcare professionals and requires a two-stage assessment, before competition and post concussion. It is therefore not appropriate as a single assessment tool for first aid providers. If an athlete with a suspected concussion has had an initial SCAT3 assessment then they should be referred to a healthcare professional for further assessment and advice.

2015 First Aid Guideline

Although a concussion scoring system would greatly assist first aid providers in the recognition of concussion, there is no simple validated scoring system in use in current practice. An individual with a suspected concussion should be evaluated by a healthcare professional.

Cooling of burns

Immediate active cooling of thermal burns, defined as any method undertaken to decrease local tissue temperature, is a common first aid recommendation for many years. Cooling thermal burns will minimise the resulting depth of the burn[120,121] and possibly decrease the number of patients that will eventually require hospital admission for treatment.[122] The other perceived benefits of cooling are pain relief and reduction of oedema, reduced infection rates and a faster wound healing process.

There are no scientifically supported recommendations for the specific cooling temperature, the method of cooling (e.g. gel pads, cold packs or water) or the duration of cooling. Clean water is readily available in many areas of the world and can therefore be used immediately for cooling of burns. Cooling of burns for 10 min is the currently perceived recommended practice.

Care must be taken when cooling large thermal burns or burns in infants and small children so as not to induce hypothermia.

2015 First Aid Guideline

Actively cool thermal burns as soon as possible for a minimum of 10 min duration using water.

Wet or dry burn dressings

A broad range of burn wound dressings are available, ranging from hydrocolloid dressings, polyurethane film dressings, hydrogel dressings, silicon-coated nylon dressings, biosynthetic skin substitute dressings, antimicrobial dressings, fibre dressings and simple wound dressing pads with or without medication.[123] Current burn wound dressings also include plastic wrap (cling film or medical commercial forms) and has the advantage that it is inexpensive, widely available, non-toxic, non-adherent, impermeable, and transparent allowing for wound monitoring without having to remove the dressing.

No scientific evidence was found to determine which type of dressings, wet or dry, is most effective. The decision about which type of burn dressing first aid providers should use, should therefore be determined by national and local burn management policies.

2015 First Aid Guideline

Subsequent to cooling, burns should be dressed according to current practice with a loose sterile dressing (Good Practice Point).

Dental avulsion

Following a fall or accident involving the face, a tooth can be injured or avulsed. Appropriate first aid in the case of an avulsed permanent tooth increases the chance of recovery after replacement of the tooth. Immediate re-implantation is the intervention of choice by the dental community, however it is often not possible for first aid providers to re-implant the tooth due to a lack of training or skills in that procedure.

If the tooth is not immediately re-implanted, the priority is to get the patient and the avulsed tooth to a dentist, who is capable of re-implanting the tooth as soon as possible. In the meanwhile store the tooth in a temporary storage solution. Hanks Balanced Salt solution is the recommended medium,[124–127] but other recommended solutions are Propolis,[126,128] egg white,[125,126] coconut water,[127] ricetral[124] when compared with survival following storage in whole milk. Saline[129,130] and Phosphate Buffered saline[131] were less effective as storage solutions than whole milk. The choice of a storage solution depends on the availability and accessibility of the solution.

2015 First Aid Guideline

If a tooth cannot be immediately re-implanted store it in Hank's Balanced Salt Solution. If this is not available use Propolis, egg white, coconut water, ricetral, whole milk, saline or Phosphate Buffered Saline (in order of preference) and refer the individual to a dentist as soon as possible.

Education

First Aid education and training

Education and training in First Aid has been shown to increase survival from trauma among those patients cared for by trained first aid providers[132] and to improve the resolution of symptoms.[133] Education in the form of a public health campaign has also improved the ability to recognise life-threatening illness, such as stroke[134] and from a prevention perspective it has been shown to reduce the incidence of burn injury.[122]

2015 First Aid Guideline

First aid education programmes, public health campaigns and formal first aid training are recommended in order to improve prevention, recognition and management of injury and illness.

Conflicts of interest

David Zideman	No conflict of interest reported
Anthony J. Handley	Medical advisor BA, Virgin, Places for people, Life saving Societies, Trading Company Secretary RCUK
Christina Hafner	No conflict of interest reported
Daniel Meyran	French Red Cross: Medical advisor
Emmy De Buck	Belgian Red Cross-Flanders: employee
Eunice Singletary	American Red Cross Advisory Council member
Pascal Cassan	French Red Cross Head Global First Aid Defence Center
Philippe Vandekerckhove	Red Cross Belgium: employee
Susanne Schunder-Tatzber	OMV Austrian Oil&Gas company: Health Manager
Thanos Chalkias	No conflict of interest reported
Tom Evans	No conflict of interest reported

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European Resuscitation Council Guidelines for Resuscitation 2015 Section 10. Education and implementation of resuscitation

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Introduction

The chain of survival[1] was extended to the formula of survival[2] because it was realised that the goal of saving more lives relies not only on solid and high quality science but also the effective education of lay people and healthcare professionals.[3] Ultimately, those who are engaged in the care of cardiac arrest victims should be able to implement resource efficient systems that can improve survival after cardiac arrest.

This chapter incorporates the 17 key educational PICO-questions (Population–Intervention–Control–Outcome) that where reviewed by the Education, Implementation and Teams (EIT) Task Force of the International Liaison Committee on Resuscitation (ILCOR) from 2011 to 2015. This evidence review and evaluation process followed the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) process described in the Consensus on Science and Training Recommendations 2015 (CoSTR).[4] It summarises the new treatment recommendations for training and implementation. This chapter also covers the ERC basic principles of training and teaching of basic life support as well as advanced level life support. There is a strong focus on non-technical skills teaching (e.g. communication skills, team and leadership training). The ERC portfolio of courses is also included in this chapter, which ends with an outlook about educational resuscitation research and future course developments.

Delays in providing training materials and freeing staff for training were cited as reasons for delays in the implementation of the last guidelines.[5–7] Therefore the ERC has carefully planned the translation and dissemination process for these guidelines and the teaching material for all courses to facilitate the implementation of the 2015 guidelines on resuscitation in a timely manner. This chapter provides the basis of a successful educational strategy for improved CPR education.

Summary of changes since the 2010 ERC guidelines

The following is a summary of the most important new reviews or changes in recommendations for education, implementation and teams since the ERC 2010 Guidelines:

Training

- High fidelity training manikins provide greater physical realism and their use is popular with learners. They are, however, more expensive than standard lower fidelity manikins. In centres that have the resources to purchase and maintain high fidelity manikins, we recommend their use. The use of lower fidelity manikins however is appropriate for all levels of training on ERC courses.
- Directive CPR feedback devices are useful for improving compression rate, depth, release, and hand position. Tonal devices improve compression rates only and may have a detrimental effect on compression depth while rescuers focus on the rate. There is no current evidence to link tonal device use with improved outcomes following an ERC course.
- The intervals for retraining will differ according to the characteristics of the participants (e.g. lay or healthcare). It is known that CPR skills deteriorate within months of training and therefore annual retraining strategies may not be frequent enough. Whilst optimal intervals are not known, frequent ‘low dose’ retraining may be beneficial.

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- Training in non-technical skills (e.g. communication skills, team leadership and team member roles) is an essential adjunct to the training of technical skills. This type of training should be incorporated into life support courses.
- Ambulance service dispatchers have an influential role to play in guiding lay rescuers how to deliver CPR. This role needs specific training in order to deliver clear and effective instructions in a stressful situation.

Implementation

- Data-driven performance-focused debriefing has been shown to improve performance of resuscitation teams. We highly recommend their use for teams managing patients in cardiac arrest,
- Regional systems including cardiac arrest centres are to be encouraged, as there is an association with increased survival and improved neurological outcome in victims of out-of-hospital cardiac arrest.
- The use of innovative technologies and social media can be beneficial for the deployment of rapid responders to victims of out-of-hospital cardiac arrest. Novel systems are also being developed to alert bystanders to the location of the nearest AED. Any technology that improves the delivery of swift bystander CPR with rapid access to an AED is to be encouraged.
- "It takes a system to save a life". [http://www.resuscitationacademy.com/] Healthcare systems with a responsibility for the management of patients in cardiac arrest (e.g. EMS organisations, cardiac arrest centres) should evaluate their processes to ensure that they are able to deliver care that ensures the best achievable survival rates.

Basic level training

Who to train

Basic Life Support (BLS) is the cornerstone of resuscitation and it is well established that bystander CPR is critical to survival in out-of-hospital cardiac arrests. Chest compressions and early defibrillation are the main determinants of survival from an out-of-hospital cardiac arrest and there is some evidence that the introduction of training for lay people has improved survival at 30 days and 1 year.[8,9]

For this reason a primary educational goal in resuscitation should be the training of lay people in CPR. There is evidence that training lay people in BLS is effective in improving the number of people willing to undertake BLS in a real situation.[10–12] The term 'lay people' includes a wide range of capabilities from those without any formal health care training to those with a role where it may be expected that they would provide CPR (e.g. lifeguards, first aiders). Despite the increase in access to training for lay people, there is still an unwillingness of some to perform CPR. The reasons identified for this include fear of infection, fear of getting it wrong, and fear of legal implications.[13]

Training of family members of high risk patients can reduce anxiety of those family members and the patient, improve emotional adjustment, and empower individuals to feel that they would be able to start CPR. For high-risk populations (e.g. areas where there is high risk of cardiac arrest and low bystander response), recent evidence shows that specific factors can be identified which will enable targeted training based on the community's unique characteristics.[14,15] There is evidence that likely rescuers in these populations are unlikely to seek training on their own but that they gain competency in BLS skills and/or knowledge after training.[16–18] They are willing to be trained and are likely to share training with others.[16,17,19–21]

Most research in the teaching of resuscitation has been based on training adult rescuers in adult resuscitation skills. However teaching children and young adults arguably requires different approaches, but more research is required into the best methods to teach these groups basic life support.[22]

One of the most important steps in increasing the rate of bystander resuscitation and improving survival worldwide is to educate all school children. The American Heart Association advocated compulsory resuscitation training in American schools in 2011.[23] Prior to this, the experience of teaching CPR to school children in Seattle over the last three decades has resulted in significantly higher bystander CPR rates and survival rate. Similarly, Scandinavian educational resuscitation school programs report significantly higher resuscitation rates.[24] This can be easily achieved by teaching children for just 2 h per year, beginning at the age of twelve.[22] At that age, school children have a positive attitude toward learning resuscitation and both medical professionals and teachers require training to enable them to maximise the potential of these children.[25] School children and their teachers are resuscitation multipliers in both private and public settings as the children have been shown to pass on their learning to family members. The proportion of trained individuals in society will markedly increase in the longer term, leading to an increase in the overall rate of lay resuscitation.[26]

Healthcare professionals working in a variety of settings including the community, emergency medical systems (EMS), general hospital wards, and critical care areas should all be taught CPR. Whilst low quality compressions are common both in terms of incorrect depth and rate, interruptions also contribute to ineffective CPR.[27] Given that poor performance is associated with lower survival rates, training on these components should be a core aspect of any resuscitation training.

It has been shown that well trained EMS dispatchers are able to improve bystander CPR and patient outcomes.[28] However there are concerns with their ability to recognise cardiac arrest particularly in relation to agonal breathing.[29] Consequently training of EMS dispatchers should include a focus on identification and the significance of agonal breathing,[30] and the importance of seizures as aspects of cardiac arrest. In addition EMS dispatchers need to be taught simplified scripts for instructing bystanders in CPR.[30]

How to train

BLS/AED curricula should be tailored to the target audience and kept as simple as possible. Increasing access to different modalities of training (e.g. the use of digital media, on line, instructor-led teaching) and self-directed learning, offer alternative means of teaching both lay and professional providers. The effectiveness of these different blended learning approaches remains unclear and further research is required not only to link the immediate outcomes of courses to the teaching approach but also ultimately to identify the impact on the outcome of real life cardiac arrest situations. Training should be tailored to the needs of different types of learners and a variety of different teaching methods should be used to ensure acquisition and retention of resuscitation knowledge and skills. Self-instruction programmes with synchronous or asynchronous hands on practice (e.g. video, DVD, on-line training, computer giving feedback during training) appear to be an effective alternative to instructor-led courses for laypeople and healthcare providers learning BLS skills.[31–35]

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).[36,37]

Basic life support and AED curriculum

Lay people are not only capable of effectively learning CPR, but evidence shows that they can be taught to use AEDs.[38] The introduction of Public Access Defibrillator (PAD) schemes has demonstrated the effectiveness of lay people in performing defibrillation,[39] but the question remains whether lay people require training to use AEDs or can use them without any prior input.[40] The curriculum for basic life support and AED training should be tailored to the target audience and kept as simple as possible. Whichever modality is chosen for the teaching, the following should be considered as core elements of the BLS and AED curriculum:

- Willingness to start CPR, including an understanding of personal and environmental risk
- Recognition of unconsciousness, gasping or agonal breathing in unresponsive individuals by assessment of responsiveness, opening of the airway and assessment of breathing to confirm cardiac arrest.[41,42]
- Good quality chest compressions (adherence to rate, depth, full recoil and minimising hands-off time) and rescue breathing (ventilation time and volume)
- Feedback/prompts (human feedback within the CPR-team and/or from devices) during CPR training to improve skill acquisition and retention during basic life support training.[43]

Standard CPR versus chest compression-only CPR teaching

The role of standard CPR versus chest compression-only CPR is discussed in the BLS Chapter of these ERC guidelines.[42] A simplified, education-based approach is suggested to allow communities to train all citizens in CPR:

- All citizens should be taught how to perform chest compressions as a minimum requirement.
- Ideally, full CPR skills (compressions and ventilation using a 30:2 ratio) should also be taught to all citizens.
- When training is time-limited or opportunistic (e.g. EMS telephone instructions to a bystander, mass events, public campaigns, internet-based viral videos), it should focus on compression-only CPR. Local communities may want to consider their approach based on their local population epidemiology, cultural norms and bystander response rates.
- For those initially trained in compression-only CPR, ventilation may be covered in subsequent training. 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 carers, should be taught standard CPR i.e. chest compressions and ventilation.
- For the resuscitation of children, rescuers should be encouraged to attempt resuscitation using whichever adult sequence they have been taught, as the outcome is worse if nothing is done. Non-specialists who wish to learn paediatric resuscitation because they have a responsibility for children (e.g. parents, teachers, school nurses, lifeguards), should be taught that it is preferable to modify adult basic life support and give five initial breaths followed by approximately 1 min of CPR before they go for help, if there is no-one to go for them.[44]

Basic life support and AED training methods

There are numerous methods to deliver basic life support and AED training. Traditionally, instructor-led training courses remain the most frequently used method for basic life support and AED training.[45] When compared with traditional instructor-led training, well designed self-instruction programmes (e.g. video, DVD, computer supported feedback) with shortened instructor coaching may be effective alternatives for laypeople and healthcare providers learning basic life support and, in particular, for the training of laypeople in AED skills. [18,33,34,46–49]

If instructor-led training is not available then self-directed training is an acceptable pragmatic option to use an AED. Short video/computer self-instruction (with minimal or no instructor coaching) that includes synchronous hands-on practice in AED use (practice-while-you-watch) may be considered as an effective alternative to instructor-led AED courses.[48,50,51]

Ultimately, it is known that rescuers can use AEDs without any formal training. It has been shown that the presence of a nearby AED is no guarantee of their usage.[52] The advantage of delivering training, therefore, is that it increases general awareness of their use and benefit, whilst also providing a forum to dispel common myths about their use (e.g. the belief that they may do harm).

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

The optimal duration of instructor-led BLS 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), 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 decay within three to six months after initial training.[33,46,53–55] AED skills are retained for longer than BLS skills alone.[56,57]

Although there is some evidence that higher frequency, short burst training could potentially enhance BLS training and reduce skill decay, more studies are needed to confirm this.[53,55–57]

Current evidence shows that performance in the use of an AED (e.g. speed of use, correct pad placement) can be further improved with brief training of laypeople and healthcare professionals.[49,58–60] Brief bedside booster CPR training of 2 min has also been shown to improve CPR quality irrespective of training content (instructor, or automated feedback or both) in Paediatric Basic Life Support providers during simulated cardiac arrest[61] and improved with further training.[62]

Peer-led resuscitation training has also been shown to be an effective means of delivering BLS training. Peer-tutors and assessors are competent, more available and less costly than clinical staff. Student instructors develop skills in teaching, assessment and appraisal, organisation and research. Sustainability is possible given succession-planning and consistent leadership. A 15 year review of peer led BLS teaching in a major University medical school demonstrated that such programmes can deliver greater participant satisfaction with learning outcomes equal to previous lecture-based sessions.[63]

As there is evidence that frequent training improves CPR skills, responder confidence and willingness to perform CPR, it is recommended that organisations and individuals review the need for more frequent retraining based on the likelihood of cardiac arrest in their area. Retraining should take place at least every 12–24 months for students who are taking BLS courses. Additional high frequency, low dose update or retraining in certain settings may be considered. It is recommended that individuals more likely to encounter cardiac arrest consider more frequent retraining, due to the evidence that skills decay within 3–12 months after BLS training[33,46,53,54,56,64] and evidence that frequent training improves CPR skills,[34,65–69] responder confidence,[65] and willingness to perform CPR.[34]

Use of CPR prompt/feedback devices during training

The use of CPR prompt/feedback devices may be considered during CPR training for lay people and healthcare professionals. Devices can be prompting (i.e. signal to perform an action e.g. metronome for compression rate or voice feedback), give feedback (i.e. after-event information based on effect of an action such as visual display of compression depth), or a combination of prompts and feedback. Training using a prompt/feedback device can improve CPR skill performance.[70] Instructors and rescuers should be made aware that a compressible support surface (e.g. mattress) may cause some prompt/feedback devices to overestimate depth of compression.[71,72]

A systematic appraisal of the literature determined in both manikin and human studies that audiovisual feedback devices during resuscitation resulted in rescuers providing chest compression parameters closer to recommendations but no evidence was found that this translates into improved patient outcomes.[73] Substantial variation in the ability of CPR feedback devices to improve performance was found.[74–76]

Advanced level training

Advanced level courses are mainly directed at healthcare personnel. In general, they cover the knowledge, skills and attitudes needed to function as part of (and ultimately lead) a resuscitation team.

Pre-course training and possible alternatives strategies to improve CPR training

A variety of methods can be used to prepare candidates before attending a life support course. These include the provision of pre-course reading, in the form of manuals and/or e-learning. Incorporating a pre-test into the preparatory work may further enhance these materials.[77–82] One such example was a CD-based pre-course e-learning program for ALS that was well received by the participants. It was rated as improving their understanding of the key learning domains of the ALS course but failed to show superiority for cognitive or psychomotor skills during a standard cardiac arrest simulation.[83]

Evidence has emerged regarding blended learning models (independent electronic learning coupled with a reduced duration instructor-led course). A pilot blended learning approach to ALS training including e-learning led to a 5.7% lower pass rate in cardiac arrest scenario testing, but similar scores on a knowledge and skills assessments, and reduced costs by more than half. There was no significant difference in overall pass rates.[84] This UK-based e-learning-ALS course was subsequently implemented and a further study of 27,170 candidates demonstrated equivalence to traditional instructor-led learning.[85] The online e-learning program of 6–8 h was to be completed by candidates prior to attending a one-day modified instructor-led ALS-course. e-ALS scores were significantly higher on the pre- and post-course MCQ and first attempt CAS-test pass rate was higher than compared to standard ALS courses (overall pass rate similar in both). Considering benefits such as increased candidate autonomy, improved cost-effectiveness, decreased instructor burden and improved standardisation of course material these reports encourage further dissemination of the e-learning courses for CPR training.

Principles of teaching skills

CPR skills can be taught in a stepwise process: dissecting the components of a skill into a real-time demonstration, explaining the facts, demonstration by the participants, and practicing to facilitate visualisation, understanding, cognitive processing and execution of a skill. No studies have showed any advantage for different stepwise approaches despite their theoretical framework.[86,87]

Basics of simulation to teach on advanced level courses

Simulation training is an integral part of resuscitation training. A systematic review and meta-analysis of 182 studies involving 16,636 participants on simulation-based training for resuscitation showed improvement in knowledge and skill performance compared to training without simulation.[88]

Simulation training can be used to train a range of roles from the first responder to the resuscitation team member and ultimately the resuscitation team leader. It can be utilised to train both individual and team behaviour. A critical adjunct to this learning is the debriefing that occurs at the conclusion of the scenario.

With the exception of simulation training using live actors, the majority of training involves the use of purpose built manikins. High-fidelity manikins can provide physical findings, display vital signs, physiologically respond to interventions (via computer interface) and enable procedures to be performed on them (e.g. bag mask ventilation, intubation, intravenous or intra-osseous vascular access).[89] Simulation training using high-fidelity versus low-fidelity manikins seems to deliver a slight improvement in training outcome on skill performance at the end of the course.[90]

When considering physical realism, these high-fidelity manikins are more popular with candidates and faculty but they are also much more expensive. Evidence that participants in ERC courses learn more or better CPR by using high-fidelity manikins is lacking. With this in mind, high-fidelity manikins can be used but if they are not available, the use of low-fidelity manikins is acceptable for standard advanced life support training.

Adherence to real-time 2-min cycles during advanced life support simulations is an important part of realistic fidelity. It is important that the duration of CPR cycles is not deliberately decreased in order to increase the number of scenarios.[91]

New teaching methods hold promise for the future but need more research before being adopted on a larger scale. Examples include specifically teaching “action-linked phrases” like “There’s no pulse, I will start chest compressions” which will generally prompt action (e.g. chest compressions) when taught on courses.[92] Another example is “Rapid cycle deliberate practice” (RPSD) training, which has been shown to increase resuscitation skills in paediatric residents.[93] After an initial uninterrupted scenario and debriefing, the next scenarios are short, and interrupted at predetermined points to give direct feedback on specific procedures or actions.

Training of non-technical skills (NTS) including leadership and team training to improve CPR outcome

Accomplishing successful resuscitation is a team performance in most instances and as with any other skill, effective teamwork and leadership skills need to be trained.[94,95] For example, the implementation of team training programmes resulted in an increase in hospital survival from paediatric cardiac arrest[96] and in surgical patients.[97]

Training in non-technical skills, such as effective communication, situational awareness, leadership and followership, using crisis resource management principles purposefully in simulations, has been shown to transfer learning from simulation into clinical practise.[98,99] Resuscitation team performance has been shown to improve in actual cardiac arrest or simulated in-hospital advanced life support scenarios, when specific team or leadership training is added to advanced level courses.[100–104] By delivering training in

an environment as close to real-life experience as possible, concepts regarding team working can be addressed at the level of the individual.[105,106]

Specific team training can increase team performance, leadership skills, and task management performance and the effect can last for up to one year.[94,95,100,101,107–111] On the other hand, leadership training in addition to CPR skills has been shown not to improve actual CPR skills.[112]

Assessment instruments (mainly checklists) have been developed, validated, and recommended for individual team members. Rating scales exist for the assessment of team performance, which can subsequently be used to deliver feedback on team performance.[113–116]

Training intervals and assessment of competences

Little evidence exists about the retention of knowledge after ALS courses.[117] It is believed that learners with increased clinical experience have improved long-term retention of knowledge and skills.[118,119] Written tests in ALS courses do not reliably predict practical skill performance and should not be used as a substitute for demonstration of clinical skill performance.[120,121] Assessment at the end of training seems to have a beneficial effect on subsequent performance and retention.[122,123]

There is emerging evidence that frequent manikin-based refresher training in the form of low-dose in-situ training may save costs, reduce the total time for retraining, and it seems to be preferred by the learners.[124,125] Refresher training is invariably required to maintain knowledge and skills; however, the optimal frequency for refresher training is unclear.[124,126–128]

A simulation-enhanced booster session nine months after a neonatal resuscitation training program demonstrated better procedural skill and teamwork behaviour at fifteen months.[129] Teamwork behaviours were further enhanced when residents were engaged in clinical resuscitation or by exposure to deliberate practice with simulation.

Use of checklists, feedback devices, and in-situ training

Cognitive aids such as checklists may improve adherence to guidelines as long as they do not cause delays in starting CPR and the correct checklist is used during simulation [130] and real patient cardiac arrest.[131] For example, the implementation of an Advanced Trauma Life Support check list improved adherence to protocol driven task performance, frequency and speed of task completion.[132]

Feedback devices that provide directive feedback in compression rate, depth, release, and hand position during training may be considered to improve the level of skill acquisition by the end of course.[61,74,76,133–137] In their absence, tonal guidance (e.g. music or metronome) during training may improve compression rates only. There is evidence that tonal guidance can reduce compression depth as the candidate focuses on the rate.[137–139] CPR prompt or feedback devices improve CPR skill acquisition and retention in BLS and might also be used to improve proper application of these basic CPR skills during advanced level training. However, the use of CPR feedback or prompt devices during CPR should only be considered as part of a broader system of care that should include comprehensive CPR quality improvement initiatives,[140] rather than as an isolated intervention.

In-situ simulations can offer opportunities to train the full team [141] as well as provide insight into the work flow on the organisational level.[142] Furthermore it might be easier to include training of a full team of care providers across disciplines in-situ and this can improve advanced life support provider knowledge,[143] skill performance,[144] confidence and preparedness,[141] familiarity with the environment[145] and identify common system and user errors.[142,146,147]

Briefing and debriefing after cardiac arrest simulation

Debriefing after cardiac arrest simulation is an essential part of the learning process. If the simulated scenario training is followed by debriefing then learning will occur, as opposed to scenario training without debriefing.[148] The ideal format of debriefing has yet to be determined. Studies have failed to show a difference with and without the use of video clips for debriefing.[149,150]

Implementation and change management

The formula for survival concludes with 'Local Implementation'.[2] The combination of medical science and educational efficiency is not sufficient to improve survival if there is poor or absent implementation. Frequently, this implementation will also require some form of change management to embed new visions into a local culture. Quite often, the 'easy fix' will not be the sustainable solution and prolonged negotiation and diplomacy may be needed. A prime example of this is the implementation of CPR training on the school curriculum–countries that have achieved this goal have sometimes spent years campaigning and persuading governments for this change to be adopted. Change can be driven from below, but to be sustainable it usually needs top down buy-in as well.

This section was not present in the 2010 ERC Guidelines and has been added in recognition of its importance in the quest to improve survival.

Impact of guidelines

In each country, implementation is largely based on the internationally agreed guidelines for cardiac resuscitation. National strategies for education are dependent upon evidence-based solutions to the management of cardiac arrest. The most important question, therefore, should be whether these guidelines actually result in any meaningful and improved outcomes. The authors freely acknowledge a conflict of interest here—if we prove that our guidelines have no tangible benefit then we call into question the resources that have been invested to generate them. The evidence suggests a positive benefit when considering survival to hospital discharge,[8,151–156] return of spontaneous circulation,[8,151–155] and CPR performance.[8,153] Irrespective, the likelihood of benefit is high relative to possible harm.

Cardiac arrest centres

In the last few years, regional healthcare systems have emerged for the management of conditions like stroke, major trauma, and myocardial infarction. These have mainly been driven by centralisation of limited resources as opposed to evidence of benefit from randomised trials. There is emerging evidence that the transport of patients with out-of-hospital cardiac arrest to a specialised cardiac arrest centre may be associated with improved neurologically intact survival.[157–170] The studies currently available had inconsistencies in terms of the specific factors that allegedly contributed to better outcomes. More research needs to be performed to identify the specific aspects of a cardiac arrest centre that improve outcome, as well as the influence of journey times and whether secondary transfers to such centres could also obtain the same benefit.

Scenario-based simulation training and re-training, regular practice and a team approach to device placement are necessary for coronary catheterisation laboratory personnel. When introducing

mechanical chest compression devices into clinical practice a significant learning curve was observed.[171] During prolonged resuscitation efforts in the coronary catheterisation laboratory, the implementation of a structured resuscitation approach improved teamwork.[172]

Use of technology and social media

The prevalence of smartphones and tablet devices has led to the generation of numerous approaches to implementation through the use of 'apps' and also social media. These fall into several categories:

(1) Simple delivery of information–apps that display resuscitation algorithms.
(2) Interactive delivery of information–apps that use the geolocation of the user to display the location of the nearest AED.
(3) Interactive delivery of education–apps that engage with the user and create an immersive and interactive means of educating the user (e.g. Lifesaver) [www.life-saver.org.uk].
(4) Blended learning packages for life support courses–an e-learning programme with abbreviated instructor-led training has been shown to be equivalent to standard training for advanced life support courses.[85]
(5) Feedback devices—real time use of the accelerometer to improve rate, depth of compressions as well as recording data for debriefing.[173]
(6) Notification and activation of bystander schemes—if individuals are willing and able to provide basic life support in a community, the use of these systems may lead to faster response times when compared with emergency service attendance.[174,175]
(7) Use of social media to disseminate information to a wider audience and assist with campaigns to effect change.

Ultimately, technology and social media are powerful vectors for implementation and change management. Their development and use should be encouraged and analysed to assess the actual impact on survival.

Measuring performance of resuscitation systems

As systems evolve to improve the outcomes from cardiac arrest, we need to accurately assess their impact. This is particularly important for larger systems with multi-factorial components any of which may be beneficial either in isolation or combination. For example, it has already been shown that further work needs to be done to evaluate the impact of cardiac arrest centres.

Measuring performance and implementing quality improvement initiatives will further enhance systems to deliver optimal results.[102,176–181]

Debriefing after resuscitation in the clinical setting

Feedback to members of an in-hospital cardiac arrest team about their performance in an actual cardiac arrest (as opposed to the training environment) can lead to improved outcomes. This can either be real-time and data-driven (e.g. use of feedback devices on cardiac compression metrics) or in a structured post event performance focused debrief.[102,182] The ideal approach to debriefing is yet to be determined, including the interval between actual performance and the debriefing event. Although it seems intuitive to provide this level of debriefing for out-of-hospital cardiac arrest performance, no evidence exists to support or refute its benefit.

Medical emergency teams for adults

When considering the chain of survival for cardiac arrest,[1] the first link is the early recognition of the deteriorating patient and prevention of cardiac arrest. A considerable amount of work has been done to evaluate the role of the Medical Emergency Team (MET) in this respect. We recommend their use and, in particular, the use of higher intensity systems (e.g. higher MET calling rates, senior medical staff on the team) as their use has been associated with a reduced incidence of cardiac/respiratory arrest[183–189] and improved survival rates.[184,186–189,183,190]

It is recommended that these systems include:

(1) staff education about the signs of patient deterioration
(2) appropriate and regular vital signs monitoring of patients
(3) clear guidance (e.g. via calling criteria or early warning scores) to assist staff in the early detection of patient deterioration
(4) a clear uniform system of calling for assistance
(5) a clinical response to calls for assistance.

Training in resource limited settings

There are many different techniques for teaching ALS and BLS in resource limited settings. These include simulation, multimedia learning, self-directed learning, limited instruction, and self-directed computer-based learning. Some of these techniques are less expensive and require less instructor resources than a traditional teaching format. Some techniques also enable wider dissemination of ALS and BLS training. It is reasonable to suggest the use of these strategies in resource limited settings, although the optimal strategy is yet to be determined and will differ from one country to another.[191–197]

Training in ethics and first aid

Insights into training health care professionals about DNAR issues and approaches to practicing procedures on the newly deceased are provided in the Ethics chapter of the ERC guidelines 2015.[198] The First Aid chapter of the 2015 ERC Guidelines provides guidelines about first aid education and training programs as well as public health campaigns.[199]

The ERC resuscitation course program

The ERC has developed a wide range of courses targeting all levels of providers, from basic life support for lay rescuers to advanced life support for health care providers. ERC courses teach the competences to undertake resuscitation in the clinical setting at the level that they would be expected to perform. Besides resuscitation skills, emphasis is given to non-technical skills and leadership training, application of ethical principles and advanced educational strategies as well as organisational improvements on a system level to improve survival after cardiac arrest. Specific courses teach these competences whilst others train how competences are to be taught.

ERC courses focus on teaching in small groups with a high instructor to candidate ratio using blended learning strategies, including interactive discussion, workshops and hands-on practice for skills and simulations using resuscitation manikins.[200,201]

Up-to-date information about ERC courses is available in the "ERC course rules" on the ERC website [https://www.erc.edu/index.php/doclibrary/en/]. The course rules describe in detail the ERC terminology and definitions; specifics of the organisation and management of different ERC course formats and quality control; the instructor development up to course director, instructor trainer and ERC educator; the ERC assessment and certification/recertification

process; and the ERC professional behavioural guides including complaints procedures.

Ethos

Instructors on ERC courses are trained in teaching and assessment. The ethos is to create a supportive, learner-centred environment that promotes learning, enhancing understanding of knowledge and retention of skills. First names are encouraged among both faculty and candidates to reduce apprehension. Interactions between faculty and candidates are driven to learn from each other's experiences. Aimed changes in behaviour are elaborated by encouragement with constructive and corrective feedback as well as debriefing on performance. A mentor/mentee system is used to enhance feedback and support for the candidate. Some stress is inevitable,[202] particularly during assessment, but instructors aim to enable the candidates to do their best. ERC courses are driven by the ultimate goal to improve resuscitation performance to increase survival of cardiac arrest victims.

Course management

ERC courses are overseen by the Joint International Course Committee (JICC) consisting of the chairpersons of the International Course Committees (ICC) for all ERC-course types (BLS/AED, Immediate Life Support (ILS), ALS, Neonatal Life Support (NLS), European Paediatric Immediate Life Support/European Paediatric Advanced Live Support (EPILS/EPALS), Generic Instructor Course (GIC)) and is led by the Board Director for Training and Education (DTE). On the national level, each National Resuscitation Council (NRC) assigns National Course Directors (NCD) for each course type.

The ERC has developed a web-based course management system [http://courses.erc.edu] for the administration of these courses. Candidates may sign up online to a course, or may contact the course organiser to register their interest in a specific course. At the end of the course the system will generate unique numbered course certificates for successful candidates and also each faculty member. For quality control an evaluation tool is available for each course and results are accessible for NRCs, NCDs and ICC members. Participants who successfully complete provider courses are referred to as 'providers'.

Language

Initially, the ERC courses were taught in English by an international faculty. As local instructors have been trained, and manuals and course materials have been translated into different languages, many NRCs are now able to deliver their courses locally in their native language. It is important that this does not compromise the quality control of courses and instructor development and the process of translation of new guidelines and course materials should not delay the implementation of new guidelines.[5]

Instructor development

Individuals who have passed and demonstrated a high level of performance during a provider course and, importantly, have shown qualities of leadership and team working, shown clinical credibility, with skills that include being articulate, supportive, and motivated may be identified by the course faculty as Instructor Potential (IP). Individuals with IP in any advanced course will be invited to take the ERC Generic Instructor Course (GIC). IPs after BLS/AED courses will be invited to take the BLS/AED instructor Course.

At the GIC, an ERC educator who has undertaken specific training in medical education and in the principles of adult learning (ERC Educator Master Class), is responsible for delivering the educational principles of ERC courses.

From the instructor candidate (IC) stage to full instructor (FI)

Following successful completion of a GIC, IPs are granted IC status and normally will teach on two provider courses, under supervision of the course faculty, receiving constructive and corrective feedback on his or her performance with the aim of being promoted to FI status. This feedback enhances teaching practice during the GIC and as an IC in the first provider courses by formulating learning goals for subsequent courses.

Course director (CD) status

An approved Course Director leads each ERC course. CDs are proposed by NCDs and approved by their NRC or the respective ICC. CDs are senior instructors who are clinically credible, have demonstrated excellent qualities as a teacher, mentor, and assessor, and possess the skills to lead a faculty of instructors.

General ERC course principles [ERC course rules on www.erc.edu]

Content of ERC courses

All ERC courses follow contemporary ERC guidelines. Each course has its specific course manual or teaching booklet providing the required pre-course knowledge. Candidates receive the manual in advance to prepare for each course with a mandatory pre-course MCQ (except for BLS/AED, ILS and EPILS) that aims to ensure that candidates read the materials before attending the course.

All ERC courses comprise interactive lecture and group discussions, small group workshops, hands-on skills teaching and, for advanced level training, clinically orientated Cardiac Arrest Simulation (CAS) and emergency case scenarios. Most course formats include options enabling instructors to tailor their teaching to the candidates' local needs.

Immediate and advanced life support courses

Immediate and advanced life support courses target the training of healthcare providers. Curricula have core content and can be tailored to match individual learning needs, patient case mix and the individual's role within the healthcare systems response to cardiac arrest. Core modules for these courses include:

- Cardiac arrest prevention.[203,204]
- High quality chest compressions (adherence to rate, depth, full recoil and minimizing hands-off time) and ventilation using basic skills (e.g. pocket mask, bag mask).
- Defibrillation, with charging during compressions for hands-free defibrillation.
- Advanced life support algorithms and cardiac arrest drugs.
- Non-technical skills (e.g. leadership and team training, communication).

Immediate life support courses. ILS courses for adults and EPILS courses for children are one-day courses focusing on the causes and prevention of cardiac arrest, the ABCDE approach to the critically ill patient, starting effective BLS/AED, initiating the chain of survival, and basic CPR skills (e.g. effective chest compression and safe delivery of a defibrillation shock, basic airway management, choking, intravenous or intra-osseous access, and drugs during cardiac arrest).[205] These courses are designed to be simple to run with small groups of candidates. The aim is to train candidates in the use of the equipment (e.g. defibrillator type) that is available in their clinical setting and the management of the first minutes of cardiac arrest until professional rescuers arrive.

Advanced life support courses. ALS courses for adults, EPALS for neonates and children, and NLS courses for newborns build upon the knowledge and skills from the respective Basic and/or Immediate Life Support courses. This provides the foundation for these 2-day advanced courses placing emphasis on safe defibrillation and ECG interpretation, the management of the airway, ventilation and vascular access, the management of peri-arrest rhythms, and special circumstances relating to severe illness, injury, and cardiac arrest. Post-resuscitation care, ethical aspects related to resuscitation and care of the bereaved are also included. These courses should enable providers to cover the first hour of critical illness or injury and cardiac arrest. They are not designed to provide instruction in advanced intensive care or cardiology.

The faculty meeting

The faculty meeting usually takes place at the start and at the end of each course day and is led by the course director. The aim is to brief the teaching faculty and to assess the performance and progress of each candidate. During the final faculty meeting each candidate's performance is reviewed to make a decision about successful course participation and whether candidates who have met the required criteria are offered instructor potential status. Instructor candidates on the courses are also assessed on their performance. Faculty meetings also provide an opportunity to debrief the faculty at the end of the course.

Assessment and feedback

Throughout the course, the faculty assesses each candidate formatively and individually. Candidates' performances and attitudes are discussed at the daily faculty meetings, with mentoring and feedback given as required. Instructors are taught to use a framework aimed at providing timely, constructive, goal orientated, student centred and action planned feedback to enable the learner to achieve the desired outcome.

The standard ERC feedback format is the Learning Conversation. The learning conversation starts with an invitation to reflect and it is primarily centred on any issue that the candidate wishes to discuss. This is followed by a discussion of any key areas that the instructor wishes to discuss, along with contributions from the group and other instructors. Any important performance issues are then summarised with specific action points for the candidate to improve their further performance.

Candidates' performances are continuously assessed throughout BLS, ILS, and GIC courses, measuring their competences against pre-determined criteria; no summative tests are required to be certified.

Towards the end of NLS and ALS courses a Cardiac Arrest Simulation Test (CAST) assesses the candidates' applied knowledge and skills during a simulated cardiac arrest including leading a cardiac arrest team. The reliability and measurement properties of CAST have been established.[121,206,207] Their core knowledge is assessed with an MCQ.

Mentoring

Mentoring is an essential part of all ERC courses and enables candidates to have a nominated role model. Group or 1:1 mentoring happens during ERC courses on a regular basis.

Specific formats of ERC resuscitation courses

Basic life support and automated external defibrillation (BLS/AED) provider courses and BLS/AED instructor course. BLS/AED courses are appropriate for all citizens including lay persons and trained first responders (first-aid workers, lifeguards), those with a duty of care for others (e.g. school teachers, care workers, security personnel) and ultimately all clinical and non-clinical healthcare professionals (including EMS systems dispatchers, general practitioners, dentists, medical and nursing students, and those who are less likely to manage a cardiac arrest). Combined BLS/AED courses are encouraged.

BLS/AED courses aim to enable each candidate to gain competency in recognising a cardiac arrest, immediate instigation of effective chest compression, calling appropriate help to the scene and safe use of an AED. These courses teach children and adults in CPR competences for children and adults in cardiac arrest.

The ERC BLS/AED instructor course offers candidates who hold a valid BLS/AED certificate and who are identified as instructor potential the opportunity to train to be BLS/AED instructors.

Immediate life support (ILS) course. The ILS course teaches the majority of healthcare professionals from all disciplines and professions who face adult cardiac arrests rarely but are potential first responders or resuscitation team members.[208] Applied ILS competences should result in successful resuscitation whilst awaiting the arrival of the resuscitation team covering the first minutes of CPR.[209] In a cohort study after implementation of an ILS-programme the number of cardiac arrest calls and true arrests decreased while pre-arrest calls increased as well as initial survival and survival to discharge.[210]

Advanced life support (ALS) course. The target candidates for the ALS course are physicians, nurses, EMS personnel, and selected hospital technicians who may be resuscitation team leaders and members for adult CPR.[211,212]

Beyond the expected BLS and ILS competences to be mastered by the candidates, this course format teaches the management of cardiac arrest from a diversity of causes and the management of peri-arrest problems and concentrates on the application of non-technical skills with emphasis on team-cooperation under clear team leadership.

Newborn life support (NLS) course. This one-day inter-professional course aims to give healthcare workers likely to be present at the birth of babies (e.g. midwives,[213] nurses, EMS personnel, physicians) the background knowledge and skills to approach the management and resuscitation of the newly born during the first 10-20 min. NLS places appropriate emphasis on airway management, chest compression, umbilical venous access and drugs for newborn CPR.[214]

European paediatric immediate life support (EPILS) course. EPILS is a one-day course (5 to 8 h) that trains nurses, EMS personnel, and doctors who are not part of a paediatric resuscitation team to recognise and treat critically-ill infants and children, to prevent cardiorespiratory arrest and to treat children in cardiorespiratory arrest during the first few minutes whilst awaiting the arrival of a resuscitation team. Short practical simulations adapted to the workplace and to the actual clinical role of candidates are used to teach the core competencies.

European paediatric advanced life support (EPALS) course. EPALS is designed for healthcare workers who are involved in the resuscitation of newborns, infants or children providing sufficient competences to manage critically ill or injured children during the first hour of illness.[215–218] Refresher training in paediatric basic life support and relief of foreign body airway obstruction is included.

EPALS puts great emphasis on the recognition and continuous assessment and timely treatment of the sick child (e.g. cardiac and respiratory failure, arrest and trauma simulations). Aspects of team working and team leadership are integrated in the training, including problem anticipation and situational awareness. Depending on local needs and circumstances EPALS may further include modules on newborn resuscitation, post-arrest care and handover, and/or

modules on more advanced knowledge or technical skills. These latter modules are being continuously developed.

Generic instructor course (GIC). The GIC is for candidates who have been recommended as instructor potential (IP) emanating from any ERC provider courses (except the BLS/AED course that has a separate instructor course) or with IP status from certain other provider courses (e.g. European Trauma Course). The GIC puts emphasis on developing teaching and constructive and corrective feedback and mentoring. Core knowledge of the original provider course is assumed.

An ERC educator leads the educational process, the discussions and provides critical feedback. The Educator delivers interactive sessions covering the theory of adult learning, effective teaching of skills and simulated scenarios, assessment and effective feedback, and leadership and non-technical skills through a series of interactive sessions. The faculty demonstrates each of these competencies, followed by opportunities for the candidates to practise.

Abbreviated material from the original provider course is used for the simulated teaching sessions. The GIC emphasises the concept of constructive and corrective feedback to develop future learning strategies thus providing an opportunity for each candidate to adopt the instructor role.

Educator master class (EMC). ERC educators are an essential mandatory component of the GIC faculty. A two-day educator master class teaches experienced provider course instructors with a demonstrable interest in education to become ERC educators. NRCs propose suitable candidates who are then shortlisted by the ERC Working Group on Education based on specific criteria (including motivation, qualification in medical education or documentation of demonstrated special commitment to educational practice over a number of years within the ERC).

EMC instructors are experienced educators assigned by the Working Group on Education and the Director of Training and Education. The EMC covers the theoretical framework for ERC educators, assessment and quality control, teaching methodologies, critical appraisal, the mentor role, multi-professional education strategies and continuous development of the ERC teaching faculty. The format of the EMC is a series of closed discussions, small breakout groups and problem solving sessions. Candidates are formatively assessed throughout the EMC.

European resuscitation academy (ERA)—"It takes a system to save a life"

The ERA aims to improve survival from cardiac arrest through a focus on healthcare system improvements that bring the individual links in the Chain of Survival and the Formula for Survival together. Entire EMS staff (managers, administrative and medical directors, physicians, EMTs and dispatchers) from different health care systems and countries are invited to learn from the ERA Program (derived from the Seattle (US) based Resuscitation Academy [http://www.resuscitationacademy.com/] ten steps for improving cardiac arrest survival) together with the local host health institutions. The ERA puts emphasis on defining the local cardiac arrest survival rate by understanding the importance of reporting data in a standardised Utstein template. Participating EMS systems are encouraged to develop concrete measures to improve cardiac arrest survival followed by appropriate measurements of these action plans.

Future direction for research and course development

The production of international guidelines for resuscitation is a constantly evolving exercise. High quality research continues to be published with evidence that may or may not suggest that the guidelines of today are acceptable.

In parallel with this, the science of education also continues to evolve. Our methods for teaching these guidelines have changed substantially over the years from the early days of didactic theoretical delivery of teaching to contemporary interactive, hands-on methods that also utilise technology and social media.

There is still a paucity of high quality evidence about the best methods of teaching, primarily because the numbers of candidates needed to produce statistical significance for meaningful outcomes (e.g. increase in patient survival) would need to be massive. There is a role therefore for international collaboration to achieve such numbers in a similar style to the collaborations used to assess some of the clinical content to the guidelines. Until the time that statistical significance is achieved, it is essential that we continue to evaluate our educational methods and assess the *educational* importance or relevance of the findings.

New insights about educational process, neuro-science impact on training and rapid developments in social media and online applications mean that our approach to education is constantly changing. This chapter highlights current changes and what may change in the near future.

Recommendations for educational research in resuscitation

Every educational intervention should be evaluated to ensure that it reliably achieves the learning objectives and at its best improves patient outcome in a cardiac arrest situation. The aim is to ensure that learners not only acquire skills and knowledge but also retain them to be able to provide adequate actions depending on the level of training. Evaluation at the level of patient outcome is difficult to achieve, as several other parameters influence patient outcome, such as changes in guidelines, changes in case-mix, and organisational changes. The level of outcome studied, should be determined during the planning phase of the educational event.[219] It is difficult to assess behaviour in the clinical setting so this attribute is more commonly assessed with simulation using manikins. Generalisability from manikin studies is questionable, though, and that is the reason why so little high-level evidence is found in the literature.

Education in resuscitation is still a relatively new field lacking high quality research. Studies are heterogenous in design and prone to risk of bias and therefore difficult to compare. A research compass to guide future studies in education has been devised at a research summit.[220]

Future course development

The educational strategy of the ERC is based on uniform instructor courses and standardised provider course curricula. This will evolve as more blended learning methods become available. Flexibility is needed in teaching CPR on all levels as different media like DVD, Internet and on-line training increase the learning benefit.

New curricula should allow this flexibility. Some core-content modules will be the 'heart' of any ERC-course which will allow the customisation of each course format with additional optional content (medical as well as non-technical aspects) to support and train learners according to local needs. Some institutions will, for some learners, have very specialised modules (e.g. cardiac arrest after cardiac surgery, advanced neonatal support at an ICU, obstetric resuscitation, resuscitation during surgery in the operation room) that can be added to the standard core-content of the course.

New teaching technology (IT-based learning like webinars, e-learning modules on the ERC virtual learning environment) will be adopted and this needs to be addressed in the GIC as well as in the supervision and mentoring of all instructors, course directors and educators.

Learners using video- or online training may no longer need a printed manual, as they will have immediate access to the content on the Internet. This will provide substantially more opportunity to integrate pictures, demonstration videos of skills and team performance, self-assessment tests with guidance of how to improve, and linked literature to deepen interests. A virtual learning environment (VLE) will furthermore monitor and support the ongoing learning trajectory of each individual in terms of knowledge, skills, attitudes and global performance from providers to instructors as well as course organisers.

Reading and learning knowledge-based facts, thinking through procedures and action strategies, and discussing open questions can all be done before candidates come to the course venue. Highly motivated course participants will come to the course centre with a high level of knowledge, a clear vision when to apply which procedures and how to interact with a team to perform quality CPR. Due to increasing constraints on study and teaching leave, the time spent at the course centre needs to be focused on the translation of the learned concepts in the simulated scenarios. This will enable candidates to try out, rehearse and execute life-saving techniques, using best medical practice and team leadership and management. This should ultimately enable providers to increase survival after cardiac arrest in the clinical setting.

High frequency training will be very short and might not necessarily need personal coaching by an instructor or mentor. The training environment should be brought to the learners, so that they can experience it during daily activities to reach the high frequency objective. A brief annual CPR competence test may be used to filter out those who do not achieve institutionally defined levels of competence. Some might need brief training under supervision to reach competence, whereas others may need a longer formal refresher process. Course organisers have to plan their courses in a flexible way, allowing a shorter duration for target groups with extra background, and more hands-on time for lay rescuers.

The use of high fidelity manikins and advanced feedback devices will be available for countries and organisations with the financial capacity, but not for all countries and organisations. When using low fidelity manikins, instructors need to be trained to deliver timely and valid feedback to the learner to increase their learning.

Ultimately, the goal of the ERC is to strengthen each component of the Chain of Survival through effective education and implementation. The aim should be to develop teaching strategies for lay people and healthcare professionals to deliver high quality BLS, swift defibrillation, effective advanced resuscitation, and high quality post resuscitation care. These strategies should be easy, accessible, well validated, and appealing. This will ensure that the scientific guidelines can effectively translate into improved survival rates.

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Conflicts of interest

Robert Greif	Editor for Trends in Anesthesia and Critical Care.
Andrew S. Lockey	Medical Advisor "First on Scene First Aid Company".
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European Resuscitation Council Guidelines for Resuscitation 2015 Section 11. The ethics of resuscitation and end-of-life decisions

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Summary of changes since the ERC 2010 guidelines

The traditional medical-centred approach with an emphasis on beneficence has shifted towards a balanced patient-centred approach with greater emphasis on patient autonomy. This has resulted in a readiness for understanding and interaction between patient and healthcare professionals. Future guidelines may benefit from involvement of all stakeholders: members of the public, patients, survivors and the society as active partners in understanding and implementing the ethical principles.

The content and implementation of the traditional ethical principles are placed in the context of a patient-centred approach to resuscitation:

- Autonomy, including respect for personal preferences expressed in advance directives, which implies correct information and communication.
- Beneficence, including prognostication, when to start, futility, ongoing CPR during transportation, special situations, with clear distinction between sudden cardiac arrest and expected cessation of cardiac function and respiration in terminal situations.
- Non-maleficence, including DNAR/DNACPR, when to stop/withhold and involvement of patient or proxy.
- Justice and equal access, including avoiding inequalities.

Whilst the sad reality is that the majority of those that sustain a cardiac arrest do not survive, recent studies provide evidence of steady improvement in outcomes particularly where the formula of survival is well implemented. Specific cases of refractory cardiac arrest, which would historically have been fatal, may benefit from additional interventional approaches. A further improvement in survival may be expected by applying clear guidance for starting, not starting, withdrawing or withholding resuscitation attempts, and by identifying refractory cases that may respond to advanced interventions.

Europe is a patchwork of 47 countries (Council of Europe) with differences in national laws, jurisdiction, culture, religion, and economic capabilities. European countries interpret the ethical recommendations of resuscitation in the context of these factors.

A survey of current ethical practice across Europe was conducted in the context of these guidelines. A significant variability in the approach to cardiopulmonary resuscitation (CPR) and end-of-life was documented. Whilst areas for improvement were identified, it highlighted a trend towards better application of ethical principles.

The need for harmonisation in legislation, jurisdiction, terminology and practice remains. The mission of the ERC and its Guidelines is to contribute to this harmonisation.

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New European Union (EU) regulation permitting deferred consent will harmonise and foster research of emergency interventions across EU Member States.

Healthcare professionals are responsible for maintaining their knowledge, understanding and skills, and to understand the ethical principles before being involved in a real situation where resuscitation decisions must be made.

Introduction

Sudden unexpected cardiac arrest (CA) is a catastrophic unexpected but potentially reversible event that involves family, friends and society. In Europe cardiac arrest occurs in 0.5–1.0 per 1000 inhabitants per year. Although a slow improvement has been observed over recent years, survival after out-of-hospital Cardiac Arrest (OHCA) remains low with an average survival to hospital discharge of 7.6%.[1–9]

Potentially reversible sudden unexpected cardiac arrest should be distinguished from the expected cessation of circulation and respiration in a terminal condition. Better medical knowledge, new and advanced interventions, and increasing expectations of the public have rendered ethical considerations an important part of any end-of-life intervention or decision. This includes optimising results for individual patients and society by appropriate allocation of resources.

In recent years there has been a shift from a doctor-centred approach with emphasis on beneficence, towards a patient-centred approach with greater emphasis on patient autonomy. This change is reflected in the 2015 ERC ethics guidelines for resuscitation and end-of-life decisions.

This chapter provides information and guidance on the principles of ethics: ethical and professional guidance for healthcare professionals responsible for providing resuscitation including when to start and when to stop resuscitation and special considerations required for children and for organ donation after an unsuccessful resuscitation attempt.

The healthcare professional should understand the ethical principles before being involved in a real situation where resuscitation decisions must be made.

We also report the initial findings from a European survey on Ethical Practices, which documented significant variation between countries in the approach to cardiopulmonary resuscitation (CPR) and end-of-life practices.

There is a clear need for harmonisation in legislation, terminology and practice. The mission of the ERC Guidelines is to contribute to this harmonisation.

Aspects of ethics for resuscitation and end-of-life decisions

Ethics is defined as the ways of examining and understanding the moral life, or the application of ethical reasoning to medical decision making. The key principles of medical ethics are: autonomy of the individual, beneficence, non-maleficence and justice. Dignity and honesty are frequently added as essential elements of ethics. [11–13]

The principle of patient autonomy

Respect for autonomy refers to a physician's obligation to respect a patient's preferences and to make decisions that accord with a patient's values and beliefs. Patient-centred healthcare places the patient at the centre of the decision-making process, rather than as a recipient of a medical decision. This requires patients to have an adequate understanding of relevant issues regarding their treatment options, thus enabling them to make informed decisions or participate in shared decision-making. Patient education has contributed significantly to this change in emphasis. The principle of autonomy is implemented through free and informed consent, and recognises that the person may change their decision at any time. Applying this principle during cardiac arrest where the patient is often unable to communicate preferences is challenging.[11,14–16] Moreover, the legally documented wishes of an individual patient may not be readily available, causing further ethical dilemma: how can healthcare professionals embrace patient-centricity when the views of the patient are unknown?[11,17–19]

The principle of beneficence

Beneficence implies that interventions must benefit the patient after assessing relevant risk and benefit. Evidence-based clinical guidelines exist to assist healthcare professionals in deciding which treatment approaches are most appropriate.[20–22] Increasingly, patients are involved as active partners in the guideline development process, ensuring that patient's views and perspective are captured in the guidance provided.[23] Such involvement, however, has not yet been witnessed in the context of resuscitation guidelines.

The principle of non-maleficence

Non-maleficence or 'primum non nocere' stems from the Hippocratic axiom 'help or at least do no harm'. CPR should not be performed in futile cases. However, it is difficult to define futility in a way that is precise, prospective and applies to the majority of cases. CPR is an invasive procedure with a low likelihood of success. Advance directives are rarely available to emergency healthcare professionals. Therefore, CPR has become the norm for most patients with acute, life-threatening conditions.[24,25]

The principle of justice and equitable access

Justice implies that health resources are distributed equally and fairly, irrespective of the patient's social status, in the absence of discrimination, with the right for each individual to receive the current standard of care. The appropriate allocation of resources has become an important consideration for invasive procedures. CPR is a procedure requiring coordinated efforts of many healthcare professionals. The ethical considerations regarding CPR and end-of-life decisions include achieving the best results for the individual patient, for relatives and for society as a whole by appropriate allocation of available resources. There is no consensus about what constitutes a just and fair method of balancing the preferences and requirements of individual patients against the diverse needs of society.[11,13,19,21,26]

Withholding specific medical care due to financial motives is not acceptable but it may be appropriate to consider the overall costs and potential benefits to the individual patient, the family and society.[13,21,27–29]

There is evidence that citizens from lower socioeconomic groups have both an increased incidence and lower chance of survival of OHCA. The likelihood of a person receiving bystander CPR after a cardiac arrest is nearly five times greater in higher income neighbourhoods compared with lower income ones. Caucasian patients are more likely to receive bystander CPR than other ethnic groups.[2,30–39]

Medical futility

The World Medical Association (WMA) defines futile medical treatment as a treatment that "offers no reasonable hope of recovery or improvement" or from which "the patient is permanently unable to experience any benefit". Resuscitation is considered futile when the chances of good quality survival are minimal.[40] The first prerequisite to consider a treatment futile is the presence or absence of a medical indication. The decision not to attempt resuscitation does not require the consent of patients or of those close

to them, who often have unrealistic expectations about the likely success and potential benefits of resuscitation.[41,42] Starting a futile treatment may offer false hope to the family and patient that may undermine the patient's ability for rational judgment and autonomy. [40,43] However, decision makers have a duty to consult the patient or a representative if the patient lacks capacity, in accordance with a "clear and accessible policy".[44–46] The medical team must explain that the decision not to attempt resuscitation does not mean giving up or that the patient will be ignored or abandoned, but rather that the intent is to protect the patient from harm and to maximise comfort and quality of life.[44,47]

Some countries allow prospective decisions to withhold CPR whilst in others countries or religions withholding CPR is not allowed or considered illegal. There is a lack of consistency in terms such as 'Do Not Attempt Resuscitation' (DNAR), 'Do Not Attempt Cardiopulmonary Resuscitation' (DNACPR) or 'Allow Natural Death' (AND). This confusing use of acronyms may generate misunderstandings in national legislation and jurisdiction [48,49]

Advance directives

Advance directives are decisions about treatment provided prospectively by an individual in case they are unable to participate directly in medical decision-making at some point in the future.[50] Advance directives can take two different but not mutually exclusive forms: (1) 'Living Wills' are written documents that express a person's preferences regarding the provision or the withholding of specified treatments in the event that they become unable to make decisions in the future; and (2) a 'Lasting power of attorney for health care' allows individuals to appoint a proxy (e.g., a trusted relative or friend) who can make health care decisions on their behalf in case they lose decision-making capacity.[51]

The advance directives must meet three criteria: existence, validity and applicability. Physicians must not delay resuscitation interventions while trying to establish if an advance directive prohibiting CPR exists.[51] Neither must CPR be attempted if it is considered more harmful than helpful, even if contrary to a valid and applicable advance decision.

In several countries advance directives have the same legal force as contemporaneous decisions. However, their applicability is complicated by the challenge of drafting a directive that accurately represents a patient's wishes at the time of writing.[52] Indeed, people often adapt to disabilities, and preferences may change over time. Therefore, periodic reviews of directives are required to ensure patients' current wishes and circumstances are accurately reflected.[41,52,53]

Article 9 of the Convention on Human Rights and Biomedicine requires physicians to "take into account" previously expressed wishes of their patients.[19] However, the legal status of advance directives in the national legislation of European countries is very disparate. Several countries have adopted specific laws assigning binding force to advance directives about end of life decisions, including resuscitation.[51]

Human Rights relevant to resuscitation and end-of-life decisions

Policies about resuscitation and individual decisions of healthcare professionals must comply with human rights. Provisions relevant to decisions about attempting CPR include the following rights: to life; to protection from inhuman or degrading treatment; to respect for privacy and family life; to freedom of expression, which includes the right to hold opinions and to receive information; and to be free from discriminatory practice in respect of these rights.[19] Failing to involve a patient at the time of writing a DNAR order breaches Article 8 of the European Convention of Human Rights.[45]

Patient-centred care

The increasing centricity of the patient within healthcare demands that we seek to understand the perspective of the survivor of cardiac arrest, with assessment seeking to be inclusive of clinical and patient-reported outcomes over the short and longer-term. This has been recognised within the updated Utstein Resuscitation Registry template for out-of-hospital cardiac arrest, which recommends the assessment of patient-reported outcomes and the quality of life of survivors.[54] However, specific assessment guidance does not currently exist. The COSCA (Core Outcome Set—Cardiac Arrest) initiative will seek international consensus on what should be measured and when in all clinical trials of cardiac arrest, and make recommendations on both clinical and patient-reported outcomes.[55,56] Such guidance may also inform patient-centred outcome assessment in routine practice and registries, informing more targeted treatment and allocation of resources for survivors of cardiac arrest.[54–58]

Ethically, we cannot ignore the patient perspective. However, ensuring that patient-centred outcomes are captured to the best effect requires an improved understanding of what matters, for whom, in what context and when: this requires a further commitment to work together with the public, with the survivors of cardiac arrest and their families as partners in this process.[59]

Practical implications for in- and out-of-hospital cardiac arrest

Outcome from sudden cardiac arrest

Resuscitation attempts are unsuccessful in 70–98% of cases. In pre-hospital systems with a well-organised implementation of the elements of the 'formula of survival' [20] about 1/3–1/2 of patients may achieve return of spontaneous circulation (ROSC) with CPR, with a smaller proportion surviving to the hospital critical care unit. Smaller proportions still survive to hospital discharge with good neurological outcome.[8]

The best resuscitation outcome is for an individual to be cognitively unimpaired and with an acceptable quality of life, or to report no significant deterioration when compared to the pre-morbid state.

However, studies have reported cognitive impairment in up to 50% of survivors.[9,60,61] Moreover, where acceptable levels of quality of life have been reported, this has been assessed using generic, preference-based utility measures such as the EuroQoL EQ-5D or Health Utility Index, or generic health status measures such as the Short Form 12-item Health Survey (SF-12).[57,62,63] Whilst providing a broad overview of health status and a useful comparator with the general population, generic measures cannot capture the complexities of specific conditions and it is unclear if they accurately assess the outcomes that really matter to the CA survivors.[55] Consequently, they may underestimate the health needs and experiences of survivors, and are often less responsive to important changes in recovery than well-developed condition or domain specific measures.[55]

Early adequate CPR may increase survival beyond 50%.[64,65] Substantial variation in survival is seen between communities.[66–69] Real improvements in global outcome will require a community-centred 'public health' approach.[8,70] Policy-level executives need to become aware of their crucial role in this.

In-hospital cardiac arrest (IHCA)

Following in-hospital cardiac arrest, the default position is to start resuscitation unless a decision was made to withhold CPR.

Decisions to withhold resuscitation are usually taken by a senior physician in collaboration with members of the multi-professional team.[71] Resuscitation decisions should be reviewed following an emergency admission to hospital, after any important changes in patient status / prognosis, following a request from the patient or their relatives, and prior to discharge / transfer to another facility.[72]Standardised systems to withhold resuscitation decrease the incidence of futile resuscitation attempts.[72] Instructions should be specific, detailed, and transferable across health care settings, and easily understood.[73,74] There may be occasions where a clinician decides it is necessary to override a prior decision to withhold CPR. Such circumstances might include a sudden arrest due to a readily reversible cause (e.g., choking, blocked tracheal tube) or where a patient is undergoing a specific procedure or general anaesthesia. Whenever possible such circumstances should be discussed in advance with the patient to establish their prior wishes.

Determining when CPR is likely to be unsuccessful or, in other words, futile, is often difficult. Two clinical decision rules have been developed using data from the AHA Get with the Guidelines Programme ($n > 50{,}000$ cases). The first developed a flow chart indicating the likelihood of survival to discharge with good neurological function. In this model, admission from a nursing facility with a cerebral performance category (CPC) of 2 or less had a very low (2.3%) chance of survival after cardiac arrest, as did admission from home or another hospital and a CPC score of 3 (2.2% survival).[75] Other important predictors of poor outcome were advancing age, presence of organ failure, malignancy and hypotension. Absence of co-morbidities, presence of arrhythmias and myocardial infarction were associated with better outcomes. The Go-FAR score, produced by the same group uses 13 pre-arrest variables to predict outcome.[75] A low score predicted good outcome (27% favourable survival) whilst a high score predicted poor outcome (0.8% favourable survival). Good neurological function at admission predicted good outcome whilst major trauma, stroke, malignancy, sepsis, non-cardiac medical admission, organ failure and advancing age were key determinants of adverse outcomes. Prediction studies are particularly dependent on system factors such as time to start of CPR and time to defibrillation. These intervals may be prolonged in the total study cohort but may not be applicable to an individual case.

Inevitably, judgements will have to be made based on all available information. Decisions should not be made based on a single element, such as age.[76] There will remain grey areas where judgement is required for individual patients.

It is difficult to define an optimal duration for resuscitation attempts. In a further study from the AHA Get With The Guidelines-Resuscitation (GWTG-R) registry, 88% of patients who achieved sustained ROSC did so within 30 min.[77] As a rule, resuscitation should be continued as long as VF persists. Asystole for more than 20 min during ALS in the absence of a reversible cause is generally accepted as an indication to abandon further resuscitation attempts. However, there are reports of exceptional cases that do not support the general rule, and each case must be assessed individually.

Presently, there are no valid prognostication tools of poor outcome during the first few hours after ROSC. The prediction of final neurological outcome in CA patients remaining comatose after ROSC is generally unreliable during the first 3 days after CA and until the first 2–3 days after termination of hypothermia.

Reliable prognostication of a poor outcome in comatose cardiac arrest survivors supports discussions with relatives and decisions to withdraw life-sustaining therapy. Guidelines for prognostication in such patients are described in detail in the post resuscitation care chapter of the 2015 ERC Guidelines.[27]

We should bear in mind that the implementation of a termination of resuscitation (ToR) protocol will inevitably introduce some self-fulfilling prophecy and must be challenged periodically as new treatments evolve.

The focus of most published studies has been on predicting poor outcomes amongst comatose survivors of cardiac arrest. Future research should also consider factors that would predict a good outcome in order to inform treatment decisions and discussions with relatives.

Out-of-hospital cardiac arrest (OHCA)

The decision to start or discontinue CPR is usually more challenging outside a hospital.[78,79] Specific challenges include the lack of sufficient, unequivocal information about a patient's wishes and values, comorbidities and baseline health status. Access to diagnostic tests to identify reversible causes is limited and teams in general are small and in many countries only comprise emergency medical technicians or paramedics. Prognostic assessment in terms of survival and subsequent quality of life carries a higher risk of bias and thus injustice.[80,81] Considering this and the proven correlation between time to BLS or first shock and outcome, the default for OHCA still needs to be to start CPR as soon as possible and address questions later. Exceptions are the conditions that enable recognition of life extinct (ROLE), namely massive cranial and cerebral destruction, decapitation, decomposition or putrefaction, incineration, dependent lividity (hypostasis) with rigor mortis, and foetal maceration. In such cases, the non-physician might be making a diagnosis of death but is not certifying death, which, in most countries, can be done only by a physician.

CPR that has no chance of success in terms of survival or acceptable quality of life is pointless and may violate the right for mercy and dignity in the face of death. Defining this 'no chance of success' is however very difficult and, in contrast to other medical interventions, it has been argued that success rates of less than 1% still justify the resuscitation effort.[78,81,82] Institutional guidelines for the Termination Of Resuscitation (ToR) in the pre-hospital environment are very much needed to reduce unwanted variability in this decision-making.

Several authors have developed and prospectively tested unequivocal termination of resuscitation (ToR) rules. One prospective study demonstrated that a basic life support ToR rule was 100% predictive of death when applied by defibrillation-only emergency medical technicians. Subsequent studies showed external generalisability of this rule, but others challenged this. The implementation of a ToR rule significantly reduced the rate of transport of futile OHCA yet also led in two separate studies to an unexpected survival of 3.4% and 9% respectively in OHCA patients without pre-hospital sustained ROSC.

Some EMS systems use just that one component, the absence of pre-hospital return of spontaneous circulation (ROSC), as the criterion to terminate resuscitation and this clearly may exclude potential survivors for transportation.[78,83–87]

Patients with refractory cardiac arrest, with ongoing CPR during transport to hospital, used to have a very poor prognosis.[88,89] In a moving vehicle, manual CPR may be difficult and the use of mechanical devices may be considered. As advanced rescue therapies and specific circumstances-related interventions become more widely available and success rates are improving, defining which patients might benefit from these becomes crucial. [90–92]

Withholding or withdrawing CPR

Healthcare professionals should consider withholding or withdrawing CPR in children and adults when:

- the safety of the provider can no longer be sufficiently assured;
- there is obvious mortal injury or irreversible death [ROLE];

- a valid and relevant advance directive becomes available;
- there is other strong evidence that further CPR would be against patient's values and preferences or is considered 'futile';
- asystole for more than 20 min despite ongoing ALS, in the absence of a reversible cause.

After stopping CPR, the possibility of ongoing support of the circulation and transport to a dedicated centre in perspective of organ donation should be considered.

Transport to hospital with ongoing CPR

Healthcare professionals should consider transport to hospital with ongoing CPR when, in the absence of the above CPR withdrawal criteria, there is one or more of the following present:

- EMS witnessed arrest;
- ROSC at any moment;
- VT/VF as presenting rhythm;
- Presumed reversible cause (e.g., cardiac, toxic, hypothermia).

This decision should be considered early in the process e.g., after 10 min of ALS without ROSC and in view of the circumstances e.g., distance, CPR delay and presumed CPR quality in view of patient characteristics e.g., presumed QoL.

Paediatric cardiac arrest

Despite differences in pathophysiology and aetiology, the ethical framework for decision-making in paediatric cardiac arrest does not differ much from that described above.[93,94] Most physicians will err even more on the side of intervention in children for emotional reasons and continue a resuscitation attempt longer, despite the overall prognosis in children often being worse than in adults. It is therefore important for clinicians to understand the factors that influence resuscitation success and the boundaries of the care they provide. As in adult practice, futile resuscitation might be considered dysthanasia (merciless prolongation of life) and should be avoided.[81] The child's best interest might sometimes conflict with parent or guardian's rights. From a societal perspective, we allow parent's decisions to differ from so-called best interest standards as long as no unacceptable harm is done to the child. Extrapolating this to the context of resuscitation, parent's rights and decision-making might prevail up to the point where there would be harm. Prolonged futile resuscitation could be an example of such harm. Providing adequate information in a clear but empathic way is crucial for this decision-making process.

Most countries have procedures for medico-legal investigation of Sudden Unexplained Death Of Infancy (SUDI). In many SUDI cases no final cause is identified and death might be related to an intrinsic vulnerability, developmental changes and environmental factors.[95] Some deaths however might be caused by infection, neuro-metabolic disease or by accidental or inflicted injury. In most countries, legal authorities are involved in cases of sudden unexplained or accidental death. In some countries systematic review of all child deaths is organised to get a better understanding and knowledge for the prevention of future children's deaths.[96] Although there are still major challenges, formal child death reviews may contribute greatly to prevention, care delivered and final outcome of paediatric cardiac arrest.

Specific circumstances

Slow code

Some prehospital providers find it difficult to stop resuscitation once started and would argue for continuing CPR, especially in young persons, until arrival to the hospital. Some defend this practice on the ground that, at a certain point, the 'best interest' of the family might start to outweigh that of the patient.[97,98] This view is not supported by evidence. In the setting of post-traumatic cardiac arrest it seemed that families of patients who die out-of-hospital adapt better to their losses when there is cessation of futile resuscitative efforts in the field. [93] Performing futile CPR to address the grief and needs of 'significant others' is ethically unsound, being both deceptive and paternalistic.[43]

Likewise, certain authors argued in favour of a 'slow code' initiating some 'symbolic' resuscitation measures but unhurriedly or omitting the most aggressive ones, sparing physician and family the helpless feeling of doing nothing and avoiding potential conflict or the need to communicate bad news, especially in those settings where there is no strong physician–patient relationship and a clear lack of information.[43] This 'slow code' is equally deceptive and paternalistic, and undermines both the patient–physician relationship and the training and education of our teams.[93]

A valuable alternative may be a 'tailored code', where high quality resuscitation is performed but clear limits are defined. Family members are informed in a transparent way what will be done and what not.[99,100]

Provider safety

Safety of the healthcare provider is vitally important. Infectious disease epidemics have raised concerns about the safety of healthcare providers involved in the care of cardiac arrest patients. Specific attention to the use of proper protective equipment is essential, especially when there is insufficient information about a patient's history and potential infectious state. To date there is little information about the precise risk of transmission when doing CPR on an infectious patient, and as such – if properly protected – providers should attempt resuscitation in these patients. Possible exemptions to this standard rule would be those infections or situations where a clear danger remains for the healthcare provider, even when protected. In these cases the provider's own safety would be priority. When attempting CPR in infectious patients' healthcare professionals must use proper protective equipment and be sufficiently trained in its use.[101,102]

Resuscitation after suicide attempts

A person with mental illness is not necessarily considered mentally incompetent and may have an equal right to reject medical treatment and opt for palliative care. Based on the concept of autonomy, one could argue that a suicide attempt may in itself be an expression of personal preferences. In an emergency it is difficult to assess mental capacity reliably even if a suicide note is found. Given that non-treatment leads to serious harm, the default remains to start CPR as soon as possible and address potential issues later.[103,104]

Organ donation

The primary goal of resuscitation is to save the patient's life.[105] Nonetheless, resuscitation efforts may result in brain death. In these cases, the aim of resuscitation could change to the preservation of organs for possible donation.[106] Several studies have shown that the outcome of organs transplanted from patients who received CPR and are brain dead is not different from the outcomes of organs transplanted from patients who have been pronounced brain dead from other causes (see section on Post Resuscitation Care).[107–109] However, the duty of resuscitation teams for the living patient should not be confused with the duty of physicians for the dead donors, where the organs are preserved to save other people's lives. On the other hand, it is reasonable to suggest that all European countries should enhance their efforts to maximise the possibility of organ donation from cardiac arrest patients who became brain

dead or after stopping resuscitation in case of CPR failure.[110] Procedures should ensure that any possible interference of the transplant team in the decision making of the resuscitation team is avoided.

Variability in ethical CPR practices in Europe

Ten years after a report by Baskett and Lim,[111] opinion leaders representing 32 European countries where the activities of the European Resuscitation Council are organised, have responded to questions regarding local ethical legislation and practice of resuscitation, and organisation of out-of-hospital and in-hospital resuscitation services. The survey methods and results are detailed and discussed elsewhere.

The survey showed that there is still a wide variability in the implementation of ethical practices in European countries.

Equal access to emergency care and to early defibrillation is now well established: the first attending ambulance arrives at the scene within 10 min in the majority of countries (18/32 in rural areas and 24/32 in urban areas). Defibrillation by the first attending ambulance is available in 29/32 countries.

The principle of patient autonomy is now legally supported in the majority of countries (advance directives in 20 countries and DNAR in 22 countries).

However, areas for improvement were identified: in less than half of countries family members are usually allowed to be present during CPR (adult in 10/32 and children in 13/32 countries). This has not substantially changed in the last 10 years.

At this time euthanasia and physician-assisted suicide are controversial subjects in many European countries and the discussion is ongoing in several European countries.

Certain forms of treatment limitations such as withholding CPR are allowed (19 countries) and practiced (21 countries) in most European countries.

Harmonisation of legislation relating to resuscitation and end-of-life would further support ethical practices.

Healthcare professionals should know and apply the established national and local legislation and policies.

Communication

Family presence during resuscitation

Since the 1980s, the concept of a family member being present at the resuscitation process became an accepted practice in many countries.[112–116] The majority of relatives and parents who were present during resuscitation attempts would wish to be so again.[113] A recent European survey reported that in only 31% of countries family members are usually allowed to be present during in-hospital resuscitation attempts of an adult and only slightly more if the victim was a child (41%).

The ERC supports relatives being offered the choice to be present during a resuscitation attempt whilst cultural and social variations must be understood and appreciated with sensitivity. Observing the resuscitation attempt may provide benefit to family members by reducing guilt or disappointment, allowing time to accept the reality of death and help the grieving process. When possible, an experienced member of staff should facilitate and support the relative during the resuscitation attempt.[114,115] Family presence during resuscitation attempts will contribute to an increasingly open attitude and appreciation of the autonomy of both patient and relatives.[111,112] No data support the concerns that family members may be traumatised witnessing CPR, or may interfere with medical care procedures.[117] We should focus our efforts on working together with the survivors of cardiac arrest, family members and the public as partners in the co-production of future guidance.

Bringing bad news and bereavement counselling

A multidisciplinary approach to the care at the end of life, including communication, taking into account cultural, social, emotional, religious, spiritual preferences and local differences needs further development and implementation in healthcare systems worldwide.

Compassionate communication with patients and loved ones is essential when dealing with end of-life-care. The aim is to understand the patient's goals and expectations of medical treatment to support the individual choice of the best care. Some patients wish to prolong life as long as possible, while others value dignity and pain relief even at the expense of a potentially shortened lifetime. Privacy and adequate time are essential for good communication about life values and significant decisions.[118]

Multidisciplinary bereavement programs are beneficial to families of patients who die in the emergency department.[119] The grieving process may be supported by allowing unrestricted visiting, provision of clear verbal and written information, providing the opportunity to visit the deceased and facilitating religious procedures.[120,121] Patients and their beloved ones deserve respect.

Clinicians should be honest about what can and cannot be achieved. Sharing the truth of the situation can act as a symbolic expression of a complex set of commitments.[29] This will allow the patients to make informed decisions about the choices available to them at the end of their lives.

Documentation of DNAR order in the patient's chart

DNAR decisions and discussions relating to DNAR should be recorded clearly in the patient's notes.[72,73,122,123] Whatever system is used it must be highly visible in order to inform personnel on the spot.

Over time the situation or the perspectives of patients might change and DNAR orders should be revised accordingly.[124] Exemptions from DNAR order should be clearly specified (e.g., cardiac arrest complicating diagnostic procedures, such as allergic shock due to radiology dye or intracardiac catheter investigation) to ensure the patient will receive appropriate treatment.

Training, research and audit

It is the individual responsibility of healthcare professionals to maintain their knowledge, understanding and skills related to resuscitation. Their knowledge about relevant national legal and organisational policies in their country should be kept up to date.

Improving public education regarding Cardiopulmonary Resuscitation

The shift from medical-centred to patient-centred practice constitutes a major ethical development. This requires that the patient is aware (and not misinformed) of the true limitations and possible outcomes of resuscitation.[125–127] Lay people may have unrealistic expectations from CPR[128,129] and exposure to realistic outcome data may affect personal preferences.[130]

Training health care professionals about DNAR issues

Healthcare professionals should receive training about the legal and ethical basis of DNAR decisions and about how to communicate effectively with patients, relatives or next of kin. Quality of life, supportive care and end-of-life decisions need to be explained as an integrative part of the medical and nursing practice.[131] Training

will need to be sensitive to personal, moral and religious beliefs and feelings.

Practicing procedures on the recently dead

There is a wide diversity of opinion about practicing on the newly dead ranging from complete non-acceptance because of an innate respect for the deceased[132] to the acceptance of non-invasive procedures not leaving major marks.[133] Others accept training of any procedure on dead bodies and justify skills training as paramount for the well-being of future patients.[134–137]

Healthcare students and teaching professionals are advised to learn and follow the established legal, regional and local hospital policies.

Research and informed consent

Research in the field of resuscitation is necessary to test commonly used interventions with uncertain efficacy or new potentially beneficial treatments.[138,139] To include participants in a study, informed consent must be obtained. In emergencies, there is often insufficient time to obtain informed consent. Deferred consent or exception to informed consent with prior community consultation, are considered ethically acceptable alternatives for respecting autonomy.[140,141] Following 12 years of ambiguity, a new European Union (EU) Regulation permitting deferred consent is expected to harmonise and foster emergency research across Member States.[139,140,142,143] Further regulatory improvements are needed for emergency surgical research [144] and for researching non-medicinal interventions.[139] Despite this progress, regulations still need to converge at an international level to harmonise multi-national emergency research.[145]

Audit of in-hospital cardiac arrests and registry analyses

Local CPR management can be improved through post-CPR debriefing and feedback to ensure a PDCA (plan-do-check-act) circle of quality improvement. Debriefing and feedback enables identification of CPR quality errors and prevents their repetition.[146–148] Submission of CPR data to national audits and/or international registries has led to outcome-prediction models, which may facilitate advance care planning[149–153], and to quantification of the frequency of resuscitation system errors and their impact on in-hospital mortality.[154] Data from registries have shown significant improvements in cardiac arrest outcomes from 2000 to 2010.[3,155–157]

Published evidence suggests that resuscitation team-based infrastructure and multilevel institutional audit,[158] accurate reporting[54] of resuscitation attempts at national audit level and/or multinational registry level, and subsequent data analysis and feedback from reported results may contribute to continuous improvement of in-hospital CPR quality and cardiac arrest outcomes.[2,3,159–161]

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