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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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[1] See Appendix 1 for the ERC 2015 Guidelines Writing Group.

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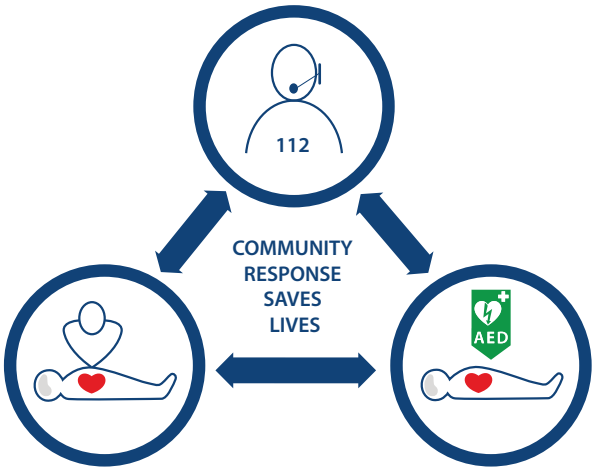


Fig. 1.1. The interactions between the emergency medical dispatcher, the bystander who provides CPR and the timely use of an automated external defibrillator are the key ingredients for improving survival from out of hospital cardiac arrest.

- 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]

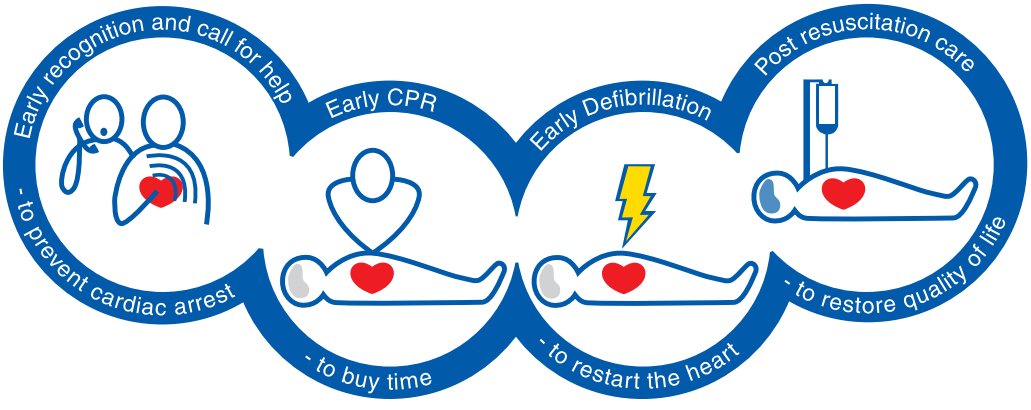


Fig. 1.2. The Chain of Survival.

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).

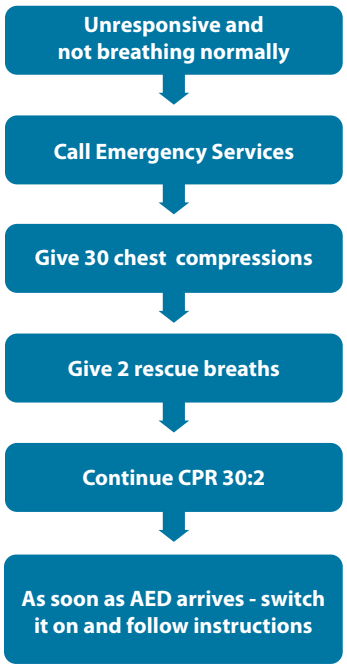


Fig. 1.3. The basic life support/automated external defibrillation (BLS/AED) algorithm.

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

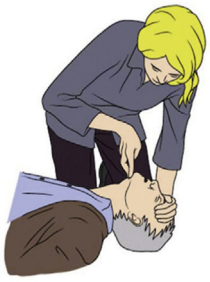
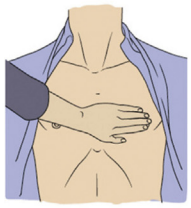
SEQUENCE / Action		Technical description
SAFETY **Make sure you, the victim and any bystanders are safe**		
RESPONSE **Check the victim for a response**		Gently shake his shoulders and ask loudly: “Are you all right?" If he responds leave him in the position in which you find him, provided there is no 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.

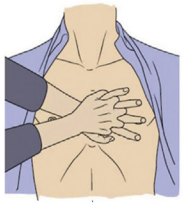
CIRCULATION

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*)

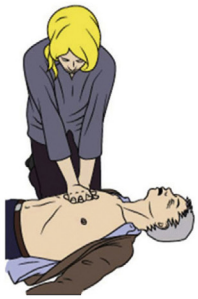
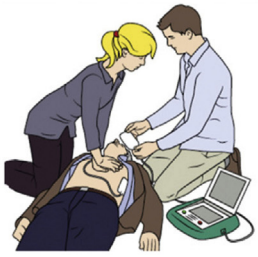
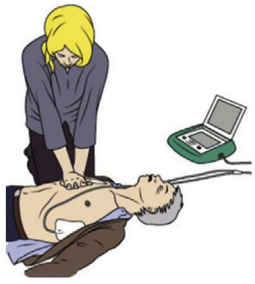
		Continue with chest compressions and rescue breaths in a ratio of 30:2
IF UNTRAINED OR UNABLE TO DO RESCUE BREATHS **Continue compression only CPR**		Give chest compressions only CPR (continuous compressions at a rate of 100-120 min^{-1})
WHEN AED ARRIVES **Switch on the AED and attach the electrode 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*)

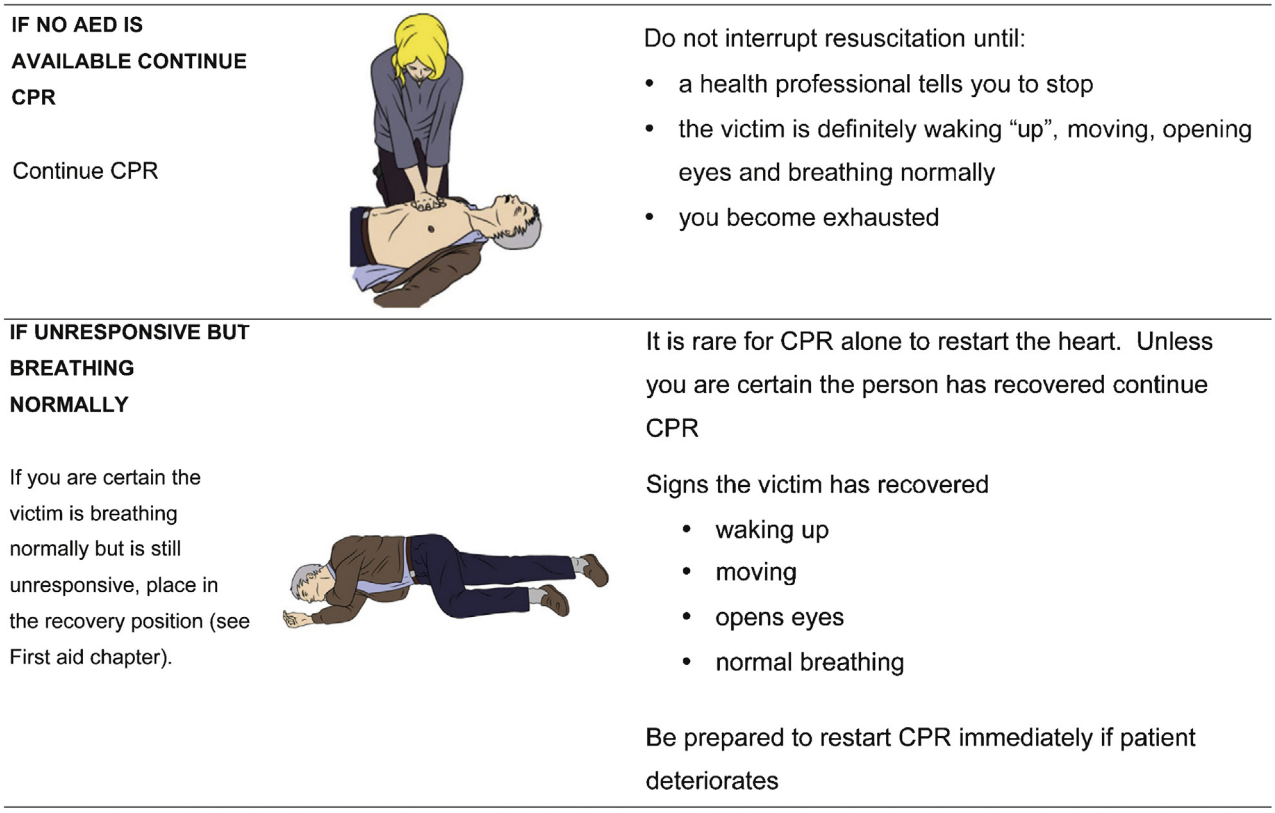


Fig. 1.4. (*Continued*)

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.

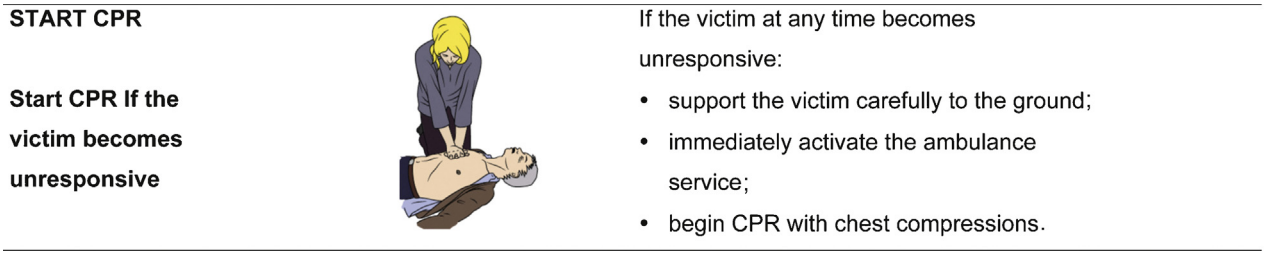


Fig. 1.5. (*Continued*)

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

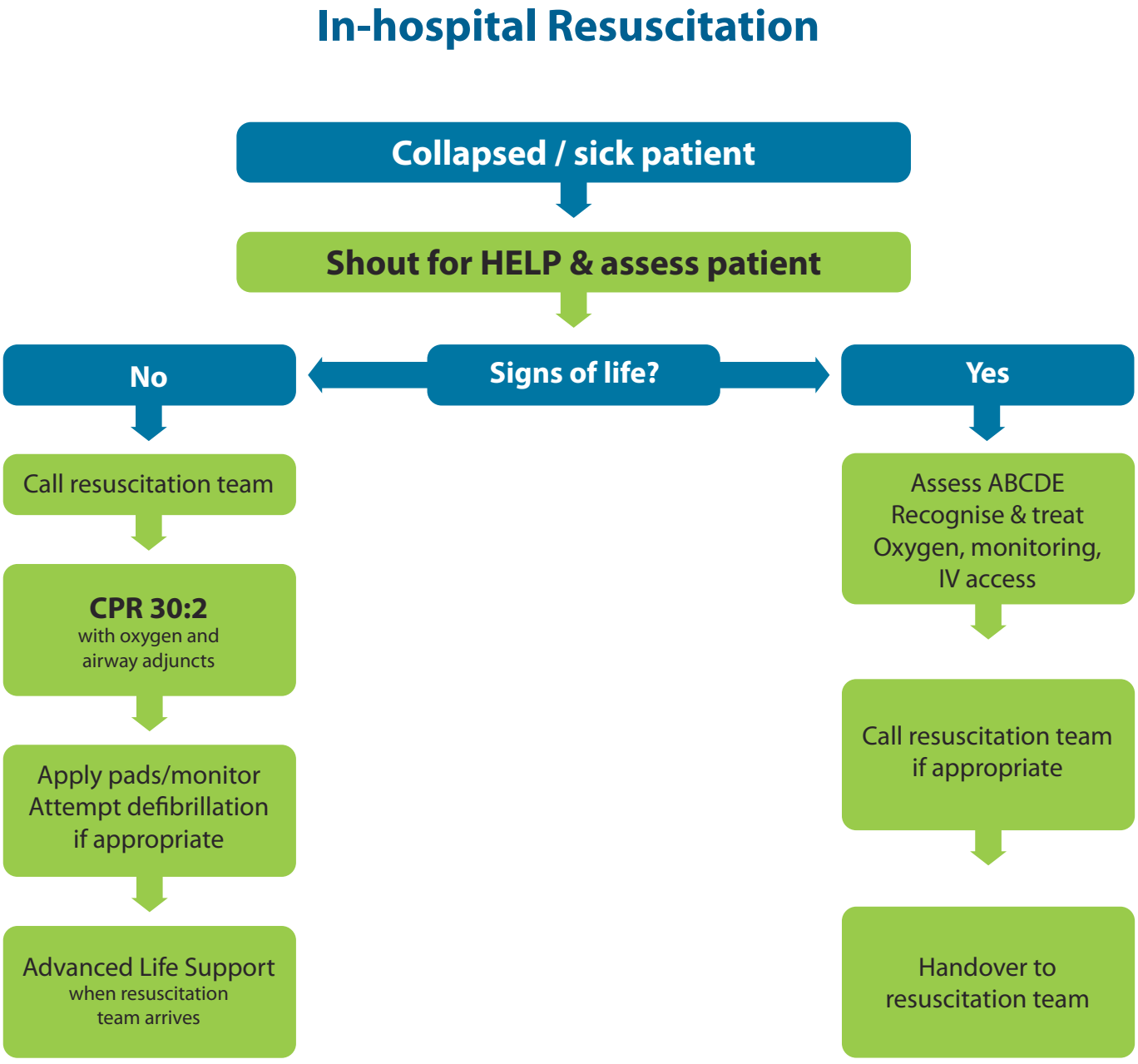


Fig. 1.6. In-hospital resuscitation algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure IV – intravenous; CPR – cardiopulmonary resuscitation.

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.

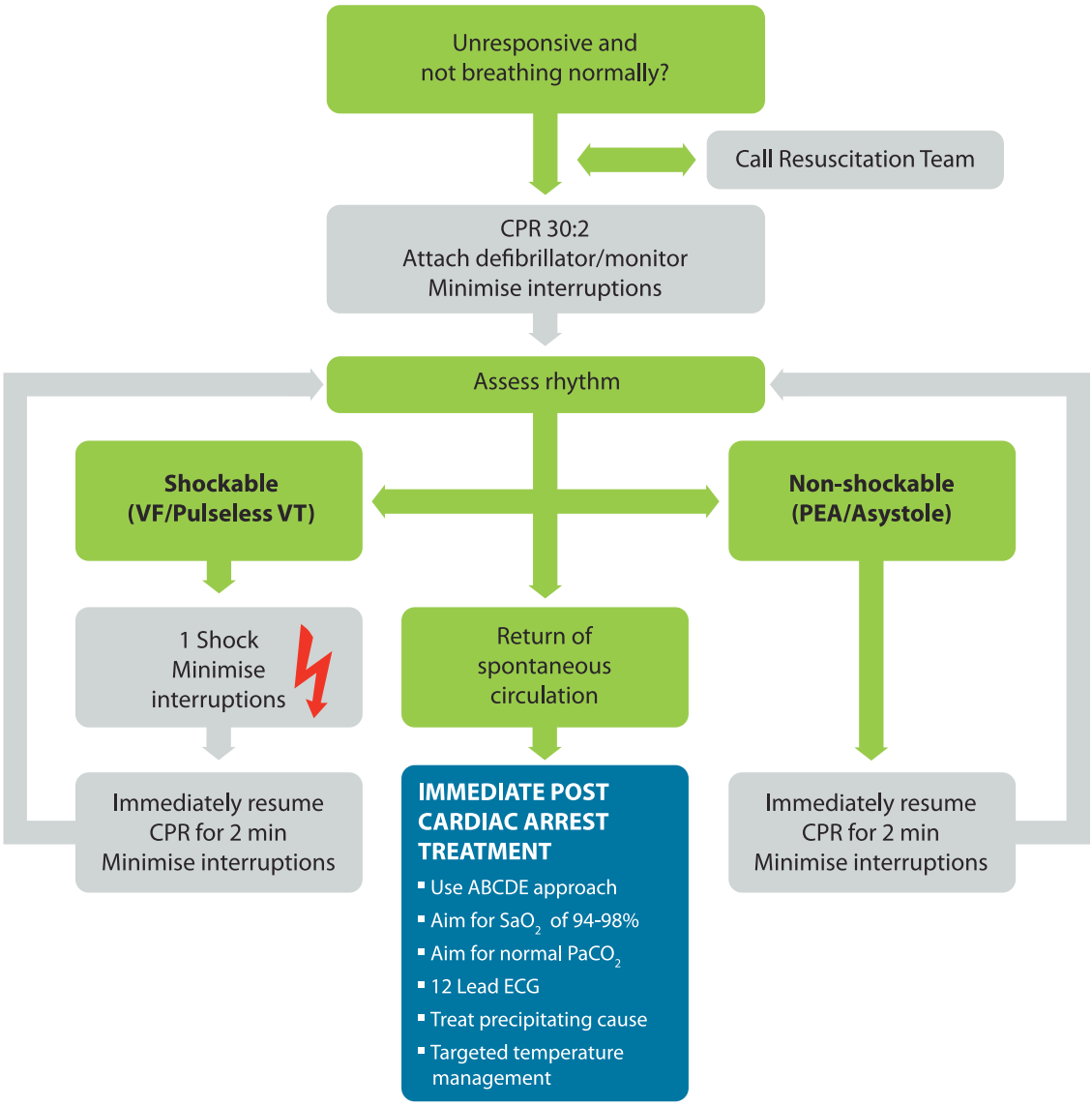
During treatment of VF/pVT, healthcare providers must practice efficient coordination between CPR and shock delivery whether using a manual defibrillator or an AED. Reduction in the peri-shock pause (the interval between stopping compressions to resuming compressions after shock delivery) by even a few 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).

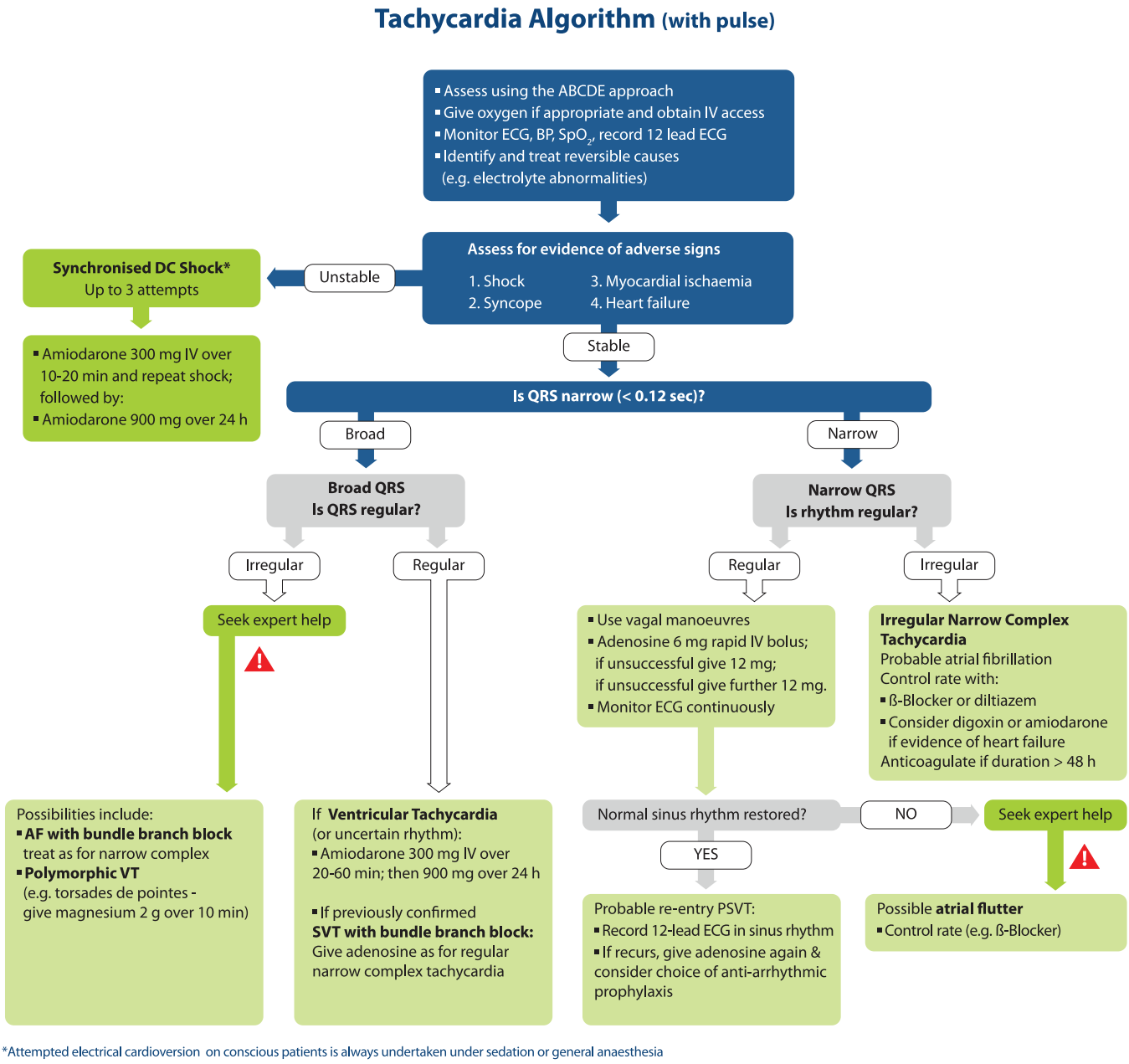


Fig. 1.8. Tachycardia algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure; IV – intravenous; SpO_2 – oxygen saturation measured by pulse oximetry; BP – blood pressure; ECG – electrocardiogram; DC – direct current; AF – atrial fibrillation; VT – ventricular tachycardia; SVT – supraventricular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

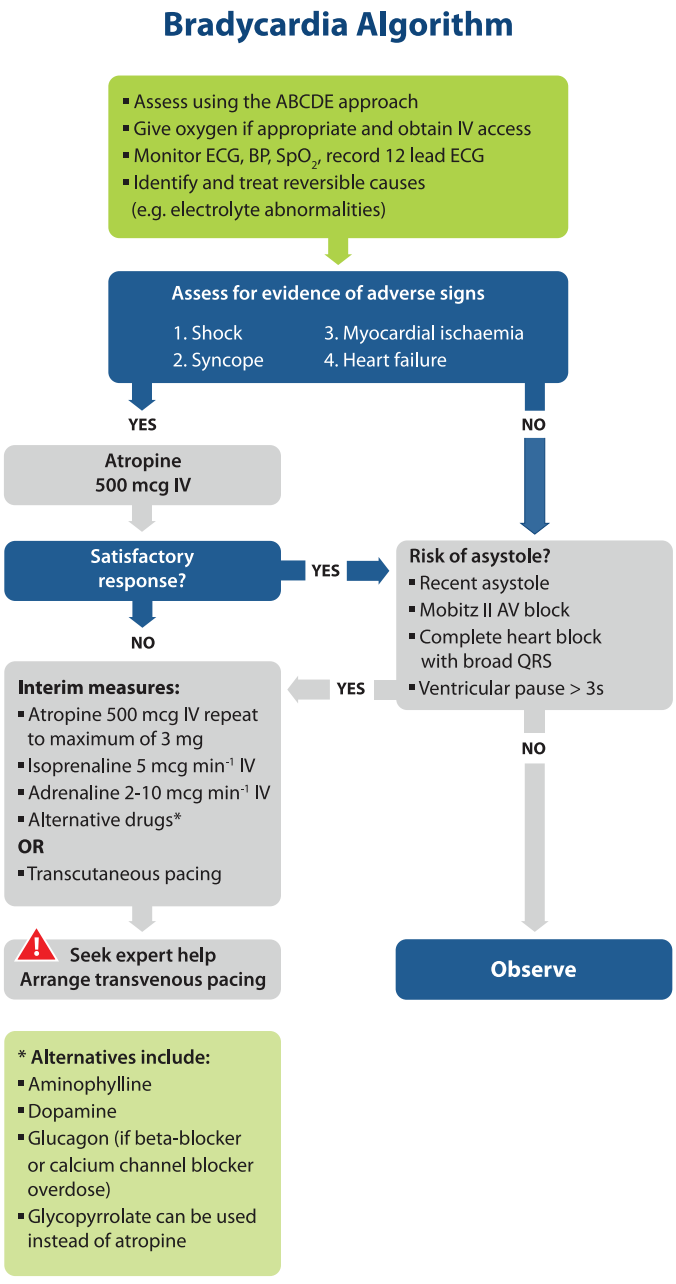


Fig. 1.9. Bradycardia algorithm. ABCDE – Airway, Breathing Circulation, Disability, Exposure; IV – intravenous; SpO_2 – oxygen saturation measured by pulse oximetry; BP – blood pressure; ECG – electrocardiogram; AV – atrioventricular.

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

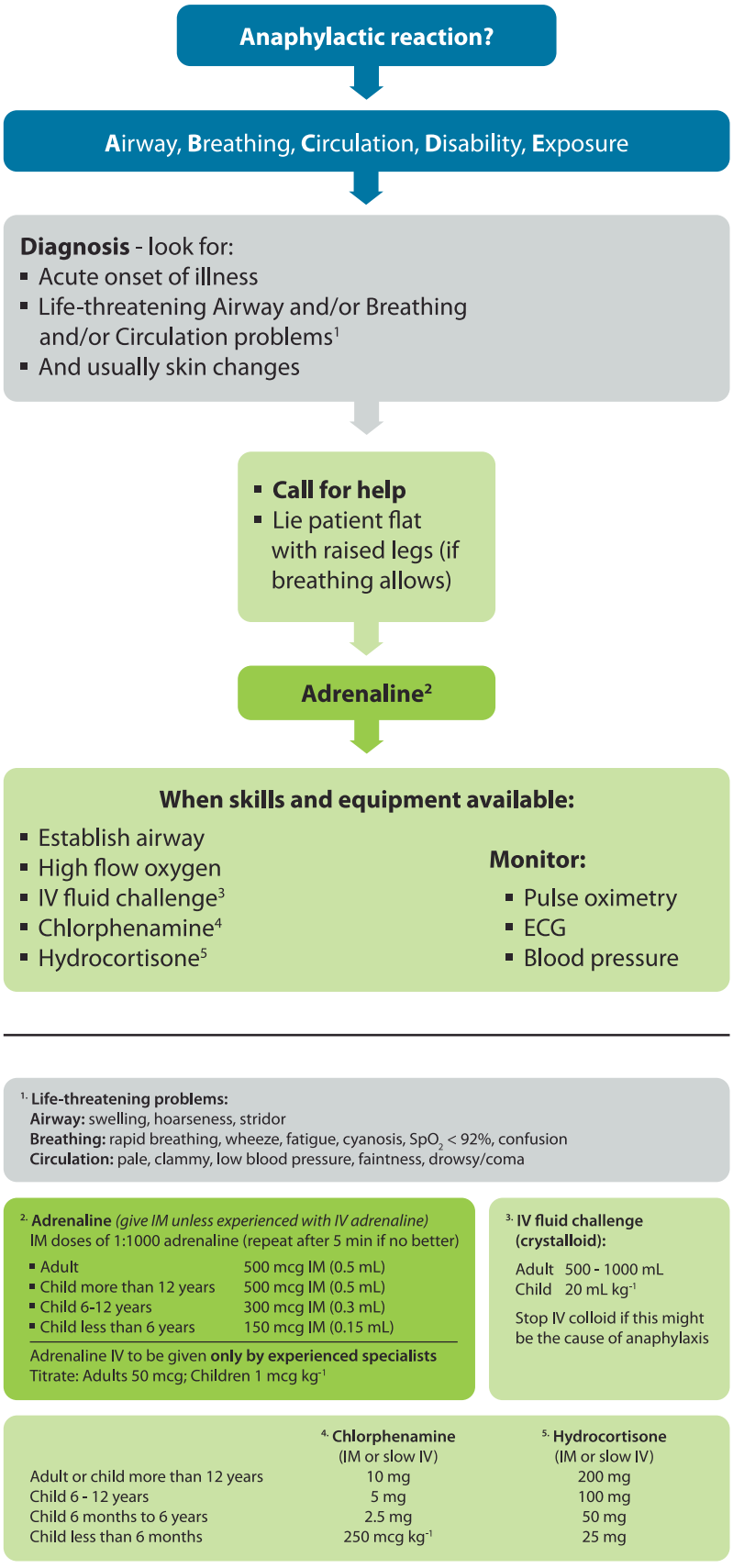


Fig. 1.10. Anaphylaxis treatment algorithm.[282]

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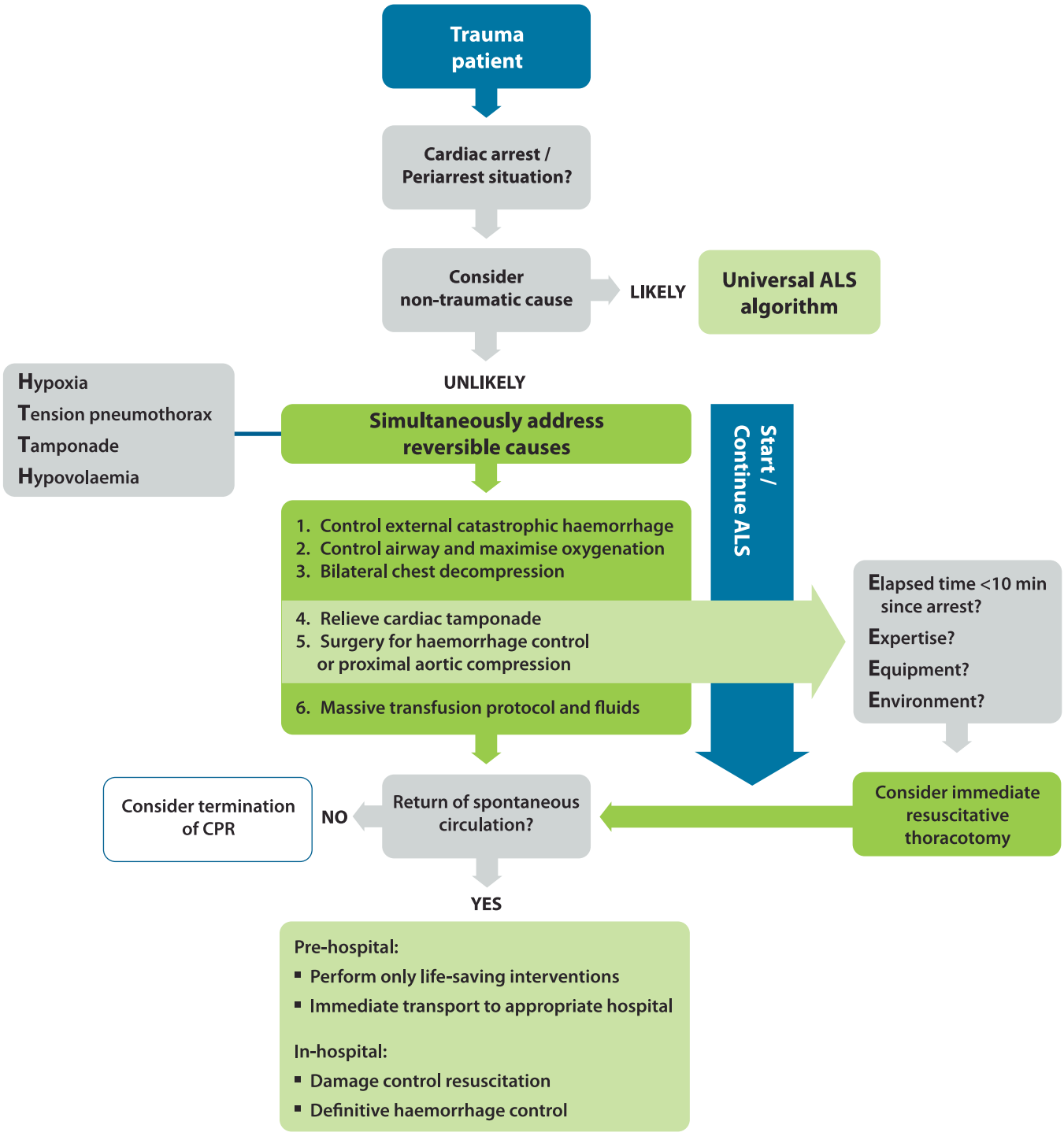

Fig. 1.11. Traumatic cardiac arrest algorithm.

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

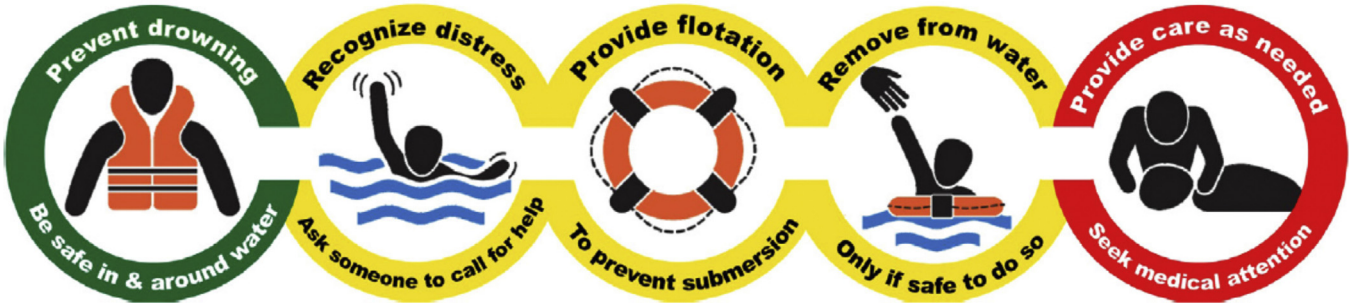

Fig. 1.12. Drowning chain of survival.[337]

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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

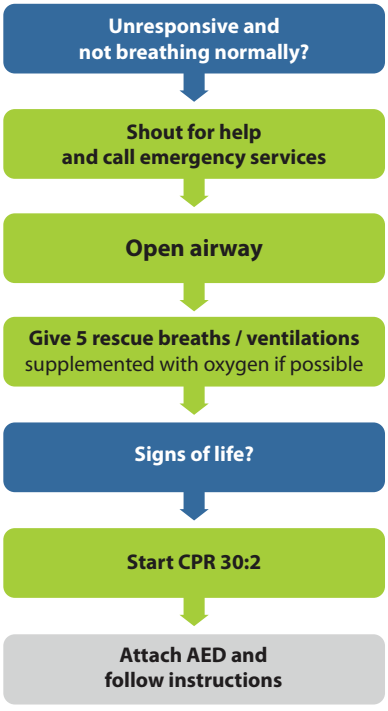


Fig. 1.13. Drowning treatment algorithm for rescuers with a duty to respond.

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

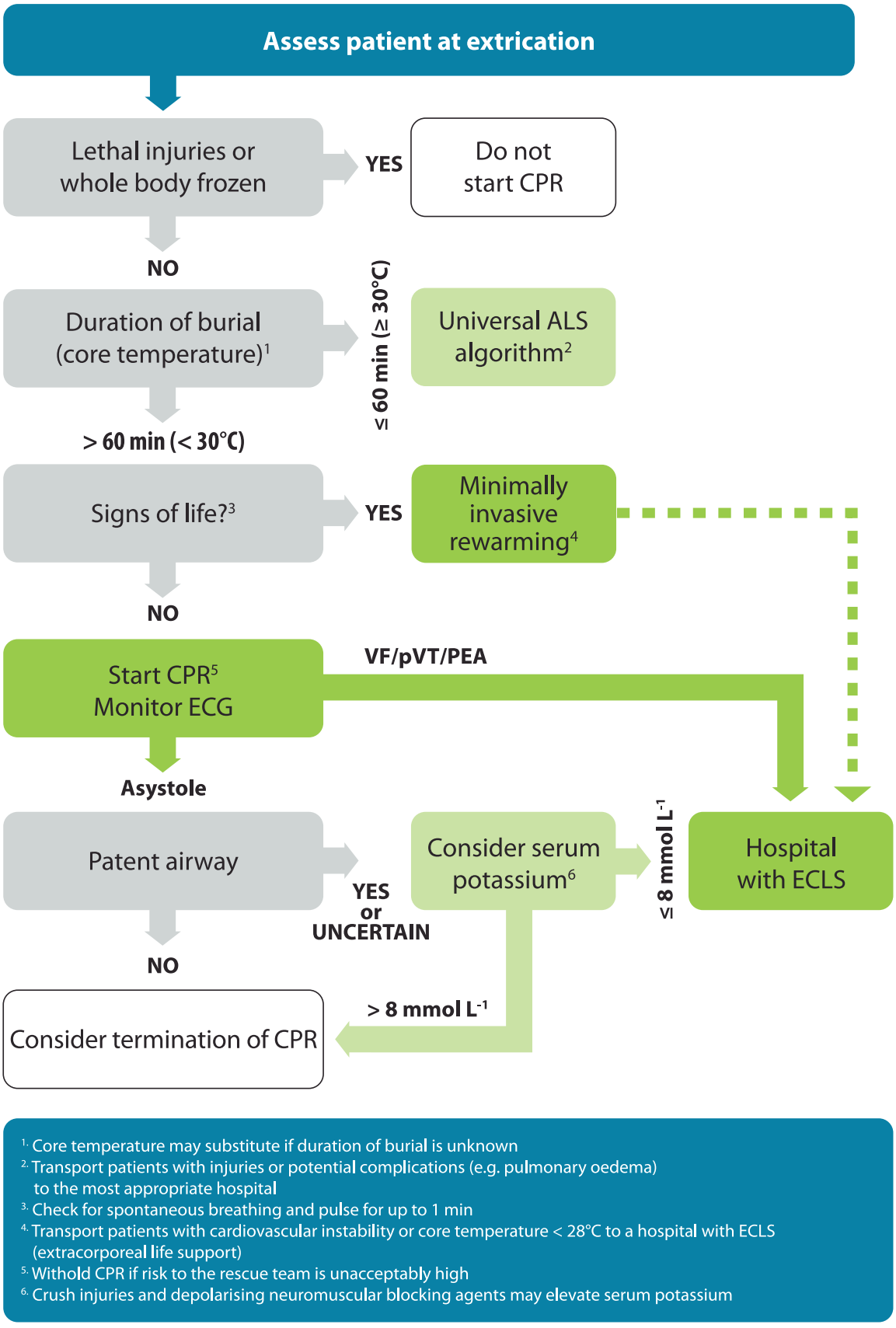
From 20 weeks' gestation, the uterus can compress both the inferior vena cava (IVC) and aorta, impeding venous return and cardiac output. The hand position for chest compressions may need to be slightly higher on the sternum for patients with advanced pregnancy e.g. third trimester.[360] Manually displace 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.

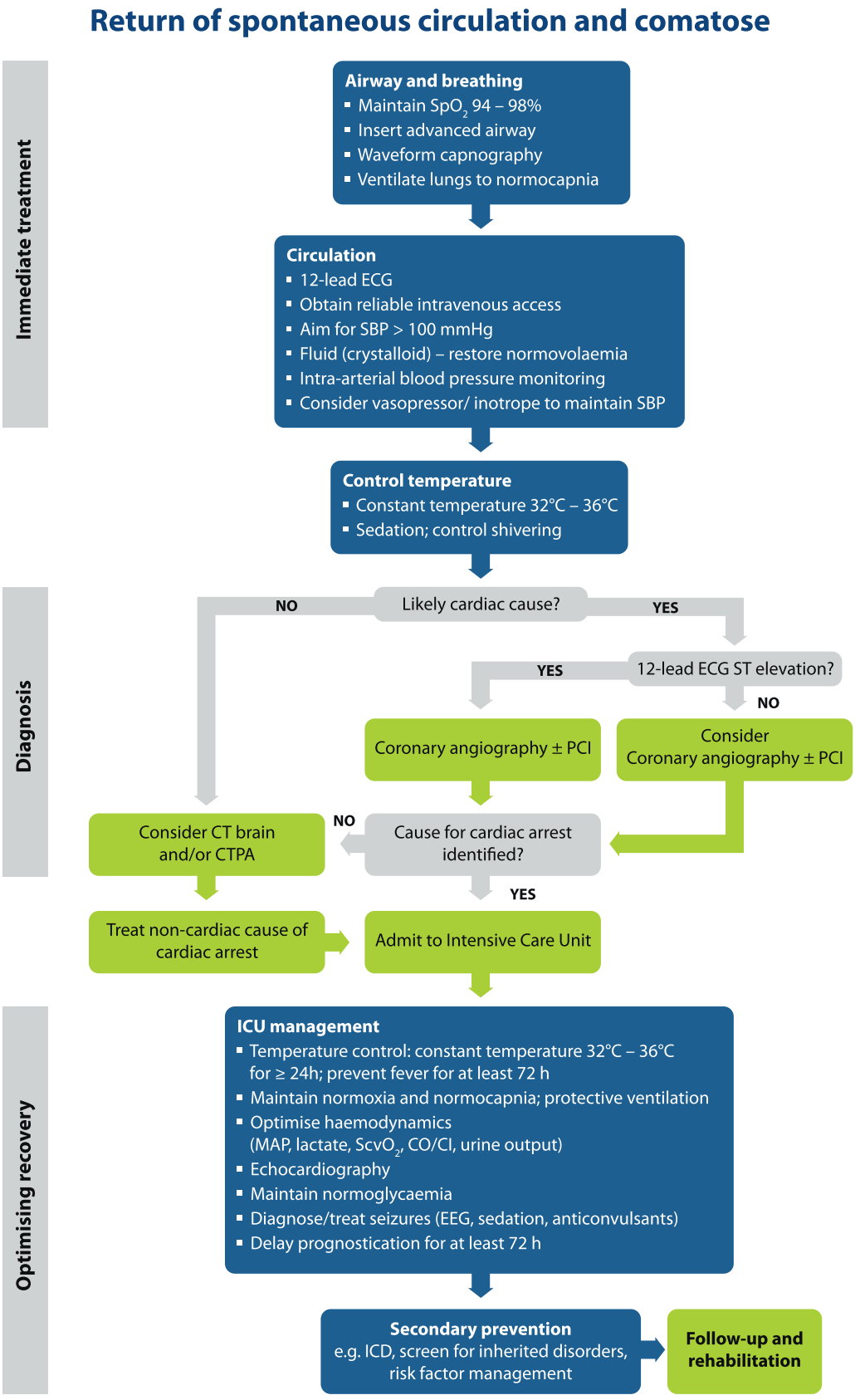


Fig. 1.15. Post resuscitation care algorithm. SBP – systolic blood pressure; PCI – percutaneous coronary intervention; CTPA – computed tomography pulmonary angiogram; ICU – intensive care unit; MAP – mean arterial pressure; $ScvO_2$ – central venous oxygenation; CO/CI – cardiac output/cardiac index;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

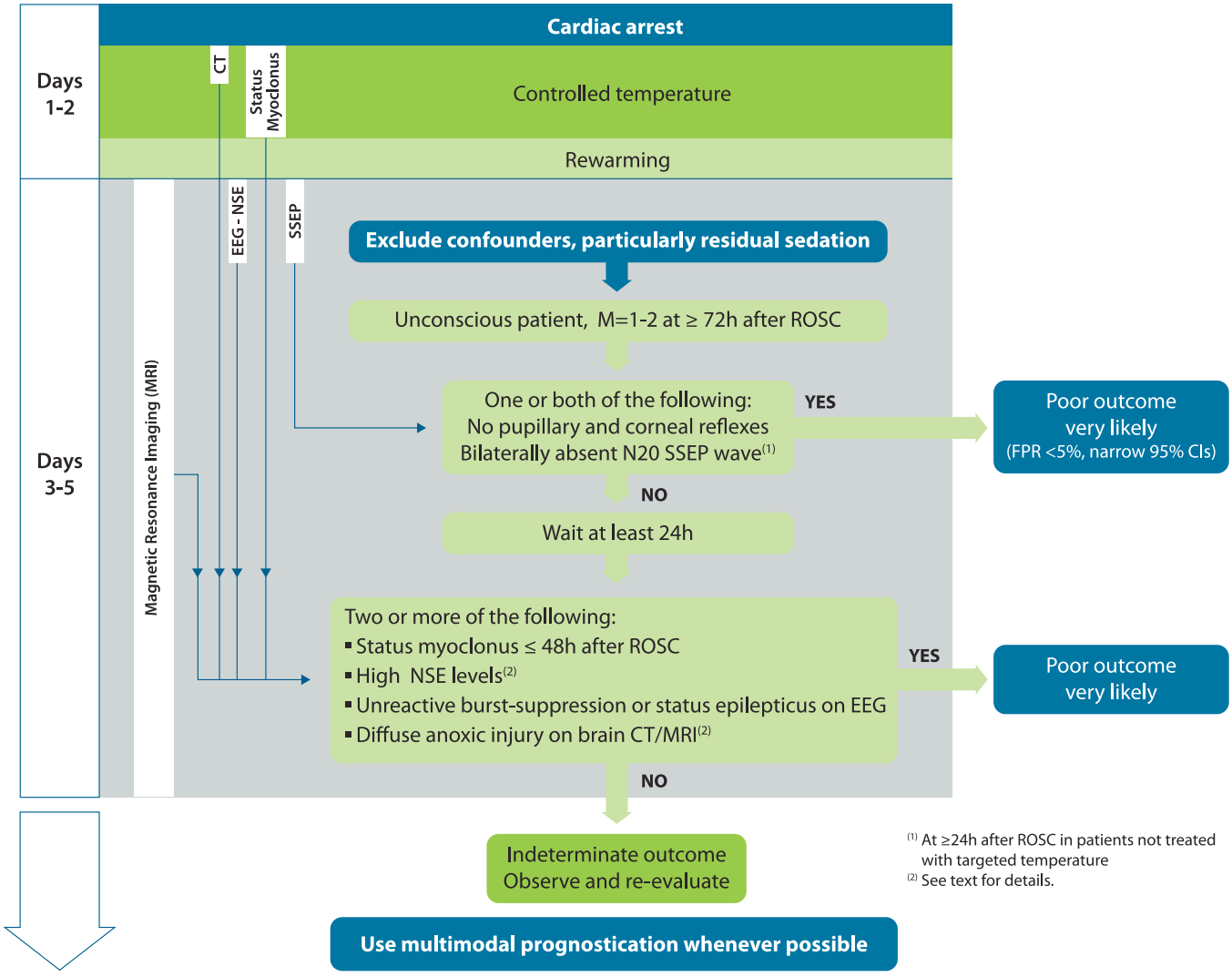


Fig. 1.16. Prognostication strategy algorithm. EEG – electroencephalography; NSE – neuron specific enolase; SSEP – somatosensory evoked potentials; ROSC – return of spontaneous circulation; M – Motor score of Glasgow Coma Scale.

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).

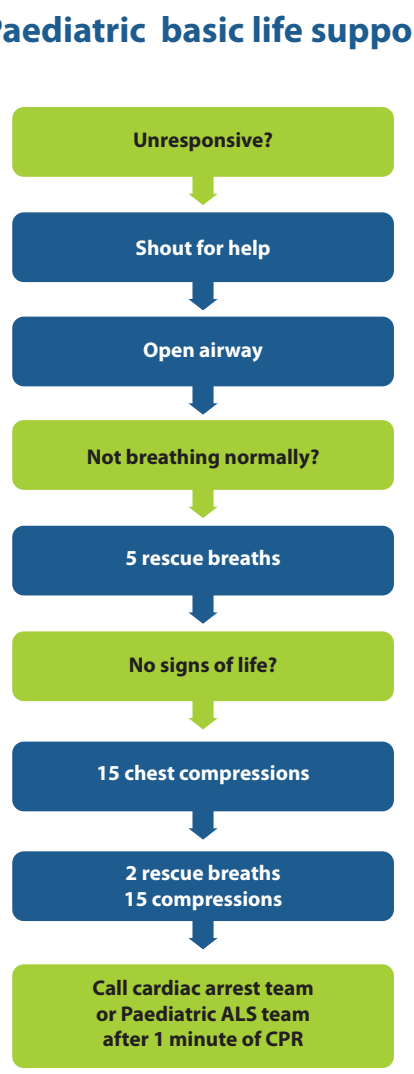


Fig. 1.17. Paediatric basic life support algorithm.

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

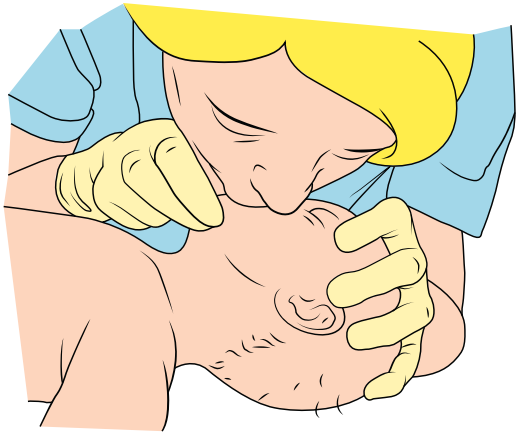


Fig. 1.18. Mouth-to-mouth-and-nose ventilation – infant.

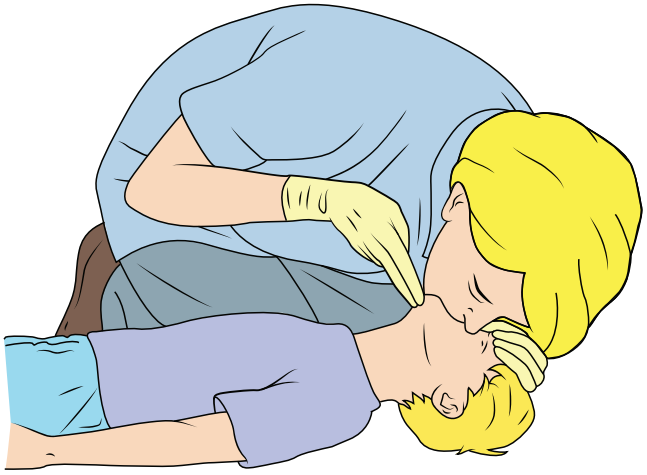


Fig. 1.19. Mouth-to-mouth ventilation – child.

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.

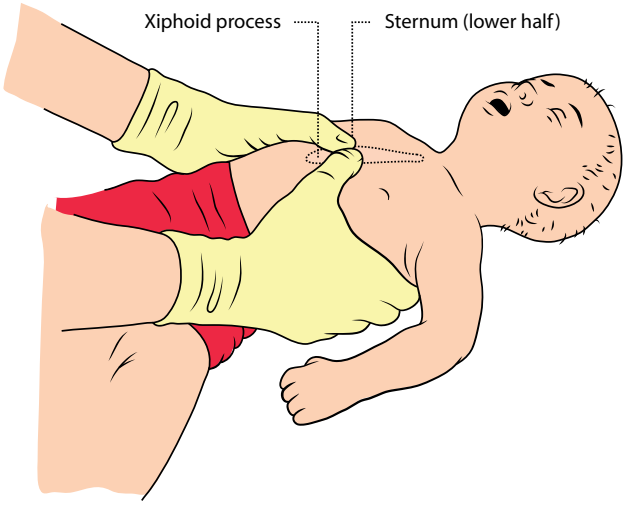


Fig. 1.20. Chest compression – infant.

- 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.

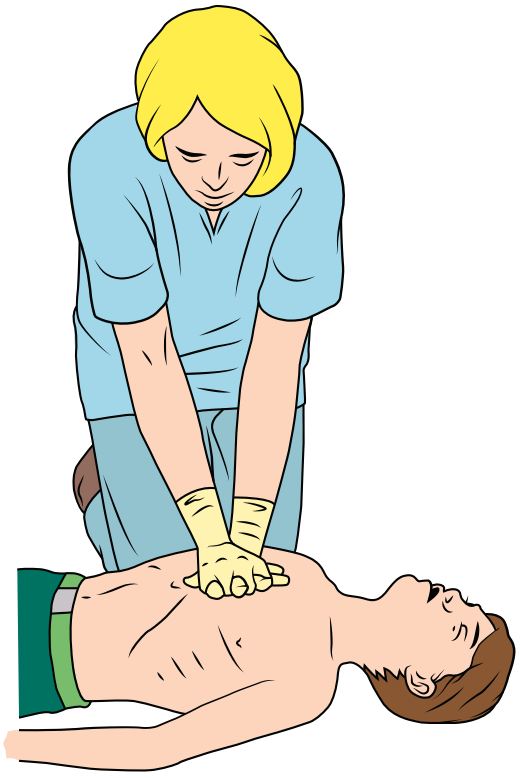


Fig. 1.22. Chest compression with two hands – child.

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

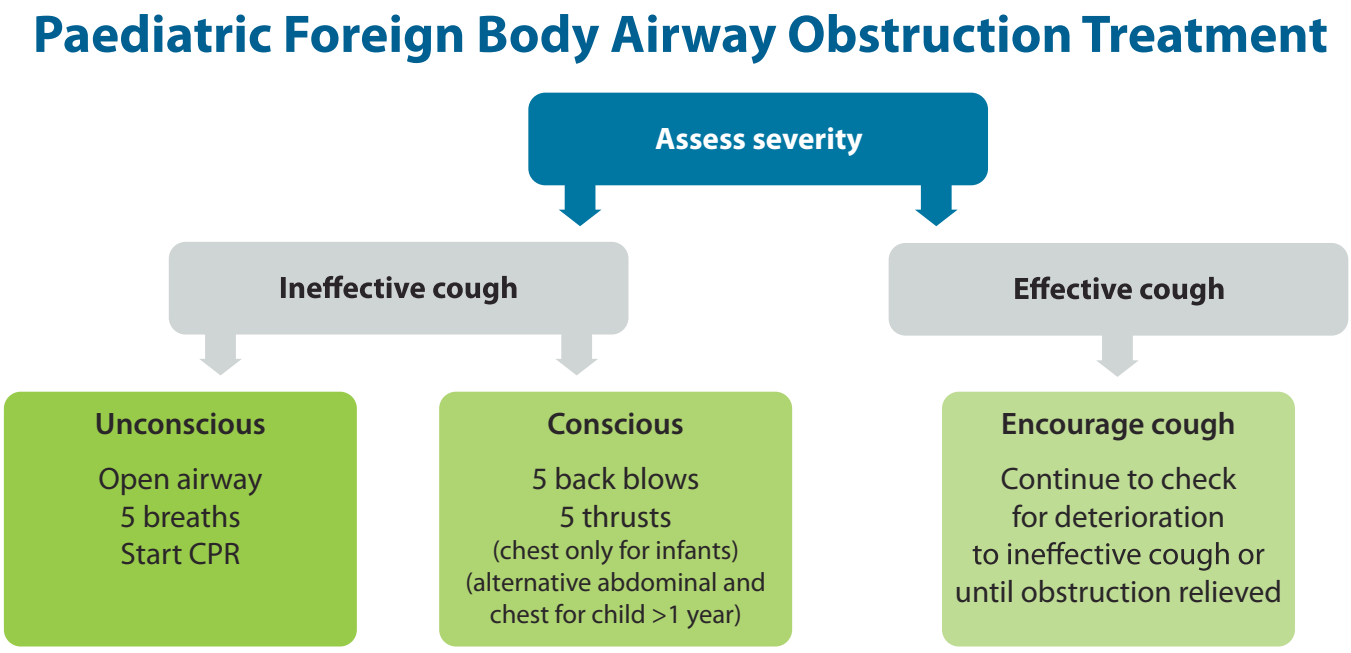


Fig. 1.23. Paediatric foreign body airway obstruction algorithm.

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

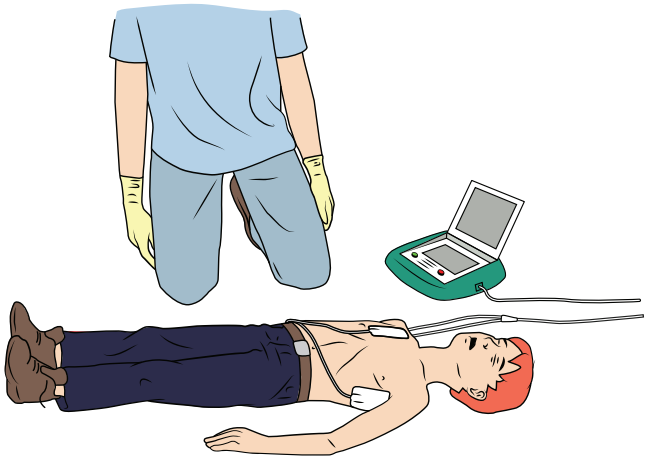


Fig. 1.24. Paddle positions for defibrillation – child.

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

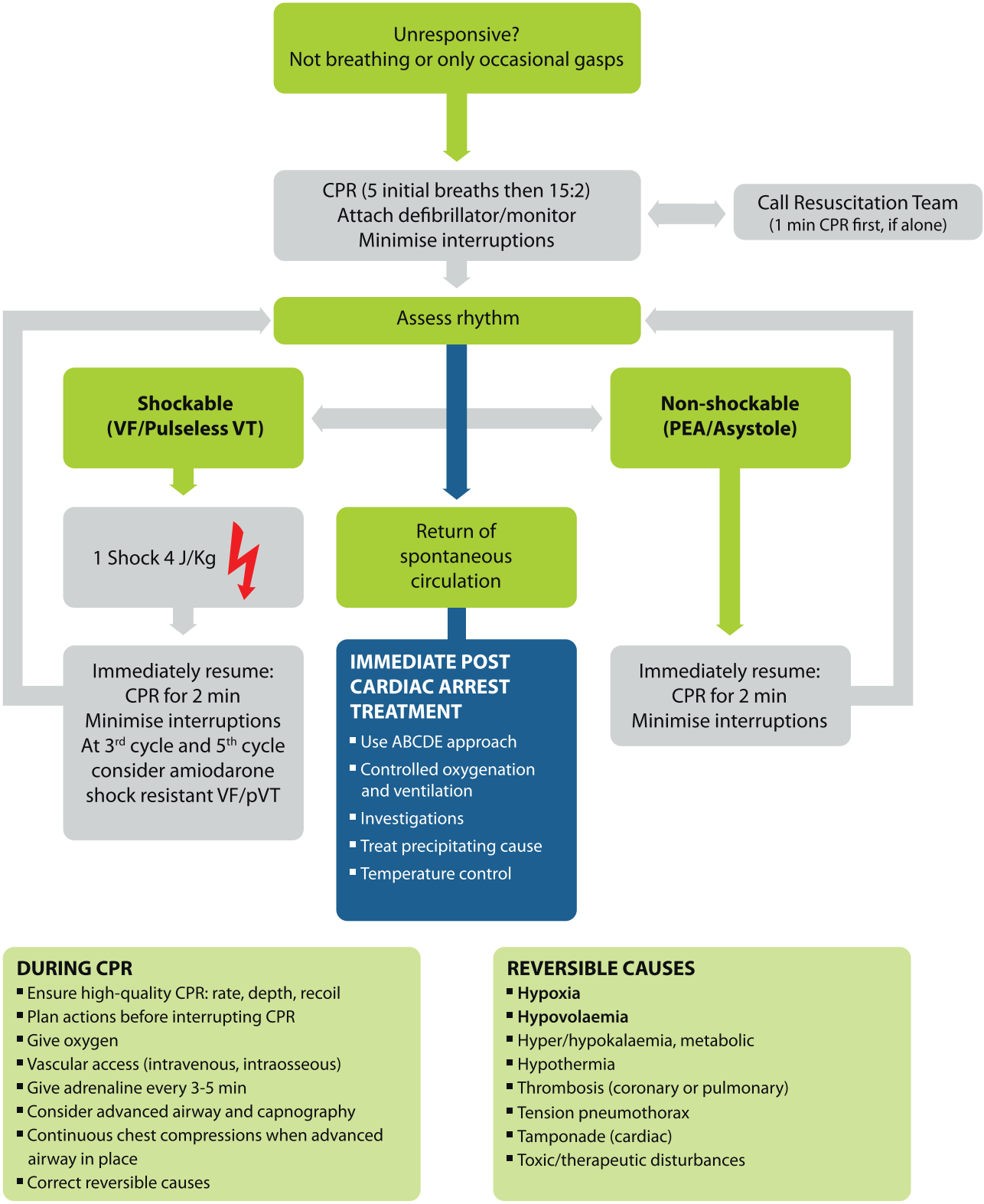


Fig. 1.25. Paediatric advanced life support algorithm.

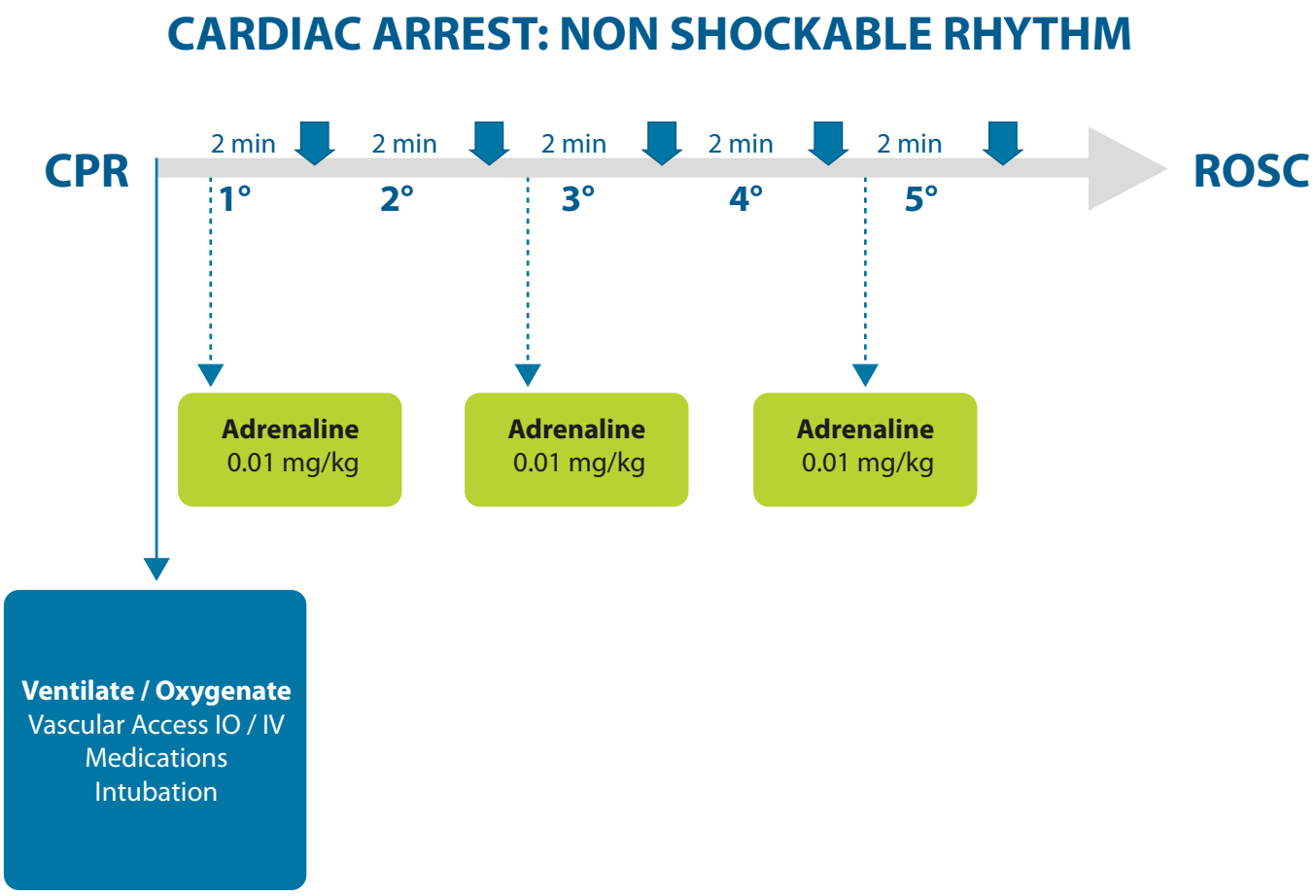


Fig. 1.26. Paediatric algorithm for non-shockable rhythm.

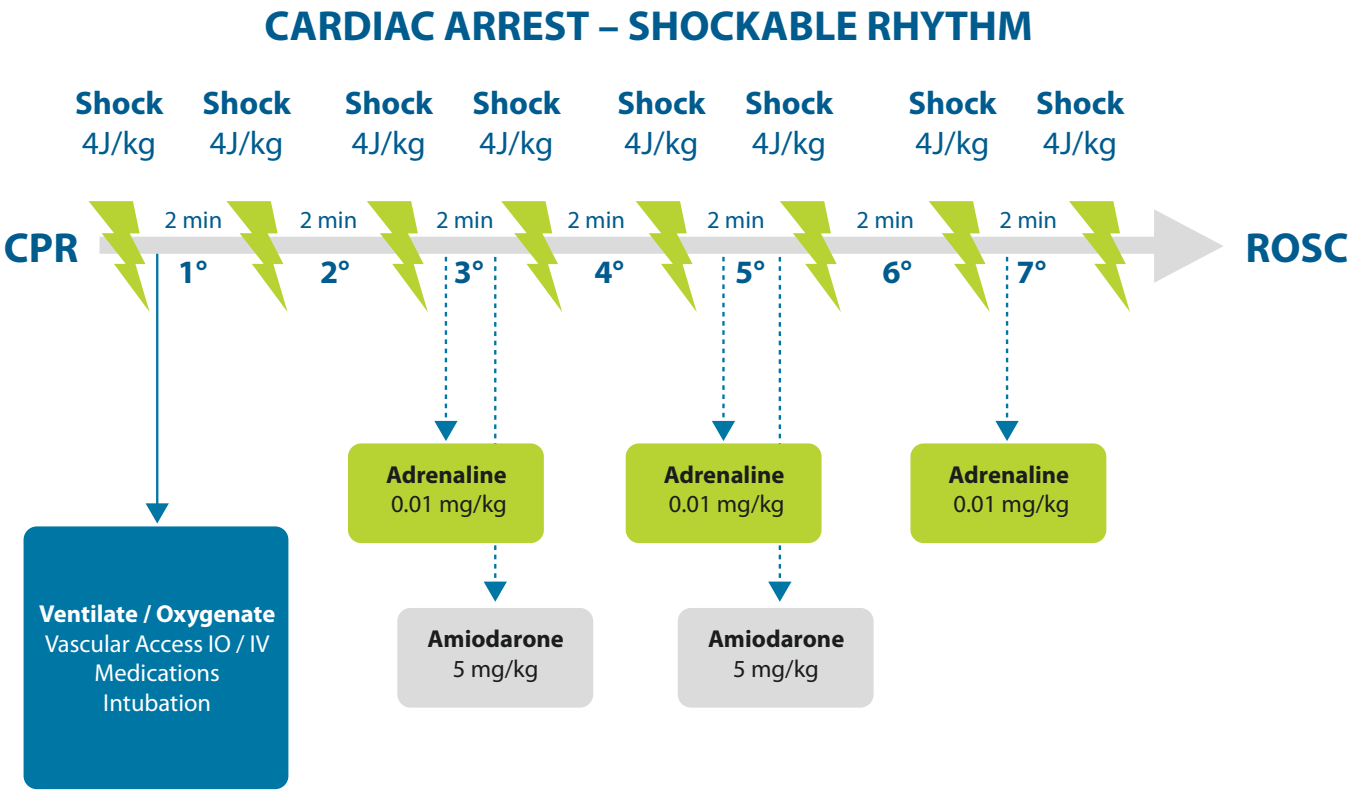


Fig. 1.27. Paediatric algorithm for shockable rhythm.

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}

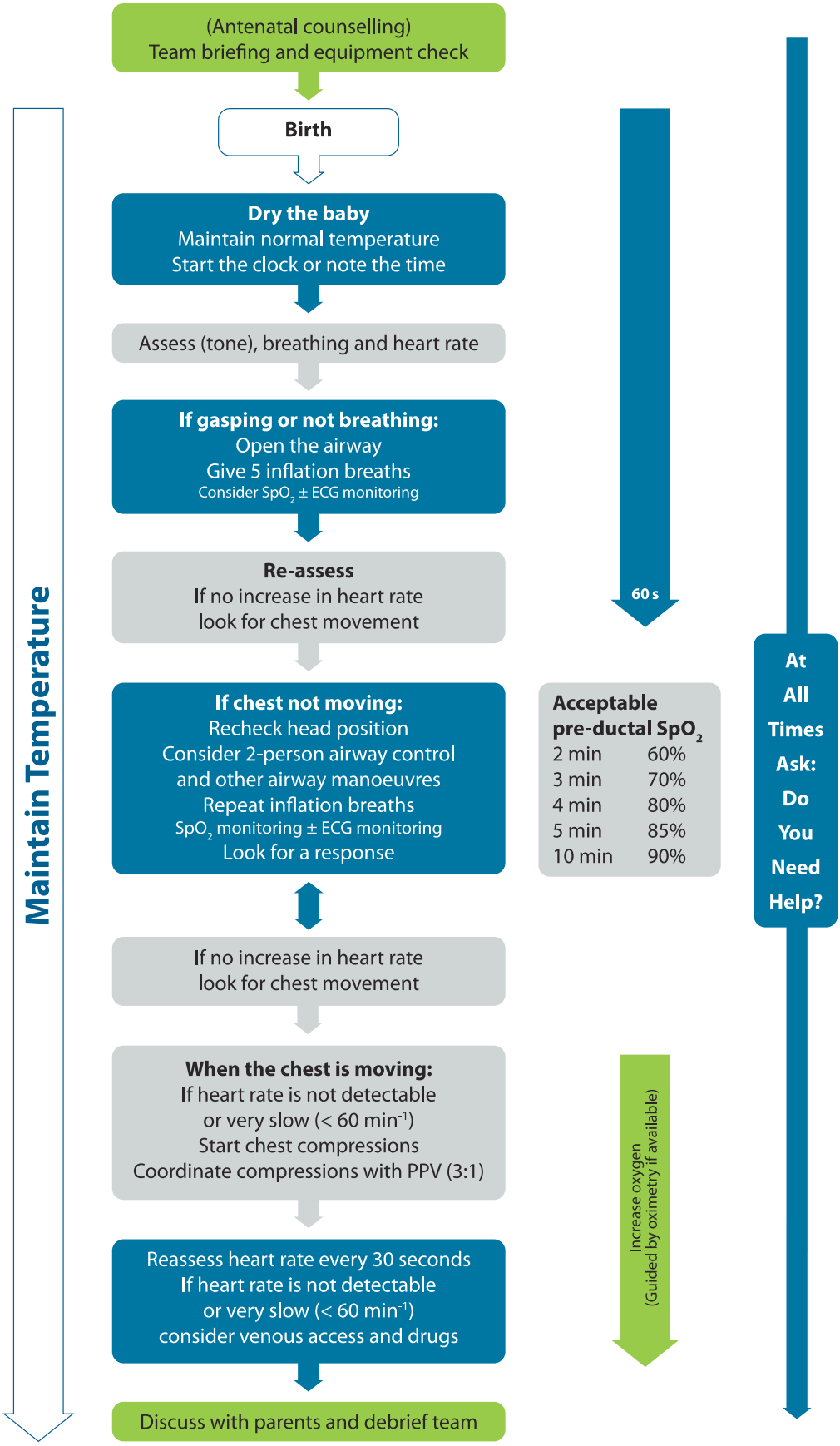


Fig. 1.28. Newborn life support algorithm (SpO_2: transcutaneous pulse oximetry, ECG: electrocardiograph, PPV: positive pressure ventilation).

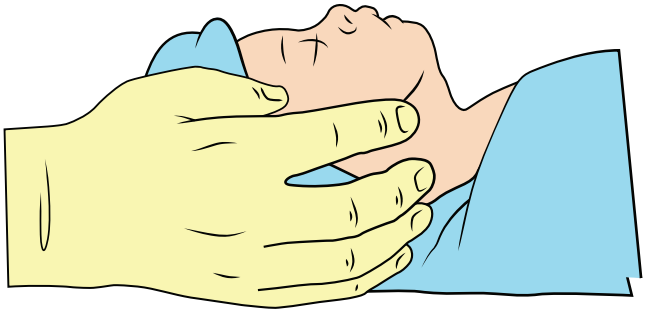


Fig. 1.29. Newborn with head in neutral position.

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

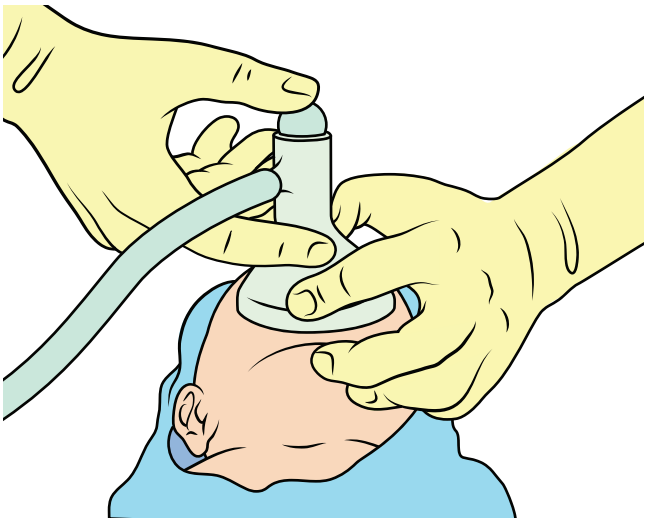


Fig. 1.30. Mask ventilation of newborn.

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]

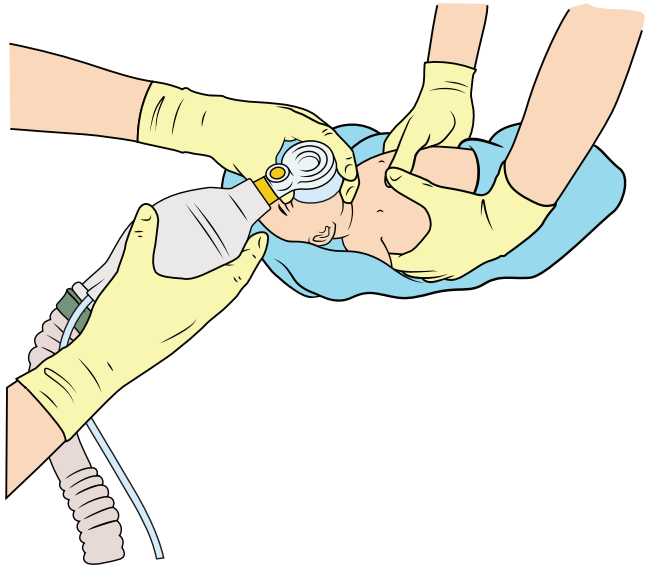


Fig. 1.31. Ventilation and chest compression of newborn.

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]

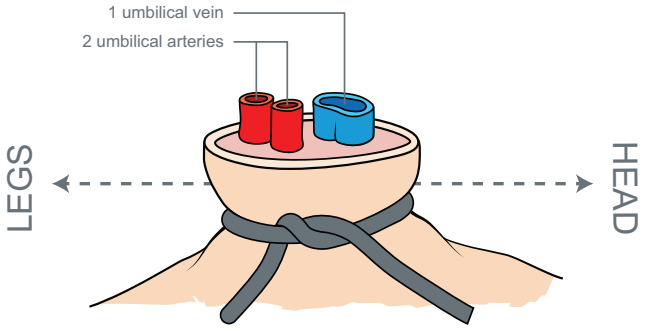


Fig. 1.32. Newborn umbilical cord showing the arteries and veins.

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

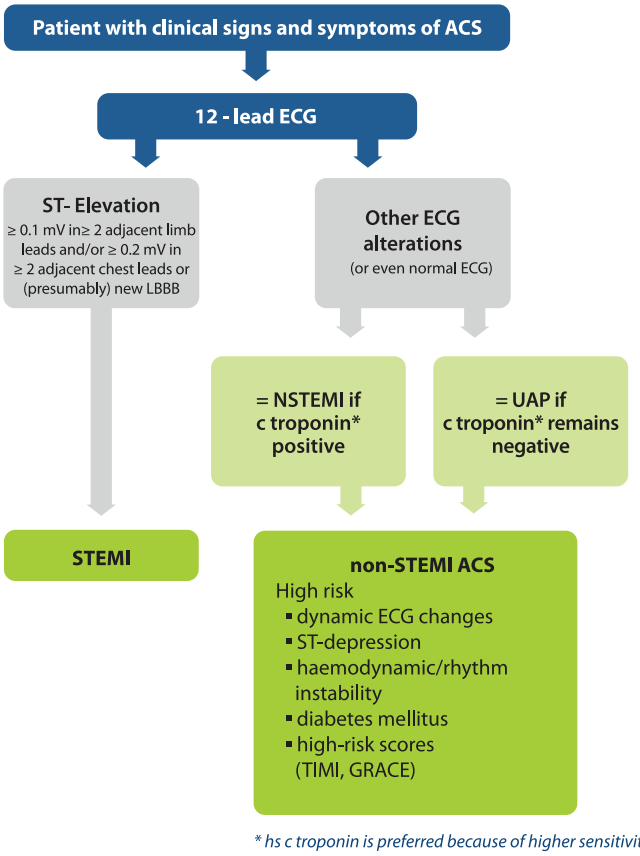


Fig. 1.33. Definitions of acute coronary syndromes (ACS); ECG, electrocardiogram; LBBB, left bundle branch block; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation acute myocardial infarction; c troponin, cardiac troponin; UAP, unstable angina pectoris; TIMI, thrombolysis in acute myocardial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

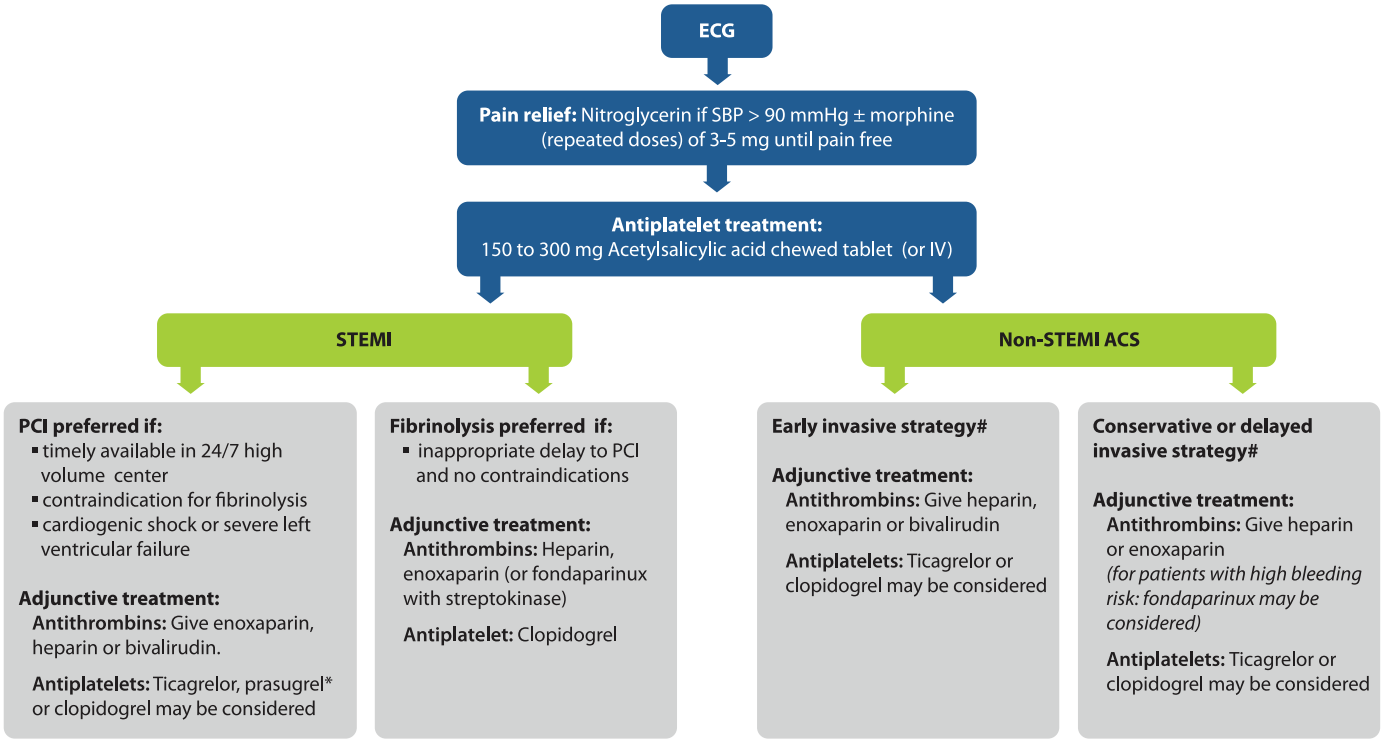


Fig. 1.34. Treatment algorithm for acute coronary syndromes; ECG, electrocardiogram; SBP, systolic blood pressure; STEMI, ST-elevation myocardial infarction; non-STEMI-ACS, non-ST-elevation acute coronary syndrome; PCI, percutaneous coronary intervention.

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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Koen Monsieurs	Section 1	Executive summary	No conflict of interest reported
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References

1. Perkins GD, Handley AJ, Koster KW, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 2. Adult basic life support and automated external defibrillation. Resuscitation 2015;95:81–98.
2. Soar J, Nolan JP, Bottiger BW, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 3. Adult advanced life support. Resuscitation 2015;95:99–146.
3. Truhlar A, Deakin CD, Soar J, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 4. Cardiac arrest in special circumstances. Resuscitation 2015;95:147–200.
4. Nolan JP, Soar J, Cariou A, et al. European Resuscitation Council and European Society of Intensive Care Medicine Guidelines for Post-resuscitation Care 2015 Section 5. Post resuscitation care. Resuscitation 2015;95:201–21.
5. Maconochie I, Bingham R, Eich C, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 6. Paediatric life support. Resuscitation 2015;95:222–47.
6. Wyllie J, Jos Bruinenberg J, Roehr CC, Rüdiger M, Trevisanuto D. B.U. European Resuscitation Council Guidelines for Resuscitation 2015 Section 7. Resuscitation and support of transition of babies at birth. Resuscitation 2015;95:248–62.
7. Nikolaou NI, Arntz HR, Bellou A, Beygui F, Bossaert LL, Cariou A. European Resuscitation Council Guidelines for Resuscitation 2015 Section 8. Initial management of acute coronary syndromes. Resuscitation 2015;95:263–76.
8. Zideman DA, De Buck EDJ, Singletary EM, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 9. First aid. Resuscitation 2015;95:277–86.
9. Greif R, Lockey AS, Conaghan P, Lippert A, De Vries W, Monsieurs KG. European Resuscitation Council Guidelines for Resuscitation 2015 Section 10. Principles of education in resuscitation. Resuscitation 2015;95:287–300.
10. Bossaert L, Perkins GD, Askitopoulou H, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 11. The ethics of resuscitation and end-of-life decisions. Resuscitation 2015;95:301–10.
11. Soreide E, Morrison L, Hillman K, et al. The formula for survival in resuscitation. Resuscitation 2013;84:1487–93.
12. Deakin CD, Nolan JP, Soar J, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 4. Adult advanced life support. Resuscitation 2010;81:1305–52.
13. Nolan J, Soar J, Eikeland H. The chain of survival. Resuscitation 2006;71:270–1.
14. Morley PT, Lang E, Aickin R, et al. Part 2: Evidence Evaluation and Management of Conflict of Interest for the ILCOR 2015. Consensus on Science and Treatment Recommendations. Resuscitation 2015;95:e33–41.
15. *GRADE handbook*. Available at: http://www.guidelinedevelopment.org/handbook/. Updated October 2013 [accessed 06.03.15].
16. Nolan JP, Hazinski MF, Aicken R, et al. Part I. Executive Summary: 2015 International Consensus on Cardiopulmonary Resudcitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2015;95:e1–32.
17. Hazinski MF, Nolan JP, Aicken R, et al. Part I. Executive Summary: 2015 International Consensus on Cardiopulmonary Resudcitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Circulation 2015.
18. Perkins GD, Travers AH, Considine J, et al. Part 3: Adult Basic Life Support and Automated External Defibrillation: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2015;95:e43–70.
19. Ringh M, Herlitz J, Hollenberg J, Rosenqvist M, Svensson L. Out of hospital cardiac arrest outside home in Sweden, change in characteristics, outcome and availability for public access defibrillation. Scand J Trauma Resusc Emerg Med 2009;17:18.
20. Hulleman M, Berdowski J, de Groot JR, et al. Implantable cardioverter-defibrillators have reduced the incidence of resuscitation for out-of-hospital cardiac arrest caused by lethal arrhythmias. Circulation 2012;126:815–21.
21. Blom MT, Beesems SG, Homma PC, et al. Improved survival after out-of-hospital cardiac arrest and use of automated external defibrillators. Circulation 2014;130:1868–75.
22. Weisfeldt ML, Sitlani CM, Ornato JP, et al. Survival after application of automatic external defibrillators before arrival of the emergency medical system: evaluation in the resuscitation outcomes consortium population of 21 million. J Am Coll Cardiol 2010;55:1713–20.
23. Berdowski J, Blom MT, Bardai A, Tan HL, Tijssen JG, Koster RW. Impact of onsite or dispatched automated external defibrillator use on survival after out-of-hospital cardiac arrest. Circulation 2011;124:2225–32.
24. Sasson C, Rogers MA, Dahl J, Kellermann AL. Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. Circ Cardiovasc Qual Outcomes 2010;3:63–81.
25. Nehme Z, Andrew E, Bernard S, Smith K. Comparison of out-of-hospital cardiac arrest occurring before and after paramedic arrival: epidemiology, survival to hospital discharge and 12-month functional recovery. Resuscitation 2015;89:50–7.
26. Takei Y, Nishi T, Kamikura T, et al. Do early emergency calls before patient collapse improve survival after out-of-hospital cardiac arrests? Resuscitation 2015;88:20–7.
27. Holmberg M, Holmberg S, Herlitz J. Factors modifying the effect of bystander cardiopulmonary resuscitation on survival in out-of-hospital cardiac arrest patients in Sweden. Eur Heart J 2001;22:511–9.
28. Wissenberg M, Lippert FK, Folke F, et al. Association of national initiatives to improve cardiac arrest management with rates of bystander intervention and patient survival after out-of-hospital cardiac arrest. JAMA 2013;310:1377–84.
29. Hasselqvist-Ax I, Riva G, Herlitz J, et al. Early cardiopulmonary resuscitation in out-of-hospital cardiac arrest. N Engl J Med 2015;372:2307–15.
30. Rea TD, Fahrenbruch C, Culley L, et al. CPR with chest compresssions alone or with rescue breathing. N Engl J Med 2010;363:423–33.
31. Svensson L, Bohm K, Castren M, et al. Compression-only CPR or standard CPR in out-of-hospital cardiac arrest. N Engl J Med 2010;363:434–42.
32. Hupfl M, Selig HF, Nagele P. Chest-compression-only versus standard cardiopulmonary resuscitation: a meta-analysis. Lancet 2010;376:1552–7.
33. Ringh M, Rosenqvist M, Hollenberg J, et al. Mobile-phone dispatch of laypersons for CPR in out-of-hospital cardiac arrest. N Engl J Med 2015;372:2316–25.
34. van Alem AP, Vrenken RH, de Vos R, Tijssen JG, Koster RW. Use of automated external defibrillator by first responders in out of hospital cardiac arrest: prospective controlled trial. BMJ 2003;327:1312.
35. Fothergill RT, Watson LR, Chamberlain D, Virdi GK, Moore FP, Whitbread M. Increases in survival from out-of-hospital cardiac arrest: a five year study. Resuscitation 2013;84:1089–92.
36. Perkins GD, Lall R, Quinn T, et al. Mechanical versus manual chest compression for out-of-hospital cardiac arrest (PARAMEDIC): a pragmatic, cluster randomised controlled trial. Lancet 2015;385:947–55.
37. Zijlstra JA, Stieglis R, Riedijk F, Smeekes M, van der Worp WE, Koster RW. Local lay rescuers with AEDs, alerted by text messages, contribute to early defibrillation in a Dutch out-of-hospital cardiac arrest dispatch system. Resuscitation 2014;85:1444–9.
38. Bahr J, Klingler H, Panzer W, Rode H, Kettler D. Skills of lay people in checking the carotid pulse. Resuscitation 1997;35:23–6.
39. Nyman J, Sihvonen M. Cardiopulmonary resuscitation skills in nurses and nursing students. Resuscitation 2000;47:179–84.
40. Tibballs J, Russell P. Reliability of pulse palpation by healthcare personnel to diagnose paediatric cardiac arrest. Resuscitation 2009;80:61–4.

41. Tibballs J, Weeranatna C. The influence of time on the accuracy of healthcare personnel to diagnose paediatric cardiac arrest by pulse palpation. Resuscitation 2010;81:671–5.
42. Moule P. Checking the carotid pulse: diagnostic accuracy in students of the healthcare professions. Resuscitation 2000;44:195–201.
43. Bobrow BJ, Zuercher M, Ewy GA, et al. Gasping during cardiac arrest in humans is frequent and associated with improved survival. Circulation 2008;118:2550–4.
44. Perkins GD, Stephenson B, Hulme J, Monsieurs KG. Birmingham assessment of breathing study (BABS). Resuscitation 2005;64:109–13.
45. Perkins GD, Walker G, Christensen K, Hulme J, Monsieurs KG. Teaching recognition of agonal breathing improves accuracy of diagnosing cardiac arrest. Resuscitation 2006;70:432–7.
46. Breckwoldt J, Schloesser S, Arntz HR. Perceptions of collapse and assessment of cardiac arrest by bystanders of out-of-hospital cardiac arrest (OOHCA). Resuscitation 2009;80:1108–13.
47. Stecker EC, Reinier K, Uy-Evanado A, et al. Relationship between seizure episode and sudden cardiac arrest in patients with epilepsy: a community-based study. Circ Arrhythm Electrophysiol 2013;6:912–6.
48. Dami F, Fuchs V, Praz L, Vader JP. Introducing systematic dispatcher-assisted cardiopulmonary resuscitation (telephone-CPR) in a non-Advanced Medical Priority Dispatch System (AMPDS): implementation process and costs. Resuscitation 2010;81:848–52.
49. Nurmi J, Pettila V, Biber B, Kuisma M, Komulainen R, Castren M. Effect of protocol compliance to cardiac arrest identification by emergency medical dispatchers. Resuscitation 2006;70:463–9.
50. Lewis M, Stubbs BA, Eisenberg MS. Dispatcher-assisted cardiopulmonary resuscitation: time to identify cardiac arrest and deliver chest compression instructions. Circulation 2013;128:1522–30.
51. Hauff SR, Rea TD, Culley LL, Kerry F, Becker L, Eisenberg MS. Factors impeding dispatcher-assisted telephone cardiopulmonary resuscitation. Ann Emerg Med 2003;42:731–7.
52. Bohm K, Stalhandske B, Rosenqvist M, Ulfvarson J, Hollenberg J, Svensson L. Tuition of emergency medical dispatchers in the recognition of agonal respiration increases the use of telephone assisted CPR. Resuscitation 2009;80:1025–8.
53. Bohm K, Rosenqvist M, Hollenberg J, Biber B, Engerstrom L, Svensson L. Dispatcher-assisted telephone-guided cardiopulmonary resuscitation: an underused lifesaving system. Eur J Emerg Med: Off J Eur Soc Emerg Med 2007;14:256–9.
54. Bång A, Herlitz J, Martinell S. Interaction between emergency medical dispatcher and caller in suspected out-of-hospital cardiac arrest calls with focus on agonal breathing. A review of 100 tape recordings of true cardiac arrest cases. Resuscitation 2003;56:25–34.
55. Roppolo LP, Westfall A, Pepe PE, et al. Dispatcher assessments for agonal breathing improve detection of cardiac arrest. Resuscitation 2009;80:769–72.
56. Vaillancourt C, Verma A, Trickett J, et al. Evaluating the effectiveness of dispatch-assisted cardiopulmonary resuscitation instructions. Acad Emerg Med: Off J Soc Acad Emerg Med 2007;14:877–83.
57. Tanaka Y, Taniguchi J, Wato Y, Yoshida Y, Inaba H. The continuous quality improvement project for telephone-assisted instruction of cardiopulmonary resuscitation increased the incidence of bystander CPR and improved the outcomes of out-of-hospital cardiac arrests. Resuscitation 2012;83:1235–41.
58. Clawson J, Olola C, Heward A, Patterson B. Cardiac arrest predictability in seizure patients based on emergency medical dispatcher identification of previous seizure or epilepsy history. Resuscitation 2007;75:298–304.
59. Eisenberg MS, Hallstrom AP, Carter WB, Cummins RO, Bergner L, Pierce J. Emergency CPR instruction via telephone. Am J Public Health 1985;75:47–50.
60. Akahane M, Ogawa T, Tanabe S, et al. Impact of telephone dispatcher assistance on the outcomes of pediatric out-of-hospital cardiac arrest. Crit Care Med 2012;40:1410–6.
61. Bray JE, Deasy C, Walsh J, Bacon A, Currell A, Smith K. Changing EMS dispatcher CPR instructions to 400 compressions before mouth-to-mouth improved bystander CPR rates. Resuscitation 2011;82:1393–8.
62. Culley LL, Clark JJ, Eisenberg MS, Larsen MP. Dispatcher-assisted telephone CPR: common delays and time standards for delivery. Ann Emerg Med 1991;20:362–6.
63. Stipulante S, Tubes R, El Fassi M, et al. Implementation of the ALERT algorithm, a new dispatcher-assisted telephone cardiopulmonary resuscitation protocol, in non-Advanced Medical Priority Dispatch System (AMPDS) Emergency Medical Services centres. Resuscitation 2014;85:177–81.
64. Rea TD, Eisenberg MS, Culley LL, Becker L. Dispatcher-assisted cardiopulmonary resuscitation and survival in cardiac arrest. Circulation 2001;104:2513–6.
65. Hallstrom AP. Dispatcher-assisted "phone" cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation. Crit Care Med 2000;28:N190–2.
66. Stromsoe A, Svensson L, Axelsson AB, et al. Improved outcome in Sweden after out-of-hospital cardiac arrest and possible association with improvements in every link in the chain of survival. Eur Heart J 2015;36:863–71.
67. Takei Y, Inaba H, Yachida T, Enami M, Goto Y, Ohta K. Analysis of reasons for emergency call delays in Japan in relation to location: high incidence of correctable causes and the impact of delays on patient outcomes. Resuscitation 2010;81:1492–8.
68. Herlitz J, Engdahl J, Svensson L, Young M, Angquist KA, Holmberg S. A short delay from out of hospital cardiac arrest to call for ambulance increases survival. Eur Heart J 2003;24:1750–5.
69. Nehme Z, Andrew E, Cameron P, et al. Direction of first bystander call for help is associated with outcome from out-of-hospital cardiac arrest. Resuscitation 2014;85:42–8.
70. Cha KC, Kim HJ, Shin HJ, Kim H, Lee KH, Hwang SO. Hemodynamic effect of external chest compressions at the lower end of the sternum in cardiac arrest patients. J Emerg Med 2013;44:691–7.
71. Qvigstad E, Kramer-Johansen J, Tomte O, et al. Clinical pilot study of different hand positions during manual chest compressions monitored with capnography. Resuscitation 2013;84:1203–7.
72. Orlowski JP. Optimum position for external cardiac compression in infants and young children. Ann Emerg Med 1986;15:667–73.
73. Chamberlain D, Smith A, Colquhoun M, Handley AJ, Kern KB, Woollard M. Randomised controlled trials of staged teaching for basic life support. 2: Comparison of CPR performance and skill retention using either staged instruction or conventional training. Resuscitation 2001;50:27–37.
74. Handley AJ. Teaching hand placement for chest compression – a simpler technique. Resuscitation 2002;53:29–36.
75. Handley AJ, Handley JA. Performing chest compressions in a confined space. Resuscitation 2004;61:55–61.
76. Perkins GD, Stephenson BT, Smith CM, Gao F. A comparison between over-the-head and standard cardiopulmonary resuscitation. Resuscitation 2004;61:155–61.
77. Hostler D, Everson-Stewart S, Rea TD, et al. Effect of real-time feedback during cardiopulmonary resuscitation outside hospital: prospective, cluster-randomised trial. BMJ 2011;342:d512.
78. Stiell IG, Brown SP, Christenson J, et al. What is the role of chest compression depth during out-of-hospital cardiac arrest resuscitation? Crit Care Med 2012;40:1192–8.
79. Stiell IG, Brown SP, Nichol G, et al. What is the optimal chest compression depth during out-of-hospital cardiac arrest resuscitation of adult patients? Circulation 2014;130:1962–70.
80. Vadeboncoeur T, Stolz U, Panchal A, et al. Chest compression depth and survival in out-of-hospital cardiac arrest. Resuscitation 2014;85:182–8.
81. Hellevuo H, Sainio M, Nevalainen R, et al. Deeper chest compression – more complications for cardiac arrest patients? Resuscitation 2013;84:760–5.
82. Idris AH, Guffey D, Pepe PE, et al. Chest compression rates and survival following out-of-hospital cardiac arrest. Crit Care Med 2015;43:840–8.
83. Idris AH, Guffey D, Aufderheide TP, et al. Relationship between chest compression rates and outcomes from cardiac arrest. Circulation 2012;125:3004–12.
84. Cheskes S, Schmicker RH, Verbeek PR, et al. The impact of peri-shock pause on survival from out-of-hospital shockable cardiac arrest during the Resuscitation Outcomes Consortium PRIMED trial. Resuscitation 2014;85:336–42.
85. Cheskes S, Schmicker RH, Christenson J, et al. Perishock pause: an independent predictor of survival from out-of-hospital shockable cardiac arrest. Circulation 2011;124:58–66.
86. Vaillancourt C, Everson-Stewart S, Christenson J, et al. The impact of increased chest compression fraction on return of spontaneous circulation for out-of-hospital cardiac arrest patients not in ventricular fibrillation. Resuscitation 2011;82:1501–7.
87. Sell RE, Sarno R, Lawrence B, et al. Minimizing pre- and post-defibrillation pauses increases the likelihood of return of spontaneous circulation (ROSC). Resuscitation 2010;81:822–5.
88. Christenson J, Andrusiek D, Everson-Stewart S, et al. Chest compression fraction determines survival in patients with out-of-hospital ventricular fibrillation. Circulation 2009;120:1241–7.
89. Delvaux AB, Trombley MT, Rivet CJ, et al. Design and development of a cardiopulmonary resuscitation mattress. J Intensive Care Med 2009;24:195–9.
90. Nishisaki A, Maltese MR, Niles DE, et al. Backboards are important when chest compressions are provided on a soft mattress. Resuscitation 2012;83:1013–20.
91. Sato H, Komasawa N, Ueki R, et al. Backboard insertion in the operating table increases chest compression depth: a manikin study. J Anesth 2011;25:770–2.
92. Perkins GD, Smith CM, Augre C, et al. Effects of a backboard, bed height, and operator position on compression depth during simulated resuscitation. Intensive Care Med 2006;32:1632–5.
93. Perkins GD, Kocierz L, Smith SC, McCulloch RA, Davies RP. Compression feedback devices over estimate chest compression depth when performed on a bed. Resuscitation 2009;80:79–82.
94. Cloete G, Dellimore KH, Scheffer C, Smuts MS, Wallis LA. The impact of backboard size and orientation on sternum-to-spine compression depth and compression stiffness in a manikin study of CPR using two mattress types. Resuscitation 2011;82:1064–70.
95. Niles DE, Sutton RM, Nadkarni VM, et al. Prevalence and hemodynamic effects of leaning during CPR. Resuscitation 2011;82:S23–6.
96. Zuercher M, Hilwig RW, Ranger-Moore J, et al. Leaning during chest compressions impairs cardiac output and left ventricular myocardial blood flow in piglet cardiac arrest. Crit Care Med 2010;38:1141–6.
97. Aufderheide TP, Pirrallo RG, Yannopoulos D, et al. Incomplete chest wall decompression: a clinical evaluation of CPR performance by EMS personnel and assessment of alternative manual chest compression–decompression techniques. Resuscitation 2005;64:353–62.

98. Yannopoulos D, McKnite S, Aufderheide TP, et al. Effects of incomplete chest wall decompression during cardiopulmonary resuscitation on coronary and cerebral perfusion pressures in a porcine model of cardiac arrest. Resuscitation 2005;64:363–72.
99. Couper K, Salman B, Soar J, Finn J, Perkins GD. Debriefing to improve outcomes from critical illness: a systematic review and meta-analysis. Intensive Care Med 2013;39:1513–23.
100. Couper K, Kimani PK, Abella BS, et al. The system-wide effect of real-time audiovisual feedback and postevent debriefing for in-hospital cardiac arrest: the cardiopulmonary resuscitation quality improvement initiative. Crit Care Med 2015 [in press].
101. Baskett P, Nolan J, Parr M. Tidal volumes which are perceived to be adequate for resuscitation. Resuscitation 1996;31:231–4.
102. Beesems SG, Wijmans L, Tijssen JG, Koster RW. Duration of ventilations during cardiopulmonary resuscitation by lay rescuers and first responders: relationship between delivering chest compressions and outcomes. Circulation 2013;127:1585–90.
103. Sayre MR, Cantrell SA, White LJ, Hiestand BC, Keseg DP, Koser S. Impact of the 2005 American Heart Association cardiopulmonary resuscitation and emergency cardiovascular care guidelines on out-of-hospital cardiac arrest survival. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2009;13:469–77.
104. Steinmetz J, Barnung S, Nielsen SL, Risom M, Rasmussen LS. Improved survival after an out-of-hospital cardiac arrest using new guidelines. Acta Anaesthesiol Scand 2008;52:908–13.
105. Olasveengen TM, Vik E, Kuzovlev A, Sunde K. Effect of implementation of new resuscitation guidelines on quality of cardiopulmonary resuscitation and survival. Resuscitation 2009;80:407–11.
106. Hinchey PR, Myers JB, Lewis R, et al. Improved out-of-hospital cardiac arrest survival after the sequential implementation of 2005 AHA guidelines for compressions, ventilations, and induced hypothermia: the Wake County experience. Ann Emerg Med 2010;56:348–57.
107. Panchal AR, Bobrow BJ, Spaite DW, et al. Chest compression-only cardiopulmonary resuscitation performed by lay rescuers for adult out-of-hospital cardiac arrest due to non-cardiac aetiologies. Resuscitation 2013;84:435–9.
108. Kitamura T, Iwami T, Kawamura T, et al. Time-dependent effectiveness of chest compression-only and conventional cardiopulmonary resuscitation for out-of-hospital cardiac arrest of cardiac origin. Resuscitation 2011;82:3–9.
109. Mohler MJ, Wendel CS, Mosier J, et al. Cardiocerebral resuscitation improves out-of-hospital survival in older adults. J Am Geriatr Soc 2011;59:822–6.
110. Bobrow BJ, Spaite DW, Berg RA, et al. Chest compression-only CPR by lay rescuers and survival from out-of-hospital cardiac arrest. JAMA 2010;304:1447–54.
111. Kitamura T, Iwami T, Kawamura T, Nagao K, Tanaka H, Hiraide A. Bystander-initiated rescue breathing for out-of-hospital cardiac arrests of noncardiac origin. Circulation 2010;122:293–9.
112. Ong ME, Ng FS, Anushia P, et al. Comparison of chest compression only and standard cardiopulmonary resuscitation for out-of-hospital cardiac arrest in Singapore. Resuscitation 2008;78:119–26.
113. Bohm K, Rosenqvist M, Herlitz J, Hollenberg J, Svensson L. Survival is similar after standard treatment and chest compression only in out-of-hospital bystander cardiopulmonary resuscitation. Circulation 2007;116:2908–12.
114. SOS-KANTO Study Group. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): an observational study. Lancet 2007;369:920–6.
115. Iwami T, Kawamura T, Hiraide A, et al. Effectiveness of bystander-initiated cardiac-only resuscitation for patients with out-of-hospital cardiac arrest. Circulation 2007;116:2900–7.
116. Bossaert L, Van Hoeyweghen R. Evaluation of cardiopulmonary resuscitation (CPR) techniques. The Cerebral Resuscitation Study Group. Resuscitation 1989;17 Suppl.:S99–109 [discussion S99–206].
117. Gallagher EJ, Lombardi G, Gennis P. Effectiveness of bystander cardiopulmonary resuscitation and survival following out-of-hospital cardiac arrest. JAMA 1995;274:1922–5.
118. Olasveengen TM, Wik L, Steen PA. Standard basic life support vs. continuous chest compressions only in out-of-hospital cardiac arrest. Acta Anaesthesiol Scand 2008;52:914–9.
119. Kitamura T, Iwami T, Kawamura T, et al. Conventional and chest-compression-only cardiopulmonary resuscitation by bystanders for children who have out-of-hospital cardiac arrests: a prospective, nationwide, population-based cohort study. Lancet 2010;375:1347–54.
120. Goto Y, Maeda T, Goto Y. Impact of dispatcher-assisted bystander cardiopulmonary resuscitation on neurological outcomes in children with out-of-hospital cardiac arrests: a prospective, nationwide, population-based cohort study. J Am Heart Assoc 2014;3:e000499.
121. Yeung J, Okamoto D, Soar J, Perkins GD. AED training and its impact on skill acquisition, retention and performance – a systematic review of alternative training methods. Resuscitation 2011;82:657–64.
122. Mitani Y, Ohta K, Yodoya N, et al. Public access defibrillation improved the outcome after out-of-hospital cardiac arrest in school-age children: a nationwide, population-based, Utstein registry study in Japan. Europace 2013;15:1259–66.
123. Johnson MA, Grahan BJ, Haukoos JS, et al. Demographics, bystander CPR, and AED use in out-of-hospital pediatric arrests. Resuscitation 2014;85:920–6.
124. Akahane M, Tanabe S, Ogawa T, et al. Characteristics and outcomes of pediatric out-of-hospital cardiac arrest by scholastic age category. Pediatr Crit Care Med: J Soc Crit Care Med World Feder Pediatr Intensive Crit Care Soc 2013;14:130–6.
125. Nichol G, Valenzuela T, Roe D, Clark L, Huszti E, Wells GA. Cost effectiveness of defibrillation by targeted responders in public settings. Circulation 2003;108:697–703.
126. Nichol G, Huszti E, Birnbaum A, et al. Cost-effectiveness of lay responder defibrillation for out-of-hospital cardiac arrest. Ann Emerg Med 2009;54:226–35, e1–2.
127. Folke F, Lippert FK, Nielsen SL, et al. Location of cardiac arrest in a city center: strategic placement of automated external defibrillators in public locations. Circulation 2009;120:510–7.
128. Hansen CM, Lippert FK, Wissenberg M, et al. Temporal trends in coverage of historical cardiac arrests using a volunteer-based network of automated external defibrillators accessible to laypersons and emergency dispatch centers. Circulation 2014;130:1859–67.
129. Weisfeldt ML, Everson-Stewart S, Sitlani C, et al. Ventricular tachyarrhythmias after cardiac arrest in public versus at home. N Engl J Med 2011;364:313–21.
130. The Public Access Defibrillation Trial Investigators. Public-access defibrillation and survival after out-of-hospital cardiac arrest. N Engl J Med 2004;351:637–46.
131. ILCOR presents a universal AED sign. European Resuscitation Council; 2008. From: https://www.erc.edu/index.php/newsItem/en/nid=204/ [accessed 28.06.15].
132. Forcina MS, Farhat AY, O'Neil WW, Haines DE. Cardiac arrest survival after implementation of automated external defibrillator technology in the in-hospital setting. Crit Care Med 2009;37:1229–36.
133. Smith RJ, Hickey BB, Santamaria JD. Automated external defibrillators and survival after in-hospital cardiac arrest: early experience at an Australian teaching hospital. Crit Care Resusc 2009;11:261–5.
134. Smith RJ, Hickey BB, Santamaria JD. Automated external defibrillators and in-hospital cardiac arrest: patient survival and device performance at an Australian teaching hospital. Resuscitation 2011;82:1537–42.
135. Chan PS, Krumholz HM, Spertus JA, et al. Automated external defibrillators and survival after in-hospital cardiac arrest. JAMA 2010;304:2129–36.
136. Gibbison B, Soar J. Automated external defibrillator use for in-hospital cardiac arrest is not associated with improved survival. Evid Based Med 2011;16:95–6.
137. Chan PS, Krumholz HM, Nichol G, Nallamothu BK. Delayed time to defibrillation after in-hospital cardiac arrest. N Engl J Med 2008;358:9–17.
138. Fingerhut LA, Cox CS, Warner M. International comparative analysis of injury mortality. Findings from the ICE on injury statistics. International Collaborative Effort on Injury Statistics. Adv Data 1998:1–20.
139. Proceedings of the 2005 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation 2005;67:157–341.
140. Langhelle A, Sunde K, Wik L, Steen PA. Airway pressure with chest compressions versus Heimlich manoeuvre in recently dead adults with complete airway obstruction. Resuscitation 2000;44:105–8.
141. Guildner CW, Williams D, Subitch T. Airway obstructed by foreign material: the Heimlich maneuver. JACEP 1976;5:675–7.
142. Ruben H, Macnaughton FI. The treatment of food-choking. Practitioner 1978;221:725–9.
143. Sandroni C, Nolan J, Cavallaro F, Antonelli M. In-hospital cardiac arrest: incidence, prognosis and possible measures to improve survival. Intensive Care Med 2007;33:237–45.
144. Nolan JP, Soar J, Smith GB, et al. Incidence and outcome of in-hospital cardiac arrest in the United Kingdom National Cardiac Arrest Audit. Resuscitation 2014;85:987–92.
145. Smith GB. In-hospital cardiac arrest: is it time for an in-hospital 'chain of prevention'? Resuscitation 2010.
146. Muller D, Agrawal R, Arntz HR. How sudden is sudden cardiac death? Circulation 2006;114:1146–50.
147. Winkel BG, Risgaard B, Sadjadieh G, Bundgaard H, Haunso S, Tfelt-Hansen J. Sudden cardiac death in children (1–18 years): symptoms and causes of death in a nationwide setting. Eur Heart J 2014;35:868–75.
148. Harmon KG, Drezner JA, Wilson MG, Sharma S. Incidence of sudden cardiac death in athletes: a state-of-the-art review. Heart 2014;100:1227–34.
149. Basso C, Carturan E, Pilichou K, Rizzo S, Corrado D, Thiene G. Sudden cardiac death with normal heart: molecular autopsy. Cardiovasc Pathol 2010;19:321–5.
150. Mazzanti A, O'Rourke S, Ng K, et al. The usual suspects in sudden cardiac death of the young: a focus on inherited arrhythmogenic diseases. Expert Rev Cardiovasc Ther 2014;12:499–519.
151. Goldberger JJ, Basu A, Boineau R, et al. Risk stratification for sudden cardiac death: a plan for the future. Circulation 2014;129:516–26.
152. Corrado D, Drezner J, Basso C, Pelliccia A, Thiene G. Strategies for the prevention of sudden cardiac death during sports. Eur J Cardiovasc Prev Rehabil: Off J Eur Soc Cardiol Work Groups Epidemiol Prev Cardiac Rehabil Exerc Physiol 2011;18:197–208.
153. Mahmood S, Lim L, Akram Y, Alford-Morales S, Sherin K, Committee APP. Screening for sudden cardiac death before participation in high school and collegiate sports: American College of Preventive Medicine position statement on preventive practice. Am J Prev Med 2013;45:130–3.
154. Skinner JR. Investigating sudden unexpected death in the young: a chance to prevent further deaths. Resuscitation 2012;83:1185–6.

155. Skinner JR. Investigation following resuscitated cardiac arrest. Arch Dis Child 2013;98:66–71.
156. Vriesendorp PA, Schinkel AF, Liebregts M, et al. Validation of the 2014 ESC guidelines risk prediction model for the primary prevention of sudden cardiac death in hypertrophic cardiomyopathy. Circ Arrhythm Electrophysiol 2015.
157. Morrison LJ, Visentin LM, Kiss A, et al. Validation of a rule for termination of resuscitation in out-of-hospital cardiac arrest. N Engl J Med 2006;355:478–87.
158. Richman PB, Vadeboncoeur TF, Chikani V, Clark L, Bobrow BJ. Independent evaluation of an out-of-hospital termination of resuscitation (TOR) clinical decision rule. Acad Emerg Med: Off J Soc Acad Emerg Med 2008;15:517–21.
159. Morrison LJ, Verbeek PR, Zhan C, Kiss A, Allan KS. Validation of a universal prehospital termination of resuscitation clinical prediction rule for advanced and basic life support providers. Resuscitation 2009;80:324–8.
160. Sasson C, Hegg AJ, Macy M, Park A, Kellermann A, McNally B. Prehospital termination of resuscitation in cases of refractory out-of-hospital cardiac arrest. JAMA 2008;300:1432–8.
161. Morrison LJ, Eby D, Veigas PV, et al. Implementation trial of the basic life support termination of resuscitation rule: reducing the transport of futile out-of-hospital cardiac arrests. Resuscitation 2014;85:486–91.
162. Skrifvars MB, Vayrynen T, Kuisma M, et al. Comparison of Helsinki and European Resuscitation Council "do not attempt to resuscitate" guidelines, and a termination of resuscitation clinical prediction rule for out-of-hospital cardiac arrest patients found in asystole or pulseless electrical activity. Resuscitation 2010;81:679–84.
163. Fukuda T, Ohashi N, Matsubara T, et al. Applicability of the prehospital termination of resuscitation rule in an area dense with hospitals in Tokyo: a single-center, retrospective, observational study: is the pre hospital TOR rule applicable in Tokyo? Am J Emerg Med 2014;32:144–9.
164. Chiang WC, Ko PC, Chang AM, et al. Predictive performance of universal termination of resuscitation rules in an Asian community: are they accurate enough? Emerg Med J 2015;32:318–23.
165. Diskin FJ, Camp-Rogers T, Peberdy MA, Ornato JP, Kurz MC. External validation of termination of resuscitation guidelines in the setting of intra-arrest cold saline, mechanical CPR, and comprehensive post resuscitation care. Resuscitation 2014;85:910–4.
166. Drennan IR, Lin S, Sidalak DE, Morrison LJ. Survival rates in out-of-hospital cardiac arrest patients transported without prehospital return of spontaneous circulation: an observational cohort study. Resuscitation 2014;85:1488–93.
167. Brennan RT, Braslow A. Skill mastery in public CPR classes. Am J Emerg Med 1998;16:653–7.
168. Chamberlain D, Smith A, Woollard M, et al. Trials of teaching methods in basic life support (3): comparison of simulated CPR performance after first training and at 6 months, with a note on the value of re-training. Resuscitation 2002;53:179–87.
169. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. Resuscitation 1996;33:107–16.
170. Lapostolle F, Le Toumelin P, Agostinucci JM, Catineau J, Adnet F. Basic cardiac life support providers checking the carotid pulse: performance, degree of conviction, and influencing factors. Acad Emerg Med: Off J Soc Acad Emerg Med 2004;11:878–80.
171. Liberman M, Lavoie A, Mulder D, Sampalis J. Cardiopulmonary resuscitation: errors made by pre-hospital emergency medical personnel. Resuscitation 1999;42:47–55.
172. Ruppert M, Reith MW, Widmann JH, et al. Checking for breathing: evaluation of the diagnostic capability of emergency medical services personnel, physicians, medical students, and medical laypersons. Ann Emerg Med 1999;34:720–9.
173. White L, Rogers J, Bloomingdale M, et al. Dispatcher-assisted cardiopulmonary resuscitation: risks for patients not in cardiac arrest. Circulation 2010;121:91–7.
174. Sheak KR, Wiebe DJ, Leary M, et al. Quantitative relationship between end-tidal carbon dioxide and CPR quality during both in-hospital and out-of-hospital cardiac arrest. Resuscitation 2015;89:149–54.
175. Soar J, Callaway CW, Aibiki M, et al. Part 4: Advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e71–122.
176. Edelson DP, Robertson-Dick BJ, Yuen TC, et al. Safety and efficacy of defibrillator charging during ongoing chest compressions: a multi-center study. Resuscitation 2010;81:1521–6.
177. Hansen LK, Mohammed A, Pedersen M, et al. Eur J Emerg Med 2015.
178. Featherstone P, Chalmers T, Smith GB. RSVP: a system for communication of deterioration in hospital patients. Br J Nurs 2008;17:860–4.
179. Marshall S, Harrison J, Flanagan B. The teaching of a structured tool improves the clarity and content of interprofessional clinical communication. Qual Saf Health Care 2009;18:137–40.
180. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. JAMA 2005;293:305–10.
181. Abella BS, Sandbo N, Vassilatos P, et al. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. Circulation 2005;111:428–34.
182. Pokorna M, Necas E, Kratochvil J, Skripsky R, Andrlik M, Franek O. A sudden increase in partial pressure end-tidal carbon dioxide (P(ET)CO(2)) at the moment of return of spontaneous circulation. J Emerg Med 2010;38:614–21.
183. Heradstveit BE, Sunde K, Sunde GA, Wentzel-Larsen T, Heltne JK. Factors complicating interpretation of capnography during advanced life support in cardiac arrest – a clinical retrospective study in 575 patients. Resuscitation 2012;83:813–8.
184. Davis DP, Sell RE, Wilkes N, et al. Electrical and mechanical recovery of cardiac function following out-of-hospital cardiac arrest. Resuscitation 2013;84:25–30.
185. Stiell IG, Wells GA, Field B, et al. Advanced cardiac life support in out-of-hospital cardiac arrest. N Engl J Med 2004;351:647–56.
186. Olasveengen TM, Sunde K, Brunborg C, Thowsen J, Steen PA, Wik L. Intravenous drug administration during out-of-hospital cardiac arrest: a randomized trial. JAMA 2009;302:2222–9.
187. Herlitz J, Ekstrom L, Wennerblom B, Axelsson A, Bang A, Holmberg S. Adrenaline in out-of-hospital ventricular fibrillation. Does it make any difference? Resuscitation 1995;29:195–201.
188. Holmberg M, Holmberg S, Herlitz J. Low chance of survival among patients requiring adrenaline (epinephrine) or intubation after out-of-hospital cardiac arrest in Sweden. Resuscitation 2002;54:37–45.
189. Jacobs IG, Finn JC, Jelinek GA, Oxer HF, Thompson PL. Effect of adrenaline on survival in out-of-hospital cardiac arrest: a randomised double-blind placebo-controlled trial. Resuscitation 2011;82:1138–43.
190. Benoit JL, Gerecht RB, Steuerwald MT, McMullan JT. Endotracheal intubation versus supraglottic airway placement in out-of-hospital cardiac arrest: a meta-analysis. Resuscitation 2015;93:20–6.
191. Perkins GD, Nolan JP. Early adrenaline for cardiac arrest. BMJ 2014;348:g3245.
192. Soar J, Nolan JP. Airway management in cardiopulmonary resuscitation. Curr Opin Crit Care 2013;19:181–7.
193. Lexow K, Sunde K. Why Norwegian 2005 guidelines differs slightly from the ERC guidelines. Resuscitation 2007;72:490–2.
194. Deakin CD, Nolan JP, Sunde K, Koster RW. European Resuscitation Council Guidelines for Resuscitation 2010 Section 3. Electrical therapies: automated external defibrillators, defibrillation, cardioversion and pacing. Resuscitation 2010;81:1293–304.
195. Koster RW, Walker RG, Chapman FW. Recurrent ventricular fibrillation during advanced life support care of patients with prehospital cardiac arrest. Resuscitation 2008;78:252–7.
196. Morrison LJ, Henry RM, Ku V, Nolan JP, Morley P, Deakin CD. Single-shock defibrillation success in adult cardiac arrest: a systematic review. Resuscitation 2013;84:1480–6.
197. Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. Resuscitation 2006;71:137–45.
198. Eftestol T, Sunde K, Steen PA. Effects of interrupting precordial compressions on the calculated probability of defibrillation success during out-of-hospital cardiac arrest. Circulation 2002;105:2270–3.
199. Karlis G, Iacovidou N, Lelovas P, et al. Effects of early amiodarone administration during and immediately after cardiopulmonary resuscitation in a swine model. Acta Anaesthesiol Scand 2014;58:114–22.
200. Bhende MS, Thompson AE. Evaluation of an end-tidal CO_2 detector during pediatric cardiopulmonary resuscitation. Pediatrics 1995;95:395–9.
201. Sehra R, Underwood K, Checchia P. End tidal CO_2 is a quantitative measure of cardiac arrest. Pacing Clin Electrophysiol 2003;26:515–7.
202. Giberson B, Uber A, Gaieski DF, et al. When to stop CPR and when to perform rhythm analysis: potential confusion among ACLS providers. J Intensive Care Med 2014.
203. Berg RA, Hilwig RW, Kern KB, Ewy GA. Precountershock cardiopulmonary resuscitation improves ventricular fibrillation median frequency and myocardial readiness for successful defibrillation from prolonged ventricular fibrillation: a randomized, controlled swine study. Ann Emerg Med 2002;40:563–70.
204. Eftestol T, Sunde K, Aase SO, Husoy JH, Steen PA. Probability of successful defibrillation" as a monitor during CPR in out-of-hospital cardiac arrested patients. Resuscitation 2001;48:245–54.
205. Kolarova J, Ayoub IM, Yi Z, Gazmuri RJ. Optimal timing for electrical defibrillation after prolonged untreated ventricular fibrillation. Crit Care Med 2003;31:2022–8.
206. Yeung J, Chilwan M, Field R, Davies R, Gao F, Perkins GD. The impact of airway management on quality of cardiopulmonary resuscitation: an observational study in patients during cardiac arrest. Resuscitation 2014;85:898–904.
207. Lee PM, Lee C, Rattner P, Wu X, Gershengorn H, Acquah S. Intraosseous versus central venous catheter utilization and performance during inpatient medical emergencies. Crit Care Med 2015;43:1233–8.
208. Reades R, Studnek JR, Vandeventer S, Garrett J. Intraosseous versus intravenous vascular access during out-of-hospital cardiac arrest: a randomized controlled trial. Ann Emerg Med 2011;58:509–16.
209. Leidel BA, Kirchhoff C, Bogner V, Braunstein V, Biberthaler P, Kanz KG. Comparison of intraosseous versus central venous vascular access in adults under resuscitation in the emergency department with inaccessible peripheral veins. Resuscitation 2012;83:40–5.
210. Helm M, Haunstein B, Schlechtriemen T, Ruppert M, Lampl L, Gassler M. EZ-IO((R)) intraosseous device implementation in German Helicopter Emergency Medical Service. Resuscitation 2015;88:43–7.

211. Wenzel V, Lindner KH, Augenstein S, et al. Intraosseous vasopressin improves coronary perfusion pressure rapidly during cardiopulmonary resuscitation in pigs. Crit Care Med 1999;27:1565–9.
212. Hoskins SL, do Nascimento Jr P, Lima RM, Espana-Tenorio JM, Kramer GC. Pharmacokinetics of intraosseous and central venous drug delivery during cardiopulmonary resuscitation. Resuscitation 2012;83:107–12.
213. Myerburg RJ, Halperin H, Egan DA, et al. Pulseless electric activity: definition, causes, mechanisms, management, and research priorities for the next decade: report from a National Heart, Lung, and Blood Institute workshop. Circulation 2013;128:2532–41.
214. Nordseth T, Edelson DP, Bergum D, et al. Optimal loop duration during the provision of in-hospital advanced life support (ALS) to patients with an initial non-shockable rhythm. Resuscitation 2014;85:75–81.
215. Narasimhan M, Koenig SJ, Mayo PH. Advanced echocardiography for the critical care physician: Part 1. Chest 2014;145:129–34.
216. Flato UA, Paiva EF, Carballo MT, Buehler AM, Marco R, Timerman A. Echocardiography for prognostication during the resuscitation of intensive care unit patients with non-shockable rhythm cardiac arrest. Resuscitation 2015;92:1–6.
217. Breitkreutz R, Price S, Steiger HV, et al. Focused echocardiographic evaluation in life support and peri-resuscitation of emergency patients: a prospective trial. Resuscitation 2010;81:1527–33.
218. Olaussen A, Shepherd M, Nehme Z, Smith K, Bernard S, Mitra B. Return of consciousness during ongoing cardiopulmonary resuscitation: a systematic review. Resuscitation 2014;86C:44–8.
219. Couper K, Smyth M, Perkins GD. Mechanical devices for chest compression: to use or not to use? Curr Opin Crit Care 2015;21:188–94.
220. Deakin CD, Low JL. Accuracy of the advanced trauma life support guidelines for predicting systolic blood pressure using carotid, femoral, and radial pulses: observational study. BMJ 2000;321:673–4.
221. Connick M, Berg RA. Femoral venous pulsations during open-chest cardiac massage. Ann Emerg Med 1994;24:1176–9.
222. Weil MH, Rackow EC, Trevino R, Grundler W, Falk JL, Griffel MI. Difference in acid–base state between venous and arterial blood during cardiopulmonary resuscitation. N Engl J Med 1986;315:153–6.
223. Meaney PA, Bobrow BJ, Mancini ME, et al. Cardiopulmonary resuscitation quality: improving cardiac resuscitation outcomes both inside and outside the hospital: a consensus statement from the American Heart Association. Circulation 2013;128:417–35.
224. Friess SH, Sutton RM, French B, et al. Hemodynamic directed CPR improves cerebral perfusion pressure and brain tissue oxygenation. Resuscitation 2014;85:1298–303.
225. Friess SH, Sutton RM, Bhalala U, et al. Hemodynamic directed cardiopulmonary resuscitation improves short-term survival from ventricular fibrillation cardiac arrest. Crit Care Med 2013;41:2698–704.
226. Sutton RM, Friess SH, Bhalala U, et al. Hemodynamic directed CPR improves short-term survival from asphyxia-associated cardiac arrest. Resuscitation 2013;84:696–701.
227. Babbs CF. We still need a real-time hemodynamic monitor for CPR. Resuscitation 2013;84:1297–8.
228. Fukuda T, Ohashi N, Nishida M, et al. Application of cerebral oxygen saturation to prediction of the futility of resuscitation for out-of-hospital cardiopulmonary arrest patients: a single-center, prospective, observational study: can cerebral regional oxygen saturation predict the futility of CPR? Am J Emerg Med 2014;32:747–51.
229. Parnia S, Nasir A, Ahn A, et al. A feasibility study of cerebral oximetry during in-hospital mechanical and manual cardiopulmonary resuscitation. Crit Care Med 2014;42:930–3.
230. Genbrugge C, Meex I, Boer W, et al. Increase in cerebral oxygenation during advanced life support in out-of-hospital patients is associated with return of spontaneous circulation. Crit Care 2015;19:112.
231. Nolan JP, Cerebral oximetry during cardiac arrest-feasible, but benefit yet to be determined. Crit Care Med 2014;42:1001–2.
232. Hamrick JL, Hamrick JT, Lee JK, Lee BH, Koehler RC, Shaffner DH. Efficacy of chest compressions directed by end-tidal CO_2 feedback in a pediatric resuscitation model of basic life support. J Am Heart Assoc 2014;3:e000450.
233. Wallmuller C, Sterz F, Testori C, et al. Emergency cardio-pulmonary bypass in cardiac arrest: seventeen years of experience. Resuscitation 2013;84:326–30.
234. Kagawa E, Dote K, Kato M, et al. Should we emergently revascularize occluded coronaries for cardiac arrest? Rapid-response extracorporeal membrane oxygenation and intra-arrest percutaneous coronary intervention. Circulation 2012;126:1605–13.
235. Xie A, Phan K, Yi-Chin Tsai M, Yan TD, Forrest P. Venoarterial extracorporeal membrane oxygenation for cardiogenic shock and cardiac arrest: a meta-analysis. J Cardiothorac Vasc Anesth 2015;29:637–45.
236. Riggs KR, Becker LB, Sugarman J. Ethics in the use of extracorporeal cardiopulmonary resuscitation in adults. Resuscitation 2015;91:73–5.
237. Gundersen K, Kvaloy JT, Kramer-Johansen J, Steen PA, Eftestol T. Development of the probability of return of spontaneous circulation in intervals without chest compressions during out-of-hospital cardiac arrest: an observational study. BMC Med 2009;7:6.
238. Perkins GD, Davies RP, Soar J, Thickett DR. The impact of manual defibrillation technique on no-flow time during simulated cardiopulmonary resuscitation. Resuscitation 2007;73:109–14.
239. Fouche PF, Simpson PM, Bendall J, Thomas RE, Cone DC, Doi SA. Airways in out-of-hospital cardiac arrest: systematic review and meta-analysis. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2014;18:244–56.
240. Voss S, Rhys M, Coates D, et al. How do paramedics manage the airway during out of hospital cardiac arrest? Resuscitation 2014;85:1662–6.
241. Lin S, Callaway CW, Shah PS, et al. Adrenaline for out-of-hospital cardiac arrest resuscitation: a systematic review and meta-analysis of randomized controlled trials. Resuscitation 2014;85:732–40.
242. Patanwala AE, Slack MK, Martin JR, Basken RL, Nolan PE. Effect of epinephrine on survival after cardiac arrest: a systematic review and meta-analysis. Miner Anestesiol 2014;80:831–43.
243. Lindner KH, Dirks B, Strohmenger HU, Prengel AW, Lindner IM, Lurie KG. Randomised comparison of epinephrine and vasopressin in patients with out-of-hospital ventricular fibrillation. Lancet 1997;349:535–7.
244. Wenzel V, Krismer AC, Arntz HR, Sitter H, Stadlbauer KH, Lindner KH. A comparison of vasopressin and epinephrine for out-of-hospital cardiopulmonary resuscitation. N Engl J Med 2004;350:105–13.
245. Stiell IG, Hebert PC, Wells GA, et al. Vasopressin versus epinephrine for inhospital cardiac arrest: a randomised controlled trial. Lancet 2001;358:105–9.
246. Ong ME, Tiah L, Leong BS, et al. A randomised, double-blind, multi-centre trial comparing vasopressin and adrenaline in patients with cardiac arrest presenting to or in the Emergency Department. Resuscitation 2012;83:953–60.
247. Mentzelopoulos SD, Zakynthinos SG, Siempos I, Malachias S, Ulmer H, Wenzel V. Vasopressin for cardiac arrest: meta-analysis of randomized controlled trials. Resuscitation 2012;83:32–9.
248. Callaway CW, Hostler D, Doshi AA, et al. Usefulness of vasopressin administered with epinephrine during out-of-hospital cardiac arrest. Am J Cardiol 2006;98:1316–21.
249. Gueugniaud PY, David JS, Chanzy E, et al. Vasopressin and epinephrine vs. epinephrine alone in cardiopulmonary resuscitation. N Engl J Med 2008;359:21–30.
250. Ducros L, Vicaut E, Soleil C, et al. Effect of the addition of vasopressin or vasopressin plus nitroglycerin to epinephrine on arterial blood pressure during cardiopulmonary resuscitation in humans. J Emerg Med 2011;41:453–9.
251. Kudenchuk PJ, Cobb LA, Copass MK, et al. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. N Engl J Med 1999;341:871–8.
252. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. N Engl J Med 2002;346:884–90.
253. Skrifvars MB, Kuisma M, Boyd J, et al. The use of undiluted amiodarone in the management of out-of-hospital cardiac arrest. Acta Anaesthesiol Scand 2004;48:582–7.
254. Petrovic T, Adnet F, Lapandry C. Successful resuscitation of ventricular fibrillation after low-dose amiodarone. Ann Emerg Med 1998;32:518–9.
255. Levine JH, Massumi A, Scheinman MM, et al. Intravenous amiodarone for recurrent sustained hypotensive ventricular tachyarrhythmias. Intravenous Amiodarone Multicenter Trial Group. J Am Coll Cardiol 1996;27:67–75.
256. Somberg JC, Bailin SJ, Haffajee CI, et al. Intravenous lidocaine versus intravenous amiodarone (in a new aqueous formulation) for incessant ventricular tachycardia. Am J Cardiol 2002;90:853–9.
257. Somberg JC, Timar S, Bailin SJ, et al. Lack of a hypotensive effect with rapid administration of a new aqueous formulation of intravenous amiodarone. Am J Cardiol 2004;93:576–81.
258. Böttiger BW, Martin E. Thrombolytic therapy during cardiopulmonary resuscitation and the role of coagulation activation after cardiac arrest. Curr Opin Crit Care 2001;7:176–83.
259. Spöhr F, Böttiger BW. Safety of thrombolysis during cardiopulmonary resuscitation. Drug Saf 2003;26:367–79.
260. Wu JP, Gu DY, Wang S, Zhang ZJ, Zhou JC, Zhang RF. Good neurological recovery after rescue thrombolysis of presumed pulmonary embolism despite prior 100 minutes CPR. J Thorac Dis 2014;6:E289–93.
261. Langhelle A, Tyvold SS, Lexow K, Hapnes SA, Sunde K, Steen PA. In-hospital factors associated with improved outcome after out-of-hospital cardiac arrest. A comparison between four regions in Norway. Resuscitation 2003;56:247–63.
262. Kramer-Johansen J, Myklebust H, Wik L, et al. Quality of out-of-hospital cardiopulmonary resuscitation with real time automated feedback: a prospective interventional study. Resuscitation 2006;71:283–92.
263. Sutton RM, Maltese MR, Niles D, et al. Quantitative analysis of chest compression interruptions during in-hospital resuscitation of older children and adolescents. Resuscitation 2009;80:1259–63.
264. Sutton RM, Niles D, Nysaether J, et al. Quantitative analysis of CPR quality during in-hospital resuscitation of older children and adolescents. Pediatrics 2009;124:494–9.
265. Wik L, Olsen JA, Persse D, et al. Manual vs. integrated automatic load-distributing band CPR with equal survival after out of hospital cardiac arrest. The randomized CIRC trial. Resuscitation 2014;85:741–8.
266. Rubertsson S, Lindgren E, Smekal D, et al. Mechanical chest compressions and simultaneous defibrillation vs conventional cardiopulmonary resuscitation in out-of-hospital cardiac arrest: the LINC randomized trial. JAMA 2014;311:53–61.
267. Aufderheide TP, Nichol G, Rea TD, et al. A trial of an impedance threshold device in out-of-hospital cardiac arrest. N Engl J Med 2011;365:798–806.

268. Plaisance P, Lurie KG, Payen D. Inspiratory impedance during active compression–decompression cardiopulmonary resuscitation: a randomized evaluation in patients in cardiac arrest. Circulation 2000;101:989–94.
269. Plaisance P, Lurie KG, Vicaut E, et al. Evaluation of an impedance threshold device in patients receiving active compression–decompression cardiopulmonary resuscitation for out of hospital cardiac arrest. Resuscitation 2004;61:265–71.
270. Aufderheide TP, Frascone RJ, Wayne MA, et al. Standard cardiopulmonary resuscitation versus active compression–decompression cardiopulmonary resuscitation with augmentation of negative intrathoracic pressure for out-of-hospital cardiac arrest: a randomised trial. Lancet 2011;377:301–11.
271. Frascone RJ, Wayne MA, Swor RA, et al. Treatment of non-traumatic out-of-hospital cardiac arrest with active compression decompression cardiopulmonary resuscitation plus an impedance threshold device. Resuscitation 2013;84:1214–22.
272. Wee JH, Park JH, Choi SP, Park KN. Outcomes of patients admitted for hanging injuries with decreased consciousness but without cardiac arrest. Am J Emerg Med 2013;31:1666–70.
273. Penney DJ, Stewart AH, Parr MJ. Prognostic outcome indicators following hanging injuries. Resuscitation 2002;54:27–9.
274. Wood S. Interactions between hypoxia and hypothermia. Annu Rev Physiol 1991;53:71–85.
275. Schneider SM. Hypothermia: from recognition to rewarming. Emerg Med Rep 1992;13:1–20.
276. Gruber E, Beikircher W, Pizzinini R, et al. Non-extracorporeal rewarming at a rate of 6.8 degrees C per hour in a deeply hypothermic arrested patient. Resuscitation 2014;85:e119–20.
277. Bouchama A, Knochel JP. Heat stroke. N Engl J Med 2002;346:1978–88.
278. Hadad E, Weinbroum AA, Ben-Abraham R. Drug-induced hyperthermia and muscle rigidity: a practical approach. Eur J Emerg Med: Off J Eur Soc Emerg Med 2003;10:149–54.
279. Halloran LL, Bernard DW. Management of drug-induced hyperthermia. Curr Opin Pediatr 2004;16:211–5.
280. Bouchama A, Dehbi M, Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical recommendations. Crit Care 2007;11:R54.
281. Brenner ML, Moore LJ, DuBose JJ, et al. A clinical series of resuscitative endovascular balloon occlusion of the aorta for hemorrhage control and resuscitation. J Trauma Acute Care Surg 2013;75:506–11.
282. Soar J, Pumphrey R, Cant A, et al. Emergency treatment of anaphylactic reactions – guidelines for healthcare providers. Resuscitation 2008;77:157–69.
283. Soar J. Emergency treatment of anaphylaxis in adults: concise guidance. Clin Med 2009;9:181–5.
284. Soar J, Perkins GD, Abbas G, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution. Resuscitation 2010;81:1400–33.
285. Muraro A, Roberts G, Worm M, et al. Anaphylaxis: guidelines from the European Academy of Allergy and Clinical Immunology. Allergy 2014;69:1026–45.
286. Simpson CR, Sheikh A. Adrenaline is first line treatment for the emergency treatment of anaphylaxis. Resuscitation 2010;81:641–2.
287. Kemp SF, Lockey RF, Simons FE. Epinephrine: the drug of choice for anaphylaxis. A statement of the World Allergy Organization. Allergy 2008;63:1061–70.
288. Bautista E, Simons FE, Simons KJ, et al. Epinephrine fails to hasten hemodynamic recovery in fully developed canine anaphylactic shock. Int Arch Allergy Immunol 2002;128:151–64.
289. Zwingmann J, Mehlhorn AT, Hammer T, Bayer J, Sudkamp NP, Strohm PC. Survival and neurologic outcome after traumatic out-of-hospital cardiopulmonary arrest in a pediatric and adult population: a systematic review. Crit Care 2012;16:R117.
290. Leis CC, Hernandez CC, Blanco MJ, Paterna PC, Hernandez Rde E, Torres EC. Traumatic cardiac arrest: should advanced life support be initiated? J Trauma Acute Care Surg 2013;74:634–8.
291. Lockey D, Crewdson K, Davies G. Traumatic cardiac arrest: who are the survivors? Ann Emerg Med 2006;48:240–4.
292. Crewdson K, Lockey D, Davies G. Outcome from paediatric cardiac arrest associated with trauma. Resuscitation 2007;75:29–34.
293. Kleber C, Giesecke MT, Lindner T, Haas NP, Buschmann CT. Requirement for a structured algorithm in cardiac arrest following major trauma: epidemiology, management errors, and preventability of traumatic deaths in Berlin. Resuscitation 2014;85:405–10.
294. Leigh-Smith S, Harris T. Tension pneumothorax – time for a re-think? Emerg Med J 2005;22:8–16.
295. Chen KY, Jerng JS, Liao WY, et al. Pneumothorax in the ICU: patient outcomes and prognostic factors. Chest 2002;122:678–83.
296. Warner KJ, Copass MK, Bulger EM. Paramedic use of needle thoracostomy in the prehospital environment. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2008;12:162–8.
297. Mistry N, Bleetman A, Roberts KJ. Chest decompression during the resuscitation of patients in prehospital traumatic cardiac arrest. Emerg Med J 2009;26:738–40.
298. Deakin CD, Davies G, Wilson A. Simple thoracostomy avoids chest drain insertion in prehospital trauma. J Trauma 1995;39:373–4.
299. Massarutti D, Trillo G, Berlot G, et al. Simple thoracostomy in prehospital trauma management is safe and effective: a 2-year experience by helicopter emergency medical crews. Eur J Emerg Med: Off J Eur Soc Emerg Med 2006;13:276–80.
300. Konstantinides SV, Torbicki A, Agnelli G, et al. 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J 2014;35:3033–69, 69a–69k.
301. Kurkciyan I, Meron G, Behringer W, et al. Accuracy and impact of presumed cause in patients with cardiac arrest. Circulation 1998;98:766–71.
302. Kurkciyan I, Meron G, Sterz F, et al. Pulmonary embolism as a cause of cardiac arrest: presentation and outcome. Arch Intern Med 2000;160:1529–35.
303. Pokorna M, Necas E, Skripsky R, Kratochvil J, Andrlik M, Franek O. How accurately can the aetiology of cardiac arrest be established in an out-of-hospital setting? Analysis by "concordance in diagnosis crosscheck tables". Resuscitation 2011;82:391–7.
304. Wallmuller C, Meron G, Kurkciyan I, Schober A, Stratil P, Sterz F. Causes of in-hospital cardiac arrest and influence on outcome. Resuscitation 2012;83:1206–11.
305. Bergum D, Nordseth T, Mjolstad OC, Skogvoll E, Haugen BO. Causes of in-hospital cardiac arrest – incidences and rate of recognition. Resuscitation 2015;87:63–8.
306. Stub D, Nehme Z, Bernard S, Lijovic M, Kaye DM, Smith K. Exploring which patients without return of spontaneous circulation following ventricular fibrillation out-of-hospital cardiac arrest should be transported to hospital? Resuscitation 2014;85:326–31.
307. Mowry JB, Spyker DA, Cantilena Jr LR, McMillan N, Ford M. 2013 annual report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 31st annual report. Clin Toxicol (Phila) 2014;52:1032–283.
308. Proudfoot AT, Krenzelok EP, Vale JA. Position paper on urine alkalinization. J Toxicol Clin Toxicol 2004;42:1–26.
309. Greene S, Harris C, Singer J. Gastrointestinal decontamination of the poisoned patient. Pediatr Emerg Care 2008;24:176–86 [quiz 87–9].
310. Benson BE, Hoppu K, Troutman WG, et al. Position paper update: gastric lavage for gastrointestinal decontamination. Clin Toxicol (Phila) 2013;51:140–6.
311. Chyka PA, Seger D, Krenzelok EP, Vale JA. Position paper: single-dose activated charcoal. Clin Toxicol (Phila) 2005;43:61–87.
312. Ellis SJ, Newland MC, Simonson JA, et al. Anesthesia-related cardiac arrest. Anesthesiology 2014;120:829–38.
313. Gonzalez LP, Braz JR, Modolo MP, de Carvalho LR, Modolo NS, Braz LG. Pediatric perioperative cardiac arrest and mortality: a study from a tertiary teaching hospital. Pediatr Crit Care Med: J Soc Crit Care Med World Feder Pediatr Intensive Crit Care Soc 2014;15:878–84.
314. Sprung J, Warner ME, Contreras MG, et al. Predictors of survival following cardiac arrest in patients undergoing noncardiac surgery: a study of 518,294 patients at a tertiary referral center. Anesthesiology 2003;99:259–69.
315. Charalambous CP, Zipitis CS, Keenan DJ. Chest reexploration in the intensive care unit after cardiac surgery: a safe alternative to returning to the operating theater. Ann Thorac Surg 2006;81:191–4.
316. LaPar DJ, Ghanta RK, Kern JA, et al. Hospital variation in mortality from cardiac arrest after cardiac surgery: an opportunity for improvement? Ann Thorac Surg 2014;98:534–9 [discussion 9–40].
317. Wagner H, Terkelsen CJ, Friberg H, et al. Cardiac arrest in the catheterisation laboratory: a 5-year experience of using mechanical chest compressions to facilitate PCI during prolonged resuscitation efforts. Resuscitation 2010;81:383–7.
318. Larsen AI, Hjornevik AS, Ellingsen CL, Nilsen DW. Cardiac arrest with continuous mechanical chest compression during percutaneous coronary intervention. A report on the use of the LUCAS device. Resuscitation 2007;75:454–9.
319. Tsao NW, Shih CM, Yeh JS, et al. Extracorporeal membrane oxygenation-assisted primary percutaneous coronary intervention may improve survival of patients with acute myocardial infarction complicated by profound cardiogenic shock. J Crit Care 2012;27:530, e1–11.
320. Alpert MA. Sudden cardiac arrest and sudden cardiac death on dialysis: epidemiology, evaluation, treatment, and prevention. Hemodial Int 2011;15:S22–9.
321. Sacchetti A, Stuccio N, Panebianco P, Torres M. ED hemodialysis for treatment of renal failure emergencies. Am J Emerg Med 1999;17:305–7.
322. Davis TR, Young BA, Eisenberg MS, Rea TD, Copass MK, Cobb LA. Outcome of cardiac arrests attended by emergency medical services staff at community outpatient dialysis centers. Kidney Int 2008;73:933–9.
323. Lafrance JP, Nolin L, Senecal L, Leblanc M. Predictors and outcome of cardiopulmonary resuscitation (CPR) calls in a large haemodialysis unit over a seven-year period. Nephrol Dial Transplant 2006;21:1006–12.
324. Bird S, Petley GW, Deakin CD, Clewlow F. Defibrillation during renal dialysis: a survey of UK practice and procedural recommendations. Resuscitation 2007;73:347–53.
325. O'Rourke MF, Donaldson E, Geddes JS. An airline cardiac arrest program. Circulation 1997;96:2849–53.
326. Page RL, Joglar JA, Kowal RC, et al. Use of automated external defibrillators by a U.S. airline. N Engl J Med 2000;343:1210–6.
327. Graf J, Stuben U, Pump S. In-flight medical emergencies. Dtsch Arztebl Int 2012;109:591–601 [quiz 2].

328. Brown AM, Rittenberger JC, Ammon CM, Harrington S, Guyette FX. In-flight automated external defibrillator use and consultation patterns. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2010;14:235–9.
329. Bertrand C, Rodriguez Redington P, Lecarpentier E, et al. Preliminary report on AED deployment on the entire Air France commercial fleet: a joint venture with Paris XII University Training Programme. Resuscitation 2004;63:175–81.
330. Skogvoll E, Bjelland E, Thorarinsson B. Helicopter emergency medical service in out-of-hospital cardiac arrest – a 10-year population-based study. Acta Anaesthesiol Scand 2000;44:972–9.
331. Lyon RM, Nelson MJ. Helicopter emergency medical services (HEMS) response to out-of-hospital cardiac arrest. Scand J Trauma Resusc Emerg Med 2013;21:1.
332. Forti A, Zilio G, Zanatta P, et al. Full recovery after prolonged cardiac arrest and resuscitation with mechanical chest compression device during helicopter transportation and percutaneous coronary intervention. J Emerg Med 2014;47:632–4.
333. Pietsch U, Lischke V, Pietsch C. Benefit of mechanical chest compression devices in mountain HEMS: lessons learned from 1 year of experience and evaluation. Air Med J 2014;33:299–301.
334. Omori K, Sato S, Sumi Y, et al. The analysis of efficacy for AutoPulse system in flying helicopter. Resuscitation 2013;84:1045–50.
335. Putzer G, Braun P, Zimmermann A, et al. LUCAS compared to manual cardiopulmonary resuscitation is more effective during helicopter rescue-a prospective, randomized, cross-over manikin study. Am J Emerg Med 2013;31:384–9.
336. Lin CY, Wang YF, Lu TH, Kawach I. Unintentional drowning mortality, by age and body of water: an analysis of 60 countries. Inj Prev 2015;21:e43–50.
337. Szpilman D, Webber J, Quan L, et al. Creating a drowning chain of survival. Resuscitation 2014;85:1149–52.
338. Vahatalo R, Lunetta P, Olkkola KT, Suominen PK. Drowning in children: Utstein style reporting and outcome. Acta Anaesthesiol Scand 2014;58:604–10.
339. Claesson A, Lindqvist J, Herlitz J. Cardiac arrest due to drowning – changes over time and factors of importance for survival. Resuscitation 2014;85:644–8.
340. Dyson K, Morgans A, Bray J, Matthews B, Smith K. Drowning related out-of-hospital cardiac arrests: characteristics and outcomes. Resuscitation 2013;84:1114–8.
341. Tipton MJ, Golden FS. A proposed decision-making guide for the search, rescue and resuscitation of submersion (head under) victims based on expert opinion. Resuscitation 2011;82:819–24.
342. Wanscher M, Agersnap L, Ravn J, et al. Outcome of accidental hypothermia with or without circulatory arrest: experience from the Danish Praesto Fjord boating accident. Resuscitation 2012;83:1078–84.
343. Kieboom JK, Verkade HJ, Burgerhof JG, et al. Outcome after resuscitation beyond 30 minutes in drowned children with cardiac arrest and hypothermia: Dutch nationwide retrospective cohort study. BMJ 2015;350:h418.
344. Tomazin I, Ellerton J, Reisten O, Soteras I, Avbelj M. International Commission for Mountain Emergency M. Medical standards for mountain rescue operations using helicopters: official consensus recommendations of the International Commission for Mountain Emergency Medicine (ICAR MEDCOM). High Alt Med Biol 2011;12:335–41.
345. Pietsch U, Lischke V, Pietsch C, Kopp KH. Mechanical chest compressions in an avalanche victim with cardiac arrest: an option for extreme mountain rescue operations. Wilderness Environ Med 2014;25:190–3.
346. Ellerton J, Gilbert H. Should helicopters have a hoist or 'long-line' capability to perform mountain rescue in the UK? Emerg Med J 2012;29:56–9.
347. Klemenc-Ketis Z, Tomazin I, Kersnik J. HEMS in Slovenia: one country, four models, different quality outcomes. Air Med J 2012;31:298–304.
348. Tomazin I, Vegnuti M, Ellerton J, Reisten O, Sumann G, Kersnik J. Factors impacting on the activation and approach times of helicopter emergency medical services in four Alpine countries. Scand J Trauma Resusc Emerg Med 2012;20:56.
349. Wang JC, Tsai SH, Chen YL, et al. The physiological effects and quality of chest compressions during CPR at sea level and high altitude. Am J Emerg Med 2014;32:1183–8.
350. Suto T, Saito S. Considerations for resuscitation at high altitude in elderly and untrained populations and rescuers. Am J Emerg Med 2014;32:270–6.
351. Narahara H, Kimura M, Suto T, et al. Effects of cardiopulmonary resuscitation at high altitudes on the physical condition of untrained and unacclimatized rescuers. Wilderness Environ Med 2012;23:161–4.
352. Boyd J, Brugger H, Shuster M. Prognostic factors in avalanche resuscitation: a systematic review. Resuscitation 2010;81:645–52.
353. Lightning-associated deaths – United States, 1980–1995. MMWR Morb Mortal Wkly Rep 1998;47:391–4.
354. Zafren K, Durrer B, Herry JP, Brugger H. Lightning injuries: prevention and on-site treatment in mountains and remote areas. Official guidelines of the International Commission for Mountain Emergency Medicine and the Medical Commission of the International Mountaineering and Climbing Federation (ICAR and UIAA MEDCOM). Resuscitation 2005;65:369–72.
355. Why asthma still kills: the national review of asthma deaths (NRAD). Confidential enquiry report 2014; 2014. From: http://www.rcplondon.ac.uk/sites/default/files/why-asthma-still-kills-full-report.pdf.
356. Hubner P, Meron G, Kurkciyan I, et al. Neurologic causes of cardiac arrest and outcomes. J Emerg Med 2014;47:660–7.
357. Skrifvars MB, Parr MJ. Incidence, predisposing factors, management and survival following cardiac arrest due to subarachnoid haemorrhage: a review of the literature. Scand J Trauma Resusc Emerg Med 2012;20:75.
358. Arnaout M, Mongardon N, Deye N, et al. Out-of-hospital cardiac arrest from brain cause: epidemiology, clinical features, and outcome in a multicenter cohort. Crit Care Med 2015;43:453–60.
359. Adabag S, Huxley RR, Lopez FL, et al. Obesity related risk of sudden cardiac death in the atherosclerosis risk in communities study. Heart 2015;101:215–21.
360. Lipman S, Cohen S, Einav S, et al. The Society for Obstetric Anesthesia and Perinatology consensus statement on the management of cardiac arrest in pregnancy. Anesth Analg 2014;118:1003–16.
361. Boyd R, Teece S. Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary. Perimortem caesarean section. Emerg Med J 2002;19:324–5.
362. McNally B, Robb R, Mehta M, et al. Out-of-hospital cardiac arrest surveillance – Cardiac Arrest Registry to Enhance Survival (CARES), United States, October 1, 2005–December 31, 2010. MMWR Surveill Summ 2011;60:1–19.
363. Black CJ, Busuttil A, Robertson C. Chest wall injuries following cardiopulmonary resuscitation. Resuscitation 2004;63:339–43.
364. Krischer JP, Fine EG, Davis JH, Nagel EL. Complications of cardiac resuscitation. Chest 1987;92:287–91.
365. Kashiwagi Y, Sasakawa T, Tampo A, et al. Computed tomography findings of complications resulting from cardiopulmonary resuscitation. Resuscitation 2015;88:86–91.
366. Nolan JP, Neumar RW, Adrie C, et al. Post-cardiac arrest syndrome: epidemiology, pathophysiology, treatment, and prognostication. A Scientific Statement from the International Liaison Committee on Resuscitation; the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiovascular Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative, and Critical Care; the Council on Clinical Cardiology; the Council on Stroke. Resuscitation 2008;79:350–79.
367. Spaite DW, Bobrow BJ, Stolz U, et al. Statewide regionalization of postarrest care for out-of-hospital cardiac arrest: association with survival and neurologic outcome. Ann Emerg Med 2014;64:496–506, e1.
368. Soholm H, Wachtell K, Nielsen SL, et al. Tertiary centres have improved survival compared to other hospitals in the Copenhagen area after out-of-hospital cardiac arrest. Resuscitation 2013;84:162–7.
369. Sunde K, Pytte M, Jacobsen D, et al. Implementation of a standardised treatment protocol for post resuscitation care after out-of-hospital cardiac arrest. Resuscitation 2007;73:29–39.
370. Gaieski DF, Band RA, Abella BS, et al. Early goal-directed hemodynamic optimization combined with therapeutic hypothermia in comatose survivors of out-of-hospital cardiac arrest. Resuscitation 2009;80:418–24.
371. Carr BG, Goyal M, Band RA, et al. A national analysis of the relationship between hospital factors and post-cardiac arrest mortality. Intensive Care Med 2009;35:505–11.
372. Oddo M, Schaller MD, Feihl F, Ribordy V, Liaudet L. From evidence to clinical practice: effective implementation of therapeutic hypothermia to improve patient outcome after cardiac arrest. Crit Care Med 2006;34:1865–73.
373. Knafelj R, Radsel P, Ploj T, Noc M. Primary percutaneous coronary intervention and mild induced hypothermia in comatose survivors of ventricular fibrillation with ST-elevation acute myocardial infarction. Resuscitation 2007;74:227–34.
374. Mongardon N, Dumas F, Ricome S, et al. Postcardiac arrest syndrome: from immediate resuscitation to long-term outcome. Ann Intensive Care 2011;1:45.
375. Stub D, Bernard S, Duffy SJ, Kaye DM. Post cardiac arrest syndrome: a review of therapeutic strategies. Circulation 2011;123:1428–35.
376. Nielsen N, Wetterslev J, Cronberg T, et al. Targeted temperature management at 33 degrees C versus 36 degrees C after cardiac arrest. N Engl J Med 2013;369:2197–206.
377. Lemiale V, Dumas F, Mongardon N, et al. Intensive care unit mortality after cardiac arrest: the relative contribution of shock and brain injury in a large cohort. Intensive Care Med 2013;39:1972–80.
378. Dragancea I, Rundgren M, Englund E, Friberg H, Cronberg T. The influence of induced hypothermia and delayed prognostication on the mode of death after cardiac arrest. Resuscitation 2013;84:337–42.
379. Tomte O, Andersen GO, Jacobsen D, Draegni T, Auestad B, Sunde K. Strong and weak aspects of an established post-resuscitation treatment protocol-A five-year observational study. Resuscitation 2011;82:1186–93.
380. Laurent I, Monchi M, Chiche JD, et al. Reversible myocardial dysfunction in survivors of out-of-hospital cardiac arrest. J Am Coll Cardiol 2002;40:2110–6.
381. Ruiz-Bailen M, Aguayo de Hoyos E, Ruiz-Navarro S, et al. Reversible myocardial dysfunction after cardiopulmonary resuscitation. Resuscitation 2005;66:175–81.
382. Chalkias A, Xanthos T. Pathophysiology and pathogenesis of post-resuscitation myocardial stunning. Heart Fail Rev 2012;17:117–28.
383. Adrie C, Monchi M, Laurent I, et al. Coagulopathy after successful cardiopulmonary resuscitation following cardiac arrest: implication of the protein C anticoagulant pathway. J Am Coll Cardiol 2005;46:21–8.
384. Adrie C, Adib-Conquy M, Laurent I, et al. Successful cardiopulmonary resuscitation after cardiac arrest as a "sepsis-like" syndrome. Circulation 2002;106:562–8.

385. Adrie C, Laurent I, Monchi M, Cariou A, Dhainaou JF, Spaulding C. Postresuscitation disease after cardiac arrest: a sepsis-like syndrome? Curr Opin Crit Care 2004;10:208–12.
386. Huet O, Dupic L, Batteux F, et al. Postresuscitation syndrome: potential role of hydroxyl radical-induced endothelial cell damage. Crit Care Med 2011;39:1712–20.
387. Fink K, Schwarz M, Feldbrugge L, et al. Severe endothelial injury and subsequent repair in patients after successful cardiopulmonary resuscitation. Crit Care 2010;14:R104.
388. van Genderen ME, Lima A, Akkerhuis M, Bakker J, van Bommel J. Persistent peripheral and microcirculatory perfusion alterations after out-of-hospital cardiac arrest are associated with poor survival. Crit Care Med 2012;40:2287–94.
389. Bro-Jeppesen J, Kjaergaard J, Wanscher M, et al. Systemic inflammatory response and potential prognostic implications after out-of-hospital cardiac arrest: a substudy of the target temperature management trial. Crit Care Med 2015;43:1223–32.
390. Sutherasan Y, Penuelas O, Muriel A, et al. Management and outcome of mechanically ventilated patients after cardiac arrest. Crit Care 2015;19:215.
391. Pilcher J, Weatherall M, Shirtcliffe P, Bellomo R, Young P, Beasley R. The effect of hyperoxia following cardiac arrest – a systematic review and meta-analysis of animal trials. Resuscitation 2012;83:417–22.
392. Wang CH, Chang WT, Huang CH, et al. The effect of hyperoxia on survival following adult cardiac arrest: a systematic review and meta-analysis of observational studies. Resuscitation 2014;85:1142–8.
393. Stub D, Smith K, Bernard S, et al. Air versus oxygen in ST-segment elevation myocardial infarction. Circulation 2015.
394. Bouzat P, Suys T, Sala N, Oddo M. Effect of moderate hyperventilation and induced hypertension on cerebral tissue oxygenation after cardiac arrest and therapeutic hypothermia. Resuscitation 2013;84:1540–5.
395. Buunk G, van der Hoeven JG, Meinders AE. Cerebrovascular reactivity in comatose patients resuscitated from a cardiac arrest. Stroke 1997;28:1569–73.
396. Buunk G, van der Hoeven JG, Meinders AE. A comparison of near-infrared spectroscopy and jugular bulb oximetry in comatose patients resuscitated from a cardiac arrest. Anaesthesia 1998;53:13–9.
397. Roberts BW, Kilgannon JH, Chansky ME, Mittal N, Wooden J, Trzeciak S. Association between postresuscitation partial pressure of arterial carbon dioxide and neurological outcome in patients with post-cardiac arrest syndrome. Circulation 2013;127:2107–13.
398. Schneider AG, Eastwood GM, Bellomo R, et al. Arterial carbon dioxide tension and outcome in patients admitted to the intensive care unit after cardiac arrest. Resuscitation 2013;84:927–34.
399. Larsen JM, Ravkilde J. Acute coronary angiography in patients resuscitated from out-of-hospital cardiac arrest – a systematic review and meta-analysis. Resuscitation 2012;83:1427–33.
400. Camuglia AC, Randhawa VK, Lavi S, Walters DL. Cardiac catheterization is associated with superior outcomes for survivors of out of hospital cardiac arrest: review and meta-analysis. Resuscitation 2014;85:1533–40.
401. Grasner JT, Meybohm P, Caliebe A, et al. Postresuscitation care with mild therapeutic hypothermia and coronary intervention after out-of-hospital cardiopulmonary resuscitation: a prospective registry analysis. Crit Care 2011;15:R61.
402. Callaway CW, Schmicker RH, Brown SP, et al. Early coronary angiography and induced hypothermia are associated with survival and functional recovery after out-of-hospital cardiac arrest. Resuscitation 2014;85:657–63.
403. Dumas F, White L, Stubbs BA, Cariou A, Rea TD. Long-term prognosis following resuscitation from out of hospital cardiac arrest: role of percutaneous coronary intervention and therapeutic hypothermia. J Am Coll Cardiol 2012;60:21–7.
404. Zanuttini D, Armellini I, Nucifora G, et al. Predictive value of electrocardiogram in diagnosing acute coronary artery lesions among patients with out-of-hospital-cardiac-arrest. Resuscitation 2013;84:1250–4.
405. Dumas F, Manzo-Silberman S, Fichet J, et al. Can early cardiac troponin I measurement help to predict recent coronary occlusion in out-of-hospital cardiac arrest survivors? Crit Care Med 2012;40:1777–84.
406. Sideris G, Voicu S, Dillinger JG, et al. Value of post-resuscitation electrocardiogram in the diagnosis of acute myocardial infarction in out-of-hospital cardiac arrest patients. Resuscitation 2011;82:1148–53.
407. Muller D, Schnitzer L, Brandt J, Arntz HR. The accuracy of an out-of-hospital 12-lead ECG for the detection of ST-elevation myocardial infarction immediately after resuscitation. Ann Emerg Med 2008;52:658–64.
408. Dumas F, Cariou A, Manzo-Silberman S, et al. Immediate percutaneous coronary intervention is associated with better survival after out-of-hospital cardiac arrest: insights from the PROCAT (Parisian Region Out of hospital Cardiac ArresT) registry. Circ Cardiovasc Interv 2010;3:200–7.
409. Radsel P, Knafelj R, Kocjancic S, Noc M. Angiographic characteristics of coronary disease and postresuscitation electrocardiograms in patients with aborted cardiac arrest outside a hospital. Am J Cardiol 2011;108:634–8.
410. Hollenbeck RD, McPherson JA, Mooney MR, et al. Early cardiac catheterization is associated with improved survival in comatose survivors of cardiac arrest without STEMI. Resuscitation 2014;85:88–95.
411. Redfors B, Ramunddal T, Angeras O, et al. Angiographic findings and survival in patients undergoing coronary angiography due to sudden cardiac arrest in Western Sweden. Resuscitation 2015;90:13–20.
412. Bro-Jeppesen J, Kjaergaard J, Wanscher M, et al. Emergency coronary angiography in comatose cardiac arrest patients: do real-life experiences support the guidelines? Eur Heart J Acute Cardiovasc Care 2012;1:291–301.
413. Dankiewicz J, Nielsen N, Annborn M, et al. Survival in patients without acute ST elevation after cardiac arrest and association with early coronary angiography: a post hoc analysis from the TTM trial. Intensive Care Med 2015;41:856–64.
414. Chelly J, Mongardon N, Dumas F, et al. Benefit of an early and systematic imaging procedure after cardiac arrest: insights from the PROCAT (Parisian Region Out of Hospital Cardiac Arrest) registry. Resuscitation 2012;83:1444–50.
415. Bro-Jeppesen J, Annborn M, Hassager C, et al. Hemodynamics and vasopressor support during targeted temperature management at 33 degrees C Versus 36 degrees C after out-of-hospital cardiac arrest: a post hoc study of the target temperature management trial. Crit Care Med 2015;43:318–27.
416. Chang WT, Ma MH, Chien KL, et al. Postresuscitation myocardial dysfunction: correlated factors and prognostic implications. Intensive Care Med 2007;33:88–95.
417. Dellinger RP, Levy MM, Rhodes A, et al. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock: 2012. Crit Care Med 2013;41:580–637.
418. Pro CI, Yealy DM, Kellum JA, et al. A randomized trial of protocol-based care for early septic shock. N Engl J Med 2014;370:1683–93.
419. Investigators A, Group ACT, Peake SL, et al. Goal-directed resuscitation for patients with early septic shock. N Engl J Med 2014;371:1496–506.
420. Mouncey PR, Osborn TM, Power GS, et al. Trial of early, goal-directed resuscitation for septic shock. N Engl J Med 2015;372:1301–11.
421. Zeiner A, Sunder-Plassmann G, Sterz F, et al. The effect of mild therapeutic hypothermia on renal function after cardiopulmonary resuscitation in men. Resuscitation 2004;60:253–61.
422. Lee DS, Green LD, Liu PP, et al. Effectiveness of implantable defibrillators for preventing arrhythmic events and death: a meta-analysis. J Am Coll Cardiol 2003;41:1573–82.
423. Vardas PE, Auricchio A, Blanc JJ, et al. Guidelines for cardiac pacing and cardiac resynchronization therapy: the Task Force for Cardiac Pacing and Cardiac Resynchronization Therapy of the European Society of Cardiology. Developed in collaboration with the European Heart Rhythm Association. Eur Heart J 2007;28:2256–95.
424. Task Force on the management of STseamiotESoC, Steg PG, James SK, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J 2012;33:2569–619.
425. Buunk G, van der Hoeven JG, Meinders AE. Cerebral blood flow after cardiac arrest. Neth J Med 2000;57:106–12.
426. Angelos MG, Ward KR, Hobson J, Beckley PD. Organ blood flow following cardiac arrest in a swine low-flow cardiopulmonary bypass model. Resuscitation 1994;27:245–54.
427. Fischer M, Bottiger BW, Popov-Cenic S, Hossmann KA. Thrombolysis using plasminogen activator and heparin reduces cerebral no-reflow after resuscitation from cardiac arrest: an experimental study in the cat. Intensive Care Med 1996;22:1214–23.
428. Sakabe T, Tateishi A, Miyauchi Y, et al. Intracranial pressure following cardiopulmonary resuscitation. Intensive Care Med 1987;13:256–9.
429. Morimoto Y, Kemmotsu O, Kitami K, Matsubara I, Tedo I. Acute brain swelling after out-of-hospital cardiac arrest: pathogenesis and outcome. Crit Care Med 1993;21:104–10.
430. Nishizawa H, Kudoh I. Cerebral autoregulation is impaired in patients resuscitated after cardiac arrest. Acta Anaesthesiol Scand 1996;40:1149–53.
431. Sundgreen C, Larsen FS, Herzog TM, Knudsen GM, Boesgaard S, Aldershvile J. Autoregulation of cerebral blood flow in patients resuscitated from cardiac arrest. Stroke 2001;32:128–32.
432. Snyder BD, Hauser WA, Loewenson RB, Leppik IE, Ramirez-Lassepas M, Gumnit RJ. Neurologic prognosis after cardiopulmonary arrest. III: Seizure activity. Neurology 1980;30:1292–7.
433. Bouwes A, van Poppelen D, Koelman JH, et al. Acute posthypoxic myoclonus after cardiopulmonary resuscitation. BMC Neurol 2012;12:63.
434. Seder DB, Sunde K, Rubertsson S, et al. Neurologic outcomes and postresuscitation care of patients with myoclonus following cardiac arrest. Crit Care Med 2015;43:965–72.
435. Benbadis SR, Chen S, Melo M. What's shaking in the ICU? The differential diagnosis of seizures in the intensive care setting. Epilepsia 2010;51:2338–40.
436. Caviness JN, Brown P. Myoclonus: current concepts and recent advances. Lancet Neurol 2004;3:598–607.
437. Ingvar M. Cerebral blood flow and metabolic rate during seizures. Relationship to epileptic brain damage. Ann N Y Acad Sci 1986;462:194–206.
438. Thomke F, Weilemann SL. Poor prognosis despite successful treatment of postanoxic generalized myoclonus. Neurology 2010;74:1392–4.
439. Mullner M, Sterz F, Binder M, Schreiber W, Deimel A, Laggner AN. Blood glucose concentration after cardiopulmonary resuscitation influences functional neurological recovery in human cardiac arrest survivors. J Cereb Blood Flow Metab: Off J Int Soc Cereb Blood Flow Metab 1997;17:430–6.
440. Nielsen N, Hovdenes J, Nilsson F, et al. Outcome, timing and adverse events in therapeutic hypothermia after out-of-hospital cardiac arrest. Acta Anaesthesiol Scand 2009;53:926–34.

441. Padkin A. Glucose control after cardiac arrest. Resuscitation 2009;80:611–2.
442. Takino M, Okada Y. Hyperthermia following cardiopulmonary resuscitation. Intensive Care Med 1991;17:419–20.
443. Hickey RW, Kochanek PM, Ferimer H, Alexander HL, Garman RH, Graham SH. Induced hyperthermia exacerbates neurologic neuronal histologic damage after asphyxial cardiac arrest in rats. Crit Care Med 2003;31:531–5.
444. Takasu A, Saitoh D, Kaneko N, Sakamoto T, Okada Y. Hyperthermia: is it an ominous sign after cardiac arrest? Resuscitation 2001;49:273–7.
445. Zeiner A, Holzer M, Sterz F, et al. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. Arch Intern Med 2001;161:2007–12.
446. Hickey RW, Kochanek PM, Ferimer H, Graham SH, Safar P. Hypothermia and hyperthermia in children after resuscitation from cardiac arrest. Pediatrics 2000;106:118–22.
447. Diringer MN, Reaven NL, Funk SE, Uman GC. Elevated body temperature independently contributes to increased length of stay in neurologic intensive care unit patients. Crit Care Med 2004;32:1489–95.
448. Gunn AJ, Thoresen M. Hypothermic neuroprotection. NeuroRx 2006;3:154–69.
449. Froehler MT, Geocadin RG. Hypothermia for neuroprotection after cardiac arrest: mechanisms, clinical trials and patient care. J Neurol Sci 2007;261:118–26.
450. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med 2002;346:549–56.
451. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med 2002;346:557–63.
452. Cronberg T, Lilja G, Horn J, et al. Neurologic function and health-related quality of life in patients following targeted temperature management at 33 degrees C vs 36 degrees C after out-of-hospital cardiac arrest: a randomized clinical trial. JAMA Neurol 2015.
453. Lilja G, Nielsen N, Friberg H, et al. Cognitive function in survivors of out-of-hospital cardiac arrest after target temperature management at 33 degrees C versus 36 degrees C. Circulation 2015;131:1340–9.
454. Nolan JP, Morley PT, Vanden Hoek TL, Hickey RW. Therapeutic hypothermia after cardiac arrest. An advisory statement by the Advancement Life support Task Force of the International Liaison committee on Resuscitation. Resuscitation 2003;57:231–5.
455. Kuboyama K, Safar P, Radovsky A, et al. Delay in cooling negates the beneficial effect of mild resuscitative cerebral hypothermia after cardia arrest in dogs: a prospective, randomized study. Crit Care Med 1993;21:1348–58.
456. Colbourne F, Corbett D. Delayed postischemic hypothermia: a six month survival study using behavioral and histological assessments of neuroprotection. J Neurosci 1995;15:7250–60.
457. Haugk M, Testori C, Sterz F, et al. Relationship between time to target temperature and outcome in patients treated with therapeutic hypothermia after cardiac arrest. Crit Care 2011;15:R101.
458. Benz-Woerner J, Delodder F, Benz R, et al. Body temperature regulation and outcome after cardiac arrest and therapeutic hypothermia. Resuscitation 2012;83:338–42.
459. Perman SM, Ellenberg JH, Grossestreuer AV, et al. Shorter time to target temperature is associated with poor neurologic outcome in post-arrest patients treated with targeted temperature management. Resuscitation 2015;88:114–9.
460. Kim F, Nichol G, Maynard C, et al. Effect of prehospital induction of mild hypothermia on survival and neurological status among adults with cardiac arrest: a randomized clinical trial. JAMA 2014;311:45–52.
461. Hoedemaekers CW, Ezzahti M, Gerritsen A, van der Hoeven JG. Comparison of cooling methods to induce and maintain normo- and hypothermia in intensive care unit patients: a prospective intervention study. Crit Care 2007;11:R91.
462. Gillies MA, Pratt R, Whiteley C, Borg J, Beale RJ, Tibby SM. Therapeutic hypothermia after cardiac arrest: a retrospective comparison of surface and endovascular cooling techniques. Resuscitation 2010;81:1117–22.
463. Bro-Jeppesen J, Hassager C, Wanscher M, et al. Post-hypothermia fever is associated with increased mortality after out-of-hospital cardiac arrest. Resuscitation 2013;84:1734–40.
464. Winters SA, Wolf KH, Kettinger SA, Seif EK, Jones JS, Bacon-Baguley T. Assessment of risk factors for post-rewarming "rebound hyperthermia" in cardiac arrest patients undergoing therapeutic hypothermia. Resuscitation 2013;84:1245–9.
465. Arrich J. Clinical application of mild therapeutic hypothermia after cardiac arrest. Crit Care Med 2007;35:1041–7.
466. Sandroni C, Cariou A, Cavallaro F, et al. Prognostication in comatose survivors of cardiac arrest: an advisory statement from the European Resuscitation Council and the European Society of Intensive Care Medicine. Resuscitation 2014;85:1779–89.
467. Stiell IG, Nichol G, Leroux BG, et al. Early versus later rhythm analysis in patients with out-of-hospital cardiac arrest. N Engl J Med 2011;365:787–97.
468. Laver S, Farrow C, Turner D, Nolan J. Mode of death after admission to an intensive care unit following cardiac arrest. Intensive Care Med 2004;30:2126–8.
469. Sandroni C, Cavallaro F, Callaway CW, et al. Predictors of poor neurological outcome in adult comatose survivors of cardiac arrest: a systematic review and meta-analysis. Part 2: Patients treated with therapeutic hypothermia. Resuscitation 2013;84:1324–38.
470. Sandroni C, Cavallaro F, Callaway CW, et al. Predictors of poor neurological outcome in adult comatose survivors of cardiac arrest: a systematic review and meta-analysis. Part 1: Patients not treated with therapeutic hypothermia. Resuscitation 2013;84:1310–23.
471. Geocadin RG, Peberdy MA, Lazar RM. Poor survival after cardiac arrest resuscitation: a self-fulfilling prophecy or biologic destiny? Crit Care Med 2012;40:979–80.
472. Samaniego EA, Mlynash M, Caulfield AF, Eyngorn I, Wijman CA. Sedation confounds outcome prediction in cardiac arrest survivors treated with hypothermia. Neurocrit Care 2011;15:113–9.
473. Sharshar T, Citerio G, Andrews PJ, et al. Neurological examination of critically ill patients: a pragmatic approach. Report of an ESICM expert panel. Intensive Care Med 2014;40:484–95.
474. Jorgensen EO, Holm S. The natural course of neurological recovery following cardiopulmonary resuscitation. Resuscitation 1998;36:111–22.
475. Wijdicks EF, Young GB. Myoclonus status in comatose patients after cardiac arrest. Lancet 1994;343:1642–3.
476. Cronberg T, Brizzi M, Liedholm LJ, et al. Neurological prognostication after cardiac arrest – recommendations from the Swedish Resuscitation Council. Resuscitation 2013;84:867–72.
477. Taccone FS, Cronberg T, Friberg H, et al. How to assess prognosis after cardiac arrest and therapeutic hypothermia. Crit Care 2014;18:202.
478. Greer DM, Yang J, Scripko PD, et al. Clinical examination for prognostication in comatose cardiac arrest patients. Resuscitation 2013;84:1546–51.
479. Dragancea I, Horn J, Kuiper M, et al. Neurological prognostication after cardiac arrest and targeted temperature management 33 degrees C versus 36 degrees C: results from a randomised controlled clinical trial. Resuscitation 2015.
480. Stammet P, Collignon O, Hassager C, et al. Neuron-specific enolase as a predictor of death or poor neurological outcome after out-of-hospital cardiac arrest and targeted temperature management at 33 degrees C and 36 degrees C. J Am Coll Cardiol 2015;65:2104–14.
481. Rossetti AO, Oddo M, Logroscino G, Kaplan PW. Prognostication after cardiac arrest and hypothermia: a prospective study. Ann Neurol 2010;67:301–7.
482. Stammet P, Wagner DR, Gilson G, Devaux Y. Modeling serum level of s100beta and bispectral index to predict outcome after cardiac arrest. J Am Coll Cardiol 2013;62:851–8.
483. Oddo M, Rossetti AO. Early multimodal outcome prediction after cardiac arrest in patients treated with hypothermia. Crit Care Med 2014;42:1340–7.
484. Lee BK, Jeung KW, Lee HY, Jung YH, Lee DH. Combining brain computed tomography and serum neuron specific enolase improves the prognostic performance compared to either alone in comatose cardiac arrest survivors treated with therapeutic hypothermia. Resuscitation 2013;84:1387–92.
485. Rittenberger JC, Popescu A, Brenner RP, Guyette FX, Callaway CW. Frequency and timing of nonconvulsive status epilepticus in comatose post-cardiac arrest subjects treated with hypothermia. Neurocrit Care 2012;16:114–22.
486. Greer DM. Unexpected good recovery in a comatose post-cardiac arrest patient with poor prognostic features. Resuscitation 2013;84:e81–2.
487. Al Thenayan E, Savard M, Sharpe M, Norton L, Young B. Predictors of poor neurologic outcome after induced mild hypothermia following cardiac arrest. Neurology 2008;71:1535–7.
488. Cronberg T, Rundgren M, Westhall E, et al. Neuron-specific enolase correlates with other prognostic markers after cardiac arrest. Neurology 2011;77:623–30.
489. Grossestreuer AV, Abella BS, Leary M, et al. Time to awakening and neurologic outcome in therapeutic hypothermia-treated cardiac arrest patients. Resuscitation 2013;84:1741–6.
490. Gold B, Puertas L, Davis SP, et al. Awakening after cardiac arrest and post resuscitation hypothermia: are we pulling the plug too early? Resuscitation 2014;85:211–4.
491. Krumnikl JJ, Bottiger BW, Strittmatter HJ, Motsch J. Complete recovery after 2 h of cardiopulmonary resuscitation following high-dose prostaglandin treatment for atonic uterine haemorrhage. Acta Anaesthesiol Scand 2002;46:1168–70.
492. Moulaert VRMP, Verbunt JA, van Heugten CM, Wade DT. Cognitive impairments in survivors of out-of-hospital cardiac arrest: a systematic review. Resuscitation 2009;80:297–305.
493. Wilder Schaaf KP, Artman LK, Peberdy MA, et al. Anxiety, depression, and PTSD following cardiac arrest: a systematic review of the literature. Resuscitation 2013;84:873–7.
494. Wachelder EM, Moulaert VR, van Heugten C, Verbunt JA, Bekkers SC, Wade DT. Life after survival: long-term daily functioning and quality of life after an out-of-hospital cardiac arrest. Resuscitation 2009;80:517–22.
495. Cronberg T, Lilja G, Rundgren M, Friberg H, Widner H. Long-term neurological outcome after cardiac arrest and therapeutic hypothermia. Resuscitation 2009;80:1119–23.
496. Torgersen J, Strand K, Bjelland TW, et al. Cognitive dysfunction and health-related quality of life after a cardiac arrest and therapeutic hypothermia. Acta Anaesthesiol Scand 2010;54:721–8.
497. Cobbe SM, Dalziel K, Ford I, Marsden AK. Survival of 1476 patients initially resuscitated from out of hospital cardiac arrest. BMJ 1996;312:1633–7.
498. Lundgren-Nilsson A, Rosen H, Hofgren C, Sunnerhagen KS. The first year after successful cardiac resuscitation: function, activity, participation and quality of life. Resuscitation 2005;66:285–9.

499. Moulaert VR, Wachelder EM, Verbunt JA, Wade DT, van Heugten CM. Determinants of quality of life in survivors of cardiac arrest. J Rehabil Med 2010;42:553–8.
500. Sandroni C, Adrie C, Cavallaro F, et al. Are patients brain-dead after successful resuscitation from cardiac arrest suitable as organ donors? A systematic review. Resuscitation 2010;81:1609–14.
501. Ranthe MF, Winkel BG, Andersen EW, et al. Risk of cardiovascular disease in family members of young sudden cardiac death victims. Eur Heart J 2013;34:503–11.
502. Engdahl J, Abrahamsson P, Bang A, Lindqvist J, Karlsson T, Herlitz J. Is hospital care of major importance for outcome after out-of-hospital cardiac arrest? Experience acquired from patients with out-of-hospital cardiac arrest resuscitated by the same Emergency Medical Service and admitted to one of two hospitals over a 16-year period in the municipality of Goteborg. Resuscitation 2000;43:201–11.
503. Liu JM, Yang Q, Pirrallo RG, Klein JP, Aufderheide TP. Hospital variability of out-of-hospital cardiac arrest survival. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2008;12:339–46.
504. Carr BG, Kahn JM, Merchant RM, Kramer AA, Neumar RW. Inter-hospital variability in post-cardiac arrest mortality. Resuscitation 2009;80:30–4.
505. Herlitz J, Engdahl J, Svensson L, Angquist KA, Silfverstolpe J, Holmberg S. Major differences in 1-month survival between hospitals in Sweden among initial survivors of out-of-hospital cardiac arrest. Resuscitation 2006;70:404–9.
506. Keenan SP, Dodek P, Martin C, Priestap F, Norena M, Wong H. Variation in length of intensive care unit stay after cardiac arrest: where you are is as important as who you are. Crit Care Med 2007;35:836–41.
507. Callaway CW, Schmicker R, Kampmeyer M, et al. Receiving hospital characteristics associated with survival after out-of-hospital cardiac arrest. Resuscitation 2010;81:524–9.
508. Stub D, Smith K, Bray JE, Bernard S, Duffy SJ, Kaye DM. Hospital characteristics are associated with patient outcomes following out-of-hospital cardiac arrest. Heart 2011;97:1489–94.
509. Marsch S, Tschan F, Semmer NK, Zobrist R, Hunziker PR, Hunziker S. ABC versus CAB for cardiopulmonary resuscitation: a prospective, randomized simulator-based trial. Swiss Med Wkly 2013;143:w13856.
510. Lubrano R, Cecchetti C, Bellelli E, et al. Comparison of times of intervention during pediatric CPR maneuvers using ABC and CAB sequences: a randomized trial. Resuscitation 2012;83:1473–7.
511. Sekiguchi H, Kondo Y, Kukita I. Verification of changes in the time taken to initiate chest compressions according to modified basic life support guidelines. Am J Emerg Med 2013;31:1248–50.
512. Maconochie I, de Caen A, Aickin R, et al. Part 6: pediatric basic life support and pediatric advanced life support. 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e149–70.
513. Sutton RM, French B, Niles DE, et al. 2010 American Heart Association recommended compression depths during pediatric in-hospital resuscitations are associated with survival. Resuscitation 2014;85:1179–84.
514. Biarent D, Bingham R, Richmond S, et al. European Resuscitation Council Guidelines for Resuscitation 2005. Section 6: Paediatric life support. Resuscitation 2005;67:S97–133.
515. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests: epidemiology and outcome. Resuscitation 1995;30:141–50.
516. Sirbaugh PE, Pepe PE, Shook JE, et al. A prospective, population-based study of the demographics, epidemiology, management, and outcome of out-of-hospital pediatric cardiopulmonary arrest. Ann Emerg Med 1999;33:174–84.
517. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. Ann Emerg Med 1995;25:495–501.
518. Young KD, Seidel JS. Pediatric cardiopulmonary resuscitation: a collective review. Ann Emerg Med 1999;33:195–205.
519. Reis AG, Nadkarni V, Perondi MB, Grisi S, Berg RA. A prospective investigation into the epidemiology of in-hospital pediatric cardiopulmonary resuscitation using the international Utstein reporting style. Pediatrics 2002;109:200–9.
520. Young KD, Gausche-Hill M, McClung CD, Lewis RJ. A prospective, population-based study of the epidemiology and outcome of out-of-hospital pediatric cardiopulmonary arrest. Pediatrics 2004;114:157–64.
521. Rajan S, Wissenberg M, Folke F, et al. Out-of-hospital cardiac arrests in children and adolescents: incidences, outcomes, and household socioeconomic status. Resuscitation 2015;88:12–9.
522. Gupta P, Tang X, Gall CM, Lauer C, Rice TB, Wetzel RC. Epidemiology and outcomes of in-hospital cardiac arrest in critically ill children across hospitals of varied center volume: a multi-center analysis. Resuscitation 2014;85:1473–9.
523. Nishiuchi T, Hayashino Y, Iwami T, et al. Epidemiological characteristics of sudden cardiac arrest in schools. Resuscitation 2014;85:1001–6.
524. Pilmer CM, Kirsh JA, Hildebrandt D, Krahn AD, Gow RM. Sudden cardiac death in children and adolescents between 1 and 19 years of age. Heart Rhythm 2014;11:239–45.
525. Moler FW, Donaldson AE, Meert K, et al. Multicenter cohort study of out-of-hospital pediatric cardiac arrest. Crit Care Med 2011;39:141–9.
526. Tibballs J, Kinney S. Reduction of hospital mortality and of preventable cardiac arrest and death on introduction of a pediatric medical emergency team. Pediatr Crit Care Med: J Soc Crit Care Med World Feder Pediatr Intensive Crit Care Soc 2009;10:306–12.
527. Chan PS, Jain R, Nallmothu BK, Berg RA, Sasson C. Rapid response teams: a systematic review and meta-analysis. Arch Intern Med 2010;170:18–26.
528. Bonafide CP, Localio AR, Song L, et al. Cost–benefit analysis of a medical emergency team in a children's hospital. Pediatrics 2014;134:235–41.
529. Hayes LW, Dobyns EL, DiGiovine B, et al. A multicenter collaborative approach to reducing pediatric codes outside the ICU. Pediatrics 2012;129:e785–91.
530. Chaiyakulsil C, Pandee U. Validation of pediatric early warning score in pediatric emergency department. Pediatr Int 2015.
531. Randhawa S, Roberts-Turner R, Woronick K, DuVal J. Implementing and sustaining evidence-based nursing practice to reduce pediatric cardiopulmonary arrest. Nurs Res 2011;33:443–56.
532. Fleming S, Thompson M, Stevens R, et al. Normal ranges of heart rate and respiratory rate in children from birth to 18 years of age: a systematic review of observational studies. Lancet 2011;377:1011–8.
533. Carcillo JA. Pediatric septic shock and multiple organ failure. Crit Care Clin 2003;19:413–40, viii.
534. Tsung JW, Blaivas M. Feasibility of correlating the pulse check with focused point-of-care echocardiography during pediatric cardiac arrest: a case series. Resuscitation 2008;77:264–9.
535. Inagawa G, Morimura N, Miwa T, Okuda K, Hirata M, Hiroki K. A comparison of five techniques for detecting cardiac activity in infants. Paediatr Anaesth 2003;13:141–6.
536. Frederick K, Bixby E, Orzel MN, Stewart-Brown S, Willett K. Will changing the emphasis from 'pulseless' to 'no signs of circulation' improve the recall scores for effective life support skills in children? Resuscitation 2002;55:255–61.
537. Maitland K, Kiguli S, Opoka RO, et al. Mortality after fluid bolus in African children with severe infection. N Engl J Med 2011;364:2483–95.
538. Maitland K, George EC, Evans JA, et al. Exploring mechanisms of excess mortality with early fluid resuscitation: insights from the FEAST trial. BMC Med 2013;11:68.
539. Kelm DJ, Perrin JT, Cartin-Ceba R, Gajic O, Schenck L, Kennedy CC. Fluid overload in patients with severe sepsis and septic shock treated with early goal-directed therapy is associated with increased acute need for fluid-related medical interventions and hospital death. Shock 2015;43:68–73.
540. Dung NM, Day NP, Tam DT, et al. Fluid replacement in dengue shock syndrome: a randomized, double-blind comparison of four intravenous-fluid regimens. Clin Infect Dis: Off Publ Infect Dis Soc Am 1999;29:787–94.
541. Ngo NT, Cao XT, Kneen R, et al. Acute management of dengue shock syndrome: a randomized double-blind comparison of 4 intravenous fluid regimens in the first hour. Clin Infect Dis: Off Publ Infect Dis Soc Am 2001;32:204–13.
542. Wills BA, Nguyen MD, Ha TL, et al. Comparison of three fluid solutions for resuscitation in dengue shock syndrome. N Engl J Med 2005;353:877–89.
543. Upadhyay M, Singhi S, Murlidharan J, Kaur N, Majumdar S. Randomized evaluation of fluid resuscitation with crystalloid (saline) and colloid (polymer from degraded gelatin in saline) in pediatric septic shock. Indian Pediatr 2005;42:223–31.
544. Santhanam I, Sangareddi S, Venkataraman S, Kissoon N, Thiruvengadamudayan V, Kasthuri RK. A prospective randomized controlled study of two fluid regimens in the initial management of septic shock in the emergency department. Pediatr Emerg Care 2008;24:647–55.
545. Carcillo JA, Davis AL, Zaritsky A. Role of early fluid resuscitation in pediatric septic shock. JAMA 1991;266:1242–5.
546. Rechner JA, Loach VJ, Ali MT, Barber VS, Young JD, Mason DG. A comparison of the laryngeal mask airway with facemask and oropharyngeal airway for manual ventilation by critical care nurses in children. Anaesthesia 2007;62:790–5.
547. Blevin AE, McDouall SF, Rechner JA, et al. A comparison of the laryngeal mask airway with the facemask and oropharyngeal airway for manual ventilation by first responders in children. Anaesthesia 2009;64:1312–6.
548. Hedges JR, Mann NC, Meischke H, Robbins M, Goldberg R, Zapka J. Assessment of chest pain onset and out-of-hospital delay using standardized interview questions: the REACT Pilot Study. Rapid Early Action for Coronary Treatment (REACT) Study Group. Acad Emerg Med: Off J Soc Acad Emerg Med 1998;5:773–80.
549. Wang HE, Kupas DF, Paris PM, Bates RR, Costantino JP, Yealy DM. Multivariate predictors of failed prehospital endotracheal intubation. Acad Emerg Med: Off J Soc Acad Emerg Med 2003;10:717–24.
550. Pepe P, Zachariah B, Chandra N. Invasive airway technique in resuscitation. Ann Emerg Med 1991;22:393–403.
551. Deakers TW, Reynolds G, Stretton M, Newth CJ. Cuffed endotracheal tubes in pediatric intensive care. J Pediatr 1994;125:57–62.
552. Newth CJ, Rachman B, Patel N, Hammer J. The use of cuffed versus uncuffed endotracheal tubes in pediatric intensive care. J Pediatr 2004;144:333–7.
553. Mhanna MJ, Zamel YB, Tichy CM, Super DM. The "air leak" test around the endotracheal tube, as a predictor of postextubation stridor, is age dependent in children. Crit Care Med 2002;30:2639–43.
554. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. Ann Emerg Med 2001;37:32–7.
555. Gausche M, Lewis RJ, Stratton SJ, et al. Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: a controlled clinical trial. JAMA 2000;283:783–90.
556. Hartrey R, Kestin IG. Movement of oral and nasal tracheal tubes as a result of changes in head and neck position. Anaesthesia 1995;50:682–7.
557. Van de Louw A, Cracco C, Cerf C, et al. Accuracy of pulse oximetry in the intensive care unit. Intensive Care Med 2001;27:1606–13.
558. Seguin P, Le Rouzo A, Tanguy M, Guillou YM, Feuillu A, Malledant Y. Evidence for the need of bedside accuracy of pulse oximetry in an intensive care unit. Crit Care Med 2000;28:703–6.

559. Del Castillo J, Lopez-Herce J, Matamoros M, et al. Hyperoxia, hypocapnia and hypercapnia as outcome factors after cardiac arrest in children. Resuscitation 2012;83:1456–61.
560. Stockinger ZT, McSwain Jr NE. Prehospital endotracheal intubation for trauma does not improve survival over bag-valve-mask ventilation. J Trauma 2004;56:531–6.
561. Pitetti R, Glustein JZ, Bhende MS. Prehospital care and outcome of pediatric out-of-hospital cardiac arrest. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2002;6:283–90.
562. Bhende MS, Thompson AE, Orr RA. Utility of an end-tidal carbon dioxide detector during stabilization and transport of critically ill children. Pediatrics 1992;89:1042–4.
563. Bhende MS, LaCovey DC. End-tidal carbon dioxide monitoring in the prehospital setting. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2001;5:208–13.
564. Ornato JP, Shipley JB, Racht EM, et al. Multicenter study of a portable, hand-size, colorimetric end-tidal carbon dioxide detection device. Ann Emerg Med 1992;21:518–23.
565. Gonzalez del Rey JA, Poirier MP, Digiulio GA. Evaluation of an ambu-bag valve with a self-contained, colorimetric end-tidal CO_2 system in the detection of airway mishaps: an animal trial. Pediatr Emerg Care 2000;16:121–3.
566. Bhende MS, Karasic DG, Karasic RB. End-tidal carbon dioxide changes during cardiopulmonary resuscitation after experimental asphyxial cardiac arrest. Emerg Med 1996;14:349–50.
567. DeBehnke DJ, Hilander SJ, Dobler DW, Wickman LL, Swart GL. The hemodynamic and arterial blood gas response to asphyxiation: a canine model of pulseless electrical activity. Resuscitation 1995;30:169–75.
568. Ornato JP, Garnett AR, Glauser FL. Relationship between cardiac output and the end-tidal carbon dioxide tension. Ann Emerg Med 1990;19:1104–6.
569. Kanter RK, Zimmerman JJ, Strauss RH, Stoeckel KA. Pediatric emergency intravenous access. Evaluation of a protocol. Am J Dis Child 1986;140:132–4.
570. Anson JA. Vascular access in resuscitation: is there a role for the intraosseous route? Anesthesiology 2014;120:1015–31.
571. Neuhaus D, Weiss M, Engelhardt T, et al. Semi-elective intraosseous infusion after failed intravenous access in pediatric anesthesia. Paediatr Anaesth 2010;20:168–71.
572. Cameron JL, Fontanarosa PB, Passalaqua AM. A comparative study of peripheral to central circulation delivery times between intraosseous and intravenous injection using a radionuclide technique in normovolemic and hypovolemic canines. J Emerg Med 1989;7:123–7.
573. Warren DW, Kissoon N, Sommerauer JF, Rieder MJ. Comparison of fluid infusion rates among peripheral intravenous and humerus, femur, malleolus, and tibial intraosseous sites in normovolemic and hypovolemic piglets. Ann Emerg Med 1993;22:183–6.
574. Buck ML, Wiggins BS, Sesler JM. Intraosseous drug administration in children and adults during cardiopulmonary resuscitation. Ann Pharmacother 2007;41:1679–86.
575. Brickman KR, Krupp K, Rega P, Alexander J, Guinness M. Typing and screening of blood from intraosseous access. Ann Emerg Med 1992;21:414–7.
576. Johnson L, Kissoon N, Fiallos M, Abdelmoneim T, Murphy S. Use of intraosseous blood to assess blood chemistries and hemoglobin during cardiopulmonary resuscitation with drug infusions. Crit Care Med 1999;27:1147–52.
577. Ummenhofer W, Frei FJ, Urwyler A, Drewe J. Are laboratory values in bone marrow aspirate predictable for venous blood in paediatric patients? Resuscitation 1994;27:123–8.
578. Ong ME, Chan YH, Oh JJ, Ngo AS. An observational, prospective study comparing tibial and humeral intraosseous access using the EZ-IO. Am J Emerg Med 2009;27:8–15.
579. Kleinman ME, Oh W, Stonestreet BS. Comparison of intravenous and endotracheal epinephrine during cardiopulmonary resuscitation in newborn piglets. Crit Care Med 1999;27:2748–54.
580. Perel P, Roberts I, Ker K. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev 2013;2:CD000567.
581. Myburgh J, Cooper DJ, Finfer S, et al. Saline or albumin for fluid resuscitation in patients with traumatic brain injury. N Engl J Med 2007;357:874–84.
582. Dellinger RP, Levy MM, Rhodes A, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock, 2012. Intensive Care Med 2013;39:165–228.
583. Levy B, Perez P, Perny J, Thivilier C, Gerard A. Comparison of norepinephrine-dobutamine to epinephrine for hemodynamics, lactate metabolism, and organ function variables in cardiogenic shock. A prospective, randomized pilot study. Crit Care Med 2011;39:450–5.
584. Burdett E, Dushianthan A, Bennett-Guerrero E, et al. Perioperative buffered versus non-buffered fluid administration for surgery in adults. Cochrane Database Syst Rev 2012;12:CD004089.
585. Shaw AD, Raghunathan K, Peyerl FW, Munson SH, Paluszkiewicz SM, Schermer CR. Association between intravenous chloride load during resuscitation and in-hospital mortality among patients with SIRS. Intensive Care Med 2014;40:1897–905.
586. Yunos NM, Bellomo R, Bailey M. Chloride-restrictive fluid administration and incidence of acute kidney injury – reply. JAMA 2013;309:543–4.
587. Yunos NM, Bellomo R, Hegarty C, Story D, Ho L, Bailey M. Association between a chloride-liberal vs chloride-restrictive intravenous fluid administration strategy and kidney injury in critically ill adults. JAMA 2012;308:1566–72.
588. Elmer J, Wilcox SR, Raja AS. Massive transfusion in traumatic shock. J Emerg Med 2013;44:829–38.
589. Kua JP, Ong GY, Ng KC. Physiologically-guided balanced resuscitation: an evidence-based approach for acute fluid management in paediatric major trauma. Ann Acad Med Singap 2014;43:595–604.
590. Patterson MD, Boenning DA, Klein BL, et al. The use of high-dose epinephrine for patients with out-of-hospital cardiopulmonary arrest refractory to prehospital interventions. Pediatr Emerg Care 2005;21:227–37.
591. Perondi MB, Reis AG, Paiva EF, Nadkarni VM, Berg RA. A comparison of high-dose and standard-dose epinephrine in children with cardiac arrest. N Engl J Med 2004;350:1722–30.
592. Carpenter TC, Stenmark KR. High-dose epinephrine is not superior to standard-dose epinephrine in pediatric in-hospital cardiopulmonary arrest. Pediatrics 1997;99:403–8.
593. Dieckmann RA, Vardis R. High-dose epinephrine in pediatric out-of-hospital cardiopulmonary arrest. Pediatrics 1995;95:901–13.
594. Enright K, Turner C, Roberts P, Cheng N, Browne G. Primary cardiac arrest following sport or exertion in children presenting to an emergency department: chest compressions and early defibrillation can save lives, but is intravenous epinephrine always appropriate? Pediatr Emerg Care 2012;28:336–9.
595. Saharan S, Balaji S. Cardiovascular collapse during amiodarone infusion in a hemodynamically compromised child with refractory supraventricular tachycardia. Ann Pediatr Cardiol 2015;8:50–2.
596. Brady WJ, Swart G, DeBehnke DJ, Ma OJ, Aufderheide TP. The efficacy of atropine in the treatment of hemodynamically unstable bradycardia and atrioventricular block: prehospital and emergency department considerations. Resuscitation 1999;41:47–55.
597. Smith I, Monk TG, White PF. Comparison of transesophageal atrial pacing with anticholinergic drugs for the treatment of intraoperative bradycardia. Anesth Analg 1994;78:245–52.
598. Chadda KD, Lichstein E, Gupta PK, Kourtesis P. Effects of atropine in patients with bradyarrhythmia complicating myocardial infarction: usefulness of an optimum dose for overdrive. Am J Med 1977;63:503–10.
599. van Walraven C, Stiell IG, Wells GA, Hebert PC, Vandemheen K. Do advanced cardiac life support drugs increase resuscitation rates from in-hospital cardiac arrest? The OTAC Study Group. Ann Emerg Med 1998;32:544–53.
600. Gupta P, Tomar M, Radhakrishnan S, Shrivastava S. Hypocalcemic cardiomyopathy presenting as cardiogenic shock. Ann Pediatr Cardiol 2011;4:152–5.
601. Kette F, Ghuman J, Parr M. Calcium administration during cardiac arrest: a systematic review. Eur J Emerg Med: Off J Eur Soc Emerg Med 2013;20:72–8.
602. Dias CR, Leite HP, Nogueira PC, Brunow de Carvalho W. Ionized hypocalcemia is an early event and is associated with organ dysfunction in children admitted to the intensive care unit. J Crit Care 2013;28:810–5.
603. Krinsley JS. Effect of an intensive glucose management protocol on the mortality of critically ill adult patients. Mayo Clin Proc 2004;79:992–1000.
604. Salter N, Quin G, Tracy E. Cardiac arrest in infancy: don't forget glucose! Emerg Med J 2010;27:720–1.
605. Topjian AA, Berg RA, Bierens JJ, et al. Brain resuscitation in the drowning victim. Neurocrit Care 2012;17:441–67.
606. Allegra J, Lavery R, Cody R, et al. Magnesium sulfate in the treatment of refractory ventricular fibrillation in the prehospital setting. Resuscitation 2001;49:245–9.
607. Reis AG, Ferreira de Paiva E, Schvartsman C, Zaritsky AL. Magnesium in cardiopulmonary resuscitation: critical review. Resuscitation 2008;77:21–5.
608. Tzivoni D, Banai S, Schuger C, et al. Treatment of torsade de pointes with magnesium sulfate. Circulation 1988;77:392–7.
609. Bar-Joseph G, Abramson NS, Kelsey SF, Mashiach T, Craig MT, Safar P. Improved resuscitation outcome in emergency medical systems with increased usage of sodium bicarbonate during cardiopulmonary resuscitation. Acta Anaesthesiol Scand 2005;49:6–15.
610. Weng YM, Wu SH, Li WC, Kuo CW, Chen SY, Chen JC. The effects of sodium bicarbonate during prolonged cardiopulmonary resuscitation. Am J Emerg Med 2013;31:562–5.
611. Raymond TT, Stromberg D, Stigall W, Burton G, Zaritsky A. American Heart Association's Get With The Guidelines-Resuscitation I. Sodium bicarbonate use during in-hospital pediatric pulseless cardiac arrest – a report from the American Heart Association Get With The Guidelines((R))-Resuscitation. Resuscitation 2015;89:106–13.
612. Duncan JM, Meaney P, Simpson P, Berg RA, Nadkarni V, Schexnayder S. Vasopressin for in-hospital pediatric cardiac arrest: results from the American Heart Association National Registry of Cardiopulmonary Resuscitation. Pediatr Crit Care Med: J Soc Crit Care Med World Feder Pediatr Intensive Crit Care Soc 2009;10:191–5.
613. Mukoyama T, Kinoshita K, Nagao K, Tanjoh K. Reduced effectiveness of vasopressin in repeated doses for patients undergoing prolonged cardiopulmonary resuscitation. Resuscitation 2009;80:755–61.
614. Matok I, Vardi A, Augarten A, et al. Beneficial effects of terlipressin in prolonged pediatric cardiopulmonary resuscitation: a case series. Crit Care Med 2007;35:1161–4.
615. Mentzelopoulos SD, Malachias S, Chamos C, et al. Vasopressin, steroids, and epinephrine and neurologically favorable survival after in-hospital cardiac arrest: a randomized clinical trial. JAMA 2013;310:270–9.
616. Daley MJ, Lat I, Mieure KD, Jennings HR, Hall JB, Kress JP. A comparison of initial monotherapy with norepinephrine versus vasopressin for resuscitation in septic shock. Ann Pharmacother 2013;47:301–10.

617. Atkins DL, Sirna S, Kieso R, Charbonnier F, Kerber RE. Pediatric defibrillation: importance of paddle size in determining transthoracic impedance. Pediatrics 1988;82:914–8.
618. Atkins DL, Kerber RE. Pediatric defibrillation: current flow is improved by using "adult" electrode paddles. Pediatrics 1994;94:90–3.
619. Gurnett CA, Atkins DL. Successful use of a biphasic waveform automated external defibrillator in a high-risk child. Am J Cardiol 2000;86:1051–3.
620. Rossano J, Quan L, Schiff MMAKDLA. Survival is not correlated with defibrillation dosing in pediatric out-of-hospital ventricular fibrillation. Circulation 2003;108. IV-320-1.
621. Atkinson E, Mikysa B, Conway JA, et al. Specificity and sensitivity of automated external defibrillator rhythm analysis in infants and children. Ann Emerg Med 2003;42:185–96.
622. Cecchin F, Jorgenson DB, Berul CI, et al. Is arrhythmia detection by automatic external defibrillator accurate for children? Sensitivity and specificity of an automatic external defibrillator algorithm in 696 pediatric arrhythmias. Circulation 2001;103:2483–8.
623. Atkins DL, Hartley LL, York DK. Accurate recognition and effective treatment of ventricular fibrillation by automated external defibrillators in adolescents. Pediatrics 1998;101:393–7.
624. Samson R, Berg R, Bingham R. Pediatric Advanced Life Support Task Force ILCoR. Use of automated external defibrillators for children: an update. An advisory statement from the Pediatric Advanced Life Support Task Force, International Liaison Committee on Resuscitation. Resuscitation 2003;57:237–43.
625. Berg RA, Samson RA, Berg MD, et al. Better outcome after pediatric defibrillation dosage than adult dosage in a swine model of pediatric ventricular fibrillation. J Am Coll Cardiol 2005;45:786–9.
626. Herlitz J, Engdahl J, Svensson L, Young M, Angquist KA, Holmberg S. Characteristics and outcome among children suffering from out of hospital cardiac arrest in Sweden. Resuscitation 2005;64:37–40.
627. Bray JE, Di Palma S, Jacobs I, Straney L, Finn J. Trends in the incidence of presumed cardiac out-of-hospital cardiac arrest in Perth, Western Australia, 1997–2010. Resuscitation 2014;85:757–61.
628. Mitani Y, Ohta K, Ichida F, et al. Circumstances and outcomes of out-of-hospital cardiac arrest in elementary and middle school students in the era of public-access defibrillation. Circ J: Off J Jpn Circ Soc 2014;78:701–7.
629. Lin YR, Wu HP, Chen WL, et al. Predictors of survival and neurologic outcomes in children with traumatic out-of-hospital cardiac arrest during the early postresuscitative period. J Trauma Acute Care Surg 2013;75:439–47.
630. Zeng J, Qian S, Zheng M, Wang Y, Zhou G, Wang H. The epidemiology and resuscitation effects of cardiopulmonary arrest among hospitalized children and adolescents in Beijing: an observational study. Resuscitation 2013;84:1685–90.
631. Cheung W, Middleton P, Davies S, Tummala S, Thanakrishnan G, Gullick J. A comparison of survival following out-of-hospital cardiac arrest in Sydney, Australia, between 2004–2005 and 2009–2010. Crit Care Resusc 2013;15:241–6.
632. Nitta M, Kitamura T, Iwami T, et al. Out-of-hospital cardiac arrest due to drowning among children and adults from the Utstein Osaka Project. Resuscitation 2013;84:1568–73.
633. De Maio VJ, Osmond MH, Stiell IG, et al. Epidemiology of out-of hospital pediatric cardiac arrest due to trauma. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2012;16:230–6.
634. Deasy C, Bray J, Smith K, et al. Paediatric traumatic out-of-hospital cardiac arrests in Melbourne, Australia. Resuscitation 2012;83:471–5.
635. Samson RA, Nadkarni VM, Meaney PA, Carey SM, Berg MD, Berg RA. Outcomes of in-hospital ventricular fibrillation in children. N Engl J Med 2006;354:2328–39.
636. Cummins RO, Graves JR, Larsen MP, et al. Out-of-hospital transcutaneous pacing by emergency medical technicians in patients with asystolic cardiac arrest. N Engl J Med 1993;328:1377–82.
637. Benson Jr D, Smith W, Dunnigan A, Sterba R, Gallagher J. Mechanisms of regular wide QRS tachycardia in infants and children. Am J Cardiol 1982;49:1778–88.
638. Lopez-Herce Cid J, Dominguez Sampedro P, Rodriguez Nunez A, et al. Cardiorespiratory arrest in children with trauma. An Pediatr (Barc) 2006;65:439–47.
639. Perron AD, Sing RF, Branas CC, Huynh T. Predicting survival in pediatric trauma patients receiving cardiopulmonary resuscitation in the prehospital setting. Prehosp Emerg Care: Off J Natl Assoc EMS Phys Natl Assoc State EMS Dir 2001;5:6–9.
640. Brindis SL, Gausche-Hill M, Young KD, Putnam B. Universally poor outcomes of pediatric traumatic arrest: a prospective case series and review of the literature. Pediatr Emerg Care 2011;27:616–21.
641. Murphy JT, Jaiswal K, Sabella J, Vinson L, Megison S, Maxson RT. Prehospital cardiopulmonary resuscitation in the pediatric trauma patient. J Pediatr Surg 2010;45:1413–9.
642. Widdel L, Winston KR. Prognosis for children in cardiac arrest shortly after blunt cranial trauma. J Trauma 2010;69:783–8.
643. Duron V, Burke RV, Bliss D, Ford HR, Upperman JS. Survival of pediatric blunt trauma patients presenting with no signs of life in the field. J Trauma Acute Care Surg 2014;77:422–6.
644. Easter JS, Vinton DT, Haukoos JS. Emergent pediatric thoracotomy following traumatic arrest. Resuscitation 2012;83:1521–4.
645. Hofbauer M, Hupfl M, Figl M, Hochtl-Lee L, Kdolsky R. Retrospective analysis of emergency room thoracotomy in pediatric severe trauma patients. Resuscitation 2011;82:185–9.
646. Polderman FN, Cohen J, Blom NA, et al. Sudden unexpected death in children with a previously diagnosed cardiovascular disorder. Int J Cardiol 2004;95:171–6.
647. Sanatani S, Wilson G, Smith CR, Hamilton RM, Williams WG, Adatia I. Sudden unexpected death in children with heart disease. Congenit Heart Dis 2006;1:89–97.
648. Morris K, Beghetti M, Petros A, Adatia I, Bohn D. Comparison of hyperventilation and inhaled nitric oxide for pulmonary hypertension after repair of congenital heart disease. Crit Care Med 2000;28:2974–8.
649. Hildebrand CA, Hartmann AG, Arcinue EL, Gomez RJ, Bing RJ. Cardiac performance in pediatric near-drowning. Crit Care Med 1988;16:331–5.
650. Mayr V, Luckner G, Jochberger S, et al. Arginine vasopressin in advanced cardiovascular failure during the post-resuscitation phase after cardiac arrest. Resuscitation 2007;72:35–44.
651. Conlon TW, Falkensammer CB, Hammond RS, Nadkarni VM, Berg RA, Topjian AA. Association of left ventricular systolic function and vasopressor support with survival following pediatric out-of-hospital cardiac arrest. Pediatr Crit Care Med 2015;16:146–54.
652. Bougouin W, Cariou A. Management of postcardiac arrest myocardial dysfunction. Curr Opin Crit Care 2013;19:195–201.
653. Guerra-Wallace MM, Casey III FL, Bell MJ, Fink EL, Hickey RW. Hyperoxia and hypoxia in children resuscitated from cardiac arrest. Pediatr Crit Care Med 2013;14:e143–8.
654. Ferguson LP, Durward A, Tibby SM. Relationship between arterial partial oxygen pressure after resuscitation from cardiac arrest and mortality in children. Circulation 2012;126:335–42.
655. Bennett KS, Clark AE, Meert KL, et al. Early oxygenation and ventilation measurements after pediatric cardiac arrest: lack of association with outcome. Crit Care Med 2013;41:1534–42.
656. Lopez-Herce J, del Castillo J, Matamoros M, et al. Post return of spontaneous circulation factors associated with mortality in pediatric in-hospital cardiac arrest: a prospective multicenter multinational observational study. Crit Care 2014;18:607.
657. Gluckman PD, Wyatt JS, Azzopardi D, et al. Selective head cooling with mild systemic hypothermia after neonatal encephalopathy: multicentre randomised trial. Lancet 2005;365:663–70.
658. Moler FW, Silverstein FS, Holubkov R, et al. Therapeutic hypothermia after out-of-hospital cardiac arrest in children. N Engl J Med 2015;372:1898–908.
659. Coimbra C, Drake M, Boris-Moller F, Wieloch T. Long-lasting neuroprotective effect of postischemic hypothermia and treatment with an anti-inflammatory/antipyretic drug. Evidence for chronic encephalopathic processes following ischemia. Stroke 1996;27:1578–85.
660. van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med 2001;345:1359–67.
661. Van den Berghe G, Wilmer A, Hermans G, et al. Intensive insulin therapy in the medical ICU. N Engl J Med 2006;354:449–61.
662. Treggiari MM, Karir V, Yanez ND, Weiss NS, Daniel S, Deem SA. Intensive insulin therapy and mortality in critically ill patients. Crit Care 2008;12:R29.
663. Losert H, Sterz F, Roine RO, et al. Strict normoglycaemic blood glucose levels in the therapeutic management of patients within 12 h after cardiac arrest might not be necessary. Resuscitation 2008;76:214–20.
664. Oksanen T, Skrifvars MB, Varpula T, et al. Strict versus moderate glucose control after resuscitation from ventricular fibrillation. Intensive Care Med 2007;33:2093–100.
665. Lopez-Herce J, Garcia C, Dominguez P, et al. Characteristics and outcome of cardiorespiratory arrest in children. Resuscitation 2004;63:311–20.
666. Idris AH, Berg RA, Bierens J, et al. Recommended guidelines for uniform reporting of data from drowning: the "Utstein style". Resuscitation 2003;59:45–57.
667. Eich C, Brauer A, Timmermann A, et al. Outcome of 12 drowned children with attempted resuscitation on cardiopulmonary bypass: an analysis of variables based on the "Utstein Style for Drowning". Resuscitation 2007;75:42–52.
668. Tinsley C, Hill JB, Shah J, et al. Experience of families during cardiopulmonary resuscitation in a pediatric intensive care unit. Pediatrics 2008;122:e799–804.
669. Vavarouta A, Xanthos T, Papadimitriou L, Kouskouni E, Iacovidou N. Family presence during resuscitation and invasive procedures: physicians' and nurses' attitudes working in pediatric departments in Greece. Resuscitation 2011;82:713–6.
670. Corniero P, Gamell A, Parra Cotanda C, Trenchs V, Cubells CL. Family presence during invasive procedures at the emergency department: what is the opinion of Spanish medical staff? Pediatr Emerg Care 2011;27:86–91.
671. Ersdal HL, Mduma E, Svensen E, Perlman JM. Early initiation of basic resuscitation interventions including face mask ventilation may reduce birth asphyxia related mortality in low-income countries: a prospective descriptive observational study. Resuscitation 2012;83:869–73.
672. Perlman JM, Risser R. Cardiopulmonary resuscitation in the delivery room: associated clinical events. Arch Pediatr Adolesc Med 1995;149:20–5.
673. Barber CA, Wyckoff MH. Use and efficacy of endotracheal versus intravenous epinephrine during neonatal cardiopulmonary resuscitation in the delivery room. Pediatrics 2006;118:1028–34.

674. Ghavam S, Batra D, Mercer J, et al. Effects of placental transfusion in extremely low birthweight infants: meta-analysis of long- and short-term outcomes. Transfusion 2014;54:1192–8.
675. Budin P [Maloney WJ, Trans.] The nursling. The feeding and hygiene of premature and full-term infants. London: The Caxton Publishing Company; 1907.
676. Wyllie J, Perlman JM, Kattwinkel J, et al. Part 7: Neonatal resuscitation: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e171–203.
677. Apgar V. A proposal for a new method of evaluation of the newborn infant. Curr Res Anesth Analg 1953;32.
678. Chamberlain G, Banks J. Assessment of the Apgar score. Lancet 1974;2:1225–8.
679. Owen CJ, Wyllie JP. Determination of heart rate in the baby at birth. Resuscitation 2004;60:213–7.
680. Dawson JA, Saraswat A, Simionato L, et al. Comparison of heart rate and oxygen saturation measurements from Masimo and Nellcor pulse oximeters in newly born term infants. Acta Paediatr 2013;102:955–60.
681. Kamlin CO, Dawson JA, O'Donnell CP, et al. Accuracy of pulse oximetry measurement of heart rate of newborn infants in the delivery room. J Pediatr 2008;152:756–60.
682. Katheria A, Rich W, Finer N. Electrocardiogram provides a continuous heart rate faster than oximetry during neonatal resuscitation. Pediatrics 2012;130:e1177–81.
683. Kamlin CO, O'Donnell CP, Everest NJ, Davis PG, Morley CJ. Accuracy of clinical assessment of infant heart rate in the delivery room. Resuscitation 2006;71:319–21.
684. Voogdt KG, Morrison AC, Wood FE, van Elburg RM, Wyllie JP. A randomised, simulated study assessing auscultation of heart rate at birth. Resuscitation 2010;81:1000–3.
685. O'Donnell CP, Kamlin CO, Davis PG, Carlin JB, Morley CJ. Clinical assessment of infant colour at delivery. Arch Dis Child Fetal Neonatal Ed 2007;92:F465–7.
686. Konstantelos D, Gurth H, Bergert R, Ifflaender S, Rudiger M. Positioning of term infants during delivery room routine handling – analysis of videos. BMC Pediatr 2014;14:33.
687. Kelleher J, Bhat R, Salas AA, et al. Oronasopharyngeal suction versus wiping of the mouth and nose at birth: a randomised equivalency trial. Lancet 2013;382:326–30.
688. Al Takroni AM, Parvathi CK, Mendis KB, Hassan S, Reddy I, Kudair HA. Selective tracheal suctioning to prevent meconium aspiration syndrome. Gynaecol Obstet 1998;63:259–63.
689. Chettri S, Adhisivam B, Bhat BV. Endotracheal suction for nonvigorous neonates born through meconium stained amniotic fluid: a randomized controlled trial. J Pediatr 2015.
690. Davis RO, Philips III JB, Harris Jr BA, Wilson ER, Huddleston JF. Fatal meconium aspiration syndrome occurring despite airway management considered appropriate. Am J Obstet Gynecol 1985;151:731–6.
691. Manganaro R, Mami C, Palmara A, Paolata A, Gemelli M. Incidence of meconium aspiration syndrome in term meconium-stained babies managed at birth with selective tracheal intubation. J Perinat Med 2001;29:465–8.
692. Yoder BA. Meconium-stained amniotic fluid and respiratory complications: impact of selective tracheal suction. Obstet Gynecol 1994;83:77–84.
693. Wyllie J, Perlman JM, Kattwinkel J, et al. Part 11: Neonatal resuscitation: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2010;81(Suppl. 1):e260–87.
694. Vyas H, Milner AD, Hopkin IE, Boon AW. Physiologic responses to prolonged and slow-rise inflation in the resuscitation of the asphyxiated newborn infant. J Pediatr 1981;99:635–9.
695. Boon AW, Milner AD, Hopkin IE. Lung expansion, tidal exchange, and formation of the functional residual capacity during resuscitation of asphyxiated neonates. J Pediatr 1979;95:1031–6.
696. Mariani G, Dik PB, Ezquer A, et al. Pre-ductal and post-ductal O_2 saturation in healthy term neonates after birth. J Pediatr 2007;150:418–21.
697. Dawson JA, Kamlin CO, Vento M, et al. Defining the reference range for oxygen saturation for infants after birth. Pediatrics 2010;125:e1340–7.
698. Davis PG, Tan A, O'Donnell CP, Schulze A. Resuscitation of newborn infants with 100% oxygen or air: a systematic review and meta-analysis. Lancet 2004;364:1329–33.
699. Vento M, Moro M, Escrig R, et al. Preterm resuscitation with low oxygen causes less oxidative stress. Inflamm Chronic Lung Dis Pediatr 2009.
700. Perlman JM, Wyllie J, Kattwinkel J, et al. Part 7: Neonatal resuscitation: 2015 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Circulation 2015.
701. Saugstad OD, Aune D, Aguar M, Kapadia V, Finer N, Vento M. Systematic review and meta-analysis of optimal initial fraction of oxygen levels in the delivery room at ≤32 weeks. Acta Paediatr 2014;103:744–51.
702. O'Donnell CP, Kamlin CO, Davis PG, Morley CJ. Feasibility of and delay in obtaining pulse oximetry during neonatal resuscitation. J Pediatr 2005;147:698–9.
703. Dawson JA, Kamlin CO, Wong C, et al. Oxygen saturation and heart rate during delivery room resuscitation of infants <30 weeks' gestation with air or 100% oxygen. Arch Dis Child Fetal Neonatal Ed 2009;94:F87–91.
704. Dildy GA, van den Berg PP, Katz M, et al. Intrapartum fetal pulse oximetry: fetal oxygen saturation trends during labor and relation to delivery outcome. Am J Obstet Gynecol 1994;171:679–84.
705. Dawson JA, Schmolzer GM, Kamlin CO, et al. Oxygenation with T-piece versus self-inflating bag for ventilation of extremely preterm infants at birth: a randomized controlled trial. J Pediatr 2011;158:912–8, e1-2.
706. Szyld E, Aguilar A, Musante GA, et al. Comparison of devices for newborn ventilation in the delivery room. J Pediatr 2014;165, 234–9.e3.
707. Hartung JC, Schmolzer G, Schmalisch G, Roehr CC. Repeated thermosterilisation further affects the reliability of positive end-expiratory pressure valves. J Paediatr Child Health 2013;49:741–5.
708. Schmolzer GM, Agarwal M, Kamlin CO, Davis PG. Supraglottic airway devices during neonatal resuscitation: an historical perspective, systematic review and meta-analysis of available clinical trials. Resuscitation 2013;84:722–30.
709. Trevisanuto D, Cavallin F, Nguyen LN, et al. Supreme laryngeal mask airway versus face mask during neonatal resuscitation: a randomized controlled trial. J Pediatr 2015;167:286–91.
710. Kempley ST, Moreiras JW, Petrone FL. Endotracheal tube length for neonatal intubation. Resuscitation 2008;77:369–73.
711. Gill I, O'Donnell CP. Vocal cord guides on neonatal endotracheal tubes. Arch Dis Child Fetal Neonatal Ed 2014;99:F344.
712. Palme-Kilander C, Tunell R. Pulmonary gas exchange during facemask ventilation immediately after birth. Arch Dis Child 1993;68:11–6.
713. Aziz HF, Martin JB, Moore JJ. The pediatric disposable end-tidal carbon dioxide detector role in endotracheal intubation in newborns. J Perinatol 1999;19:110–3.
714. Bhende MS, LaCovey D. A note of caution about the continuous use of colorimetric end-tidal CO_2 detectors in children. Pediatrics 1995;95:800–1.
715. Repetto JE, Donohue P-CP, Baker SF, Kelly L, Nogee LM. Use of capnography in the delivery room for assessment of endotracheal tube placement. J Perinatol 2001;21:284–7.
716. Roberts WA, Maniscalco WM, Cohen AR, Litman RS, Chhibber A. The use of capnography for recognition of esophageal intubation in the neonatal intensive care unit. Pediatr Pulmonol 1995;19:262–8.
717. Hosono S, Inami I, Fujita H, Minato M, Takahashi S, Mugishima H. A role of end-tidal CO(2) monitoring for assessment of tracheal intubations in very low birth weight infants during neonatal resuscitation at birth. J Perinat Med 2009;37:79–84.
718. Garey DM, Ward R, Rich W, Heldt G, Leone T, Finer NN. Tidal volume threshold for colorimetric carbon dioxide detectors available for use in neonates. Pediatrics 2008;121:e1524–7.
719. Morley CJ, Davis PG, Doyle LW, Brion LP, Hascoet JM, Carlin JB. Nasal CPAP or intubation at birth for very preterm infants. N Engl J Med 2008;358:700–8.
720. SUPPORT Study Group of the Eunice Kennedy Shriver NICHD Neonatal Research Network, Finer NN, Carlo WA, et al. Early CPAP versus surfactant in extremely preterm infants. N Engl J Med 2010;362:1970–9.
721. Dunn MS, Kaempf J, de Klerk A, et al. Randomized trial comparing 3 approaches to the initial respiratory management of preterm neonates. Pediatrics 2011;128:e1069–76.
722. Hishikawa K, Goishi K, Fujiwara T, Kaneshige M, Ito Y, Sago H. Pulmonary air leak associated with CPAP at term birth resuscitation. Arch Dis Child Fetal Neonatal Ed 2015.
723. Poets CF, Rudiger M, Mask CPAP. during neonatal transition: too much of a good thing for some term infants? Arch Dis Child Fetal Neonatal Ed 2015.
724. Houri PK, Frank LR, Menegazzi JJ, Taylor R. A randomized, controlled trial of two-thumb vs two-finger chest compression in a swine infant model of cardiac arrest [see comment]. Prehosp Emerg Care 1997;1:65–7.
725. Dellimore K, Heunis S, Gohier F, et al. Development of a diagnostic glove for unobtrusive measurement of chest compression force and depth during neonatal CPR. Conf Proc IEEE Eng Med Biol Soc 2013;2013:350–3.
726. Martin PS, Kemp AM, Theobald PS, Maguire SA, Jones MD. Do chest compressions during simulated infant CPR comply with international recommendations? Arch Dis Child 2013;98:576–81.
727. Martin P, Theobald P, Kemp A, Maguire S, Maconochie I, Jones M. Real-time feedback can improve infant manikin cardiopulmonary resuscitation by up to 79% – a randomised controlled trial. Resuscitation 2013;84:1125–30.
728. Park J, Yoon C, Lee JC, et al. Manikin-integrated digital measuring system for assessment of infant cardiopulmonary resuscitation techniques. IEEE J Biomed Health Inform 2014;18:1659–67.
729. Saini SS, Gupta N, Kumar P, Bhalla AK, Kaur H. A comparison of two-fingers technique and two-thumbs encircling hands technique of chest compression in neonates. J Perinatol 2012;32:690–4.
730. You Y. Optimum location for chest compressions during two-rescuer infant cardiopulmonary resuscitation. Resuscitation 2009;80:1378–81.
731. Christman C, Hemway RJ, Wyckoff MH, Perlman JM. The two-thumb is superior to the two-finger method for administering chest compressions in a manikin model of neonatal resuscitation. Arch Dis Child Fetal Neonatal Ed 2011;96:F99–101.
732. Meyer A, Nadkarni V, Pollock A, et al. Evaluation of the Neonatal Resuscitation Program's recommended chest compression depth using computerized tomography imaging. Resuscitation 2010;81:544–8.
733. Dannevig I, Solevag AL, Saugstad OD, Nakstad B. Lung injury in asphyxiated newborn pigs resuscitated from cardiac arrest – the impact of supplementary oxygen, longer ventilation intervals and chest compressions at different compression-to-ventilation ratios. Open Respir Med J 2012;6:89–96.

734. Dannevig I, Solevag AL, Sonerud T, Saugstad OD, Nakstad B. Brain inflammation induced by severe asphyxia in newborn pigs and the impact of alternative resuscitation strategies on the newborn central nervous system. Pediatr Res 2013;73:163–70.
735. Hemway RJ, Christman C, Perlman J. The 3:1 is superior to a 15:2 ratio in a newborn manikin model in terms of quality of chest compressions and number of ventilations. Arch Dis Child Fetal Neonatal Ed 2013;98: F42–5.
736. Solevag AL, Dannevig I, Wyckoff M, Saugstad OD, Nakstad B. Extended series of cardiac compressions during CPR in a swine model of perinatal asphyxia. Resuscitation 2010;81:1571–6.
737. Solevag AL, Dannevig I, Wyckoff M, Saugstad OD, Nakstad B. Return of spontaneous circulation with a compression:ventilation ratio of 15:2 versus 3:1 in newborn pigs with cardiac arrest due to asphyxia. Arch Dis Child Fetal Neonatal Ed 2011;96:F417–21.
738. Solevag AL, Madland JM, Gjaerum E, Nakstad B. Minute ventilation at different compression to ventilation ratios, different ventilation rates, and continuous chest compressions with asynchronous ventilation in a newborn manikin. Scand J Trauma Resuscitation Emerg Med 2012;20:73.
739. Berkowitz ID, Chantarojanasiri T, Koehler RC, et al. Blood flow during cardiopulmonary resuscitation with simultaneous compression and ventilation in infant pigs. Pediatr Res 1989;26:558–64.
740. Wyckoff MH, Perlman JM, Laptook AR. Use of volume expansion during delivery room resuscitation in near-term and term infants. Pediatrics 2005;115:950–5.
741. Harrington DJ, Redman CW, Moulden M, Greenwood CE. The long-term outcome in surviving infants with Apgar zero at 10 minutes: a systematic review of the literature and hospital-based cohort. Am J Obstet Gynecol 2007;196, 463.e1–5.
742. Kopelman LM, Irons TG, Kopelman AE. Neonatologists judge the "Baby Doe" regulations. N Engl J Med 1988;318:677–83.
743. Sanders MR, Donohue PK, Oberdorf MA, Rosenkrantz TS, Allen MC. Perceptions of the limit of viability: neonatologists' attitudes toward extremely preterm infants. J Perinatol 1995;15:494–502.
744. Costeloe KL, Hennessy EM, Haider S, Stacey F, Marlow N, Draper ES. Short term outcomes after extreme preterm birth in England: comparison of two birth cohorts in 1995 and 2006 (the EPICure studies). BMJ 2012;345: e7976.
745. Manktelow BN, Seaton SE, Field DJ, Draper ES. Population-based estimates of in-unit survival for very preterm infants. Pediatrics 2013;131:e425–32.
746. Marlow N, Bennett C, Draper ES, Hennessy EM, Morgan AS, Costeloe KL. Perinatal outcomes for extremely preterm babies in relation to place of birth in England: the EPICure 2 study. Arch Dis Child Fetal Neonatal Ed 2014;99:F181–8.
747. Fulbrook P, Latour J, Albarran J, et al. The presence of family members during cardiopulmonary resuscitation: European federation of Critical Care Nursing associations. European Society of Paediatric and Neonatal Intensive Care and European Society of Cardiology Council on Cardiovascular Nursing and Allied Professions Joint Position Statement. Eur J Cardiovasc Nurs 2007;6:255–8.
748. Edwards AD, Brocklehurst P, Gunn AJ, et al. Neurological outcomes at 18 months of age after moderate hypothermia for perinatal hypoxic ischaemic encephalopathy: synthesis and meta-analysis of trial data. BMJ 2010;340:c363.
749. Azzopardi D, Strohm B, Marlow N, et al. Effects of hypothermia for perinatal asphyxia on childhood outcomes. N Engl J Med 2014;371:140–9.
750. Iliodromiti S, Mackay DF, Smith GC, Pell JP, Nelson SM. Apgar score and the risk of cause-specific infant mortality: a population-based cohort study. Lancet 2014;384:1749–55.
751. Rudiger M, Braun N, Aranda J, et al. Neonatal assessment in the delivery room – Trial to Evaluate a Specified Type of Apgar (TEST-Apgar). BMC Pediatr 2015;15:18.
752. Dalili H, Nili F, Sheikh M, Hardani AK, Shariat M, Nayeri F. Comparison of the four proposed Apgar scoring systems in the assessment of birth asphyxia and adverse early neurologic outcomes. PLOS ONE 2015;10:e0122116.
753. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. J Am Coll Cardiol 2012;60:1581–98.
754. Roffi M, Patrono C, Collet JP, et al. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). Eur Heart J 2015, http://dx.doi.org/10.1093/eurheartj/ehv320.
755. Henrikson CA, Howell EE, Bush DE, et al. Chest pain relief by nitroglycerin does not predict active coronary artery disease. Ann Intern Med 2003;139:979–86.
756. American College of Emergency P, Society for Cardiovascular A, Interventions, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;61:e78–140.
757. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2014;130:2354–94.
758. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC Guideline for the Management of Patients with Non-ST-Elevation Acute Coronary Syndromes: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014;64: e139–228.
759. Canto JG, Rogers WJ, Bowlby LJ, French WJ, Pearce DJ, Weaver WD. The prehospital electrocardiogram in acute myocardial infarction: is its full potential being realized? National Registry of Myocardial Infarction 2 Investigators. J Am Coll Cardiol 1997;29:498–505.
760. Terkelsen CJ, Lassen JF, Norgaard BL, et al. Reduction of treatment delay in patients with ST-elevation myocardial infarction: impact of pre-hospital diagnosis and direct referral to primary percutanous coronary intervention. Eur Heart J 2005;26:770–7.
761. Carstensen S, Nelson GC, Hansen PS, et al. Field triage to primary angioplasty combined with emergency department bypass reduces treatment delays and is associated with improved outcome. Eur Heart J 2007;28:2313–9.
762. Brown JP, Mahmud E, Dunford JV, Ben-Yehuda O. Effect of prehospital 12-lead electrocardiogram on activation of the cardiac catheterization laboratory and door-to-balloon time in ST-segment elevation acute myocardial infarction. Am J Cardiol 2008;101:158–61.
763. Martinoni A, De Servi S, Boschetti E, et al. Importance and limits of pre-hospital electrocardiogram in patients with ST elevation myocardial infarction undergoing percutaneous coronary angioplasty. Eur J Cardiovasc Prev Rehabil 2011;18:526–32.
764. Sorensen JT, Terkelsen CJ, Norgaard BL, et al. Urban and rural implementation of pre-hospital diagnosis and direct referral for primary percutaneous coronary intervention in patients with acute ST-elevation myocardial infarction. Eur Heart J 2011;32:430–6.
765. Chan AW, Kornder J, Elliott H, et al. Improved survival associated with prehospital triage strategy in a large regional ST-segment elevation myocardial infarction program. JACC Cardiovasc Interv 2012;5:1239–46.
766. Quinn T, Johnsen S, Gale CP, et al. Effects of prehospital 12-lead ECG on processes of care and mortality in acute coronary syndrome: a linked cohort study from the Myocardial Ischaemia National Audit Project. Heart 2014;100:944–50.
767. Ong ME, Wong AS, Seet CM, et al. Nationwide improvement of door-to-balloon times in patients with acute ST-segment elevation myocardial infarction requiring primary percutaneous coronary intervention with out-of-hospital 12-lead ECG recording and transmission. Ann Emerg Med 2013;61:339–47.
768. Swor R, Hegerberg S, McHugh-McNally A, Goldstein M, McEachin CC. Prehospital 12-lead ECG: efficacy or effectiveness? Prehosp Emerg Care 2006;10:374–7.
769. Masoudi FA, Magid DJ, Vinson DR, et al. Implications of the failure to identify high-risk electrocardiogram findings for the quality of care of patients with acute myocardial infarction: results of the Emergency Department Quality in Myocardial Infarction (EDQMI) study. Circulation 2006;114: 1565–71.
770. Kudenchuk PJ, Ho MT, Weaver WD, et al. Accuracy of computer-interpreted electrocardiography in selecting patients for thrombolytic therapy. MITI Project Investigators. J Am Coll Cardiol 1991;17:1486–91.
771. Dhruva VN, Abdelhadi SI, Anis A, et al. ST-Segment Analysis Using Wireless Technology in Acute Myocardial Infarction (STAT-MI) trial. J Am Coll Cardiol 2007;50:509–13.
772. Bhalla MC, Mencl F, Gist MA, Wilber S, Zalewski J. Prehospital electrocardiographic computer identification of ST-segment elevation myocardial infarction. Prehosp Emerg Care 2013;17:211–6.
773. Clark EN, Sejersten M, Clemmensen P, Macfarlane PW. Automated electrocardiogram interpretation programs versus cardiologists' triage decision making based on teletransmitted data in patients with suspected acute coronary syndrome. Am J Cardiol 2010;106:1696–702.
774. de Champlain F, Boothroyd LJ, Vadeboncoeur A, et al. Computerized interpretation of the prehospital electrocardiogram: predictive value for ST segment elevation myocardial infarction and impact on on-scene time. CJEM 2014;16:94–105.
775. Squire BT, Tamayo-Sarver JH, Rashi P, Koenig W, Niemann JT. Effect of prehospital cardiac catheterization lab activation on door-to-balloon time, mortality, and false-positive activation. Prehosp Emerg Care 2014;18: 1–8.
776. Youngquist ST, Shah AP, Niemann JT, Kaji AH, French WJ. A comparison of door-to-balloon times and false-positive activations between emergency department and out-of-hospital activation of the coronary catheterization team. Acad Emerg Med 2008;15:784–7.
777. van't Hof AW, Rasoul S, van de Wetering H, et al. Feasibility and benefit of prehospital diagnosis, triage, and therapy by paramedics only in patients who are candidates for primary angioplasty for acute myocardial infarction. Am Heart J 2006;151, 1255.e1–5.
778. Keller T, Zeller T, Peetz D, et al. Sensitive troponin I assay in early diagnosis of acute myocardial infarction. N Engl J Med 2009;361:868–77.
779. Goldstein JA, Gallagher MJ, O'Neill WW, Ross MA, O'Neil BJ, Raff GL. A randomized controlled trial of multi-slice coronary computed tomography for evaluation of acute chest pain. J Am Coll Cardiol 2007;49:863–71.
780. Forberg JL, Hilmersson CE, Carlsson M, et al. Negative predictive value and potential cost savings of acute nuclear myocardial perfusion imaging in low risk patients with suspected acute coronary syndrome: a prospective single blinded study. BMC Emerg Med 2009;9:12.
781. Nucifora G, Badano LP, Sarraf-Zadegan N, et al. Comparison of early dobutamine stress echocardiography and exercise electrocardiographic testing for

management of patients presenting to the emergency department with chest pain. Am J Cardiol 2007;100:1068–73.
782. Wei K. Utility contrast echocardiography in the emergency department. JACC Cardiovasc Imaging 2010;3:197–203.
783. Gaibazzi N, Squeri A, Reverberi C, et al. Contrast stress-echocardiography predicts cardiac events in patients with suspected acute coronary syndrome but nondiagnostic electrocardiogram and normal 12-hour troponin. J Am Soc Echocardiogr 2011;24:1333–41.
784. Douglas PS, Khandheria B, Stainback RF, et al. ACCF/ASE/ACEP/ASNC/SCAI/SCCT/SCMR 2007 appropriateness criteria for transthoracic and transesophageal echocardiography: a report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American Society of Echocardiography, American College of Emergency Physicians, American Society of Nuclear Cardiology, Society for Cardiovascular Angiography and Interventions, Society of Cardiovascular Computed Tomography, and the Society for Cardiovascular Magnetic Resonance endorsed by the American College of Chest Physicians and the Society of Critical Care Medicine. J Am Coll Cardiol 2007;50:187–204.
785. Hamm CW, Bassand JP, Agewall S, et al. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2011;32:2999–3054.
786. Samad Z, Hakeem A, Mahmood SS, et al. A meta-analysis and systematic review of computed tomography angiography as a diagnostic triage tool for patients with chest pain presenting to the emergency department. J Nucl Cardiol 2012;19:364–76.
787. Kearney PM, Baigent C, Godwin J, Halls H, Emberson JR, Patrono C. Do selective cyclo-oxygenase-2 inhibitors and traditional non-steroidal anti-inflammatory drugs increase the risk of atherothrombosis? Meta-analysis of randomised trials. BMJ 2006;332:1302–8.
788. Rawles JM, Kenmure AC. Controlled trial of oxygen in uncomplicated myocardial infarction. Br Med J 1976;1:1121–3.
789. Wijesinghe M, Perrin K, Ranchord A, Simmonds M, Weatherall M, Beasley R. Routine use of oxygen in the treatment of myocardial infarction: systematic review. Heart 2009;95:198–202.
790. Cabello JB, Burls A, Emparanza JI, Bayliss S, Quinn T. Oxygen therapy for acute myocardial infarction. Cochrane Database Syst Rev 2013;8:CD007160.
791. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 2013;127:e362–425.
792. Mega JL, Braunwald E, Wiviott SD, et al. Rivaroxaban in patients with a recent acute coronary syndrome. N Engl J Med 2012;366:9–19.
793. Keeley EC, Boura JA, Grines CL. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet 2003;361:13–20.
794. Pinto DS, Kirtane AJ, Nallamothu BK, et al. Hospital delays in reperfusion for ST-elevation myocardial infarction: implications when selecting a reperfusion strategy. Circulation 2006;114:2019–25.
795. Le May MR, So DY, Dionne R, et al. A citywide protocol for primary PCI in ST-segment elevation myocardial infarction. N Engl J Med 2008;358:231–40.
796. Bradley EH, Herrin J, Wang Y, et al. Strategies for reducing the door-to-balloon time in acute myocardial infarction. N Engl J Med 2006;355:2308–20.
797. Nikolaou N, Welsford M, Beygui F, et al. Part 5: Acute coronary syndromes: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e123–48.
798. Bonnefoy E, Lapostolle F, Leizorovicz A, et al. Primary angioplasty versus prehospital fibrinolysis in acute myocardial infarction: a randomised study. Lancet 2002;360:825–9.
799. Armstrong PW. A comparison of pharmacologic therapy with/without timely coronary intervention vs. primary percutaneous intervention early after ST-elevation myocardial infarction: the WEST (Which Early ST-elevation myocardial infarction Therapy) study. Eur Heart J 2006;27:1530–8.
800. Thiele H, Eitel I, Meinberg C, et al. Randomized comparison of pre-hospital-initiated facilitated percutaneous coronary intervention versus primary percutaneous coronary intervention in acute myocardial infarction very early after symptom onset: the LIPSIA-STEMI trial (Leipzig immediate prehospital facilitated angioplasty in ST-segment myocardial infarction). JACC Cardiovasc Interv 2011;4:605–14.
801. Armstrong PW, Gershlick AH, Goldstein P, et al. Fibrinolysis or primary PCI in ST-segment elevation myocardial infarction. N Engl J Med 2013;368:1379–87.
802. Van de Werf F, Barron HV, Armstrong PW, et al. Incidence and predictors of bleeding events after fibrinolytic therapy with fibrin-specific agents: a comparison of TNK-tPA and rt-PA. Eur Heart J 2001;22:2253–61.
803. Ellis SG, Tendera M, de Belder MA, et al. Facilitated PCI in patients with ST-elevation myocardial infarction. N Engl J Med 2008;358:2205–17.
804. Itoh T, Fukami K, Suzuki T, et al. Comparison of long-term prognostic evaluation between pre-intervention thrombolysis and primary coronary intervention: a prospective randomized trial: five-year results of the IMPORTANT study. Circ J 2010;74:1625–34.
805. Kurihara H, Matsumoto S, Tamura R, et al. Clinical outcome of percutaneous coronary intervention with antecedent mutant t-PA administration for acute myocardial infarction. Am Heart J 2004;147:E14.
806. Thiele H, Scholz M, Engelmann L, et al. ST-segment recovery and prognosis in patients with ST-elevation myocardial infarction reperfused by prehospital combination fibrinolysis, prehospital initiated facilitated percutaneous coronary intervention, or primary percutaneous coronary intervention. Am J Cardiol 2006;98:1132–9.
807. Gershlick AH, Stephens-Lloyd A, Hughes S, et al. Rescue angioplasty after failed thrombolytic therapy for acute myocardial infarction. N Engl J Med 2005;353:2758–68.
808. Thiele H, Zeymer U, Neumann FJ, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. N Engl J Med 2012;367:1287–96.
809. Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization and long-term survival in cardiogenic shock complicating acute myocardial infarction. JAMA 2006;295:2511–5.
810. Rab T, Kern KB, Tamis-Holland JE, et al. Cardiac arrest: a treatment algorithm for emergent invasive cardiac procedures in the resuscitated comatose patient. J Am Coll Cardiol 2015;66:62–73.
811. Zideman D, Singletary EM, De Buck E, et al. Part 9: First aid: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e229–65.
812. Adnet F, Borron SW, Finot MA, Minadeo J, Baud FJ. Relation of body position at the time of discovery with suspected aspiration pneumonia in poisoned comatose patients. Crit Care Med 1999;27:745–8.
813. Rathgeber J, Panzer W, Gunther U, et al. Influence of different types of recovery positions on perfusion indices of the forearm. Resuscitation 1996;32:13–7.
814. Del Rossi G, Dubose D, Scott N, et al. Motion produced in the unstable cervical spine by the HAINES and lateral recovery positions. Prehosp Emerg Care 2014;18:539–43.
815. Wong DH, O'Connor D, Tremper KK, Zaccari J, Thompson P, Hill D. Changes in cardiac output after acute blood loss and position change in man. Crit Care Med 1989;17:979–83.
816. Jabot J, Teboul JL, Richard C, Monnet X. Passive leg raising for predicting fluid responsiveness: importance of the postural change. Intensive Care Med 2009;35:85–90.
817. Gaffney FA, Bastian BC, Thal ER, Atkins JM, Blomqvist CG. Passive leg raising does not produce a significant or sustained autotransfusion effect. J Trauma 1982;22:190–3.
818. Bruera E, de Stoutz N, Velasco-Leiva A, Schoeller T, Hanson J. Effects of oxygen on dyspnoea in hypoxaemic terminal-cancer patients. Lancet 1993;342:13–4.
819. Philip J, Gold M, Milner A, Di Iulio J, Miller B, Spruyt O. A randomized, double-blind, crossover trial of the effect of oxygen on dyspnea in patients with advanced cancer. J Pain Symptom Manage 2006;32:541–50.
820. Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiberger JJ. First aid normobaric oxygen for the treatment of recreational diving injuries. Undersea Hyperb Med 2007;34:43–9.
821. Wijesinghe M, Perrin K, Healy B, et al. Pre-hospital oxygen therapy in acute exacerbations of chronic obstructive pulmonary disease. Intern Med J 2011;41:618–22.
822. Bentur L, Canny GJ, Shields MD, et al. Controlled trial of nebulized albuterol in children younger than 2 years of age with acute asthma. Pediatrics 1992;89:133–7.
823. van der Woude HJ, Postma DS, Politiek MJ, Winter TH, Aalbers R. Relief of dyspnoea by beta2-agonists after methacholine-induced bronchoconstriction. Respir Med 2004;98:816–20.
824. Lavorini F. The challenge of delivering therapeutic aerosols to asthma patients. ISRN Allergy 2013;2013:102418.
825. Lavorini F. Inhaled drug delivery in the hands of the patient. J Aerosol Med Pulm Drug Deliv 2014;27:414–8.
826. Conner JB, Buck PO. Improving asthma management: the case for mandatory inclusion of dose counters on all rescue bronchodilators. J Asthma 2013;50:658–63.
827. Cheung RT. Hong Kong patients' knowledge of stroke does not influence time-to-hospital presentation. J Clin Neurosci 2001;8:311–4.
828. Fonarow GC, Smith EE, Saver JL, et al. Improving door-to-needle times in acute ischemic stroke: the design and rationale for the American Heart Association/American Stroke Association's Target: stroke initiative. Stroke 2011;42:2983–9.
829. Lin CB, Peterson ED, Smith EE, et al. Emergency medical service hospital prenotification is associated with improved evaluation and treatment of acute ischemic stroke. Circ Cardiovasc Qual Outcomes 2012;5:514–22.
830. Nazliel B, Starkman S, Liebeskind DS, et al. A brief prehospital stroke severity scale identifies ischemic stroke patients harboring persisting large arterial occlusions. Stroke 2008;39:2264–7.
831. Wojner-Alexandrov AW, Alexandrov AV, Rodriguez D, Persse D, Grotta JC. Houston paramedic and emergency stroke treatment and outcomes study (HoPSTO). Stroke 2005;36:1512–8.
832. You JS, Chung SP, Chung HS, et al. Predictive value of the Cincinnati Prehospital Stroke Scale for identifying thrombolytic candidates in acute ischemic stroke. Am J Emerg Med 2013;31:1699–702.
833. O'Brien W, Crimmins D, Donaldson W, et al. FASTER (Face, Arm, Speech, Time, Emergency Response): experience of Central Coast Stroke Services implementation of a pre-hospital notification system for expedient management of acute stroke. J Clin Neurosci 2012;19:241–5.

834. Barbash IM, Freimark D, Gottlieb S, et al. Outcome of myocardial infarction in patients treated with aspirin is enhanced by pre-hospital administration. Cardiology 2002;98:141–7.
835. Freimark D, Matetzky S, Leor J, et al. Timing of aspirin administration as a determinant of survival of patients with acute myocardial infarction treated with thrombolysis. Am J Cardiol 2002;89:381–5.
836. Quan D, LoVecchio F, Clark B, Gallagher III JV. Prehospital use of aspirin rarely is associated with adverse events. Prehosp Disaster Med 2004;19:362–5.
837. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Lancet 1988;2:349–60.
838. Verheugt FW, van der Laarse A, Funke-Kupper AJ, Sterkman LG, Galema TW, Roos JP. Effects of early intervention with low-dose aspirin (100 mg) on infarct size, reinfarction and mortality in anterior wall acute myocardial infarction. Am J Cardiol 1990;66:267–70.
839. Elwood PC, Williams WO. A randomized controlled trial of aspirin in the prevention of early mortality in myocardial infarction. J R Coll Gen Pract 1979;29:413–6.
840. Frilling B, Schiele R, Gitt AK, et al. Characterization and clinical course of patients not receiving aspirin for acute myocardial infarction: results from the MITRA and MIR studies. Am Heart J 2001;141:200–5.
841. Simons FE, Ardusso LR, Bilo MB, et al. World allergy organization guidelines for the assessment and management of anaphylaxis. World Allergy Organ J 2011;4:13–37.
842. Chong LK, Morice AH, Yeo WW, Schleimer RP, Peachell PT. Functional desensitization of beta agonist responses in human lung mast cells. Am J Respir Cell Mol Biol 1995;13:540–6.
843. Korenblat P, Lundie MJ, Dankner RE, Day JH. A retrospective study of epinephrine administration for anaphylaxis: how many doses are needed? Allergy Asthma Proc 1999;20:383–6.
844. Rudders SA, Banerji A, Corel B, Clark S, Camargo Jr CA. Multicenter study of repeat epinephrine treatments for food-related anaphylaxis. Pediatrics 2010;125:e711–8.
845. Rudders SA, Banerji A, Katzman DP, Clark S, Camargo Jr CA. Multiple epinephrine doses for stinging insect hypersensitivity reactions treated in the emergency department. Ann Allergy Asthma Immunol 2010;105:85–93.
846. Inoue N, Yamamoto A. Clinical evaluation of pediatric anaphylaxis and the necessity for multiple doses of epinephrine. Asia Pac Allergy 2013;3:106–14.
847. Ellis BC, Brown SG. Efficacy of intramuscular epinephrine for the treatment of severe anaphylaxis: a comparison of two ambulance services with different protocols. Ann Emerg Med 2013;62:S146.
848. Oren E, Banerji A, Clark S, Camargo Jr CA. Food-induced anaphylaxis and repeated epinephrine treatments. Ann Allergy Asthma Immunol 2007;99:429–32.
849. Tsuang A, Menon N, Setia N, Geyman L, Nowak-Wegrzyn AH. Multiple epinephrine doses in food-induced anaphylaxis in children. J Allergy Clin Immunol 2013;131:AB90.
850. Banerji A, Rudders SA, Corel B, Garth AM, Clark S, Camargo Jr CA. Repeat epinephrine treatments for food-related allergic reactions that present to the emergency department. Allergy Asthma Proc 2010;31:308–16.
851. Noimark L, Wales J, Du Toit G, et al. The use of adrenaline autoinjectors by children and teenagers. Clin Exp Allergy 2012;42:284–92.
852. Jarvinen KM, Sicherer SH, Sampson HA, Nowak-Wegrzyn A. Use of multiple doses of epinephrine in food-induced anaphylaxis in children. J Allergy Clin Immunol 2008;122:133–8.
853. Slama G, Traynard PY, Desplanque N, et al. The search for an optimized treatment of hypoglycemia. Carbohydrates in tablets, solutin, or gel for the correction of insulin reactions. Arch Intern Med 1990;150:589–93.
854. Husband AC, Crawford S, McCoy LA, Pacaud D. The effectiveness of glucose, sucrose, and fructose in treating hypoglycemia in children with type 1 diabetes. Pediatr Diabetes 2010;11:154–8.
855. McTavish L, Wiltshire E. Effective treatment of hypoglycemia in children with type 1 diabetes: a randomized controlled clinical trial. Pediatr Diabetes 2011;12:381–7.
856. Osterberg KL, Pallardy SE, Johnson RJ, Horswill CA. Carbohydrate exerts a mild influence on fluid retention following exercise-induced dehydration. J Appl Physiol 2010;108:245–50.
857. Kalman DS, Feldman S, Krieger DR, Bloomer RJ. Comparison of coconut water and a carbohydrate-electrolyte sport drink on measures of hydration and physical performance in exercise-trained men. J Int Soc Sports Nutr 2012;9:1.
858. Chang CQ, Chen YB, Chen ZM, Zhang LT. Effects of a carbohydrate-electrolyte beverage on blood viscosity after dehydration in healthy adults. Chin Med J 2010;123:3220–5.
859. Seifert J, Harmon J, DeClercq P. Protein added to a sports drink improves fluid retention. Int J Sport Nutr Exerc Metab 2006;16:420–9.
860. Wong SH, Chen Y. Effect of a carbohydrate-electrolyte beverage, lemon tea, or water on rehydration during short-term recovery from exercise. Int J Sport Nutr Exerc Metab 2011;21:300–10.
861. Shirreffs SM, Watson P, Maughan RJ. Milk as an effective post-exercise rehydration drink. Br J Nutr 2007;98:173–80.
862. Gonzalez-Alonso J, Heaps CL, Coyle EF. Rehydration after exercise with common beverages and water. Int J Sports Med 1992;13:399–406.
863. Ismail I, Singh R, Sirisinghe RG. Rehydration with sodium-enriched coconut water after exercise-induced dehydration. Southeast Asian J Trop Med Public Health 2007;38:769–85.
864. Saat M, Singh R, Sirisinghe RG, Nawawi M. Rehydration after exercise with fresh young coconut water, carbohydrate-electrolyte beverage and plain water. J Physiol Anthropol Appl Hum Sci 2002;21:93–104.
865. Miccheli A, Marini F, Capuani G, et al. The influence of a sports drink on the postexercise metabolism of elite athletes as investigated by NMR-based metabolomics. J Am Coll Nutr 2009;28:553–64.
866. Kompa S, Redbrake C, Hilgers C, Wustemeyer H, Schrage N, Remky A. Effect of different irrigating solutions on aqueous humour pH changes, intraocular pressure and histological findings after induced alkali burns. Acta Ophthalmol Scand 2005;83:467–70.
867. King NA, Philpott SJ, Leary A. A randomized controlled trial assessing the use of compression versus vasoconstriction in the treatment of femoral hematoma occurring after percutaneous coronary intervention. Heart Lung 2008;37:205–10.
868. Levy AS, Marmar E. The role of cold compression dressings in the postoperative treatment of total knee arthroplasty. Clin Orthop Rel Res 1993:174–8.
869. Kheirabadi BS, Edens JW, Terrazas IB, et al. Comparison of new hemostatic granules/powders with currently deployed hemostatic products in a lethal model of extremity arterial hemorrhage in swine. J Trauma 2009;66:316–26, discussion 27-8.
870. Ward KR, Tiba MH, Holbert WH, et al. Comparison of a new hemostatic agent to current combat hemostatic agents in a Swine model of lethal extremity arterial hemorrhage. J Trauma 2007;63:276–83, discussion 83-4.
871. Carraway JW, Kent D, Young K, Cole A, Friedman R, Ward KR. Comparison of a new mineral based hemostatic agent to a commercially available granular zeolite agent for hemostasis in a swine model of lethal extremity arterial hemorrhage. Resuscitation 2008;78:230–5.
872. Arnaud F, Parreno-Sadalan D, Tomori T, et al. Comparison of 10 hemostatic dressings in a groin transection model in swine. J Trauma 2009;67:848–55.
873. Kheirabadi BS, Acheson EM, Deguzman R, et al. Hemostatic efficacy of two advanced dressings in an aortic hemorrhage model in Swine. J Trauma 2005;59:25–34, discussion 34-5.
874. Brown MA, Daya MR, Worley JA. Experience with chitosan dressings in a civilian EMS system. J Emerg Med 2009;37:1–7.
875. Cox ED, Schreiber MA, McManus J, Wade CE, Holcomb JB. New hemostatic agents in the combat setting. Transfusion 2009;49(Suppl. 5):248S–55S.
876. Ran Y, Hadad E, Daher S, et al. QuikClot Combat Gauze use for hemorrhage control in military trauma: January 2009 Israel Defense Force experience in the Gaza Strip – a preliminary report of 14 cases. Prehosp Disaster Med 2010;25:584–8.
877. Wedmore I, McManus JG, Pusateri AE, Holcomb JB. A special report on the chitosan-based hemostatic dressing: experience in current combat operations. J Trauma 2006;60:655–8.
878. Engels PT, Rezende-Neto JB, Al Mahroos M, Scarpelini S, Rizoli SB, Tien HC. The natural history of trauma-related coagulopathy: implications for treatment. J Trauma 2011;71:S448–55.
879. Sauaia A, Moore FA, Moore EE, et al. Epidemiology of trauma deaths: a reassessment. J Trauma 1995;38:185–93.
880. Beekley AC, Sebesta JA, Blackbourne LH, et al. Prehospital tourniquet use in Operation Iraqi Freedom: effect on hemorrhage control and outcomes. J Trauma 2008;64:S28–37, discussion S37.
881. Lakstein D, Blumenfeld A, Sokolov T, et al. Tourniquets for hemorrhage control on the battlefield: a 4-year accumulated experience. J Trauma 2003;54:S221–5.
882. Passos E, Dingley B, Smith A, et al. Tourniquet use for peripheral vascular injuries in the civilian setting. Injury 2014;45:573–7.
883. King DR, van der Wilden G, Kragh Jr JF, Blackbourne LH. Forward assessment of 79 prehospital battlefield tourniquets used in the current war. J Spec Oper Med 2012;12:33–8.
884. Kragh Jr JF, Littrel ML, Jones JA, et al. Battle casualty survival with emergency tourniquet use to stop limb bleeding. J Emerg Med 2011;41:590–7.
885. Kragh Jr JF, Cooper A, Aden JK, et al. Survey of trauma registry data on tourniquet use in pediatric war casualties. Pediatr Emerg Care 2012;28:1361–5.
886. Tien HC, Jung V, Rizoli SB, Acharya SV, MacDonald JC. An evaluation of tactical combat casualty care interventions in a combat environment. J Am Coll Surg 2008;207:174–8.
887. Kragh Jr JF, Nam JJ, Berry KA, et al. Transfusion for shock in US military war casualties with and without tourniquet use. Ann Emerg Med 2015;65:290–6.
888. Brodie S, Hodgetts TJ, Ollerton J, McLeod J, Lambert P, Mahoney P. Tourniquet use in combat trauma: UK military experience. J R Army Med Corps 2007;153:310–3.
889. Kue RC, Temin ES, Weiner SG, et al. Tourniquet use in a civilian emergency medical services setting: a descriptive analysis of the Boston EMS experience. Prehosp Emerg Care 2015;19:399–404.
890. Ayling J. An open question. Emerg Med Serv 2004;33:44.
891. Sundstrom T, Asbjornsen H, Habiba S, Sunde GA, Wester K. Prehospital use of cervical collars in trauma patients: a critical review. J Neurotrauma 2014;31:531–40.
892. Kwan I, Bunn F, Roberts I. Spinal immobilisation for trauma patients. Cochrane Database Syst Rev 2001:CD002803.
893. Davies G, Deakin C, Wilson A. The effect of a rigid collar on intracranial pressure. Injury 1996;27:647–9.

894. Hunt K, Hallworth S, Smith M. The effects of rigid collar placement on intracranial and cerebral perfusion pressures. Anaesthesia 2001;56:511–3.
895. Mobbs RJ, Stoodley MA, Fuller J. Effect of cervical hard collar on intracranial pressure after head injury. ANZ J Surg 2002;72:389–91.
896. Kolb JC, Summers RL, Galli RL. Cervical collar-induced changes in intracranial pressure. Am J Emerg Med 1999;17:135–7.
897. Raphael JH, Chotai R. Effects of the cervical collar on cerebrospinal fluid pressure. Anaesthesia 1994;49:437–9.
898. McCrory P, Meeuwisse W, Johnston K, et al. Consensus Statement on Concussion in Sport: the 3rd International Conference on Concussion in Sport held in Zurich, November 2008. Br J Sports Med 2009;43(Suppl. 1):i76–90.
899. Nguyen NL, Gun RT, Sparnon AL, Ryan P. The importance of immediate cooling – a case series of childhood burns in Vietnam. Burns 2002;28:173–6.
900. Yava A, Koyuncu A, Tosun N, Kilic S. Effectiveness of local cold application on skin burns and pain after transthoracic cardioversion. Emerg Med J: EMJ 2012;29:544–9.
901. Skinner AM, Brown TLH, Peat BG, Muller MJ. Reduced Hospitalisation of burns patients following a multi-media campaign that increased adequacy of first aid treatment. Burns 2004;30:82–5.
902. Wasiak J, Cleland H, Campbell F, Spinks A. Dressings for superficial and partial thickness burns. Cochrane Database Syst Rev 2013;3:CD002106.
903. Murad MK, Husum H. Trained lay first responders reduce trauma mortality: a controlled study of rural trauma in Iraq. Prehosp Disaster Med 2010;25:533–9.
904. Wall HK, Beagan BM, O'Neill J, Foell KM, Boddie-Willis CL. Addressing stroke signs and symptoms through public education: the Stroke Heroes Act FAST campaign. Prev Chronic Dis 2008;5:A49.
905. Chamberlain DA, Hazinski MF. Education in resuscitation. Resuscitation 2003;59:11–43.
906. Kudenchuk PJ, Redshaw JD, Stubbs BA, et al. Impact of changes in resuscitation practice on survival and neurological outcome after out-of-hospital cardiac arrest resulting from nonshockable arrhythmias. Circulation 2012;125:1787–94.
907. Steinberg MT, Olsen JA, Brunborg C, et al. Minimizing pre-shock chest compression pauses in a cardiopulmonary resuscitation cycle by performing an earlier rhythm analysis. Resuscitation 2015;87:33–7.
908. Swor R, Khan I, Domeier R, Honeycutt L, Chu K, Compton S. CPR training and CPR performance: do CPR-trained bystanders perform CPR? Acad Emerg Med 2006;13:596–601.
909. Tanigawa K, Iwami T, Nishiyama C, Nonogi H, Kawamura T. Are trained individuals more likely to perform bystander CPR? An observational study. Resuscitation 2011;82:523–8.
910. Nielsen AM, Isbye DL, Lippert FK, Rasmussen LS. Can mass education and a television campaign change the attitudes towards cardiopulmonary resuscitation in a rural community? Scand J Trauma Resuscitation Emerg Med 2013;21:39.
911. Sasson C, Haukoos JS, Bond C, et al. Barriers and facilitators to learning and performing cardiopulmonary resuscitation in neighborhoods with low bystander cardiopulmonary resuscitation prevalence and high rates of cardiac arrest in Columbus, OH. Circ Cardiovasc Qual Outcomes 2013;6:550–8.
912. King R, Heisler M, Sayre MR, et al. Identification of factors integral to designing community-based CPR interventions for high-risk neighborhood residents. Prehosp Emerg Care 2015;19:308–12.
913. Greenberg MR, Barr Jr GC, Rupp VA, et al. Cardiopulmonary resuscitation prescription program: a pilot randomized comparator trial. J Emerg Med 2012;43:166–71.
914. Blewer AL, Leary M, Esposito EC, et al. Continuous chest compression cardiopulmonary resuscitation training promotes rescuer self-confidence and increased secondary training: a hospital-based randomized controlled trial. Crit Care Med 2012;40:787–92.
915. Brannon TS, White LA, Kilcrease JN, Richard LD, Spillers JG, Phelps CL. Use of instructional video to prepare parents for learning infant cardiopulmonary resuscitation. Proc (Bayl Univ Med Cent) 2009;22:133–7.
916. Haugk M, Robak O, Sterz F, et al. High acceptance of a home AED programme by survivors of sudden cardiac arrest and their families. Resuscitation 2006;70:263–74.
917. Knight LJ, Wintch S, Nichols A, Arnolde V, Schroeder AR. Saving a life after discharge: CPR training for parents of high-risk children. J Healthc Qual 2013;35:9–16, quiz 7.
918. Barr Jr GC, Rupp VA, Hamilton KM, et al. Training mothers in infant cardiopulmonary resuscitation with an instructional DVD and manikin. J Am Osteopath Assoc 2013;113:538–45.
919. Plant N, Taylor K. How best to teach CPR to schoolchildren: a systematic review. Resuscitation 2013;84:415–21.
920. Bohn A, Van Aken HK, Mollhoff T, et al. Teaching resuscitation in schools: annual tuition by trained teachers is effective starting at age 10. A four-year prospective cohort study. Resuscitation 2012;83:619–25.
921. Song KJ, Shin SD, Park CB, et al. Dispatcher-assisted bystander cardiopulmonary resuscitation in a metropolitan city: a before-after population-based study. Resuscitation 2014;85:34–41.
922. Mancini ME, Cazzell M, Kardong-Edgren S, Cason CL. Improving workplace safety training using a self-directed CPR-AED learning program. AAOHN J 2009;57:159–67, quiz 68-9.
923. Cason CL, Kardong-Edgren S, Cazzell M, Behan D, Mancini ME. Innovations in basic life support education for healthcare providers: improving competence in cardiopulmonary resuscitation through self-directed learning. J Nurses Staff Dev 2009;25:E1–13.
924. Einspruch EL, Lynch B, Aufderheide TP, Nichol G, Becker L. Retention of CPR skills learned in a traditional AHA Heartsaver course versus 30-min video self-training: a controlled randomized study. Resuscitation 2007;74:476–86.
925. Lynch B, Einspruch EL, Nichol G, Becker LB, Aufderheide TP, Idris A. Effectiveness of a 30-min CPR self-instruction program for lay responders: a controlled randomized study. Resuscitation 2005;67:31–43.
926. Chung CH, Siu AY, Po LL, Lam CY, Wong PC. Comparing the effectiveness of video self-instruction versus traditional classroom instruction targeted at cardiopulmonary resuscitation skills for laypersons: a prospective randomised controlled trial. Xianggang yi xue za zhi/Hong Kong Acad Med 2010;16:165–70.
927. Roppolo LP, Pepe PE, Campbell L, et al. Prospective, randomized trial of the effectiveness and retention of 30-min layperson training for cardiopulmonary resuscitation and automated external defibrillators: The American Airlines Study. Resuscitation 2007;74:276–85.
928. Smith KK, Gilcreast D, Pierce K. Evaluation of staff's retention of ACLS and BLS skills. Resuscitation 2008;78:59–65.
929. Woollard M, Whitfeild R, Smith A, et al. Skill acquisition and retention in automated external defibrillator (AED) use and CPR by lay responders: a prospective study. Resuscitation 2004;60:17–28.
930. Woollard M, Whitfield R, Newcombe RG, Colquhoun M, Vetter N, Chamberlain D. Optimal refresher training intervals for AED and CPR skills: a randomised controlled trial. Resuscitation 2006;71:237–47.
931. Andresen D, Arntz HR, Grafling W, et al. Public access resuscitation program including defibrillator training for laypersons: a randomized trial to evaluate the impact of training course duration. Resuscitation 2008;76:419–24.
932. Beckers SK, Fries M, Bickenbach J, et al. Retention of skills in medical students following minimal theoretical instructions on semi and fully automated external defibrillators. Resuscitation 2007;72:444–50.
933. Kirkbright S, Finn J, Tohira H, Bremner A, Jacobs I, Celenza A. Audiovisual feedback device use by health care professionals during CPR: a systematic review and meta-analysis of randomised and non-randomised trials. Resuscitation 2014;85:460–71.
934. Mundell WC, Kennedy CC, Szostek JH, Cook DA. Simulation technology for resuscitation training: a systematic review and meta-analysis. Resuscitation 2013;84:1174–83.
935. Andreatta P, Saxton E, Thompson M, Annich G. Simulation-based mock codes significantly correlate with improved pediatric patient cardiopulmonary arrest survival rates. Pediatr Crit Care Med 2011;12:33–8.
936. Neily J, Mills PD, Young-Xu Y, et al. Association between implementation of a medical team training program and surgical mortality. JAMA 2010;304:1693–700.
937. Thomas EJ, Taggart B, Crandell S, et al. Teaching teamwork during the Neonatal Resuscitation Program: a randomized trial. J Perinatol 2007;27:409–14.
938. Gilfoyle E, Gottesman R, Razack S. Development of a leadership skills workshop in paediatric advanced resuscitation. Med Teacher 2007;29:e276–83.
939. Edelson DP, Litzinger B, Arora V, et al. Improving in-hospital cardiac arrest process and outcomes with performance debriefing. Arch Intern Med 2008;168:1063–9.
940. Hayes CW, Rhee A, Detsky ME, Leblanc VR, Wax RS. Residents feel unprepared and unsupervised as leaders of cardiac arrest teams in teaching hospitals: a survey of internal medicine residents. Crit Care Med 2007;35:1668–72.
941. Marsch SC, Muller C, Marquardt K, Conrad G, Tschan F, Hunziker PR. Human factors affect the quality of cardiopulmonary resuscitation in simulated cardiac arrests. Resuscitation 2004;60:51–6.
942. Raemer D, Anderson M, Cheng A, Fanning R, Nadkarni V, Savoldelli G. Research regarding debriefing as part of the learning process. Simul Healthc 2011;6(Suppl.):S52–7.
943. Byrne AJ, Sellen AJ, Jones JG, et al. Effect of videotape feedback on anaesthetists' performance while managing simulated anaesthetic crises: a multicentre study. Anaesthesia 2002;57:176–9.
944. Savoldelli GL, Naik VN, Park J, Joo HS, Chow R, Hamstra SJ. Value of debriefing during simulated crisis management: oral versus video-assisted oral feedback. Anesthesiology 2006;105:279–85.
945. Kurosawa H, Ikeyama T, Achuff P, et al. A randomized, controlled trial of in situ pediatric advanced life support recertification ("pediatric advanced life support reconstructed") compared with standard pediatric advanced life support recertification for ICU frontline providers. Crit Care Med 2014;42:610–8.
946. Patocka C, Khan F, Dubrovsky AS, Brody D, Bank I, Bhanji F. Pediatric resuscitation training-instruction all at once or spaced over time? Resuscitation 2015;88:6–11.
947. Stross JK. Maintaining competency in advanced cardiac life support skills. JAMA 1983;249:3339–41.
948. Jensen ML, Mondrup F, Lippert F, Ringsted C. Using e-learning for maintenance of ALS competence. Resuscitation 2009;80:903–8.
949. Kaczorowski J, Levitt C, Hammond M, et al. Retention of neonatal resuscitation skills and knowledge: a randomized controlled trial. Fam Med 1998;30:705–11.
950. Rea TD, Helbock M, Perry S, et al. Increasing use of cardiopulmonary resuscitation during out-of-hospital ventricular fibrillation arrest: survival implications of guideline changes. Circulation 2006;114:2760–5.

951. Aufderheide TP, Yannopoulos D, Lick CJ, et al. Implementing the 2005 American Heart Association Guidelines improves outcomes after out-of-hospital cardiac arrest. Heart Rhythm 2010;7:1357–62.
952. Garza AG, Gratton MC, Salomone JA, Lindholm D, McElroy J, Archer R. Improved patient survival using a modified resuscitation protocol for out-of-hospital cardiac arrest. Circulation 2009;119:2597–605.
953. Deasy C, Bray JE, Smith K, et al. Cardiac arrest outcomes before and after the 2005 resuscitation guidelines implementation: evidence of improvement? Resuscitation 2011;82:984–8.
954. Bigham BL, Koprowicz K, Rea T, et al. Cardiac arrest survival did not increase in the Resuscitation Outcomes Consortium after implementation of the 2005 AHA CPR and ECC guidelines. Resuscitation 2011;82:979–83.
955. Jiang C, Zhao Y, Chen Z, Chen S, Yang X. Improving cardiopulmonary resuscitation in the emergency department by real-time video recording and regular feedback learning. Resuscitation 2010;81:1664–9.
956. Stiell IG, Wells GA, Field BJ, et al. Improved out-of-hospital cardiac arrest survival through the inexpensive optimization of an existing defibrillation program: OPALS study phase II. Ontario Prehospital Advanced Life Support. JAMA 1999;281:1175–81.
957. Olasveengen TM, Tomlinson AE, Wik L, et al. A failed attempt to improve quality of out-of-hospital CPR through performance evaluation. Prehosp Emerg Care 2007;11:427–33.
958. Clarke S, Lyon R, Milligan D, Clegg G. Resuscitation feedback and targeted education improves quality of pre-hospital resuscitation in Scotland. Emerg Med J 2011;28(Suppl. 1):A6.
959. Fletcher D, Galloway R, Chamberlain D, Pateman J, Bryant G, Newcombe RG. Basics in advanced life support: a role for download audit and metronomes. Resuscitation 2008;78:127–34.
960. Rittenberger JC, Guyette FX, Tisherman SA, DeVita MA, Alvarez RJ, Callaway CW. Outcomes of a hospital-wide plan to improve care of comatose survivors of cardiac arrest. Resuscitation 2008;79:198–204.
961. Wolfe H, Zebuhr C, Topjian AA, et al. Interdisciplinary ICU cardiac arrest debriefing improves survival outcomes. Crit Care Med 2014;42:1688–95.
962. Hillman K, Chen J, Cretikos M, et al. Introduction of the medical emergency team (MET) system: a cluster-randomised controlled trial. Lancet 2005;365:2091–7.
963. Buist MD, Moore GE, Bernard SA, Waxman BP, Anderson JN, Nguyen TV. Effects of a medical emergency team on reduction of incidence of and mortality from unexpected cardiac arrests in hospital: preliminary study. BMJ 2002;324:387–90.
964. Beitler JR, Link N, Bails DB, Hurdle K, Chong DH. Reduction in hospital-wide mortality after implementation of a rapid response team: a long-term cohort study. Crit Care 2011;15:R269.
965. Chan PS, Khalid A, Longmore LS, Berg RA, Kosiborod M, Spertus JA. Hospital-wide code rates and mortality before and after implementation of a rapid response team. JAMA 2008;300:2506–13.
966. Konrad D, Jaderling G, Bell M, Granath F, Ekbom A, Martling CR. Reducing in-hospital cardiac arrests and hospital mortality by introducing a medical emergency team. Intensive Care Med 2010;36:100–6.
967. Lighthall GK, Parast LM, Rapoport L, Wagner TH. Introduction of a rapid response system at a United States veterans affairs hospital reduced cardiac arrests. Anesth Analg 2010;111:679–86.
968. Santamaria J, Tobin A, Holmes J. Changing cardiac arrest and hospital mortality rates through a medical emergency team takes time and constant review. Crit Care Med 2010;38:445–50.
969. Priestley G, Watson W, Rashidian A, et al. Introducing Critical Care Outreach: a ward-randomised trial of phased introduction in a general hospital. Intensive Care Med 2004;30:1398–404.
970. Kaldjian LC, Weir RF, Duffy TP. A clinician's approach to clinical ethical reasoning. J Gen Intern Med 2005;20:306–11.
971. O'Neill O. Autonomy and trust in bioethics. Cambridge/New York: Cambridge University Press; 2002.
972. Beauchamp TL, Childress JF. Principles of biomedical ethics. 6th ed. New York: Oxford University Press; 2009.
973. World Medical Association. Medical ethics manual. 2nd ed. World Medical Association; 2009.
974. Lippert FK, Raffay V, Georgiou M, Steen PA, Bossaert L. European Resuscitation Council Guidelines for Resuscitation 2010 Section 10. The ethics of resuscitation and end-of-life decisions. Resuscitation 2010;81:1445–51.
975. Morrison LJ, Kierzek G, Diekema DS, et al. Part 3: ethics: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122:S665–75.
976. Brody BA, Halevy A. Is futility a futile concept? J Med Philos 1995;20:123–44.
977. Swig L, Cooke M, Osmond D, et al. Physician responses to a hospital policy allowing them to not offer cardiopulmonary resuscitation. J Am Geriatr Soc 1996;44:1215–9.
978. Waisel DB, Truog RD. The cardiopulmonary resuscitation-not-indicated order: futility revisited. Ann Intern Med 1995;122:304–8.
979. British Medical Association the Resuscitation Council (UK) and the Royal College of Nursing. Decisions relating to cardiopulmonary resuscitation. A joint statment from the British Medical Association, the Resuscitation Council (UK) and the Royal College of Nursing. London: British Medical Association; 2014.
980. Soholm H, Bro-Jeppesen J, Lippert FK, et al. Resuscitation of patients suffering from sudden cardiac arrests in nursing homes is not futile. Resuscitation 2014;85:369–75.
981. Committee on Bioethics (DH-BIO) of the Council of Europe. Guide on the Decision-Making Process Regarding Medical Treatment in End-of-Life Situations; 2014.
982. Fritz Z, Cork N, Dodd A, Malyon A. DNACPR decisions: challenging and changing practice in the wake of the Tracey judgment. Clin Med 2014;14:571–6.
983. Etheridge Z, Gatland E. When and how to discuss "do not resuscitate" decisions with patients. BMJ 2015;350:h2640.
984. Xanthos T. 'Do not attempt cardiopulmonary resuscitation' or 'allowing natural death'? The time for resuscitation community to review its boundaries and its terminology. Resuscitation 2014;85:1644–5.
985. Salkic A, Zwick A. Acronyms of dying versus patient autonomy. Eur J Health Law 2012;19:289–303.
986. Johnston C, Liddle J. The Mental Capacity Act 2005: a new framework for healthcare decision making. J Med Ethics 2007;33:94–7.
987. Shaw D. A direct advance on advance directives. Bioethics 2012;26:267–74.
988. Resuscitation Council (UK). Quality Standards for cardiopulmonary resuscitation practice and training. Acute Care. London: Resuscitation Council (UK); 2013.
989. Andorno R, Biller-Andorno N, Brauer S. Advance health care directives: towards a coordinated European policy? Eur J Health Law 2009;16:207–27.
990. Staniszewska S, Haywood KL, Brett J, Tutton L. Patient and public involvement in patient-reported outcome measures: evolution not revolution. Patient 2012;5:79–87.
991. Lannon R, O'Keeffe ST. Cardiopulmonary resuscitation in older people – a review. Rev Clin Gerontol 2010;20:20–9.
992. Becker TK, Gausche-Hill M, Aswegan AL, et al. Ethical challenges in Emergency Medical Services: controversies and recommendations. Prehosp Disaster Med 2013;28:488–97.
993. Nordby H, Nohr O. The ethics of resuscitation: how do paramedics experience ethical dilemmas when faced with cancer patients with cardiac arrest? Prehosp Disaster Med 2012;27:64–70.
994. Fraser J, Sidebotham P, Frederick J, Covington T, Mitchell EA. Learning from child death review in the USA, England, Australia, and New Zealand. Lancet 2014;384:894–903.
995. Ulrich CM, Grady C. Cardiopulmonary resuscitation for Ebola patients: ethical considerations. Nurs Outlook 2015;63:16–8.
996. Torabi-Parizi P, Davey Jr RT, Suffredini AF, Chertow DS. Ethical and practical considerations in providing critical care to patients with ebola virus disease. Chest 2015;147:1460–6.
997. Zavalkoff SR, Shemie SD. Cardiopulmonary resuscitation: saving life then saving organs? Crit Care Med 2013;41:2833–4.
998. Orioles A, Morrison WE, Rossano JW, et al. An under-recognized benefit of cardiopulmonary resuscitation: organ transplantation. Crit Care Med 2013;41:2794–9.
999. Gillett G. Honouring the donor: in death and in life. J Med Ethics 2013;39:149–52.
1000. Deleted in proofs.
1001. Hurst SA, Becerra M, Perrier A, Perron NJ, Cochet S, Elger B. Including patients in resuscitation decisions in Switzerland: from doing more to doing better. J Med Ethics 2013;39:158–65.
1002. Gorton AJ, Jayanthi NV, Lepping P, Scriven MW. Patients' attitudes towards "do not attempt resuscitation" status. J Med Ethics 2008;34:624–6.
1003. Freeman K, Field RA, Perkins GD. Variation in local trust Do Not Attempt Cardiopulmonary Resuscitation (DNACPR) policies: a review of 48 English healthcare trusts. BMJ Open 2015;5:e006517.
1004. Field RA, Fritz Z, Baker A, Grove A, Perkins GD. Systematic review of interventions to improve appropriate use and outcomes associated with do-not-attempt-cardiopulmonary-resuscitation decisions. Resuscitation 2014;85:1418–31.
1005. Micallef S, Skrifvars MB, Parr MJ. Level of agreement on resuscitation decisions among hospital specialists and barriers to documenting do not attempt resuscitation (DNAR) orders in ward patients. Resuscitation 2011;82:815–8.
1006. Pitcher D, Smith G, Nolan J, Soar J. The death of DNR. Training is needed to dispel confusion around DNAR. BMJ 2009;338:b2021.
1007. Davies H, Shakur H, Padkin A, Roberts I, Slowther AM, Perkins GD. Guide to the design and review of emergency research when it is proposed that consent and consultation be waived. Emerg Med J: EMJ 2014;31:794–5.
1008. Mentzelopoulos SD, Mantzanas M, van Belle G, Nichol G. Evolution of European Union legislation on emergency research. Resuscitation 2015;91:84–91.
1009. Booth MG. Informed consent in emergency research: a contradiction in terms. Sci Eng Ethics 2007;13:351–9.
1010. World Medical Association. Guidance on good clinical practice CPMP/ICH/135/95. World Medical Association; 2013.
1011. Perkins GD, Bossaert L, Nolan J, et al. Proposed revisions to the EU clinical trials directive – comments from the European Resuscitation Council. Resuscitation 2013;84:263–4.
1012. Lemaire F. Clinical research in the ICU: response to Kompanje et al. Intensive Care Med 2014;40:766.
1013. McInnes AD, Sutton RM, Nishisaki A, et al. Ability of code leaders to recall CPR quality errors during the resuscitation of older children and adolescents. Resuscitation 2012;83:1462–6.
1014. Gabbott D, Smith G, Mitchell S, et al. Cardiopulmonary resuscitation standards for clinical practice and training in the UK. Resuscitation 2005;64:13–9.
1015. Perkins GD, Jacobs IG, Nadkarni VM, et al. Cardiac arrest and cardiopulmonary resuscitation outcome reports: update of the Utstein resuscitation registry templates for out-of-hospital cardiac arrest. Resuscitation 2014.

1016. Daya MR, Schmicker RH, Zive DM, et al. Out-of-hospital cardiac arrest survival improving over time: results from the Resuscitation Outcomes Consortium (ROC). Resuscitation 2015;91:108–15.
1017. Grasner JT, Herlitz J, Koster RW, Rosell-Ortiz F, Stamatakis L, Bossaert L. Quality management in resuscitation – towards a European cardiac arrest registry (EuReCa). Resuscitation 2011;82:989–94.
1018. Grasner JT, Bossaert L. Epidemiology and management of cardiac arrest: what registries are revealing. Best Pract Res Clin Anaesthesiol 2013;27:293–306.
1019. Wnent J, Masterson S, Grasner JT, et al. EuReCa ONE – 27 Nations, ONE Europe, ONE Registry: a prospective observational analysis over one month in 27 resuscitation registries in Europe – the EuReCa ONE study protocol. Scand J Trauma Resuscitation Emerg Med 2015;23:7.