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Part 1: Executive summary
2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations☆

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Toward International Consensus on Science

The International Liaison Committee on Resuscitation (ILCOR) was founded on November 22, 1992, and currently includes representatives from the American Heart Association (AHA), the European Resuscitation Council (ERC), the Heart and Stroke Foundation of Canada (HSFC), the Australian and New Zealand Committee on Resuscitation (ANZCOR), Resuscitation Council of Southern Africa (RCSA), the InterAmerican Heart Foundation (IAHF), and the Resuscitation Council of Asia (RCA). Its mission is to identify and review international science and knowledge relevant to cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) and when there is consensus to offer treatment recommendations. Emergency cardiovascular care includes all responses necessary to treat sudden life-threatening events affecting the cardiovascular and respiratory systems, with a particular focus on sudden cardiac arrest.

In 1999, the AHA hosted the first ILCOR conference to evaluate resuscitation science and develop common resuscitation guidelines. The conference recommendations were published in the *International Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*.[1] Since 2000, researchers from the ILCOR member councils have evaluated resuscitation science in 5-year cycles. The conclusions and recommendations of the 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care With Treatment Recommendations were published at the end of 2005.[2,3] The most recent International Consensus Conference was held in Dallas in February 2010, and this publication contains the consensus science statements and treatment recommendations developed with input from the invited participants.

The goal of every resuscitation organisation and resuscitation expert is to prevent premature cardiovascular death. When cardiac arrest or life-threatening emergencies occur, prompt and skillful response can make the difference between life and death and between intact survival and debilitation. This document summarises the 2010 evidence evaluation of published science about the recognition and response to sudden life-threatening events, particularly sudden cardiac arrest and peri-arrest events in victims of all ages. The broad range and number of topics reviewed necessitated succinctness in the consensus science statements and brevity in treatment recommendations. This supplement is not a comprehensive review of every aspect of resuscitation medicine; not all topics reviewed in 2005 were reviewed in 2010. This executive summary highlights the evidence evaluation and treatment recommendations of the 2010 evidence evaluation process. More detailed information is available in other parts of this publication.

Evidence evaluation process

To begin the current evidence evaluation process, ILCOR representatives established 6 task forces: basic life support (BLS); advanced life support (ALS); acute coronary syndromes (ACS); paediatric life support; neonatal life support; and education, implementation, and teams (EIT). Separate writing groups were formed to coordinate evidence evaluation for defibrillation and mechan-

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Table 1
Levels of Evidence.

C2010 Levels of Evidence for Studies of Therapeutic Interventions
LOE 1: Randomized controlled trials (RCTs) (or meta-analyses of RCTs)
LOE 2: Studies using concurrent controls without true randomization (eg, "pseudo"-randomized)
LOE 3: Studies using retrospective controls
LOE 4: Studies without a control group (eg, case series)
LOE 5: Studies not directly related to the specific patient/population (eg, different patient/population, animal models, mechanical models, etc)
C2010 Levels of Evidence for Prognostic Studies
LOE P1: Inception (prospective) cohort studies (or meta-analyses of inception cohort studies), or validation of Clinical Decision Rule (CDR)
LOE P2: Follow-up of untreated control groups in RCTs (or meta-analyses of follow-up studies), or derivation of CDR, or validated on split-sample only
LOE P3: Retrospective cohort studies
LOE P4: Case series
LOE P5: Studies not directly related to the specific patient/population (eg, different patient/population, animal models, mechanical models, etc)
C2010 Levels of Evidence for Diagnostic Studies
LOE D1: Validating cohort studies (or meta-analyses of validating cohort studies) or validation of Clinical Decision Rule (CDR)
LOE D2: Exploratory cohort study (or meta-analyses of follow-up studies), or derivation of CDR, or a CDR validated on a split-sample only
LOE D3: Diagnostic case-control study
LOE D4: Study of diagnostic yield (no reference standard)
LOE D5: Studies not directly related to the specific patient/population (eg, different patient/population, animal models, mechanical models, etc)

ical devices because these overlapped with both BLS and ALS. Each task force identified topics requiring evidence evaluation and invited international experts to review them. To ensure a consistent and thorough approach, a worksheet template was created with step-by-step directions to help the experts document their literature reviews, evaluate studies, determine levels of evidence (Table 1), and develop treatment recommendations (see Part 3: Evidence Evaluation Process).[4] When possible, 2 expert reviewers were invited to perform independent evaluations for each topic. The worksheet authors submitted their search strategies to 1 of 3 worksheet review experts. The lead evidence evaluation expert also reviewed all worksheets and assisted the worksheet authors in ensuring consistency and quality in the evidence evaluation. This process is described in detail in Part 3.[4] In conjunction with the International First Aid Science Advisory Board, the AHA established an additional task force to review evidence on first aid. This topic is summarised in Part 13. The evidence review followed the same process but was not part of the formal ILCOR review.

The evidence evaluation process from 2007 to 2009 initially involved 509 worksheet authors with 569 worksheets. Some of the worksheets were merged while in other cases there was no new evidence and the worksheets/topics were deleted. The 2010 International Consensus Conference in February, 2010 involved 313 experts from 30 countries. A final total of 277 specific resuscitation questions, each in standard PICO (Population, Intervention, Comparison, Outcome) format, were considered by 356 worksheet authors who reviewed thousands of relevant, peer-reviewed publications. Many of these worksheets were presented and discussed at monthly or semimonthly task force international web conferences (i.e., "webinars" that involved conference calls with simultaneous internet conferencing). Beginning in May 2009 the evidence review and summary portions of the evidence evaluation worksheets, with worksheet author conflict of interest (COI) statements, were posted on the ILCOR Web site (www.ilcor.org). Journal advertisements and emails invited public comment. Persons who submitted comments were required to indicate their potential conflicts of interest. Public comments and potential conflicts of interest were sent to the appropriate ILCOR task force chair and worksheet author for consideration.

To provide the widest possible dissemination of the science reviews performed for the 2010 International Consensus Conference, the worksheets prepared for the conference are linked from this document and can be accessed by clicking on the superscript worksheet numbers (each begins with a W) located adjacent to headings.

During the 2010 Consensus Conference, wireless Internet access was available to all conference participants to facilitate real-time verification of the literature. Expert reviewers presented summaries of their evidence evaluation in plenary and concurrent sessions. Presenters and participants then debated the evidence, conclusions, and draft summary statements. The ILCOR task forces met daily during the conference to discuss and debate the experts' recommendations and develop interim consensus science statements. Each science statement summarised the experts' interpretation of all relevant data on a specific topic, and included consensus draft treatment recommendations. The wording of science statements and treatment recommendations was revised after further review by ILCOR member organisations and the editorial board. This format ensures that the final document represents a truly international consensus process.

At the time of submission this document represented the state-of-the-art science of resuscitation medicine. With the permission of the relevant journal editors, several papers were circulated among task force members if they had been accepted for publication in peer-reviewed journals but had not yet been published. These peer-reviewed and accepted manuscripts were included in the consensus statements.

This manuscript was ultimately approved by all ILCOR member organisations and an international editorial board (listed on the title page of this supplement). Reviewers solicited by the editor of *Circulation* and the AHA Science Advisory and Coordinating Committee performed parallel peer reviews of this document before it was accepted for publication. This document is being published online simultaneously by *Circulation* and *Resuscitation*, although the version in the latter publication does not include the section on first aid.

Management of potential conflicts of interest

In order to ensure the evidence evaluation process was free from commercial bias, extensive conflict of interest management principles were instituted immediately following the completion of the 2005 Consensus on CPR and ECC Science and Treatment Recommendations (CoSTR), concurrent with the start of the 2010 CoSTR process. All of the participants were governed by the COI management principles regardless of their role in the CoSTR process. COI disclosure was required from all participants and was updated annually or when changes occurred. Commercial relationships were considered at every stage of the evidence evaluation process

and, depending on the nature of the relationship and their role in the evidence evaluation process, participants were restricted from some activities (i.e., leading, voting, deciding, writing, discussing) that directly or indirectly related to that commercial interest. While the focus of the process was the evaluation of the scientific evidence, attention was given to potential COI throughout the CoSTR process.[5–7] This policy is described in detail in Part 4: "Management of Potential Conflicts of Interest."[8]

Applying science to improve survival

From consensus on science to guidelines

This document presents international consensus statements that summarise the science of resuscitation and, wherever possible, treatment recommendations. ILCOR member organisations will subsequently publish resuscitation guidelines that are consistent with the science in this consensus document, but the organisations will also take into account geographic, economic, and system differences in practice; availability of medical devices and drugs (e.g., not all devices and drugs reviewed in this publication are available and approved for use in all countries); and ease or difficulty of training. All ILCOR member organisations are committed to minimising international differences in resuscitation practice and optimising the effectiveness of resuscitation practice, instructional methods, teaching aids, training networks and outcomes (see Part 2: ILCOR Collaboration).

The recommendations of the 2010 International Consensus Conference confirm the safety and effectiveness of current approaches, acknowledge other approaches as ineffective, and introduce new treatments resulting from evidence-based evaluation. *New and revised treatment recommendations do not imply that clinical care that involves the use of previously published guidelines is either unsafe or ineffective*. Implications for education and retention were also considered when developing the final treatment recommendations.

Ischaemic heart disease is the leading cause of death in the world.[9,10] In addition, many newly born infants die worldwide as the result of respiratory distress immediately after birth. However, most out-of-hospital victims die without receiving the interventions described in this publication.

The actions linking the adult victim of sudden cardiac arrest with survival are called the adult Chain of Survival. The links in the Chain of Survival used by many resuscitation councils include prevention of the arrest, early recognition of the emergency and activation of the emergency medical services (EMS) system, early and high-quality CPR, early defibrillation, rapid ALS, and postresuscitation care. The links in the infant and child Chain of Survival are prevention of conditions leading to cardiopulmonary arrest, early and high-quality CPR, early activation of the EMS system, and early ALS.

The most important determinant of survival from sudden cardiac arrest is the presence of a trained lay rescuer who is ready, willing, and able to act. Although some ALS techniques improve survival,[11,12] these improvements are usually less significant than the increase in survival rates that can result from higher rates of lay rescuer (bystander) CPR and establishment of automated external defibrillation programs in the community.[13–17] Thus, our greatest challenges remain the education of the lay rescuer and understanding and overcoming the barriers that prevent even trained rescuers from performing high-quality CPR. We must increase the effectiveness and efficiency of instruction, improve skills retention, and reduce barriers to action for both basic and ALS providers. Similarly, the placement and use of automated external defibrillators (AEDs) in the community should be encouraged to enable defibrillation within the first minutes after a ventricular fibrillation (VF) sudden cardiac arrest.

The Universal Algorithm

Several of the new treatment recommendations cited in this document are included in the updated ILCOR Universal Cardiac Arrest Algorithm (Fig. 1). This algorithm is intended to apply to attempted resuscitation of infant, child, and adult victims of cardiac arrest (excluding newly borns). Every effort has been made to keep this algorithm simple yet make it applicable to treatment of cardiac arrest victims of all ages and in most circumstances. Modification will be required in some situations, and these exceptions are highlighted elsewhere in this document. Each resuscitation organisation has based its guidelines on this ILCOR algorithm, although there will be regional modifications.

Rescuers begin CPR if the adult victim is unresponsive with absent or abnormal breathing, such as an occasional gasp. A single compression–ventilation ratio of 30:2 is used for the lone lay rescuer of an infant, child, or adult victim (excluding newly borns). This single ratio is designed to simplify teaching, promote skills retention, increase the number of compressions given, and decrease interruptions in compressions. The most significant adult BLS change in this document is a recommendation for a CAB (compressions, airway, breathing) sequence instead of an ABC (airway, breathing, compressions) sequence to minimise delay to initiation of compressions and resuscitation. In other words, rescuers of adult victims should begin resuscitation with compressions rather than opening the airway and delivery of breaths.

Once a defibrillator is attached, if a shockable rhythm is confirmed, a single shock is delivered. Irrespective of the resultant rhythm, CPR starting with chest compressions should resume immediately after each shock to minimise the "no-flow" time (i.e., time during which compressions are not delivered, for example, during rhythm analysis). ALS interventions are outlined in a box at the center of the algorithm. Once an advanced airway (tracheal tube or supraglottic airway) has been inserted, rescuers should provide continuous chest compressions (without pauses for ventilations) and ventilations at a regular rate (avoiding hyperventilation).

The 2005 International Consensus on Science emphasised the importance of minimal interruption of chest compressions because 2005 evidence documented the frequency of interruptions in chest compressions during both in-hospital and out-of-hospital CPR and the adverse effects of such interruptions in attaining resumption of spontaneous circulation (ROSC).[18–20] In 2010, experts agree that rescuers should be taught to adhere to all four metrics of CPR: adequate rate, adequate depth, allowing full chest recoil after each compression and minimising pauses (e.g., hands off time) in compressions.

Most significant developments in resuscitation from 2005 to 2010

Although resuscitation practices are usually studied as single interventions, they are actually performed as a large sequence of actions, each with its own timing and quality of performance. It may be difficult or impossible to assess the contribution of any one action (energy level for defibrillation, airway maneuver, drug) on the most important outcomes, such as neurologically intact survival to discharge. In fact, it is likely that it is the combination of actions, each performed correctly, in time and in order, that results in optimal survival and function. A few studies give insight into this necessary shift from studying of changes in individual actions (*point improvements*) to studying the effects of changing the entire sequence of actions (*flow improvement*).[21,22]

Fig. 1. The Universal Algorithm.

The compression–ventilation ratio was one of the most controversial topics of the 2005 International Consensus Conference. The experts began the 2005 conference acknowledging that rates of survival from cardiac arrest to hospital discharge were low, averaging ≤6% internationally,[23,24] and that survival rates had not increased substantially in recent years. That observation led to the 2005 change to a universal compression–ventilation ratio for all lone rescuers of victims of all ages and to an emphasis on the importance of CPR quality throughout the 2005 Consensus on CPR and ECC Science With Treatment Recommendations (CoSTR) document and subsequent ILCOR member council guidelines.[25]

Resuscitation outcomes vary considerably among regions.[26,27] In recent studies the outcome from cardiac arrest, particularly from shockable rhythms, is improved.[28–33] Moreover, there is an association between implementation of new resuscitation guidelines and improved outcome.[31,33] However, there is also evidence that new guidelines can take from 1.5 to 4 years to implement.[34,35]

There have been many developments in resuscitation science since 2005 and these are highlighted below.

Factors affecting lay rescuer CPR performance

During the past 5 years, there has been an effort to simplify CPR recommendations and emphasise the importance of high-quality CPR. Large observational studies from investigators in member countries of the RCA, the newest member of ILCOR,[36–39] and other studies[40,41] have provided significant data about the effects of bystander CPR.

CPR quality

Strategies to reduce the interval between stopping chest compressions and delivery of a shock (the preshock pause) will improve the chances of shock success.[42,43] These data are driving major

changes in training of resuscitation teams. Data downloaded from CPR-sensing and feedback-enabled defibrillators can be used to debrief resuscitation teams and improve CPR quality.[44]

In-hospital CPR registries

The National Registry of CPR (NRCPR) and other registries are providing valuable information about the epidemiology and outcomes of in-hospital resuscitation in adults and children.[45–52]

Insufficient evidence on devices and ALS drugs

Many devices remain under investigation, and at the time of the 2010 Consensus Conference there was insufficient evidence to recommend for or against the use of any mechanical devices. There are still no data showing that any drugs improve long-term outcome after cardiac arrest.[21] Clearly further information is needed.

Importance of post-cardiac arrest care

It is now clear that organised post-cardiac arrest care with emphasis on protocols for optimising cardiovascular and neurological care, including therapeutic hypothermia, can improve survival to hospital discharge among victims who achieve ROSC after cardiac arrest.[22,53,54] Although it is not yet possible to determine the individual effect of many of these therapies, it is clear that this "bundle of care" can improve outcome. Therapeutic hypothermia has been shown independently to improve outcome after adult witnessed out-of-hospital VF cardiac arrest and after neonatal hypoxic-ischaemic insult. Since 2005, two non-randomised studies with concurrent controls indicated possible benefit of hypothermia after cardiac arrest from other initial rhythms in-hospital and out-of-hospital,[55,56] and other studies with historic controls have shown benefit for therapeutic hypothermia after out-of-hospital all-rhythm cardiac arrests in adults.[22,57–60]

Studies of newborns with birth asphyxia[61,62] showed that therapeutic hypothermia (33.5–34.5 °C) up to 72 h after resuscitation has an acceptable safety profile and was associated with better survival and long-term neurological outcome. Retrospective studies of children following cardiac arrest failed to demonstrate benefit of therapeutic hypothermia, but a well-designed multicentre prospective randomised trial is in progress.

Many studies in recent years have attempted to identify comatose post-cardiac arrest patients who have no prospects of good neurological recovery.[63] It is now recognised that the use of therapeutic hypothermia invalidates the prognostication decision criteria that were established before hypothermia therapy was implemented: recent studies have documented occasional good outcomes in patients who would previously have met criteria predicting poor outcome (Cerebral Performance Category 3, 4, or 5).[64,65]

Education and implementation, including retraining

Basic and advanced life support knowledge and skills can deteriorate in as little as 3–6 months. Quality of education, frequent assessments and, when needed, refresher training are recommended to maintain resuscitation knowledge and skills.

Summary of the 2010 ILCOR Consensus on Science With Treatment Recommendations

Adult BLS

The 2010 International Consensus Conference addressed many questions related to the performance of BLS. These have been grouped into (1) epidemiology and recognition of cardiac arrest, (2) chest compressions, (3) airway and ventilation, (4) compression–ventilation sequence, (5) special situations, (6) EMS system, and (7) risks to the victim. Defibrillation is discussed separately in Part 6 because it is both a basic and an ALS skill.

There have been several important advances in the science of resuscitation since the 2005 ILCOR review. The following is a summary of the most important evidence-based recommendations for performance of BLS:

- Lay rescuers begin CPR if the adult victim is unresponsive and not breathing normally (ignoring occasional gasps) without assessing the victim's pulse.
- Following initial assessment, rescuers begin CPR with chest compressions rather than opening the airway and delivering rescue breathing.
- All rescuers, trained or not, should provide chest compressions to victims of cardiac arrest. A strong emphasis on delivering high-quality chest compressions remains essential: push hard to a depth of at least 2 in. or 5 cm at a rate of at least 100 compressions per minute, allow full chest recoil after each compression, and minimise interruptions in chest compressions.
- Trained rescuers should also provide ventilations with a compression–ventilation ratio of 30:2.
- EMS dispatchers should provide telephone instruction in chest compression-only CPR.

Epidemiology and recognition of cardiac arrest

Early recognition is a key step in initiating early treatment of cardiac arrest; this recognition requires identification of the most accurate method of determining cardiac arrest. In general rescuers should begin CPR if an adult is unresponsive and not breathing normally (disregarding occasional gasps). Healthcare providers cannot reliably determine the presence or absence of a pulse, so CPR should not be delayed if a pulse is not immediately found in the unresponsive adult victim who is not breathing normally. Lay rescuers cannot reliably determine the cause of an arrest, so it is not realistic to expect them to alter the response sequence to the likely aetiology of each arrest.

Chest compressions

Several components of chest compressions can alter effectiveness: hand position, position of the rescuer, position of the victim, compression depth, chest recoil, and duty cycle (see definition, below). Compression depth should at least be 2 in. (5 cm). Evidence for these techniques was reviewed in an attempt to define the optimal method.

Compressions only and compressions plus ventilations

All rescuers should perform chest compressions for all patients in cardiac arrest. Chest compressions alone are recommended for untrained laypersons responding to victims of cardiac arrest. Performing chest compressions alone is reasonable for trained laypersons if they are incapable of delivering airway and breathing

maneuver to cardiac arrest victims. Providing chest compressions with ventilations is reasonable for trained laypersons who are capable of giving CPR with ventilations to cardiac arrest victims.

Professional rescuers should provide chest compressions with ventilations for cardiac arrest victims. There is insufficient evidence to support or refute the effectiveness of the combination of chest compressions plus airway opening and oxygen inflation (compared with conventional CPR) by professional rescuers during the first few minutes of resuscitation from cardiac arrest.

Airway and ventilation

The best method of obtaining an open airway and the optimum frequency and volume of artificial ventilation were reviewed. The recommendations are unchanged from 2005.

Compression–ventilation sequence

In the 2005 International Consensus Conference recommendations, the recommended sequence of CPR actions was: airway, breathing, and circulation/chest compressions (ABC). In this 2010 document, in an attempt to shorten the delay to first chest compressions for adult victims, experts came to the consensus that rescuers may consider starting CPR with chest compressions rather than ventilations (the sequence will then be "CAB"). Rescuers should minimise interruptions in chest compressions during the resuscitation attempt.

Any recommendation for a specific CPR compression–ventilation ratio represents a compromise between the need to generate blood flow and the need to supply oxygen to the lungs and remove CO_2 from the blood. At the same time any such ratio must be taught to would-be rescuers, so the effect of compression–ventilation ratios on skills acquisition and retention must be considered. A compression–ventilation ratio of 30:2 remains reasonable for an adult victim of cardiac arrest when no advanced airway is in place.

Special situations (cervical spine injury, facedown)

It is reasonable to roll a victim who is facedown and unresponsive to the supine position to assess breathing and initiate resuscitation. Concern for protecting the neck should not hinder the evaluation process or life saving procedures.

EMS system

Recognition of cardiac arrest as the cause of collapse is rarely simple and requires EMS dispatchers to elicit critical information from the caller. Failure to recognise the true cause of the collapse occurs in as many as 50% of cases of cardiac arrest; this failure precludes the implementation of bystander CPR and lowers the victim's chance of survival.[66]

When attempting to identify a cardiac arrest victim, the EMS dispatcher should inquire about the victim's absence of consciousness and quality of breathing (normal/not normal). Dispatchers should be specifically educated about identification of abnormal breathing in order to improve recognition of adult cardiac arrest. The correct identification of cardiac arrest may be increased by careful attention to the caller's spontaneous comments and by focused questions, including questions about seizures and gasping.

Bystanders who call their local emergency response number should receive initial instructions on performing CPR. Dispatchers should provide compression-only CPR instructions to untrained rescuers for adults with suspected sudden cardiac arrest. If a dispatcher suspects asphyxial arrest, it is reasonable to provide instructions for rescue breathing followed by chest compressions. Quality-improvement efforts should assess the accuracy and timeliness of dispatcher recognition of cardiac arrest and the delivery of CPR instructions.

Risks to the victim

Many rescuers are concerned that delivering chest compressions to a victim who is not in cardiac arrest will lead to serious complications, and thus they do not initiate CPR for some victims of cardiac arrest. In individuals with presumed cardiac arrest, bystander CPR rarely leads to serious harm in victims who are eventually found not to be cardiac arrest; therefore, performance of bystander CPR should be strongly encouraged.[67]

Defibrillation

The Defibrillation Task Force considered many questions related to adult defibrillation. In general, the 2010 International Consensus Conference recommendations contain no major differences from the 2005 recommendations. The questions have been grouped into the following categories: (1) CPR before defibrillation, (2) electrode–patient interface, (3) defibrillation strategy, (4) special situations, and (5) related defibrillation topics.

There are several knowledge gaps created by the lack of high-quality, large clinical studies. These include the minimal acceptable first-shock success rate, characteristics of the optimal biphasic waveform, optimal energy levels for specific waveforms, and the best shock strategy (fixed versus escalating).

CPR before defibrillation

Whether a period of CPR should be performed before defibrillation in VF, especially after long response times, continues to be the subject of intense debate. The theoretical rationale for performing CPR before shock delivery is to improve coronary perfusion and thereby the chances of achieving sustained ROSC; however, there is inconsistent evidence to support or refute a delay in defibrillation to provide a period (90 s to 3 min) of CPR for patients in VF/pulseless ventricular tachycardia (VT) cardiac arrest. If more than one rescuer is present, one rescuer should provide chest compressions while the other activates the emergency response system, retrieves the AED and prepares to use it.

Electrode–patient interface

There are only a few studies comparing differences in outcome associated with use of different electrode–patient interfaces; many studies compare secondary end points such as the effect on transthoracic impedance. In ventricular arrhythmias there is no evidence to suggest that transthoracic impedance affects shock success. When using biphasic defibrillators for both pulseless VT/VF defibrillation and conversion of atrial fibrillation, self-adhesive defibrillator pads are safe and effective and are an acceptable alternative to standard defibrillation paddles. Hand-held paddles are preferable when using monophasic defibrillators for cardioversion of atrial fibrillation.

It is reasonable to place paddles or pads on the exposed chest in an anterolateral position. Acceptable alternative positions are the anteroposterior (paddles and pads) and apex-posterior (pads). There is insufficient evidence to make specific recommendations for the optimal electrode size for external defibrillation; however, it is reasonable to use a pad size <8 cm for adults. In terms of cardiac arrest outcomes there is insufficient evidence to recommend a specific composition of the conductive material of defibrillation electrodes.

Defibrillation strategy

All new defibrillators deliver shocks using a variety of biphasic waveforms. Although it has not been demonstrated conclusively in randomised clinical studies that biphasic defibrillators save more lives than monophasic defibrillators, biphasic defibrillators achieve higher first-shock success rates. Shock success is usually defined as termination of VF 5 s after the shock. There is insufficient evidence to recommend any specific biphasic waveform. In the absence of biphasic defibrillators, monophasic defibrillators are acceptable.

Several different biphasic waveforms exist, but no human studies have compared different biphasic waveforms and different energy levels related to defibrillation success or survival. For all waveforms insufficient evidence exists to make clear recommendations; however, it is reasonable to start at an energy level of 150–200 J for biphasic truncated exponential waveform for defibrillation of pulseless VT/VF cardiac arrest. There is insufficient evidence to determine the initial energy levels for any other biphasic waveform. Although evidence is limited, because of the lower total shock success for monophasic defibrillation, initial and subsequent shocks using this waveform should be at 360 J.

When defibrillation is required, a single shock should be provided with resumption of chest compressions/CPR immediately after the shock. Chest compressions should not be delayed for rhythm reanalysis or a pulse check immediately after a shock. CPR should not be interrupted until rhythm reanalysis is undertaken. For second and subsequent biphasic shocks the same initial energy level is acceptable. It is reasonable to increase the energy level when possible.

There are no survival differences between defibrillation in semiautomatic and manual modes during in-hospital or out-of-hospital resuscitation; however, the semiautomatic mode is preferred because it is easier to use and may deliver fewer inappropriate shocks. Trained personnel may deliver defibrillation in manual mode. Use of the manual mode enables chest compressions to be continued during charging, thereby minimising the preshock pause. For rescuers using the defibrillator in manual mode, electrocardiographic recognition skills are essential and frequent team training is helpful. The defibrillation mode (semiautomatic versus manual) that results in the best outcome will be influenced by the system, the provider's skills and training, and accuracy of electrocardiographic recognition.

Biphasic defibrillators are preferred for cardioversion of atrial fibrillation. There is no evidence to recommend a specific waveform, energy level, or strategy (fixed versus escalating) for cardioversion when using biphasic defibrillators. For cardioversion using monophasic defibrillators a high initial energy (360 J) seems preferable.

Electrical therapy in special situations

Electric pacing is not effective as a routine treatment in patients with asystolic cardiac arrest. Percussion pacing is not recommended in cardiac arrest in general; however, fist pacing may be considered in haemodynamically unstable bradyarrhythmias until an electric pacemaker (transcutaneous or transvenous) is available. The use of epicardial wires to pace the myocardium after cardiac surgery is effective.

In patients with an implantable cardioverter-defibrillator (ICD) or a permanent pacemaker, the placement of pads/paddles should not delay defibrillation. The defibrillator pad/paddle should be placed on the chest wall ideally at least 8 cm from the generator position. Anterior–posterior and anterior–lateral pad/paddle placements on the chest are acceptable in patients with an ICD or a permanent pacemaker.

Related defibrillation topics

There is insufficient evidence to support routine use of VF waveform analysis to guide defibrillation management in adult in-hospital and out-of-hospital cardiac arrest.

Rescuers should take precautions to minimise sparking (by careful pad/paddle placement, prevention of contact, etc.) during attempted defibrillation. Rescuers should try to ensure that defibrillation is not attempted in an oxygen-enriched atmosphere.

CPR techniques and devices

The success of any technique or device depends on the education and training of the rescuers. A device or technique that provides good-quality CPR and potentially better outcome when used by a highly trained team or in a test setting may result in frequent interruptions in CPR when used in an actual clinical setting.[18] As with any clinical practice intervention, the process must be monitored to assess for unintended adverse consequences.

Although no circulatory adjunct is currently recommended as preferable to manual CPR for routine use, some circulatory adjuncts are being used in both out-of-hospital and in-hospital resuscitation attempts. If a circulatory adjunct is used, rescuers should be well-trained and a program of continuous surveillance should be in place to ensure that use of the adjunct does not adversely affect survival.

The following CPR techniques and devices were reviewed during the 2010 International Consensus Conference: interposed abdominal compression CPR, active compression–decompression CPR, open-chest CPR, load-distributing band CPR, mechanical piston (thumper) CPR, Lund University Cardiac Arrest System (LUCAS) CPR, and the impedance threshold device (ITD). Interposed abdominal compression CPR has not been studied in humans since 1994. Active compression–decompression (ACD) CPR has not been studied in humans since 1999, although a meta-analysis comparing ACD CPR with standard CPR was published in 2004 and showed no significant increase in rates of immediate survival or hospital discharge.[68]

There are insufficient data to support or refute the routine use of open-chest CPR, load-distributing band CPR, LUCAS CPR, mechanical piston CPR, or the ITD instead of standard CPR. On the basis of case reports and case series[69] it may be reasonable to consider load-distributing band or LUCAS CPR to maintain continuous chest compressions while the patient undergoes percutaneous coronary intervention (PCI) or computed tomography (CT) or similar diagnostic studies when provision of manual CPR would be difficult.

ALS

The ILCOR ALS Task Force reviewed the topics of (1) airway and ventilation, (2) support of circulation during cardiac arrest, (3) peri-arrest arrhythmias, (4) cardiac arrest in special situations, (5) identification of reversible causes, (6) post-cardiac arrest care, (7) prognostication, and (8) organ donation.

The most important developments and recommendations in ALS since the 2005 ILCOR review have been:

- The use of capnography to confirm and continually monitor tracheal tube placement and quality of CPR.
- More precise guidance on control of glucose in adults with sustained ROSC. Blood glucose values >180 mg dL^{-1} (>10 mmol L^{-1}) should be treated and hypoglycaemia avoided.
- Additional evidence, albeit lower level, for use of therapeutic hypothermia for comatose survivors of cardiac arrest initially associated with nonshockable rhythms.
- Recognition that many accepted predictors of poor outcome in comatose survivors of cardiac arrest are unreliable, especially if

the patient has been treated with therapeutic hypothermia. There is inadequate evidence to recommend a specific approach to predicting poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia.
- The recognition that adults who progress to brain death after resuscitation from out-of-hospital cardiac arrest (OHCA) should be considered for organ donation.
- The recommendation that implementation of a comprehensive, structured treatment protocol may improve survival after cardiac arrest.

Airway and ventilation

Consensus conference topics related to the management of airway and ventilation are categorised as basic airway devices, cricoid pressure, advanced airway devices, confirmation of advanced airway placement, oxygenation, and strategies for ventilation. The use of oropharyngeal and nasopharyngeal airways has never been studied in cardiac arrest, but their use in this context remains reasonable.

The routine use of cricoid pressure to prevent aspiration in cardiac arrest is not recommended. If cricoid pressure is used during cardiac arrest, the pressure should be adjusted, relaxed, or released if it impedes ventilation or placement of an advanced airway.

The tracheal tube was once considered the optimal method of managing the airway during cardiac arrest. There is considerable evidence that without adequate training or ongoing skills maintenance, the incidence of failed intubations and complications (e.g., unrecognised oesophageal intubation or unrecognised dislodgment) is unacceptably high.[70–75] Prolonged attempts at tracheal intubation are harmful because the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Alternatives to the tracheal tube that have been studied during actual and manikin CPR include the bag and mask and supraglottic airway devices such as the laryngeal mask airway (LMA), Combitube, the laryngeal tube, and the I-gel. Studies comparing the supraglottic airway with tracheal intubation have generally compared insertion time and ventilation success rates. No study has shown an effect of the method of ventilation on survival.

There are no data to support the routine use of any specific approach to airway management during cardiac arrest. The best approach depends on the precise circumstances of the cardiac arrest and the competence of the rescuer. There is inadequate evidence to define the optimal timing of advanced airway placement during cardiac arrest. Healthcare professionals trained to use supraglottic airway devices may consider their use for airway management during cardiac arrest and as a backup or rescue airway in a difficult or failed tracheal intubation.

Waveform capnography is recommended to confirm and continuously monitor the position of a tracheal tube in victims of cardiac arrest and it should be used in addition to clinical assessment (auscultation and direct visualization are suggested).

If waveform capnography is not available, a nonwaveform CO_2 detector or oesophageal detector device in addition to clinical assessment can be used. Thoracic impedance may be used as an adjunctive measure to diagnose airway placement in patients with cardiac arrest; however, clinical decisions should not be based solely on thoracic impedance measurement until further study has confirmed its utility and accuracy in this population.

There is insufficient evidence to support or refute the use of a titrated oxygen concentration or constant 21% oxygen (room air) when compared with 100% oxygen during adult cardiac arrest. In the absence of other data, there is no reason to change the current treatment algorithm, which includes use of 100% oxygen during adult cardiac arrest.

There is insufficient evidence to support or refute the use of passive oxygen delivery during CPR to improve outcomes (ROSC, hospital discharge rate, and improved neurological survival) when compared with oxygen delivery by positive-pressure ventilation.

There is insufficient evidence to support or refute monitoring peak pressure and minute ventilation to improve outcome from cardiac arrest. There is indirect evidence that monitoring the respiratory rate with real-time feedback is effective in avoiding hyperventilation and achieving ventilation rates closer to recommended values, but there is no evidence that ROSC or survival is improved. Continuous capnography or capnometry monitoring if available may be beneficial by providing feedback on the effectiveness of chest compressions.

Support of circulation during cardiac arrest

Questions related to circulatory support during cardiac arrest that were discussed during the 2010 International Consensus Conference were categorised as (1) timing of drug delivery, (2) use of vasopressors during cardiac arrest, (3) use of other drugs during cardiac arrest, (4) use of intravenous (IV) fluids, and (5) provision of extracorporeal support. It is recognised that the vast majority of studies assessing the effects of drugs on survival have not been able to control for the quality of CPR. Furthermore, most drug evaluations to date have been conducted before recent advances in post-cardiac arrest care, including therapeutic hypothermia. Because most drug trials have, at most, demonstrated only short-term outcome advantage, it may be important to evaluate long-term outcome when these drugs are combined with optimised post-cardiac arrest care. One study compared the use of IV access and drugs (epinephrine, amiodarone, atropine, vasopressin, without isolating the effect of each individual drug alone), with no IV access and no drugs in adult out-of-hospital CPR. There was demonstrated improvement in ROSC and survival to hospital and intensive care unit admission but no difference in survival to discharge or neurological outcomes at discharge and 1-year follow-up.[21] However, this study was not powered to detect clinically meaningful differences in long-term outcome. Similarly, one study[76] with a "before and after" design compared various outcomes after OHCA and was not able to demonstrate any improvements after introduction of ALS (epinephrine, atropine, lidocaine). Neither of these studies is able to isolate outcomes specifically related to individual drug administration.

There is inadequate evidence to define the optimal timing or order for drug administration. Despite the continued widespread use of epinephrine and increased use of vasopressin during resuscitation in some countries, there is no placebo-controlled study that shows that the routine use of any vasopressor during human cardiac arrest increases survival to hospital discharge. There is no evidence that the routine use of other drugs (e.g., atropine, amiodarone, lidocaine, procainamide, magnesium, buffers, calcium, hormones, or fibrinolytics) during human CPR increases survival to hospital discharge. There is insufficient evidence to recommend for or against the routine infusion of IV fluids during resuscitation from cardiac arrest. There is also insufficient evidence to support or refute the routine use of extracorporeal CPR in cardiac arrest.

Peri-arrest arrhythmias

Narrow-complex tachycardia (excluding atrial fibrillation). There are 4 options for the treatment of narrow-complex tachycardia in the peri-arrest setting: electric conversion, physical maneuvers, pharmacological conversion, and rate control. The treatment choice depends on the stability of the patient and the

rhythm. In a haemodynamically unstable patient, narrow-complex tachycardia is best treated with electric cardioversion; otherwise, vagal maneuvers, IV adenosine, verapamil, and diltiazem are recommended as first-line treatment strategies. Nadolol, sotalol, propafenone, and amiodarone may be considered.

Atrial fibrillation. Patients with atrial fibrillation who are haemodynamically unstable should receive prompt electric cardioversion.

Rate control. β-Blockers and diltiazem are the drugs of choice for acute rate control in most patients with atrial fibrillation and rapid ventricular response. Digoxin and amiodarone may be used in patients with congestive heart failure; amiodarone may also result in cardioversion to normal sinus rhythm. Magnesium and clonidine have rate-controlling effects, although there are fewer supporting data for their use.

Rhythm control. Chemical cardioversion can be achieved with ibutilide, dofetilide, and flecainide. Amiodarone can also be used for chemical cardioversion but is less effective. Quinidine or procainamide may be useful for cardioversion, but their use is less well-established. Propafenone is more effective than placebo but not as effective as amiodarone, procainamide, or flecainide. There is no role for digoxin in chemical cardioversion.

Wide-complex tachycardia. There are 2 options for treatment of wide-complex tachycardia in the peri-arrest setting: electric conversion and chemical conversion. The choice depends on the stability of the patient and the rhythm. In a haemodynamically unstable patient, wide-complex tachycardia is best treated with electric cardioversion.

Procainamide is recommended for patients with haemodynamically stable monomorphic VT (mVT) who do not have severe congestive heart failure or acute myocardial infarction (AMI). Amiodarone is recommended for patients with haemodynamically stable mVT with and without either severe congestive heart failure or AMI. Sotalol may be considered for patients with haemodynamically stable sustained mVT, including patients with AMI.

In undifferentiated regular stable wide-complex tachycardia, IV adenosine may be considered relatively safe, may convert the rhythm to sinus, and may help diagnose the underlying rhythm.

Polymorphic wide-complex tachycardia associated with familial long QT syndrome may be treated with IV magnesium, pacing, and β-blockers; however, isoproterenol should be avoided. Polymorphic wide-complex tachycardia associated with acquired long QT syndrome may be treated with IV magnesium. The addition of pacing or IV isoproterenol may be considered when polymorphic wide-complex tachycardia is accompanied by bradycardia or appears to be precipitated by pauses in rhythm. Polymorphic wide-complex tachycardia without long QT syndrome may be responsive to IV β-blockers (ischaemic VT; catecholaminergic VT) or isoproterenol.

Bradycardia. In the peri-arrest setting the rescuer should seek and treat reversible causes of bradycardia. In the absence of immediately reversible causes, atropine remains the first-line drug for acute symptomatic bradycardia. If not effective, then consider isoproterenol, epinephrine, dopamine, or transcutaneous pacing.

Cardiac arrest in special situations

The ALS Task Force reviewed special situations associated with cardiac arrest, including avalanche, pregnancy, asthma, anaphylaxis, drug overdose and poisoning, coronary catheterisation, heart surgery, cardiac tamponade, pulmonary embolus, and electrolyte disorders.

Cardiac arrest caused by avalanche

Avalanches occur in areas that are difficult for rescuers to access and frequently involve multiple victims. The decision to initiate full resuscitative measures should be determined by the number of victims and the resources available and should be informed by the likelihood of survival. A victim buried by an avalanche is unlikely to survive if the victim has been buried for >35 min, the airway is obstructed on extrication and the initial core temperature is <32°, or the victim has an initial serum potassium level >7 mmol L^{-1}.

Cardiac arrest associated with pregnancy

There is insufficient evidence to support or refute the use of specialised obstetric resuscitation techniques in maternal cardiac arrest or the use of therapeutic hypothermia in the postarrest period. Treatment may be guided by understanding the physiology of pregnancy, the importance of releasing aortocaval compression, the increased risk for hypovolaemia, the optimal positioning for compressions, and the value of perimortem caesarean section early in maternal cardiac arrest.

Cardiac arrest caused by asthma, anaphylaxis, or electrolyte disorders

There is insufficient evidence to suggest any routine change to resuscitation treatment algorithms for patients with cardiac arrest caused by asthma, anaphylaxis, or electrolyte disorders.

Cardiac arrest caused by drug overdose and poisoning

The majority of questions concerning cardiac arrest caused by drug toxicity remain unanswered. The 2010 International Consensus Conference reviewed treatment of cardiac arrest caused by local anesthesia, benzodiazepines, β-blockers, calcium channel blockers, carbon monoxide, cocaine, cyanide, tricyclic antidepressants, digoxin, and opioids.

Cardiac arrest during coronary catheterisation

There are no randomised controlled trials evaluating alternative treatment strategies versus standard care for cardiac arrest during PCI. Evidence is limited to case studies for all interventions; thus, the data are insufficient to support or refute the use of mechanical chest compression, cough CPR, or emergency cardiopulmonary bypass to improve outcome of cardiac arrest during PCI.

Cardiac arrest after open or closed heart surgery

Resternotomy for patients with cardiac arrest following cardiac surgery should be considered in an appropriately staffed and equipped intensive care unit or in the operating suite. Resternotomy performed outside of these specialised environments has poor results. Chest compressions should not be withheld while preparing for emergency resternotomy. Mechanical circulatory support may be considered in the setting of cardiac arrest following cardiac surgery. There is insufficient evidence to make any recommendations about epinephrine dose, use of antiarrhythmics, or any other intervention separate from those recommended in standard protocols.

Cardiac arrest caused by cardiac tamponade

Pericardiocentesis guided by echocardiography should be considered for the treatment of cardiac arrest associated with cardiac tamponade. Non-image-guided pericardiocentesis is an acceptable alternative if echocardiography is not available. Emergency department thoracotomy and pericardiotomy are acceptable alternatives to operating suite thoracotomy and pericardiotomy for treatment of traumatic cardiac arrest associated with cardiac tamponade and can be considered for use in the treatment of nontraumatic cardiac

arrest when pericardiocentesis is unsuccessful in relieving cardiac tamponade.

Cardiac arrest caused by pulmonary embolus

Fibrinolytic therapy may be considered when pulmonary embolism is suspected as the cause of the cardiac arrest.

Identification of reversible causes

Ultrasound during cardiac arrest

Although there are several case series, no studies specifically examine the impact of ultrasound or echocardiography on patient outcomes in cardiac arrest.

Post-cardiac arrest care

Post-cardiac arrest treatment protocol

Before-and-after studies report an increase in survival of comatose patients with sustained ROSC after OHCA with implementation of a comprehensive treatment protocol.[22,53,54] Protocols include multiple elements such as hypothermia, glucose control, goal-directed haemodynamic optimisation, ventilation, and PCI. The independent effect of each element of the bundle of care could not be established.

Treatment of pulmonary embolism after ROSC

In patients with diagnosed or suspected pulmonary embolism after ROSC following cardiac arrest, there is inadequate evidence to recommend for or against the use of fibrinolytic therapy in addition to heparin. The mortality with surgical embolectomy for suspected or diagnosed pulmonary embolism is high if it follows cardiac arrest. Surgical embolectomy should be avoided in patients who have received CPR. There are few data on percutaneous mechanical thromboembolectomy, but it may be beneficial and may be considered in patients with cardiac arrest resulting from a pulmonary embolism who are not candidates for fibrinolytic therapy.

Ventilation

After restoration of circulation, routine hyperventilation leading to hypocapnia should be avoided to prevent additional cerebral ischaemia.

Controlled oxygenation

There is insufficient clinical evidence to support or refute the use of titrated inspired oxygen content in the early care of cardiac arrest patients following sustained ROSC.

Support of circulation

Fluid therapy. There is insufficient evidence to support or refute the routine use of IV fluids following sustained ROSC after cardiac arrest. Rapid infusion with cold 0.9% saline or lactated Ringer's solution appears to be well-tolerated when used to induce therapeutic hypothermia. On the basis of the pathophysiology of post-cardiac arrest syndrome, it is reasonable to use IV fluids as part of a package of post-cardiac arrest care.

Haemodynamic optimisation. There are no published randomised controlled trials of early haemodynamic optimisation after cardiac arrest. Despite limited clinical data, the known pathophysiology of post-cardiac arrest syndrome provides a rationale for titrating haemodynamic support to optimise organ perfusion.

Cardioactive drugs. No clinical trials have determined or compared the independent effect of vasopressor or inotrope use in the post-cardiac arrest period on cardiovascular dysfunction and survival to discharge. There is insufficient evidence to support or refute the routine use of vasopressors and inotropes for improving survival in adult patients with cardiovascular dysfunction after resuscitation from cardiac arrest.

Antiarrhythmic drugs. No controlled studies have specifically addressed the use of amiodarone, lidocaine, or β-blockers early or immediately after resuscitation from cardiac arrest. There is no evidence to support or refute continued administration of amiodarone or lidocaine in post-cardiac arrest patients following ROSC.

Mechanical circulatory support. There are no studies directly addressing the use of mechanical circulatory support in patients with sustained ROSC who have cardiovascular dysfunction.

Temperature control

Prevention and treatment of hyperthermia. There are no randomised controlled trials evaluating the effect of treatment of pyrexia (defined as $\geq$37.6 °C) compared with no temperature control in patients after cardiac arrest. However, it is well-established that patients who develop hyperthermia after cardiac arrest have a worse prognosis. Despite the lack of evidence, it is reasonable to treat hyperthermia if it occurs in the postresuscitation period.

Therapeutic hypothermia. Adult patients who are comatose (not responding in a meaningful way to verbal commands) with spontaneous circulation after out-of-hospital VF cardiac arrest should be cooled to 32–34 °C for 12–24 h. Induced hypothermia might also benefit comatose adult patients with spontaneous circulation after OHCA from a nonshockable rhythm or in-hospital cardiac arrest. Rapid infusion of ice-cold IV fluid at 30 mL kg^{-1} is a safe, feasible, and simple method for initially lowering core temperature by up to 1.5 °C, as is application of ice packs. When IV fluids are used to induce hypothermia, additional cooling strategies will be required to maintain hypothermia. Limited available evidence suggests that PCI during therapeutic hypothermia is feasible and safe and may be associated with improved outcome.

Seizure control

No controlled clinical trials directly addressed prophylactic treatment for seizures after cardiac arrest; consequently, there are insufficient data to support or refute the use of specific antiseizure medication in the prevention or treatment of seizures after ROSC.

Other supportive therapies

Blood glucose control. Strategies to treat hyperglycaemia that is >180 mg dL^{-1} (>10 mmol L^{-1}) should be considered in adult patients with sustained ROSC after cardiac arrest. Hypoglycaemia should be avoided.

Neuroprotective therapy. The value of routine use of coenzyme Q10 in patients treated with hypothermia is not certain. There are insufficient data to recommend for or against the use of neuroprotective drugs (thiopental, glucocorticoids, nimodipine, lidoflazine, or diazepam) alone or as an adjunct to therapeutic hypothermia in comatose cardiac arrest after ROSC.

Prognostication

Prognostication during cardiac arrest

End-tidal CO_2 and prediction of outcome. Quantitative measurement of end-tidal CO_2 may be a safe and effective noninvasive indicator of cardiac output during CPR and an abrupt increase in end-tidal CO_2 may be an early indicator of ROSC in intubated patients. Although low values of end-tidal CO_2 are associated with

a low probability of survival, there are insufficient data to support or refute a specific threshold of end-tidal CO_2 at different time intervals as a prognostic indicator of outcome during adult cardiac arrest.

Prognostication after resuscitation

Clinical examination. There are no clinical neurological signs that reliably predict poor outcome <24 h after cardiac arrest. In adult patients who are comatose after cardiac arrest, have not been treated with hypothermia and have no confounding factors (e.g., hypotension, sedatives or neuromuscular blockers), the absence of both pupillary light and corneal reflex at ≥72 h reliably predicts poor outcome. The absence of vestibulo-ocular reflexes at ≥24 h and a Glasgow Coma Scale (GCS) motor score of 2 or less at ≥72 h are less reliable predictors. Other clinical signs, including myoclonus, are not recommended for predicting poor outcome.

Biochemical markers. Evidence does not support the use of serum or cerebrospinal fluid biomarkers alone as predictors of poor outcome in comatose patients after cardiac arrest with or without treatment with therapeutic hypothermia. Limitations of studies included small numbers of patients or inconsistency in threshold values for predicting poor outcome.

Electrophysiological studies. No electrophysiological study reliably predicts outcome of comatose patients in the first 24 h after cardiac arrest when therapeutic hypothermia is not used. After 24 h, bilateral absence of the N20 cortical response to median nerve stimulation predicts poor outcome in comatose cardiac arrest survivors not treated with therapeutic hypothermia. In the absence of confounding circumstances such as use of sedatives or the presence of hypotension, hypothermia, or hypoxaemia, it is reasonable to use unprocessed electroencephalography (EEG) interpretation (specifically identifying generalised suppression to <20 μV, burst suppression pattern with generalised epileptic activity, or diffuse periodic complexes on a flat background) observed between 24 and 72 h after sustained ROSC to assist in prediction of a poor outcome in comatose survivors of cardiac arrest not treated with hypothermia.

Imaging studies. Many imaging modalities have been studied to determine their utility for prediction of outcome in survivors of adult cardiac arrest. There are no level 1 or level 2 studies that support the use of any imaging modality to predict outcome of comatose cardiac arrest survivors. In general, published imaging studies were limited by small sample sizes, variable time of imaging (many very late in the course), lack of comparison with a standardised method of prognostication, and early withdrawal of care. Despite tremendous potential, neuroimaging has yet to be proven as an independently accurate modality for prediction of outcome in individual comatose cardiac arrest survivors, and at this time there is insufficient evidence to recommend for or against the routine use of imaging studies used for this purpose.

Impact of therapeutic hypothermia on accuracy of post-cardiac arrest prognostication. There is inadequate evidence to recommend a specific approach to prognosticating poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia. There are no clinical neurological signs, electrophysiological studies, biomarkers, or imaging modalities that can reliably predict neurological outcome in the first 24 h after cardiac arrest. Beyond 24 h no single parameter for predicting poor neurological outcome in post-cardiac arrest patients treated with hypothermia is sufficiently specific.

On the basis of the limited available evidence, potentially reliable prognosticators of poor outcome in patients treated with therapeutic hypothermia after cardiac arrest include bilateral absence of N20 peak on somatosensory evoked potential ≥24 h after cardiac arrest, unreactive EEG background at 36–72 h, and the absence of both corneal and pupillary reflexes >72 h after cardiac arrest. Limited available evidence also suggests that (1) a GCS motor score of 2 or less at 3 days after sustained ROSC and (2) the presence of status epilepticus are potentially unreliable prognosticators of poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia. Serum biomarkers such as neuron-specific enolase are potentially valuable as adjunctive studies in prognostication of poor outcome in patients treated with hypothermia, but their reliability is limited by the relatively few patients who have been studied and lack of assay standardisation. Given the limited available evidence, decisions to limit care should not be made based on the results of a single prognostication tool.

Organ donation

Several studies have suggested no difference in functional outcomes of organs transplanted from patients who were determined to be brain-dead as a consequence of cardiac arrest when compared with organs recovered from donors who were brain-dead from other causes.[77–79] Thus, adult patients who progress to brain death after resuscitation from OHCA should be considered for organ donation.

Acute coronary syndromes

The Acute Coronary Syndromes Task Force reviewed the evidence related to the diagnosis and treatment of ACS in the out-of-hospital setting and during the first hours of care in hospital, typically in the emergency department (ED). The ACS Task Force reviewed the following topics: (1) diagnostic tests in ACS, (2) initial therapeutic interventions, (3) reperfusion strategies, (4) additional medical therapy, and (5) healthcare system interventions for ACS.

The following are the most important 2010 changes in recommendations for diagnosis and treatment of ACS.[80,81]

- The history and physical examination, initial ECG, and initial serum biomarkers, even when used in combination, cannot be used to reliably exclude ACS in the prehospital and ED settings.
- In contrast, chest pain observation protocols are useful for identifying patients with suspected ACS who require admission or may be referred for provocative testing for coronary artery disease (CAD) to identify reversible ischaemia. Such strategies also reduce cost by reducing unnecessary hospital admissions and improve patient safety through accurate identification of non-ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).
- The acquisition of a prehospital 12-lead ECG is essential for identification of STEMI patients before hospital arrival and should be used in conjunction with prearrival hospital notification and concurrent activation of the catheterisation laboratory.
- Nonphysicians can be trained to independently interpret 12-lead ECGs to identify patients with STEMI. This skill is of particular value in the prehospital setting, where paramedics can independently identify STEMI, thus reducing the need for ECG transmission, which is not always possible.
- Computer-assisted ECG interpretation can be used to increase diagnostic accuracy of diagnosis for STEMI when used alone or in combination with ECG interpretation by a trained healthcare provider.
- STEMI systems of care can be implemented to improve the time to treatment. The following measures have been shown to reduce

the time to primary PCI (PPCI): institutional commitment, use of a team-based approach, arranging single-call activation of the catheterisation laboratory by the emergency physician or prehospital provider, requiring the catheterisation laboratory to be ready in 20 min, having an experienced cardiologist always available, and providing real-time data feedback.
- IV β-blockers should not be given routinely in the ED or prehospital setting but rather should be reserved for a subset of patients with hypertension or tachycardia in the setting of ACS.
- The routine use of high-flow supplementary oxygen in ACS is not recommended. Instead oxygen administration should be guided by arterial oxyhaemoglobin saturation.
- Reinforce the need for time targets for reperfusion beginning from the time of first medical contact. The clinical circumstances that favor fibrinolysis and PCI are discussed, including the role of prehospital fibrinolytics.
- The prophylactic use of antiarrhythmics is discouraged.
- Immediate angiography and PCI should be considered in patients with OHCA and ROSC. It is reasonable to perform immediate angiography and PCI in selected patients, despite the absence of ST-segment elevation on the ECG or prior clinical findings such as chest pain.

Diagnostic tests in ACS

Risk stratification

Various factors may impede patients from rapidly seeking treatment. These factors include older age, race and ethnicity, female sex, low socioeconomic status, and whether the patient lives alone. Signs and symptoms alone are neither sensitive nor specific and should not be used without other data for diagnosing ACS. Signs and symptoms may be useful in combination with other important information (biomarkers, risk factors, ECG, and other diagnostic tests) in making triage and some treatment and investigational decisions for ACS in the out-of hospital and ED settings. A reduction in chest pain after administration of nitroglycerin may be unrelated to the presence or absence of ACS and should not be used as a diagnostic test or strategy in the prehospital or ED setting.

ED interpretation of 12-lead ECG for STEMI

In patients with suspected ACS, a 12-lead-ECG should be acquired and interpreted by prehospital or emergency providers as soon as possible after first patient contact. The interpretation should be used for diagnosis and triage, including destination decisions and activation of the cardiac catheterisation laboratory. If interpretation of the prehospital ECG is not available on-site, field transmission of the ECG for expert interpretation may be reasonable. It is reasonable for paramedics and nurses to independently identify STEMI on a 12-lead ECG provided there is a program of mandatory initial training followed by ongoing concurrent medical oversight of all interpretations. Prehospital ECG interpretation should be augmented with computer interpretation. Computer interpretation of the ECG may increase the specificity of diagnosis of STEMI, especially for clinicians less experienced in reading ECGs. The computer interpretation should be considered in the clinical context.

Diagnostic and prognostic test characteristics of cardiac biomarkers for ACS

Clinicians should consider the time of symptom onset, sensitivity, precision and institutional norms of the assay, and release kinetics and clearance of the measured biomarker. For all patients presenting to the ED with symptoms suggestive of cardiac ischaemia, cardiac biomarker testing should be part of the initial evaluation. A cardiac-specific troponin is the preferred biomarker. For patients who present within 6 h of onset of symptoms suggestive of cardiac ischaemia with initially negative cardiac troponin, it is recommended that the troponin level be remeasured between 6 and 12 h after symptom onset. Multimarker evaluation with creatine kinase MB (CK-MB) or myoglobin in conjunction with troponin in patients with symptoms suggestive of cardiac ischaemia may be considered to improve the sensitivity of AMI diagnosis. There is no evidence to support the use of troponin point-of-care testing (POCT) in isolation as a primary test in the prehospital setting to evaluate patients with symptoms suggestive of cardiac ischaemia.

There is insufficient evidence to support the use of myoglobin, brain natriuretic peptide (BNP), NT-proBNP, D-dimer, C-reactive protein, ischaemia-modified albumin pregnancy-associated plasma protein A (PAPP-A), or interleukin-6 in isolation as primary tests to evaluate patients with symptoms suggestive of cardiac ischaemia.

None of the currently reported clinical decision rules is adequate and appropriate for identifying ED chest pain patients who can be safely discharged from the ED. Patients who are less than 40 years of age with nonclassical presentations and lacking significant past medical history and normal serial biomarkers and 12-lead ECGs have a very low rate of short-term events.

In ED patients with suspected ACS, normal initial biomarkers, and a nonischaemic ECG, chest pain observation protocols may be recommended as a safe and effective strategy for evaluation. Chest pain observation protocols should include a history and physical examination, a period of observation, serial ECGs, serial measurement of serum cardiac markers, and either an evaluation for anatomic coronary disease or inducible myocardial ischaemia some time after AMI is excluded. These protocols may be used to improve accuracy in differentiating patients requiring inpatient admission or further diagnostic testing from those who may be discharged. Chest pain protocols may be recommended as a means to reduce length of stay, reduce hospital admissions, reduce healthcare costs, improve diagnostic accuracy, and improve quality of life. There is no direct evidence demonstrating that chest pain units (CPUs) or observation protocols reduce adverse cardiovascular outcomes, particularly mortality, for patients presenting with possible ACS, normal serum cardiac biomarkers, and a nondiagnostic ECG.

Imaging techniques

For ED patients with suspected ACS, nonischaemic ECGs, and negative biomarkers, a noninvasive test (CT angiography, cardiac magnetic resonance imaging [MRI], myocardial perfusion imaging, and echocardiography) can be useful in making the diagnosis of ACS. Diagnostic imaging may be considered as an adjunct to serial ECGs and biomarkers to identify patients who either require admission or are suitable for discharge from the ED. These noninvasive tests decrease costs, length of stay, and time to diagnosis and can provide valuable short- and long-term prognostic information on future major cardiac events. However, there are insufficient data on mortality.

Initial therapeutic interventions

Oxygen therapy

There is insufficient evidence to support or refute the empirical use of high-flow oxygen therapy in patients with uncomplicated AMI without signs of hypoxaemia or heart failure. There are insufficient data to determine if high-flow oxygen therapy might be harmful in this setting. Oxygen therapy should be initiated if dyspnoea, hypoxaemia, or signs of heart failure or shock are present. Noninvasive monitoring of arterial blood oxygen saturation may be used to determine the need for oxygen administration.

ACS and nitroglycerin

Although it is reasonable to consider the early administration of nitroglycerin in selected patients without contraindications, insufficient evidence exists to support or refute the routine administration of nitroglycerin in patients with suspected ACS. There may be some benefit if nitroglycerin administration results in pain relief.

Analgesics and sedation

Morphine should be given IV and titrated to pain relief in patients with STEMI. Morphine may be considered for pain relief in subjects with suspected NSTEMI. Some form of analgesia should be considered for patients with active chest discomfort. Although anxiolytics may be administered to patients with ACS to alleviate apprehension and anxiety, there is no evidence that anxiolytics facilitate ECG resolution, reduce infarct size, or decrease mortality in undifferentiated patients with suspected ACS. Lorazepam with nitroglycerin may be considered to alleviate pain in patients with cocaine-associated chest pain. Nonsteroidal anti-inflammatory drugs (NSAIDs) other than aspirin should not be administered and may be harmful in patients with suspected ACS; NSAIDs should be discontinued in such patients when feasible.

Aspirin

In the absence of true allergy, aspirin should be given as soon as possible to patients with suspected ACS. It is reasonable to consider EMS- or dispatcher-guided administration of aspirin by bystanders despite limited direct evidence to support or refute the practice.

Clopidogrel and other platelet ADP-receptor antagonists

Clopidogrel is recommended in addition to standard care (aspirin, anticoagulants, and/or reperfusion) for patients determined to have moderate to high-risk non-ST-elevation ACS and STEMI.

Prasugrel may be administered after angiography to patients with NSTEMI presenting with stenoses amenable to PCI. ED or prehospital administration of clopidogrel should be withheld even in patients who are not at high risk for bleeding pending consideration of prasugrel administration following angiography. In patients who are not at high risk for bleeding with planned PCI and who are determined to have STEMI less than 12 h after initial symptoms, prasugrel may be substituted for clopidogrel. Prasugrel is not recommended for STEMI patients receiving fibrinolysis.

Ticagrelor (not yet approved for administration in some countries) may be given instead of clopidogrel in addition to standard care (aspirin, anticoagulants, and/or reperfusion) to patients determined to have non-ST-elevation ACS or STEMI managed with early invasive strategy by hospital personnel.

Anticoagulants

For patients with non-ST-elevation ACS managed with a planned initial conservative approach, either fondaparinux or enoxaparin are reasonable alternatives to unfractionated heparin (UFH). For patients with non-ST-elevation ACS managed with a planned invasive approach, either enoxaparin or UFH are reasonable choices. Bivalirudin may be considered as an alternative but does not appear to offer an advantage over UFH. Fondaparinux may be used in the setting of PCI but requires coadministration of UFH and does not appear to offer an advantage over UFH alone.

For patients with non-ST-elevation ACS and renal insufficiency, bivalirudin or UFH may be considered. For patients with non-ST-elevation ACS and increased bleeding risk and for whom anticoagulant therapy is not contraindicated, fondaparinux or bivalirudin are reasonable and UFH may be considered.

For patients with STEMI managed with fibrinolysis (including those in the out-of-hospital setting), it is reasonable to administer enoxaparin instead of UFH. Patients initially treated with enoxaparin should not be switched to UFH (or those on UFH should not be switched to enoxaparin) to avoid increased bleeding risk. Fondaparinux may be considered in the hospital for patients treated specifically with nonfibrin-specific thrombolytics (i.e., streptokinase), provided the creatinine level is $<3\ mg\ dL^{-1}$.

For patients with STEMI undergoing contemporary PCI, enoxaparin may be considered a safe and effective alternative to UFH. To avoid increased risk of bleeding, patients initially treated with enoxaparin should not be switched to UFH (and those treated with UFH should not be switched to enoxaparin). In comparison with UFH, fondaparinux reduces risk of bleeding in STEMI patients undergoing PCI. There is an increased risk of catheter thrombi with fondaparinux alone. Bivalirudin may be superior to UFH plus glycoprotein IIb/IIIa blockers with respect to bleeding and mortality in STEMI patients undergoing PCI. An increased rate of stent thromboses has been observed with bivalirudin in the first 24 h after PCI.

Glycoprotein IIb/IIIa Inhibitors

There were insufficient data to support the routine use of glycoprotein IIb/IIIa inhibitors in patients with suspected STEMI or non-ST-elevation ACS in the out-of-hospital or ED settings. For selected high-risk patients with non-ST-elevation ACS, administration of abciximab, eptifibatide, or tirofiban may be acceptable, provided PCI is planned. There is an increased risk of bleeding with routine administration of glycoprotein IIb/IIIa blockers when used with heparins. Alternatives for anticoagulation and antiplatelet treatment might be considered instead.

Reperfusion strategies

In the majority of patients STEMI occurs as the result of a recent acute occlusion of a major epicardial coronary artery due to the disruption of atherosclerotic plaque and thrombus formation. Strategies aimed at restoring myocardial perfusion are an important part of the management of these patients. Restoring coronary blood flow and myocardial perfusion either by pharmacological (fibrinolytics) or mechanical therapy (PCI) has been demonstrated to improve outcomes in patients presenting within 12 h of symptom onset and later in other patient groups, such as those with cardiogenic shock. There is evidence that prehospital fibrinolysis reduces delay to treatment, especially in rural areas with long transit times. In these settings prehospital fibrinolysis is a reasonable treatment strategy.

Prehospital fibrinolysis

In patients with STEMI diagnosed in the out-of-hospital setting, reperfusion may be achieved by healthcare provider administration of fibrinolytics in the field. Alternatively fibrinolytic therapy may be administered on arrival at the hospital. If fibrinolysis is chosen as the reperfusion strategy, it should be started as soon as possible, ideally in the out-of-hospital setting, and should be administered by paramedics, nurses, or doctors under well-established protocols, competency training programs, and programs of continuous quality improvement with medical oversight.

Choice of in-hospital reperfusion strategy

Programs should be implemented to reduce the time to PCI. Shorter intervals to reperfusion increase myocardial salvage, whereas delays to reperfusion increase morbidity and mortality. The precise threshold of PPCI-related delays that should trigger the decision for fibrinolysis has not been definitively established, but time to PCI should be as short as possible. Individual Councils will determine the acceptable limit or target interval from first medical contact to PCI in light of likely patient factors and available

healthcare system resources, and the reader is referred to those Council-specific guidelines for more detailed information.

For patients presenting within 12 h of symptom onset and with ECG findings consistent with STEMI, reperfusion should be initiated as soon as possible, independently of the method chosen. The benefit of mechanical intervention over fibrinolysis varies considerably depending on the patient's condition and the duration of PPCI-related delays.

The precise threshold of PPCI-related delays that should trigger the decision for fibrinolyisis has not been definitively established, and individual Councils will determine the acceptable limits from first medical contact to PCI in light of likely patient factors and local healthcare system variables and resources and the reader is referred to those Council-specific guidelines. For those patients with a contraindication to fibrinolysis, PCI should still be pursued despite the delay, rather than offering no reperfusion therapy.

For those STEMI patients presenting in shock, PCI (or coronary artery bypass surgery) is the preferred reperfusion treatment. Fibrinolysis should only be considered if there is a substantial delay to PCI.

Combined PCI and fibrinolysis

The routine use of fibrinolysis-facilitated PPCI, compared with PPCI alone, is not recommended in patients with suspected STEMI. It is reasonable to perform angiography and possible PCI in patients with failed fibrinolysis according to clinical signs or insufficient ST-segment resolution or both.

Additional medical therapy

Several additional medical therapies have been proposed for ACS patients with the goal of reducing complications from myocardial ischaemia, decreasing major adverse cardiac events, and ultimately increasing long-term survival. Therapeutic options include antiarrhythmics, β-blockers, angiotensin-converting enzyme (ACE) inhibitors, and HMG-CoA reductase inhibitors (statins). Most data regarding the usefulness of these therapies have not been derived from patients in the out-of-hospital or ED settings. Traditional preventive interventions usually start with the first admission with a confirmed diagnosis of ACS. The current evidence indicates that none play a significant role in out-of-hospital and ED management of ACS.

Healthcare system interventions for ACS

Several systems-related strategies have been developed to improve quality of care for patients with ACS and to reduce reperfusion delay for patients with STEMI in the out-of-hospital setting and in the ED. These strategies focus on the use of prehospital 12-lead ECG and time-saving strategies to facilitate early diagnosis and rapid treatment for patients with STEMI.

Out-of-hospital 12-lead ECGs performed by out-of-hospital personnel facilitate earlier diagnosis of STEMI and provide the opportunity for rapid out-of-hospital reperfusion or rapid triage of patients to institutions able to provide such reperfusion. EMS personnel should acquire a 12-lead out-of-hospital ECG for all patients exhibiting signs and symptoms of ACS and provide advance notification to receiving institutions for patients diagnosed with STEMI. Advance notification may be achieved by direct transmission of the ECG or interpretation of the ECG by out-of-hospital personnel and advance notification. Advance notification should prompt preparations at the receiving institution to provide rapid reperfusion for the arriving STEMI patient.

Hospitals should implement out-of-hospital activation of the catheterisation laboratory for patients suspected of having STEMI who arrive by EMS transport, and first-physician contact activation of the catheterisation laboratory for suspected STEMI patients who arrive by other means. Hospitals may implement additional institution-specific techniques to improve systems of care for STEMI; however, there is little evidence to support widespread implementation. These techniques include arranging single-call activation of the catheterisation laboratory, requiring that the catheterisation laboratory be ready in 20 min, having the interventional cardiologist immediately available at the hospital, providing real-time data feedback, fostering senior management commitment, and encouraging a team-based approach.

It is reasonable to consider direct transport to PCI-capable facilities for PPCI for patients diagnosed with STEMI by EMS in the out-of-hospital setting, bypassing closer EDs as necessary, in systems where time intervals between first medical contact and balloon time are brief. In patients presenting early after onset of chest pain (<2 h) and in certain clinical subsets (age <65 years, anterior STEMI), out-of-hospital fibrinolysis may offer similar outcomes compared with PPCI.

In patients with STEMI or new left bundle branch block (LBBB) on ECG following ROSC after OHCA, immediate angiography and PCI should be considered. It is reasonable to perform immediate angiography and PCI in selected patients despite the absence of ST-segment elevation on the ECG or prior clinical findings such as chest pain. Clinical findings of coma before PCI are common in patients with OHCA and are not a contraindication to consideration for immediate angiography and PCI. It is reasonable to include cardiac catheterisation in standardised post-cardiac arrest protocols as part of an overall strategy to improve neurologically intact survival in this patient group. Therapeutic hypothermia is recommended in combination with PPCI and should be started as early as possible, preferably before initiation of PCI.

Paediatric BLS and ALS

The following is a list of changes and issues that required reemphasis in paediatric basic and advanced life support.

Systems

The use of medical emergency teams (MET) or rapid response teams (RRT) has been shown to be effective in preventing respiratory and cardiac arrests in selected paediatric inpatient settings.

Family presence during resuscitations has been shown to be beneficial for the grieving process and in general was not found to be disruptive. Thus, family presence is supported if it does not interfere with the resuscitative effort.

Assessment

Many healthcare providers find it difficult to rapidly and accurately determine the presence or absence of a pulse. On the basis of the available evidence, the task force decided to de-emphasise but not eliminate the pulse check as part of the healthcare provider assessment. Task force members recognised that healthcare providers who work in specialised settings may have enhanced skills in accurate and rapid pulse checks, although this has not been scientifically verified.

There are considerable data on the use of end-tidal CO_2 measurement, capnography, and capnometry during CPR as a measure of CPR quality and as a predictive measure of outcome. Although capnography/capnometry may reflect the quality of CPR, there is insufficient evidence of its reliability in predicting resuscitation success in infants and children.

Airway and ventilation

Opening and maintaining a patent airway and providing ventilations are fundamental elements of paediatric CPR, especially because cardiac arrest often results from, or is complicated by, asphyxia. There are no new data to change the 2005 ILCOR recommendation to use manual airway maneuver (with or without an oropharyngeal airway) and bag-mask ventilation for children who require airway control or positive-pressure ventilation for short periods in the out-of-hospital setting. When airway control or bag-mask ventilation is not effective, placement of a supraglottic airway may be helpful when performed by properly trained personnel.

Data suggest that the routine use of cricoid pressure (Sellick maneuver) when performing tracheal intubation may not protect against aspiration and may make intubation more difficult.

Routine confirmation of tracheal tube position with capnography/capnometry is recommended with the caveat that infants and children in cardiac arrest may have concentrations of exhaled CO_2 below detection limits for colorimetric devices.

After ROSC, toxic oxygen byproducts (reactive oxygen species, free radicals) are produced that may damage cell membranes, proteins, and DNA (reperfusion injury). Although there are no clinical studies in children outside the newborn period comparing different concentrations of inspired oxygen during and immediately after resuscitation, animal data from newborn resuscitation studies suggest that it is prudent to titrate inspired oxygen after return of a perfusing rhythm to prevent hyperoxaemia.

Chest compressions

Chest compression-only CPR is very attractive because it is easier to teach than conventional CPR and immediate chest compressions may be beneficial for resuscitation from sudden death due to VF/pulseless VT. Animal studies showed that conventional CPR, including ventilations and chest compressions, is best for resuscitation from asphyxial cardiac arrest. In a large study of out-of-hospital paediatric cardiac arrest,[38] children with asphyxial arrest who received chest compressions plus ventilations had a significantly better survival than paediatric cardiac arrest victims who were treated with chest compressions alone; the few children with asphyxial arrest who received compression-only CPR had no better outcome than the children who received no CPR.

To be effective, chest compressions must be deep, but it is difficult to determine the optimal depth in infants and children; should it be expressed as a fraction of the depth of the chest or an absolute measurement? How can this be made practical and teachable? After much discussion the task force decided that the best current data support a recommended compression depth of at least one third of the chest anterior–posterior dimension or approximately 4 cm (1.5 in.) in infants and 5 cm (2 in.) in children.

Compression–ventilation ratio in infants

The ILCOR Neonatal Task Force continues to recommend a compression–ventilation ratio of 3:1 for resuscitation of the newly born in the delivery room, with a pause for ventilation whether or not the infant has an advanced tracheal airway in place. The Paediatric Task Force reaffirmed its recommendation for a 15:2 ratio for 2-rescuer infant or child CPR with a pause for ventilation in patients without an advanced airway, and continuous compressions without a pause for ventilation plus a ventilation rate of about 8–10 breaths per minute for patients with an advanced airway. No previous recommendations were made for hospitalised newborns who received care in areas other than the delivery area or when arrest aetiology is primary cardiac rather than asphyxial. For example, consider the case of a 3-week-old infant who has a cardiac arrest after cardiac surgery. In the neonatal intensive care unit such an infant would be resuscitated according to the protocol for the newly born, but if the same newborn were in the paediatric intensive care unit, resuscitation would be performed according to the paediatric (infant/child) protocol. A resolution to this dilemma is suggested on the basis of the arrest aetiology and ease of training.

Vascular access and drug delivery

There is no new evidence to change the 2005 ILCOR recommendations regarding vascular access, including continued emphasis on the early use of intraosseous access and de-emphasis of the tracheal route of drug delivery. Epidemiological data, largely from the National Registry of CPR, reported an association between administration of vasopressin, calcium, or sodium bicarbonate and an increased likelihood of death following in-hospital cardiac arrest. These data, however, cannot be interpreted as establishing a cause-and-effect relationship. The association may be due to the greater likelihood of use of these drugs in children who fail to respond to standard BLS and ALS interventions. These studies and data in adult victims raise questions regarding the benefit of IV medications during resuscitation and reaffirm the emphasis on the performance of high-quality CPR.

Defibrillation

The Paediatric Task Force evaluated a number of issues related to defibrillation, including safe and effective energy dosing, stacked versus single shocks, use of AEDs in infants <1 year of age and paddle/pad type, size, and position. There were a few new human and animal studies on these topics, but the Level of Evidence was generally 3–5.

No new data are available to support a change in drug treatment of recurrent or refractory VT/VF. There were several human and animal publications on defibrillation-energy dosing for VF, but the data were contradictory, and the optimal safe and effective energy dose remains unknown. The new recommendation of an initial dose of 2–4 J kg^{-1} (in 2005, the recommended initial dose was 2 J kg^{-1}) is based on cohort studies showing low success in termination of VF in paediatric patients with 2 J kg^{-1}. However, these studies do not provide data on the success or safety of higher energy doses. The continued recommendation for a single initial shock rather than stacked shocks is extrapolated from the ever-increasing adult data that the long pauses in chest compressions that are required for stacked shocks lower resuscitation success rates, and the initial shock success rate is relatively high with biphasic defibrillation. No changes are recommended in pad/paddle size or position.

Although the safety of AEDs in infants <1 year of age is unknown, case reports have documented successful defibrillations in infants. A manual defibrillator or an AED with paediatric attenuation capabilities is preferred for use in infants and small children.

Emergency medications for arrhythmias

The literature on emergency drug treatment of arrhythmias was reviewed and the only change was the addition of procainamide as therapy for refractory supraventricular tachycardia.

Management of shock

The evidence reviewed was related to several key questions regarding the management of shock in children. There is ongoing uncertainty about the indications for using colloid versus crystalloid in resuscitation from shock. Data from a large adult trial

suggest that effectiveness of normal saline (isotonic crystalloid) is equivalent to albumin, although subgroup analysis suggested harm associated with the use of albumin in patients with traumatic brain injury. There were insufficient data to change the 2005 recommendations.

The optimal timing for tracheal intubation of children in shock remains unclear, although reports in children and adults with septic shock suggest that early intubation (before signs of respiratory failure develop) combined with a protocol-driven management approach may be beneficial. When children in septic shock were treated with a protocol that included therapy directed to normalizing central venous oxygen saturation, patient outcome appeared to improve.

Administering stress-dose corticosteroids in septic shock remains controversial, with recent trials in adults failing to show a beneficial effect.

Performing rapid sequence intubation of a child with shock can result in acute cardiovascular collapse. Etomidate typically causes less haemodynamic compromise than other induction drugs and is therefore often used in this setting. However, data suggest that the use of this drug in children and adults with septic shock is associated with increased mortality that may be secondary to the inhibitory effects of etomidate on corticosteroid synthesis.

Medications for cardiac arrest and bradycardia

The literature on medications used during cardiac arrest and bradycardia was reviewed and updated, but no new recommendations were made. It was again emphasised that calcium and sodium bicarbonate should not be routinely used in paediatric cardiac arrest (i.e., they should not be used without specific indications).

Extracorporeal cardiac life support

There is increasing evidence that extracorporeal cardiac life support (ECLS) can act as a bridge to maintain oxygenation and circulation in selected infants and children who are transplant candidates or who have a self-limited or treatable illness. ECLS can only be used if the cardiac arrest occurs in a monitored environment with protocols and personnel for its rapid initiation.

Post-cardiac arrest care

The literature on the benefit of hypothermia for patients who remain comatose after resuscitation from cardiac arrest was reviewed. There is clear benefit for adult patients who remain comatose after VF arrest, but the evidence is not as strong for infants and young children whose arrest is most commonly asphyxial.

Some patients with sudden death in whom an obvious cause of death is not found have a genetic abnormality of ionic channels, which presumably leads to fatal arrhythmia. Because this is an inherited abnormality, family members might be affected, but special tests are required for detection of this inherited genetic defect.

Special situations

New topics introduced include resuscitation of infants and children with certain congenital cardiac abnormalities, namely single ventricle following stage I procedure and following the Fontan or bidirectional Glenn procedures, as well as resuscitation of infants and children with cardiac arrest and pulmonary hypertension.

Prognosis and decision to terminate CPR

The literature on this important topic was reviewed and the task force concluded that there is insufficient evidence to allow a reliable prediction of success or failure to achieve ROSC or survival from cardiac arrest in infants and children.

Neonatal resuscitation

Since publication of the 2005 Guidelines several controversial neonatal resuscitation issues have been identified. The literature was researched and a consensus was reached on the assessment of oxygenation and role of supplementary oxygen, peripartum management of meconium, ventilation strategies, devices to confirm placement of an advanced airway (e.g., tracheal tube or LMA), medications, maintenance of body temperature, post-cardiac arrest care, and considerations for withholding and discontinuing resuscitation. Educational techniques for teaching, assessing, and maintaining resuscitation knowledge and skills and personnel needed at cesarean sections were also debated. The following are the major new recommendations:

- Progression to the next step after the initial evaluation is now directed by the simultaneous assessment of 2 vital characteristics, heart rate and respirations. The use of a third assessment—that of color is now replaced by oximetry assessment of oxyhaemoglobin saturation.
- For babies born at term, it is best to begin resuscitation with air rather than 100% oxygen.
- Administration of supplementary oxygen should be regulated by blending oxygen and air and the amount delivered to be guided by oximetry.
- The available evidence does not support or refute the routine tracheal suctioning of infants born through meconium-stained amniotic fluid, even when the infant is depressed.
- The compression–ventilation ratio should remain at 3:1 for neonates unless arrest is known to be of cardiac aetiology, in which case a higher ratio should be considered.
- Infants born at term or near term with evolving moderate to severe hypoxic-ischaemic encephalopathy should be offered therapeutic hypothermia, which should be initiated and conducted under clearly-defined protocols with treatment in neonatal intensive care facilities and the capabilities for multidisciplinary care and follow-up.
- It is appropriate to consider discontinuance of resuscitation if there has been no detectable heart rate for 10 min. The decision to continue resuscitation efforts beyond 10 min of no heart rate is often complex and may be influenced by many factors such as the presumed aetiology of the arrest, the gestation of the baby, the presence or absence of complications.
- Cord clamping should be delayed for at least 1 min in babies who do not require resuscitation. Evidence is insufficient to recommend a time for clamping for those who require resuscitation.

Education, implementation, and teams

The Education, Implementation, and Teams Task Force reviewed 5 major topics: (1) education, (2) risks and effects on the rescuer of CPR training and actual CPR performance, (3) rescuer willingness to respond, (4) implementation and teams, and (5) ethics and outcomes.

The key 2010 recommendations related to EIT include

- Efforts to implement new resuscitation guidelines are likely to be more successful if a carefully planned, multifaceted imple-

mentation strategy is used. Education, while essential, is only one element of a comprehensive implementation strategy.
- All courses should be evaluated to ensure that they reliably achieve the program objectives. Training should aim to ensure that learners acquire and retain the skills and knowledge that will enable them to act correctly in an actual cardiac arrest.
- BLS and ALS knowledge and skills can deteriorate in as few as 3–6 months after training. Frequent assessments and, when needed, refresher training is recommended to maintain resuscitation knowledge and skills.
- Short video/computer self-instruction courses, with minimal or no instructor coaching, combined with hands-on practice can be considered as an effective alternative to instructor-led BLS (CPR and AED) courses.
- Laypersons and healthcare providers should be trained to start CPR with chest compressions for adult victims of cardiac arrest. If they are trained to do so, they should also perform ventilations. Performing chest compressions alone is reasonable for trained rescuers if they are incapable of delivering airway and breathing maneuvers to cardiac arrest victims.
- AED use should not be restricted to trained personnel. Allowing the use of AEDs by persons without prior formal training can be beneficial and may be lifesaving. Because even brief training improves performance (e.g., speed of use, correct pad placement), it is recommended that training in the use of AEDs be provided.
- CPR prompt or feedback devices improve CPR skills acquisition and retention and may be considered during CPR training for laypeople and healthcare professionals. These devices may be considered for clinical use as part of an overall strategy to improve the quality of CPR.
- It is reasonable to wear personal protective equipment (e.g., gloves) when performing CPR. CPR should not be delayed or withheld if personal protective equipment is not available unless there is a clear risk to the rescuer.
- Manual chest compressions should not continue during delivery of a shock because safety has not been established.

Education

Effective and efficient resuscitation education is one of the essential elements in the translation of guidelines into clinical practice. Educational interventions need to be population-specific (e.g., lay rescuers, healthcare providers) and evaluated to ensure that they achieve the desired educational outcomes—not just at the end of the course but also during actual resuscitation events. Retention of knowledge and skills should be confirmed through assessment rather than assumption that they persist for preestablished time intervals.

Populations

This section includes who should be trained and how they should prepare for training.

There is insufficient evidence to support or refute the use of training interventions that focus on high-risk populations. Training with social support reduces anxiety in patients and family members, improves emotional adjustment, and increases feelings of empowerment.

ALS courses should incorporate precourse preparation including, but not limited to, use of computer-assisted learning tutorials, written self-instruction materials, video-based learning, textbook reading, and pretests. Any method of precourse preparation aimed at improving knowledge and skills or reducing instructor-to-learner face-to-face time should be formally assessed to ensure equivalent or improved learning outcomes compared with standard instructor-led courses.

Instructional methods

There are multiple methods for delivering course content. This section examines specific instructional methods and strategies that may have an impact on course outcomes. Short video/computer self-instruction (with minimal or no instructor coaching) that includes synchronous hands-on practice in BLS can be considered as an effective alternative to instructor-led courses.

AED use should not be restricted to trained personnel. Allowing the use of AEDs by persons without prior formal training can be beneficial and may be lifesaving. Because even brief training improves performance (e.g., speed of use, correct pad placement), it is recommended that training in the use of AEDs be provided. Laypersons can serve as AED instructors. Short video/computer self-instruction (with minimal or no instructor coaching) that includes synchronous hands-on practice in AED use may be considered as an effective alternative to instructor-led AED courses.

CPR prompt/feedback devices may be considered during CPR training for laypersons and healthcare providers. CPR prompt/feedback devices may be considered for clinical use as part of an overall strategy to improve the quality of CPR. Instructors and rescuers should be made aware that a compressible support surface (e.g., mattress) may cause a feedback device to overestimate depth of compression.

Specific teamwork training, including leadership skills, should be included in ALS courses. There is insufficient evidence to recommend any specific training intervention, compared with traditional lecture/practice sessions, to improve learning, retention, and use of ALS skills.

There is insufficient evidence to recommend teaching a specific technique to optimise complete chest recoil during actual CPR.

There is insufficient evidence to support or refute the use of more realistic techniques (e.g., high-fidelity manikins, in situ training) to improve outcomes (e.g., skills performance on a manikin, skills performance in a real arrest, willingness to perform) when compared with standard training (e.g., low-fidelity manikins, education center) in BLS and ALS courses.

Course format and duration

Resuscitation training courses vary widely in their duration and delivery of content. It is reasonable to consider shortening the duration of traditional instructor-led BLS courses. Brief reassessment (e.g., at 6 months) should be considered to improve skills and retention. The optimal duration of an instructor-led BLS course has not been determined. New course formats should be assessed to ensure that they achieve their objectives. There is insufficient evidence to support or refute alternative ALS course scheduling formats compared with the traditional 2-day provider course format.

Retraining intervals

It is recognised that knowledge and skills retention decline within weeks after initial resuscitation training. Refresher training is invariably required to maintain knowledge and skills; however, the optimal frequency for refresher training is unclear. For BLS providers (laypersons and healthcare providers), skills assessment and, if required, a skills refresher should be undertaken more often than the current commonly recommended training interval of 12–24 months. For ALS providers, there should be more frequent assessment of skills performance or refresher training or both than is currently recommended in established ALS programs. There is insufficient evidence to recommend an optimal interval and form of assessment or refresher training.

Assessment

A written test in an ALS course should not be used as a substitute for demonstration of clinical skills performance. Summative

assessment at the end of ALS training should be considered as a strategy to improve learning outcomes. There is insufficient evidence to recommend an optimal method of assessment during life support training.

Risks and effects on the rescuer of CPR training and actual CPR performance

The safety of rescuers is essential during training and actual CPR performance.

Physical effects

CPR training and actual performance is safe in most circumstances. Learners and rescuers should consider personal and environmental risks before starting CPR. Learners undertaking CPR training should be advised of the nature and extent of the physical activity required during the training program. Learners who develop significant symptoms (e.g., chest pain, severe shortness of breath) during CPR should be advised to stop. Rescuers who develop significant symptoms during actual CPR should consider stopping CPR.

Rescuer fatigue

When performing chest compressions, if feasible, it is reasonable to consider changing rescuers after about 2 min to prevent rescuer fatigue (demonstrated by deterioration in chest compression quality, in particular, depth of compressions). The change of rescuers performing chest compressions should be done with minimal interruption in compressions.

Risks during defibrillation attempts

The risks associated with defibrillation are less than previously thought. There is insufficient evidence that it is safe for the rescuer to continue manual chest compressions during shock delivery for VF (defibrillation). It is reasonable for rescuers to wear gloves when performing CPR and attempting defibrillation (manual or AED), but resuscitation should not be delayed or withheld if gloves are not available. There is insufficient evidence to make a recommendation regarding the safety of physical contact with a patient during ICD discharge. There is insufficient evidence to make a recommendation about the best method for a rescuer to avoid receiving shocks from an ICD discharge during CPR. Although there are no reports of harm to rescuers, there is insufficient evidence to make a recommendation regarding the safety of defibrillation in wet environments.

Psychological effects

There are few reports of psychological harm to rescuers after they are involved in a resuscitation attempt. There is insufficient evidence to support or refute any recommendation on minimising the incidence of psychological harm to rescuers.

Disease transmission

The risk of disease transmission during training and actual CPR performance is very low. Rescuers should take appropriate safety precautions, especially if a victim is known to have a serious infection (e.g., human immunodeficiency virus [HIV], tuberculosis, hepatitis B virus, or severe acute respiratory syndrome [SARS]).

Rescuer willingness to respond

Increasing the willingness of individuals to respond to a cardiac arrest with early recognition, calling for help, and starting CPR is essential to improve survival rates.

To increase willingness to perform CPR, laypersons should receive training in CPR that includes recognition of gasping or abnormal breathing as a sign of adult cardiac arrest when other signs of life are absent. Laypersons should be trained to start resuscitation with chest compressions in adult and paediatric victims. If unwilling or unable to perform ventilations, rescuers should be instructed to continue compression-only CPR. EMS dispatchers should provide chest compression-only CPR instructions to callers who report adult cardiac arrest and these instructions should include recognition of gasping and abnormal breathing (see Part 5: Adult Basic Life Support, for further information).

Implementation and teams

The best scientific evidence for resuscitation interventions will have little impact on patient outcomes if it is not effectively translated into clinical practice. Successful implementation is dependent on effective educational strategies to ensure that resuscitation providers have the necessary knowledge and skills in combination with the necessary infrastructure and resources. Education itself is only one strategy for implementing changes.

Implementation strategies

Institutions or communities planning to implement complex guidelines, such as therapeutic hypothermia, should consider using a comprehensive, multifaceted approach, including clinical champions; a consensus-building process; multidisciplinary involvement; written protocols; detailed process description; practical logistic support; multimodality, multilevel education; and rapid cycle improvement methods.

Individual and team factors

Individual and team factors affect performance during resuscitation attempts. It is reasonable to use cognitive aids (e.g., checklists) during resuscitation, provided that their use does not delay the start of resuscitative efforts. Aids should be validated using simulation or patient trials both before and after implementation to guide rapid cycle improvement. It is reasonable to recommend the use of briefings and debriefings during both learning and actual clinical activities.

There is insufficient evidence to recommend for or against physicians versus nonphysician providers of ALS during prehospital CPR.

System factors

Implementation of AED programs in public settings should be based on the characteristics of published reports of successful programs in similar settings. Home AED use for high-risk individuals who do not have an ICD has not been shown to change overall survival rates.

Because population (e.g., rates of witnessed arrest) and program (e.g., response time) characteristics affect survival, when implementing an AED program, community and program leaders should consider factors such as location, development of a team with responsibility for monitoring and maintaining the devices, training and retraining programs for those who are likely to use the AED, coordination with the local EMS agency, and identification of a group of paid or volunteer individuals who are committed to providing CPR and using the AED for victims of arrest.

Although extrapolation from randomised and observational studies of systems of care for other acute time-sensitive conditions (trauma, STEMI, stroke) suggests that specialised cardiac arrest centers and systems of care may be effective, there is insufficient direct evidence to recommend for or against their use. There is insufficient evidence to make recommendations supporting or refuting the effectiveness of specific performance measurement interventions to improve processes of care and clinical outcomes in resuscitation systems.

There is insufficient evidence to recommend for or against paediatric or adult basic or advanced level life support training programs in low-income countries. However, there is evidence that emergency medical training programs in neonatal and trauma resuscitation should be considered in these countries. When delivering programs in low-income countries, consideration should be given to local adaptation of training, use of existing and sustainable resources for both care and training, and development of a dedicated local infrastructure.

Recognition and prevention

Patients who have cardiac arrest often have unrecognised or untreated warning signs. This section describes strategies to predict, recognise, and prevent cardiorespiratory arrest, including the role of education.

Children and young adults presenting with characteristic symptoms of arrhythmic syncope should be assessed by a cardiology specialist. The assessment should include an ECG and in most cases an echocardiogram and exercise test. Characteristics of arrhythmic syncope include syncope in the supine position, during or after exercise, with no or only brief prodromal symptoms, repetitive episodes, or in persons with a family history of sudden death. In addition, nonpleuritic chest pain, palpitations associated with syncope, seizures (when resistant to treatment, occurring at night, or precipitated by exercise, syncope, or loud noise), and drowning by a competent swimmer should raise suspicion of increased risk. Systematic evaluation in a clinic specialising in the care of those at risk for sudden cardiac death is recommended in family members of young victims of sudden cardiac death or those with a known cardiac disorder resulting in an increased risk of sudden cardiac death.

In adults admitted to the hospital, there is insufficient evidence to support or refute the use of early warning systems/RRT systems or MET systems (compared with no such systems) to reduce cardiac and respiratory arrests and hospital mortality. However, it is reasonable for hospitals to provide a system of care that includes (1) staff education about the signs of patient deterioration, (2) appropriate and regular monitoring of the patient's vital signs, (3) clear guidance (e.g., via calling criteria or early warning scores) to assist staff in early detection of patient deterioration, (4) a clear, uniform system to call for assistance, and (5) a clinical response to calls for assistance. There is insufficient evidence to identify the best methods for delivery of these components and, based on current evidence, this should be based on local circumstances.

Hospitals should use a system validated for their specific patient population to identify individuals at increased risk of serious clinical deterioration, *respiratory arrest*, or cardiac arrest, both on admission and during hospital stay. There is insufficient evidence to identify specific educational strategies that improve outcomes (e.g., early recognition and rescue of the deteriorating patient at risk of cardiac/respiratory arrest). Educational efforts have a positive impact on knowledge, skills, and attitudes/confidence and increase the frequency of activation of a response and should therefore be considered.

Ethics and outcomes

The decision to start, continue, and terminate resuscitative efforts is based on the balance of the risks, benefits, and burdens these interventions place on patients, family members, and healthcare providers. There are circumstances where resuscitation is inappropriate and should not be provided. These include when there is clear evidence that to start resuscitation would be futile or against the expressed wishes of the patient. Systems should be established to communicate these prospective decisions, and simple algorithms should be developed to assist rescuers in limiting the burden of unnecessary, potentially painful treatments.

Decisions before cardiac arrest

Standardised orders for limitations on life-sustaining treatments (e.g., do not attempt resuscitation [DNAR], physician orders for life-sustaining treatment [POLST]) should be considered to decrease the incidence of futile resuscitation attempts and to ensure that the adult patient's wishes are honored. Instructions should be specific, detailed, transferable across healthcare settings, and easily understood. Processes, protocols, and systems should be developed that fit within local cultural norms and legal limitations to allow providers to honor patient wishes regarding resuscitative efforts.

Termination-of-resuscitation rules

Termination-of-resuscitation rules such as the "BLS termination of resuscitation rule" have been prospectively validated in the out-of-hospital setting for use by paramedics and are recommended to guide termination of out-of-hospital CPR in adults. Other rules for various provider levels, including in-hospital providers, may be helpful to reduce variability in decision making; however, rules should be prospectively validated before implementation.

Quality of life

Part of the decision-making process in deciding for or against the decision to initiate resuscitation is the likelihood of success of the resuscitation attempt and the quality of life that can be expected after discharge from the hospital.

Resuscitation after cardiac arrest produces a good quality of life in most survivors. There is little evidence to suggest that resuscitation leads to a large number of survivors with an unacceptable quality of life. Survivors may experience postarrest problems, including anxiety, depression, posttraumatic stress, and difficulties with cognitive function. Clinicians should be aware of these potential problems, screen for them, and, if found, treat them. Interventional resuscitation studies should be encouraged to include a follow-up evaluation (ideally at least 6 months after the event) that assesses general health-related quality of life with a validated instrument, affective disorder (anxiety and depression), posttraumatic stress disorder, and cognitive function.

Future directions

The science of resuscitation is evolving rapidly. It will not be in the best interests of patients if we wait 5 or more years to inform healthcare professionals of therapeutic advances in this field. ILCOR members will continue to review new science and, when necessary, publish interim advisory statements to update treatment guidelines so that resuscitation practitioners may provide state-of-the-art treatment. Existing gaps in knowledge will be closed only by continuing high-quality research into all facets of CPR.

CoSTR Part 1: Executive Summary Writing Group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/Honoraria	Ownership interest	Consultant/ Advisory Board	Other
Mary Fran Hazinski	Vanderbilt University School of Nursing; University and medical center; Professor; AHA ECC Product Development Senior Science Editor; I receive significant AHA compensation to co-edit, the publication of the 2010 CoSTR and the 2010 AHA Guidelines for CPR and ECC	None	None	None	None	None	None
Jerry P. Nolan	Royal United Hospital NHS Trust; National Health Service Hospital; Consultant in Anaesthesia and Intensive Care	None	None	None	None	None	None
John E. Billi	University of Michigan: Medical School – Professor	None	None	None	None	None	None
Bernd W. Boettiger	Uniklinik Köln – MD, DEAA	None	None	None	None	None	None
Leo Bossaert	University of Antwerp; Professor	None	None	None	None	None	None
Allan R. de Caen	Self-employed Paediatric Intensivist	None	None	None	None	None	[*] Medical expert for Canadian Medical Protective Association
Charles D. Deakin	South Hampton University Hospital	None	None	None	None	None	None
Saul Drajer	Clinica de la Esperanza: General Director of a 130 bed general hospital (Clinica de la Esperanza) located in Buenos Aires, Argentina – General Director	None	None	None	None	None	None
Brian Eigel	American Heart Association Director of Science, ECC Programs	None	None	None	None	None	None
Robert W. Hickey	University of Pittsburgh – Associate Professor	[†] Salary support from NIH for grant examining cyclopentenone prostaglandin effects in ischaemic brain injury	None	None	None	None	[*] 1–2X/year medical malpractice expert

Ian Jacobs	University of Western Australia: Discipline of Emergency Medicine Teaching/Research academic – Professor; American Heart Association – Evaluation of evidence worksheets for C2010 – Work Sheet Expert	†Chief investigator on numerous grants awarded by: (a) National Health and Medical Research Council (b) The Department of Health – Western Australia (c) The National Heart Foundation of Australia These funds are awarded to the University of Western Australia and none are used to provide any direct or indirect salary or other financial support	†Funds are received into the Discipline of Emergency Medicine – University of Western Australia from the Ambulance Service – Western Australia and Laerdal (Australia) to maintain the Cardiac Arrest Registry for Western Australia. Our role is to independently maintain, analyze, and report outcomes of CA in Western Australia. I oversee the operation of the registry and reporting of outcomes. These funds are not used to provide any direct or indirect salary or other financial support	None	None	None	None
Monica E. Kleinman	Children's Hospital Anesthesia Foundation, Sr Associate in Critical Care Medicine	None	None	None	None	None	None
Walter Kloeck	Academy of Advanced Life Support: Basic and advanced life support training – Medical Director	None	None	None	None	None	None
Rudolph W. Koster	Academic Medical Center – full-time employee-staff cardiologist	None	†Jolife covers cost of CT scans, autopsy and a 50 Euro per patient contribution to the institution (times 120 patients) †Zoll covers cost of CT scans, autopsy and a 50 Euro per patient contribution to the institution (times 120 patients) *Jolife: Lucas chest compression device on loan to institution for research purposes *Zoll: Autopulse chest compression device on loan to institution for research purposes *Phillips: Chest compression feedback device on loan to institution for research purposes	None	None	None	None
Swee Han Lim	Singapore General Hospital Tertiary Public Hospital; Sr Consultant ED	None	None	None	None	None	None
Mary E. Mancini	University of Texas at Arlington; Professor	None	None	None	None	None	None

CoSTR Part 1 (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/Honoraria	Ownership interest	Consultant/ Advisory Board	Other
William H. Montgomery	Self-employed anesthesiology: Practice of anesthesiology; Self-employed Consultant – Coordinating and planning the AHA C2010 Conference	None	None	None	None	None	None
Peter T. Morley	Royal Melbourne Hospital: Director of Medical Education; University of Melbourne: Clinical Dean, Royal Melbourne Hospital; AHA – Evidence Evaluation Expert	None	None	None	None	None	None
Laurie J. Morrison	St. Michaels; clinician scientist	*Laerdal Foundation Centre Grant – infrastructure support without salary support	None	None	None	None	None
Vinay M. Nadkarni	University of Pennsylvania School of Medicine, Children's Hospital of Philadelphia – Non-profit, Academic, University Hospital – Attending Physician, Anesthesia, Critical Care and Paediatrics	†NIH RO1: Co-Investigator, Therapeutic hypothermia after paediatric cardiac arrest †AHRQ: Agency for Healthcare Research and Quality: PI, Safety of Tracheal Intubation in the Paediatric ICU †Laerdal Foundation for Acute Care Medicine, Center of Excellence Grant, PI; *NHTSA: Co-I, Characteristics of chest compression in children *NIH RO1: Co-I, Neuroprotection during Deep Hypothermic Cardiac Arrest and Cardiopulmonary Bypass	None	None	None	None	None
Robert E. O'Connor	University of Virginia Health System – Professor and Chair of Emergency Medicine	None	None	None	None	None	None
Kazuo Okada	JRC – Administration of JRC President	None	None	None	None	None	None
Jeffrey M. Perlman	Weill Cornell: Professor of Paediatrics	†NIH – Improving antimicrobial prescribing practices in the NICU	None	None	None	None	None
Michael R. Sayre	The Ohio State University – Associate Professor	None	None	None	None	None	None
Michael Shuster	Self-employed – Emergency Physician	None	None	None	None	None	None
Jasmeet Soar	North Bristol NHS Trust – Government Hospital in UK – Consultant in Anaesthetics & Intensive Care Medicine	None	None	None	None	None	None

Kjetil Sunde	Oslo University Hospital Ulleval	None	None	None	None	None	None
Andrew H. Travers	Emergency Health Services, NS Department of Health, Nova Scotia Provincial Medical Director	†Lead Principal Investigator for the Public Access Defibrillation Trial for Edmonton, Alberta, Canada and received grant funding from NHLBI through contracts at the University of Washington which acted as the CRC; Lead Principal Investigator for the Public Access Defibrillation Trial for Edmonton, Alberta, Canada and received grant funding from †NHLBI through contracts at the University of Washington which acted as the CRC	None	None	None	None	None
Jonathan Wyllie	South Tees Foundation NHS Trust Health Service Provider NHS UK Consultant Neonatologist and Clinical Director of Neonatology	None	None	*Spoke for "Trouble Up North" sponsored by Chiesi Other invited lectures for no cost *Will receive small honorarium for speaking at the Middlesbrough Neonatal meeting Honorarium as above from Chiesi	None	*Consultant Advisory Board European Resuscitation Council Board Member and ICC co-chair. Voluntary expenses only Resuscitation Council (UK) *Executive member and Chair of Newborn Life Support Working Group. Voluntary expenses only *HEMS Clinical Governance Group. Voluntary expenses only *North east Ambulance Clinical Governance Group. Voluntary expenses only	*Expert Witness: Occasional case. Nothing recent but one pending report in the UK now
David Zideman	Imperial College NHS Trust: United Kingdom Healthcare Provider – Consultant Anaesthetist; London Organising Committee of the Olympic Games – Lead Clinician for Emergency Medical Services	None	None	None	None	None	*Expert witness for Her Majesty's Coroner for Surrey – expert advice on cardiac arrests under general anesthesia – fee for providing written testimony and personal attendance at court – fee less than 1500 US dollars

* Modest.

† Significant.

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

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Part 2: International collaboration in resuscitation science 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations

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With the founding of the International Liaison Committee on Resuscitation (ILCOR) in 1992, an international collaboration of clinicians and researchers was convened to identify, evaluate, and interpret the most valid resuscitation science. This supplement to *Resuscitation* (simultaneously published in *Circulation*) presents the results of ILCOR's most recent and extensive efforts to reach consensus on interpretation of resuscitation science and treatment recommendations. ILCOR continues to strive to reach a common goal of universal resuscitation guidelines. Building on the *2005 International Consensus on CPR and ECC Science*,[1,2] the 2010 International Consensus Conference held in Dallas, Texas, in February 2010 involved 313 experts from 30 countries. During the 2 years leading up to this conference, over 350 worksheet authors reviewed several thousand relevant, peer-reviewed publications to address more than 400 specific resuscitation questions, each in standard PICO (Population, Intervention, Comparison, Outcome) format. The experts reviewed, summarised, and categorised several thousand relevant, peer-reviewed publications into level of evidence grids, proposed consensus on science statements, and where possible, provided evidence-based treatment recommendations.[3] Key knowledge gaps were also identified and documented, with the purpose of stimulating further research in those areas. Ultimately scientific consensus was achieved by continuous discussion and debate in multiple face-to-face meetings and webinars, and finally through focused discussions of the evidence evaluation worksheets during the 3 days of the International Consensus Conference. Particular attention was paid to recognizing applicable advances in resuscitation science, while managing potential conflicts of interest among participants and identifying topics that lacked good evidence to support or change current practice.

The year 2010 marks the 50th Anniversary of cardiopulmonary resuscitation (CPR). The original reports of rescue breathing,[4] closed-chest compressions,[5] and the effective combination of the two[6] created an immediate demand for CPR training and resuscitation guidelines. In 1966, the Institute of Medicine (in the United States) convened the first conference to specifically review available evidence and to recommend standards for CPR and emergency cardiovascular care (ECC) techniques.[7] The American Heart Association (AHA) sponsored subsequent conferences in 1973 and 1979.[8,9] Parallel efforts occurred internationally as other resuscitation organisations faced a growing demand for CPR training.[10] Inevitably variations in resuscitation techniques and training methods began to emerge from countries and regions of the world.

Increasing awareness of these variations in resuscitation practices sparked interest in gathering international experts at a single location with the aim of achieving consensus in resuscitation techniques. The AHA convened such a meeting in 1985, inviting resuscitation leaders from many countries to observe the process by which the AHA reviewed evidence to create guidelines for CPR and ECC.[11] Observation by these international guests, many of whom were passionately devoted to improving resuscitation outcomes in their own countries, soon led to the realization that much could be learned from international collaboration.

By 1992, when the AHA convened their next Guidelines Conference, more than 40% of the participants were from outside the United States.[12] During this 1992 conference, a panel on international cooperation on CPR and ECC endorsed the need to foster a multinational base of evidence for resuscitation practices. What was lacking was a focused and structured mechanism with which to capture and assess this growing body of published evidence. That panel strongly recommended that an expanded group of international experts initiate a systematic review of the world's resuscitation literature. At a milestone meeting following the European Resuscitation Council's (ERC) first International CPR Conference in Brighton, England, on November 22, 1992, ILCOR was formed.[10] The inaugural cochairs were Douglas Chamberlain and Richard Cummins, and the founding member organisations were the AHA, the ERC, the Heart and Stroke Foundation of Canada, the Resuscitation Council of Southern Africa (RCSA), and the Aus-

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[1] Co-chairs and equal first co-authors.

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tralian Resuscitation Council (ARC). These organisations were later joined by the Consejo Latino-Americano de Resuscitatión (which now forms part of the InterAmerican Heart Foundation), the New Zealand Resuscitation Council (which now forms part of the Australia and New Zealand Committee on Resuscitation [ANZCOR]), and the Resuscitation Council of Asia (RCA).

With the shared vision of international cooperation, ILCOR began to systematically assess the supportive evidence for resuscitation standards and guidelines. During this process, ILCOR experts identified numerous national and regional differences in the practices of basic life support, advanced life support, paediatric life support, newborn resuscitation, acute coronary syndromes as well as in education and implementation. To date, ILCOR has published 22 scientific advisory statements with the goal of endorsing evidence-based resuscitation science that can be adopted by regional councils to formulate resuscitation guidelines.[2,13–33] While some regional differences in guidelines are inevitable because of varying implementation issues or resources, the overarching goal of ILCOR is to provide a unified consensus on the science of resuscitation and the science of resuscitation education/implementation. Compared with the 2005 International Consensus, the 2010 International Consensus Conference paid considerably more attention to the science of education/implementation.

Between 1992 and 2010, ILCOR convened 30 official international meetings, rotating its meeting sites among its member organisations. The goal of ILCOR meetings was to support the evaluation of international science by a dedicated group of experts with the aim of achieving the single best set of evidence-based resuscitation guidelines and practices. This goal defined the international CPR and ECC evidence evaluation conferences held in 2000, 2005, and 2010, as well as several interim international consensus statements.[30,34,35] The 2000 Guidelines Conference,[25,36] the first major assembly under the auspices of ILCOR, adopted a sophisticated process for gathering and assessing evidence; this process evolved further in 2005[37] and was refined for 2010.[3]

The mission statement of ILCOR was updated in 2005: "*ILCOR will provide a mechanism by which the international science and knowledge relevant to CPR and ECC is identified and reviewed. ILCOR will periodically develop and publish a consensus on resuscitation science. When possible, ILCOR will publish treatment recommendations applicable to all member organisations. This consensus mechanism may be used by member organisations to provide consistent guidelines on resuscitation. ILCOR will encourage coordination of guideline development and publication by its member organisations. While the major focus will be on evaluation of CPR and ECC science, ILCOR will also address the effectiveness of education and training, and approaches to the organisation and implementation of emergency cardiovascular care.*"

The goal of a single best set of international CPR and ECC guidelines has not yet been achieved. In general, consensus on resuscitation science has been reached, but local variations in treatment recommendations are inevitable because of differences in epidemiology, models of care, implementation, and cultural or economic factors. These variations will be reflected by some subtle differences in regional and national resuscitation guidelines. Undoubtedly, international cooperation has enabled a more thorough collection and analysis of the scientific evidence, even though that evidence has not always led to standardised training and practice. Reasons for failure to achieve truly universal guidelines include

1. Absence of high-level evidence in the form of randomised controlled clinical trials: For some interventions, such evidence may never be available, because of the nature of resuscitation and its treatment.
2. Inconsistent or contradictory evidence: Ventilation during CPR is one example of this obstacle. Discussion of the optimal timing and provision of oxygen and ventilation in relation to chest compressions consumed considerable time and energy at the 2010 Guidelines Conference. The experts debated numerous ventilation variables and airway devices for field and hospital use by lay rescuers and professionals, respectively. Many resuscitation experts argued that compression-only CPR may be more effective and that perhaps rescue breathing should be eliminated from the initial resuscitation actions of bystanders when they witness sudden cardiac arrest in adults. The need for ventilation during initial resuscitation actions for other groups of patients, e.g., adult cardiac arrest of noncardiac aetiology and child cardiac arrest, remains under debate, generating further consideration on the need and method of ventilation training for lay rescuers.
3. Evolution of resuscitation practice over 50 years: Many of the practices originally recommended were based on the best available evidence and the opinions of experts at the time. Unfortunately this has resulted in the promulgation of some unproven scientific methodologies. The resuscitation experts in ILCOR 2010 had many long discussions about what was acceptable as "grandfather practice" and what should now be removed. These interventions were often supported by minimal, contradictory, or ambiguous evidence. These discussions strengthened the resolve of all involved to continue to encourage research in all aspects of resuscitation.

Failure to translate research findings into daily practice is a well-recognised problem.[38,39] The development of good guidelines does not ensure that they will be adopted in clinical practice, and passive methods of disseminating and implementing guidelines (e.g., publication in journals) are unlikely to change professional behaviour.[40] Recent evidence suggests that full implementation of new resuscitation guidelines can take 18 months to 5 years.[41,42] Resuscitation organisations have a primary responsibility for disseminating and implementing resuscitation guidelines; this will require significant resources. Resuscitation guidelines can be disseminated effectively through the Internet, through national scientific meetings, and by local meetings held in hospitals and in the community. Resuscitation training materials should be updated as rapidly as possible to reflect the new guidelines. Standardised courses play a crucial role in disseminating resuscitation guidelines. Evaluation and verification of the implementation of new guidelines is achieved through audit.

The science of resuscitation is evolving rapidly. It would not be in the best interests of patients if resuscitation experts were to wait five or more years to inform healthcare professionals of therapeutic advances in this field. Some groups have advocated reviewing guidelines as frequently as every 2 years, or even establishing a continuous evidence evaluation process with guideline updates.[43] However, frequent changes in recommendations that do not have a major impact on outcome might undermine the process, because teaching and learning new guidelines takes time and resources. New science must be reviewed continually; if major new research evidence is published, groups such as ILCOR should publish interim consensus advisory statements to update treatment guidelines. Large, multicenter registries that use Utstein-style consensus definitions of the process of care and outcomes following resuscitation will track the dissemination of new techniques and interventions from science to guidelines to practice, and lead to further refinements in the guidelines.[30,44–46] Ideally, important interventions and practices could be taught and reviewed rapidly, giving feedback on quality of performance for all healthcare providers. The interface between resuscitation research and continuous quality improvement (audit) is becoming more blurred.

ILCOR and international collaboration has continued to mature. The quest for a single set of universal guidelines is idealistic: many problems in resuscitation require local modifications and solutions. The common goal of the resuscitation community is to reduce the rates of morbidity and mortality from cardiovascular disease. The consensus statements and treatment recommendations in this publication are based on the most comprehensive review of resuscitation science ever undertaken, and this has been achieved by active and effective international collaboration.

Disclosures.

CoSTR Part 2: writing group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Jerry P. Nolan	Royal United Hospital NHS Trust; National Health Service Hospital; Consultant in Anaesthesia and Intensive Care	None	None	None	None	None	None
Vinay M. Nadkarni	University of Pennsylvania School of Medicine, Children's Hospital of Philadelphia Non-profit, Academic, University Hospital Attending Physician, Anesthesia, Critical Care and Pediatrics	None	None	None	None	None	None
John E. Billi	University of Michigan: Medical School—Professor	None	None	None	None	None	None
Leo Bossaert	University of Antwerp; Professor	None	None	None	None	None	None
Bernd W. Boettiger	Uniklinik Köln: MD, DEAA	None	None	None	None	None	None
Douglas Chamberlain	Honorary Professor, Brighton and Sussex Medical School, University of Sussex	None	None	None	None	None	None
							[a]For several years I had a grant for EXPENSES only from Laerdal Foundation (not the company) to allow me to travel to Cardiff to pursue research interests
Saul Drajer	Clinica de la Esperanza: General Director of a 130 bed general hospital (Clinica de la Esperanza) located in Buenos Aires, Argentina—General Director	None	None	None	None	None	None
Brian Eigel	American Heart Association Director of Science, ECC Programs	None	None	None	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing: UMC Professor; AHA ECC Product Development—Senior Science Editor. I receive significant compensation as co-editor of the 2010 CoSTR and 2010 AHA Guidelines	None	None	None	None	None	None
Robert W. Hickey	University of Pittsburgh: Associate Professor	[b]Salary support from NIH for grant examining cyclopentenone prostaglandin effects in ischemic brain injury	None	None	None	None	[a]1-2X/year medical malpractice expert

Disclosures (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Ian Jacobs	University of Western Australia: Discipline of Emergency Medicine Teaching/Research academic—Professor; American Heart Association: Evaluation of evidence worksheets for C2010—Work Sheet Expert	[b]Chief investigator on numerous grants awarded by: (a) National Health and Medical Research Council (b) The Department of Health – Western Australia (c) The National Heart Foundation of Australia These funds are awarded to the University of Western Australia and none are used to provide any direct or indirect salary or other financial support	[b]Funds are received into the Discipline of Emergency Medicine – University of Western Australia from the Ambulance Service – Western Australia and Laerdal (Australia) to maintain the Cardiac Arrest (CA) Registry for Western Australia. Our role is to independently maintain, analyze and report outcomes of CA in Western Australia. I oversee the operation of the registry and reporting of outcomes. These funds are not used to provide any direct or indirect salary or other financial support	None	None	None	None
Walter Kloeck	Academy of Advanced Life Support: Basic and advanced life support training—Medical Director	None	None	None	None	None	None
William H. Montgomery	Self-employed anesthesiologist: Practice of Anesthesiology—Staff Anesthesiologist; Self Employed Consultant: Paid consultant to AHA—C2010 Conference Coordinator	None	None	None	None	None	None
Peter T. Morley	Royal Melbourne Hospital: Director of Medical Education; University of Melbourne: Clinical Dean, Royal Melbourne Hospital; AHA—Evidence Evaluation Expert	None	None	None	None	None	None
Robert E. O'Connor	University of Virginia Health System: Professor and Chair of Emergency Medicine	None	None	None	None	None	None
Kazuo Okada	Japan Resuscitation Council: President	None	None	None	None	None	None
Michael Shuster	Self-employed: emergency physician	None	None	None	None	None	None
Andrew H. Travers	Emergency Health Services: Nova Scotia Department of Health—Provincial Medical Director	[b]Lead Principal Investigator for the Public Access Defibrillation Trial for Edmonton, Alberta, Canada and received grant funding from NHLBI through contracts at the University of Washington which acted as the CRC	None	None	None	None	None

Disclosures (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
David Zideman	Imperial College NHS Trust: United Kingdom Healthcare provider—Consultant Anaesthetist; London Organising Committee of the Olympic Games: Lead Clinician for Emergency Medical Services	None	None	None	None	None	[a]Expert witness reviews for Her Majesty's Coroner for Surrey on cardiac arrests under anaesthesia – direct fee for written documentation and court attendance – less than 1500 US Dollars

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

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Part 3: Evidence evaluation process
2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations

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Introduction

> "There are known knowns. These are things we know that we know. There are known unknowns. That is to say, there are things that we now know we don't know. But there are also unknown unknowns. These are things we do not know we don't know."
> United States Secretary of Defense Donald Rumsfeld (February 2002)

> "Thought is the wind, knowledge the sail, and mankind the vessel."
> August Hare (1792–1834)

The international resuscitation community, under the auspices of the International Liaison Committee on Resuscitation (ILCOR), has performed an exhaustive review of the published literature related to resuscitation and emergency cardiovascular care (ECC). We had the opportunity to build on the detailed process developed over the past decade that facilitated the publication of the international consensus statement on therapeutic hypothermia[1] and the Consensus on Science and Treatment Recommendations in 2005 (C2005).[2] The overall process has been informed by the increasing evidence base behind good, systematic literature reviews.[3,4] There are, however, many questions that have never been addressed in a randomised controlled trial, and much of the supporting scientific literature is at lower levels of evidence (LOEs). In total, 356 worksheet authors from 29 countries throughout the world completed 411 worksheets on 277 topics. The information from these worksheets, with additional iterations from the writing groups and editorial board, makes up the evidence base from which the 2010 Consensus on Science with Treatment Recommendations (CoSTR) document is derived.

Worksheet template

As has been the process since the evidence evaluation for Guidelines 2000,[5] volunteers from around the world were asked to perform systematic reviews of the literature. These reviews were conducted according to standardised instructions in completing the evidence worksheet template, and these worksheets were subsequently reviewed in a detailed iterative process (see http://www.ILCOR.org).[6] A revised worksheet template (based on that used for the C2005 process) was created and accompanied by detailed instruction documents and an example of a completed worksheet.

Identifying the questions to ask

As with the previous evidence evaluation processes, the specific questions to be asked were informed by priorities identified by task forces and individual councils/organisations, review of the research gaps analysis,[7] and a thorough systematic approach called "evidence mapping" based on the previous guidelines (http://www.evidencemap.org/about).[8]

The questions were allocated to worksheet authors by the relevant task forces, with 2 authors initially allocated to each worksheet. All potential reviewers completed a detailed conflict of interest disclosure form, and worksheet authors were selected to avoid significant conflicts whenever possible (see Part 4).[9] Authors also listed specific potential conflicts of interest on the individual worksheets, thus ensuring transparency of the review process.[9]

It was recognised that not every question could be incorporated, and some areas were therefore not reviewed in the 2010 consensus process. In the absence of a detailed review of all areas, there are, therefore, still some "unknown unknowns."

Formatting the questions (PICO)

The questions for each worksheet topic were structured into a standardised format (PICO: Population/Patient, Intervention, Com-

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parison, Outcome; http://www.cebm.net/?o=1036).[10] This process provided a clear statement about the components of the proposed topic, which facilitated the literature search, guided the accurate allocation of the LOEs, and identified areas of overlap (e.g., in-hospital versus prehospital, during arrest versus after arrest, cardiac arrest versus hemodynamic instability, adult versus paediatric). Outcomes of relevance were documented, but in many cases these could be determined only after the initial review of the retrieved articles.

Clarifying the search strategy

Generic instructions were provided on the types of search strategy to be considered and databases to be searched. The initial search strategy was submitted electronically via the internet for initial review by one of the task force chairs and one of the worksheet experts. This was to ensure that the search appeared to be on track (e.g., addressing the desired question, reasonable combination of search terms), and the search strategy was subsequently returned to the author with comments. The authors were expected to search multiple databases, including the Cochrane database for systematic reviews and the Central Register of Controlled Trials (http://www.cochrane.org/), MEDLINE (http://www.ncbi.nlm.nih.gov/PubMed/), EMBASE (http://www.embase.com), and the master EndNote reference library collated by the American Heart Association (AHA). Many articles were not identified on the initial search but rather through a combination of additional strategies, including a review of the references from and forward searching on key articles.

Selecting studies for further review

The authors were asked to review the titles and abstracts of all articles identified by their preliminary searches and assess the relevance of the articles to the question being asked. The worksheet author then retrieved the appropriate studies for further comparison with the previously developed inclusion and exclusion criteria. This allowed a reproducible final decision on articles to include in the worksheet.[3] Inclusion of all relevant evidence including animal and manikin/model studies, as well as human studies, was encouraged, unless substantive human data were available. Only manuscripts published in full (or accepted for publication) in a peer-reviewed journal were included. Unpublished data or manuscripts published only in abstract form were again excluded.

Allocating levels of evidence

The LOEs used by any review process are a tool to create order and simplicity from the heterogeneity of published studies. There are many published classifications of LOEs. The international resuscitation community used seven LOEs in the preparation of the 2005 CoSTR.[11,12] For the 2010 CoSTR, we reviewed the literature on available classifications (Evidence Report/Technology Assessment No. 47, Systems to Rate the Strength of Scientific Evidence; Agency for Healthcare Research and Quality [AHRQ] publication No. 02-E016, available at: http://www.ahrq.gov/clinic/epcsums/strengthsum.htm)[13,14] and created a simplified list of 5 LOEs.

Worksheet authors were educated about the LOEs, and instruction material was developed and made available on the internet (see http://www.ILCOR.org). Specific LOEs based on the likelihood for bias were developed for therapeutic interventions, diagnostic questions, and prognosis (Table 1). The principles of allocation for studies related to therapeutic interventions were based on the likelihood of eliminating bias in the control group (Fig. 1): true randomisation (LOE 1), concurrent (LOE 2) versus historic (LOE 3) controls, absence of controls (LOE 4), or studies that were related to the worksheet question but that did not directly answer it (LOE 5). LOE 5 studies include studies in related populations (e.g., nonarrest), animal studies (designated in tables with an asterisk), and bench and mathematical models. Systematic reviews and meta-analyses were considered in addition to the original studies. If they added information beyond that of the studies they included,

Table 1
Levels of evidence.

C2010 LOEs for studies of therapeutic interventions
LOE 1: RCTs (or meta-analyses of RCTs)
LOE 2: Studies using concurrent controls without true randomisation (e.g., "pseudo"-randomised)
LOE 3: Studies using retrospective controls
LOE 4: Studies without a control group (e.g., case series)
LOE 5: Studies not directly related to the specific patient/population (e.g., different patient/population, animal models, mechanical models)
C2010 LOEs for prognostic studies
LOE P1: Inception (prospective) cohort studies (or meta-analyses of inception cohort studies), or validation of CDR
LOE P2: Follow-up of untreated control groups in RCTs (or meta-analyses of follow-up studies), or derivation of CDR, or validated on split-sample only
LOE P3: Retrospective cohort studies
LOE P4: Case series
LOE P5: Studies not directly related to the specific patient/population (e.g., different patient/population, animal models, mechanical models)
C2010 LOEs for diagnostic studies
LOE D1: Validating cohort studies (or meta-analyses of validating cohort studies), or validation of CDR
LOE D2: Exploratory cohort study (or meta-analyses of follow-up studies), or derivation of CDR, or a CDR validated on a split-sample only
LOE D3: Diagnostic case-control study
LOE D4: Study of diagnostic yield (no reference standard)
LOE D5: Studies not directly related to the specific patient/population (e.g., different patient/population, animal models, mechanical models)

RCTs indicates randomised controlled trials; CDR, clinical decision rule.

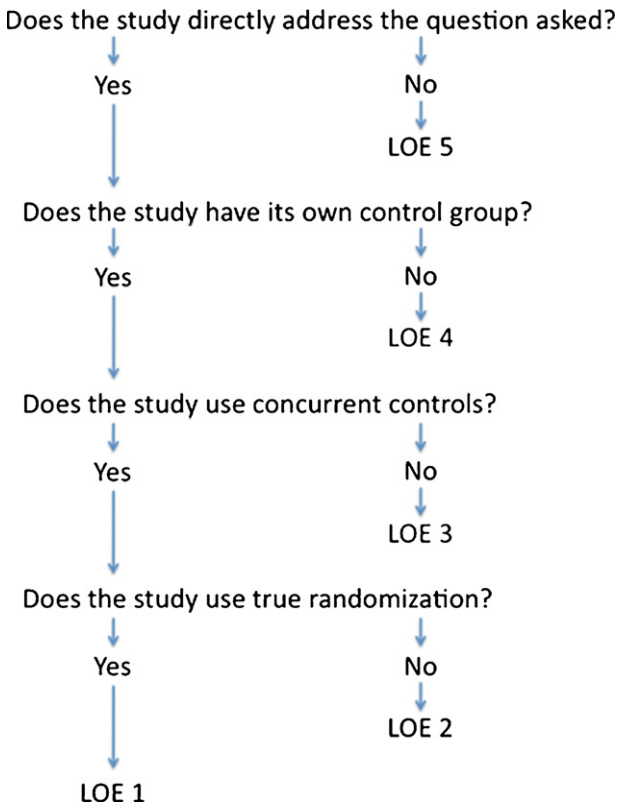

Fig. 1. Decision tree for allocation of LOE (LOE) to therapeutic intervention studies.

Table 2
Quality Factors for LOE 1, LOE P1, and LOE D1.

The 7 factors that were included as the relevant quality items for RCTs (LOE 1): Was the assignment of patients to treatment randomised? Was the randomisation list concealed? Were all patients who entered the trial accounted for at its conclusion? Were the patients analyzed in the groups to which they were randomised? Were patients and clinicians "blinded" to which treatment was being received? Aside from the experimental treatment, were the groups treated equally? Were the groups similar at the start of the trial? Good studies = have most/all of the relevant quality items. Fair studies = have some of the relevant quality items. Poor studies = have few of the relevant quality items (but sufficient value to include for further review).
The 4 factors that were included as the relevant quality items for studies of LOE P1 (as well as P2 and P3): Were comparison groups clearly defined? Were outcomes measured in the same (preferably blinded) objective way in both groups? Were known confounders identified and appropriately controlled for? Was follow-up of patients sufficiently long and complete (e.g., >80%)? For these studies it would be reasonable to consider the presence of all 4 factors=Good, only 3 factors=Fair, and only 2 factors=Poor. A study with only 1 factor would be considered of insufficient quality to include in the next step of the review.
The 3 factors that were included as the relevant quality items for studies of LOE D1 (as well as D2 and D3): Was the diagnostic test evaluated in an appropriate spectrum of patients (e.g., in those in whom it would be used in practice)? (Minimizing "spectrum bias")? Was there an independent, blind comparison with a reference ("gold") standard of diagnosis? (Minimizing "review bias") Was the reference standard applied regardless of the test result? (Minimizing "verification bias") For these studies it would be reasonable to consider the presence of all 3 factors = good, only 2 factors = fair, and only 1 factor = either poor or of insufficient quality to include in the next step of the review.

RCTs indicates randomised controlled trials.

they were allocated the same LOE as that of the studies included in the review (http://www.cebm.net/index.aspx?o=1025)[13]; otherwise, they were allocated LOE 5.

Allocating quality

The process for assessing methodological quality was also reviewed. Many different techniques had been proposed (Evidence Report/Technology Assessment No. 47, Systems to Rate the Strength of Scientific Evidence; AHRQ publication No. 02-E016, available at: http://www.ahrq.gov/clinic/epcsums/strengthsum.htm).[4,13] None of these approaches (largely involving checklists) are appropriate for all settings, and the use of different approaches could result in apparently conflicting results. Several independent factors have been reported to have an impact on the outcome of individual studies, and a modified version based on consensus was created for use.

A list of quality factors was provided for each LOE (1 through 5, including systematic reviews) and for the different types of LOEs (intervention, diagnostic, and prognostic). Three quality terms were defined on the basis of these lists: good, fair, and poor. Studies were designated as "good" if they had most or all of the relevant quality items, "fair" if they had some of the relevant quality items, and "poor" if they had only a few of the relevant quality items but sufficient quality to include for further review (Table 2). Worksheet authors were also asked to comment on the sources of funding for the individual studies, given the concerns about conflict of interest and the association between funding and outcome.[15]

Tabulating the evidence

All of the evidence identified and evaluated was displayed in standardised evidence tables. In each of three tables (supporting evidence, neutral evidence, and opposing evidence), studies were displayed according to LOE, methodological quality, and outcomes identified (Fig. 2).

Interpreting the evidence

The worksheet authors were asked to consider the internal and external validity of each study included and then to summarise the information they reviewed under the section entitled "Reviewer's Final Comments." This is where authors, who had the most intricate understanding of the literature, could succinctly describe the results of their review, start to synthesise the information, tease out the contradictions, make observations, and propose solutions. To facilitate interpretation of the results of the studies identified, the authors were asked to include the magnitude of any differences in the outcomes with an expression of their precision (i.e., 95% confidence interval) whenever possible. Absolute as well as relative changes in proportions were also requested.

The authors formulated draft Consensus on Science (CoS) statements and Treatment Recommendations (TRs) using standard formats.[13,16] Within the treatment recommendations, authors were asked to consider the magnitude of the effect, the outcome affected, the generalizability from the specific population studied, and the potential barriers to implementation (including cost, education, and logistics).[13] The recommended generic format for the CoS statement was:

> Evidence from X# type of study in adults [insert study design and highest-quality design] and X# additional studies [insert range of LOE] document consistent improvement in [insert relevant clinical outcome] when [insert treatment] is administered by [insert provider] to patients with [insert clinical condition] in the [insert prehospital, hospital, etc.] setting.[16]

The generic format for the treatment recommendation statement was:

> Therefore, administration of [therapy] for patients with [condition, setting by personnel] is recommended/should be considered.

Identifying the gaps

Worksheet authors were also asked to identify critical gaps in the literature, which were then incorporated into the final CoSTR document.

Iterative review process

Each worksheet was submitted electronically and underwent a rigorous iterative evaluation process. The layers of review before final acceptance of the worksheet included the task force chair and task force (in addition to a "domain leader" in the advanced life support task force), a worksheet expert, and the evidence evaluation expert. When the worksheet was finalised, it was posted on the Internet for public comment (http://www.americanheart.org/ILCOR; Fig. 3).

In general, multiple worksheets were prepared for each topic, but the authors were encouraged to submit a combined final worksheet whenever possible. The CoS statements and TRs drafted by the worksheet authors were also iteratively reviewed by the relevant task force(s) and writing groups in face-to-face meetings or by Web conferences ("Webinars"). These versions of the CoS and TRs were used to create the draft of the CoSTR document, which was then reviewed by the international councils and the editorial board to create the final manuscript (members of the editorial board are writing group members of Part 1, Executive Summary[17,18]).

Summary of evidence

Evidence Supporting Clinical Question

	1	2	3	4	5
Good	Arrich 2009 CD# Hypothermia After Cardiac Arrest Study Group, 2002 CD* Tiainen, 2003 E*		Bernard, 1997 C, D	Hovdenes, 2007 CD Wolff, 2009 DE Nielsen, 2009 CD	
Fair	Holzer, 2005 CD#	Bernard, 2002 D Holzer, 2006 CD	Knafelj, 2007 CD Busch, 2006 C Belliard, 2007 CD Oddo, 2006 D Sunde, 2007 CD Storm, 2008 CDE Don, 2009 CD Bro-Jeppesen, 2009 D	Oksanen, 2007 C Sagalyn, 2009 #	
Poor	Hachimi-Idrissi, 2001 E Cheung, 2006 CD#	Arrich, 2007 CD	Castrejon, 2009 D	Williams, 1958 D	
Level of evidence					

A = Return of spontaneous circulation
B = Survival of event
* = overlapping patients
C = Survival to hospital discharge
D = Intact neurological survival
E = Other endpoint
# = meta-analysis

Fig. 2. Example of supportive evidence grid for therapeutic hypothermia worksheet.

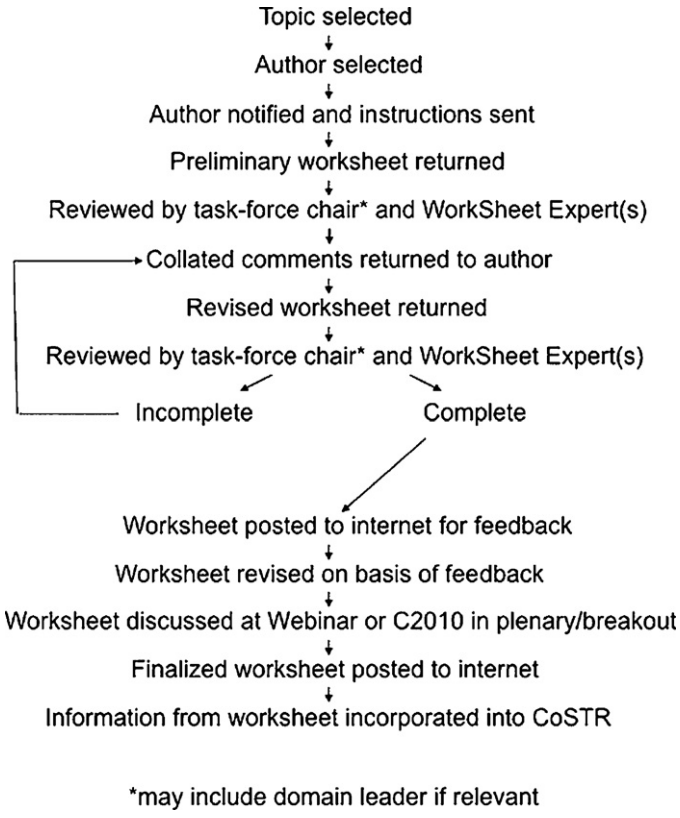

Fig. 3. Worksheet flow for C2010 process.

Controversies in the evidence evaluation process

Four main issues arose from the evidence evaluation process. First, a recurrent theme was the allocation of an LOE based on study type rather than whether the study addressed the specific PICO question. Randomised controlled trials involving a therapeutic intervention in patients who were not in cardiac arrest were often initially allocated an incorrect LOE. If the worksheet PICO question specified the population as patients who were in cardiac arrest, then all randomised controlled studies in nonarrest patients were allocated as "Good LOE 5" rather than LOE 1. Second, there was confusion about the classification of studies that looked retrospectively at data (e.g., registry studies). If the population was appropriate for the worksheet (PICO) question, and the appropriate intervention was compared with a control group from the same time period, these studies were classified as having concurrent controls (LOE 2). The methodological quality allocation was based on the quality items for LOE 2. The third issue related to the choice of LOEs (intervention, diagnosis, or prognosis). The precise wording of the PICO question for the worksheet determined the correct LOE allocation. The comparison of techniques with the outcome to diagnose or predict was graded using diagnostic or prognostic LOEs, respectively (refer, again, to Table 1). If instead the question related to a diagnostic or prognostic tool compared with another tool or standard therapy, and its effect on an outcome (e.g., return of spontaneous circulation) was evaluated, it was considered an intervention and was graded with intervention LOEs.

The final issue relates to the definition of outcomes for educational interventions. For the purposes of the worksheets, the worksheet authors customised outcomes (e.g., E1 = improved score on written test, E2 = skill retention at 6 months). There is a clear need to use a well-defined hierarchy of educational outcomes.

Summary

The C2010 evidence evaluation process used the best evidence on critical appraisal to develop a unique process to incorporate all the peer-reviewed, published science underpinning resuscitation and emergency cardiovascular care. A detailed systematic, multilayered, iterative review of the individual topics (worksheets) has informed the final product: the 2010 CoSTR.

Disclosures.

CoSTR Part 3: writing group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Peter T. Morley	Royal Melbourne Hospital: Director of Medical Education; University of Melbourne – Clinical Dean, Royal Melbourne Hospital; AHA – Evidence Evaluation Expert	None	None	None	None	None	None
Dianne L. Atkins	University of Iowa: University and Medical School – Professor. Compensated worsksheet editor for the Guidelines 2010 Process. Money is paid approximately 2/3 to my institution and 1/3 to directly me. My salary from my institution is not changed by this reimbursement	None	None	None	None	None	[a]Serving as defense expert witness in a case involving a cardiac arrest in a young child who had ventricular fibrillation. Money is paid directly to me. Law firm is Buckly and Theroux in Princeton, NJ.
John E. Billi	University of Michigan: Medical School – Professor	None	None	None	None	None	None
Leo Bossaert	University of Antwerp; Professor	None	None	None	None	None	None
Clifton W. Callaway	University of Pittsburgh: Associate Professor, UPMC; Evidence evaluation expert for AHA	[b]Grants to University of Pittsburgh: NHLBI – Resuscitation Outcomes Consortium HRSA – Development and Dissemination of Program Tools for Uncontrolled Donation After Cardiac Death (UDCD)	[b]Loan of an Arctic Sun cooling device (without disposables) to human physiology laboratory for experiments on hypothermia by Medivance, Inc.	None	[b]Co-inventor on patent about ventricular fibrillation waveform analysis, licensed by University of Pittsburgh to Medtronic ERS, Inc.	None	None
Allan R. de Caen	Self-employed: Paediatric Intensivist	None	None	None	None	None	[a]Expert witness for the Canadian Medical Protective Association
Charles D. Deakin	South Hampton University Hospital	None	None	None	None	None	None
Brian Eigel	American Heart Association Director of Science, ECC Programs	None	None	None	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing. Professor. I'm an AHA consultant t I'm compensated to co-edit the international consensus on CPR and ECC Science with Treatment and to co-edit the AHA Guidelines on CPR and ECC	None	None	None	None	None	None

Disclosures (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Robert W. Hickey	CHP	[b]NIH – funded study examining the role of cyclopentenones in hypoxic ischemic brain injury	None	None	None	None	[a]Expert witness 1–2 per year
Ian Jacobs	University of Western Australia: Discipline of Emergency Medicine Teaching/Research academic – Professor; American Heart Association – Work Sheet Expert	[b]Chief investigator on numerous grants awarded by: (a) National Health and Medical Research Council (b) The Department of Health - Western Australia (c) The National Heart Foundation of Australia These funds are awarded to the University of Western Australia and none are used to provide any direct or indirect salary or other financial support	[b]Funds are received into the Discipline of Emergency Medicine – University of Western Australia from the Ambulance Service –Western Australia and Laerdal (Australia) to maintain the Cardiac Arrest Registry for Western Australia. Our role is to independently maintain, analyze, and report outcomes of cardiac arrest in Western Australia. I oversee the operation of the registry and reporting of outcomes. These funds are not used in any way to provide any direct or indirect salary or other financial support	None	None	None	None
Monica E. Kleinman	Children's Hospital Anesthesia Foundation: Senior Associate in Critical Care Medicine	None	None	None	None	None	None

Disclosures (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Rudolph W. Koster	Academic Medical Center: Staff cardiologist-Full time employee	None	[b]Jolife covers cost of CT scans, autopsy and a 50 Euro per patient contribution to the institution (times 120 patients) [b]Zoll covers cost of CT scans, autopsy and a 50 Euro per patient contribution to the institution (times 120 patients); [a]Jolife: Lucas chest compression device on loan to institution for research purposes [a]Zoll: Autopulse chest compression device on loan to institution for research purposes [a]Philps: chest compression feedback device on loan to institution for research purposes	None	None	None	None
Mary E. Mancini	University of Texas at Arlington University: Professor	None	None	None	None	None	None
William H. Montgomery	Self-employed anesthesiologist; AHA consultant-Conference: C2010 Conference Coordinator	None	None	None	None	None	None
Laurie J. Morrison	St. Michaels; clinician scientist	[a]Laerdal Foundation Centre Grant – infrastructure support without salary support	None	None	None	None	None
Vinay M. Nadkarni	University of Pennsylvania School of Medicine; attending physician	[b]Laerdal Foundation for Acute Care Medicine: Paediatric Cardiac Arrest Learning Laboratory PI [a]NIH: THAPCA (Therapeutic Hypothermia After Paediatric Cardiac Arrest) [a]NIH: NHTSA: Thoracic Compliance in Paediatric Cardiac Arrest and Car Crashes	None	None	None	None	Expert Witness: Cardiac Arrest Facts

Disclosures (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Jerry P. Nolan	Royal United Hospital NHS Trust: Consultant in Anaesthesia and Critical Care	None	None	None	None	None	None
Robert E. O'Connor	University of Virginia Health System: Professor and Chair of Emergency Medicine	None	None	None	None	None	None
Jeffrey M. Perlman	Weill Cornell: Professor of Paediatrics	[b] NIH – Improving antimicrobial prescribing practices in the NICU	None	None	None	None	None
Michael R. Sayre	The Ohio State University: Associate Professor	None	None	None	None	None	None
Tanya I. Semenko	American Heart Association: Science Publications Manager	None	None	None	None	None	None
Michael Shuster	Self-employed physician	None	None	None	None	None	None
Jasmeet Soar	North Bristol NHS Trust: Government Hospital in UK –Consultant in Anaesthetics & Intensive Care Medicine	None	None	None	None	None	None
Jonathan Wyllie	South Tees Foundation NHS Trust Health Service Provider NHS UK Consultant Neonatologist and Clinical Director of Neonatology	None	None	[a]Spoke for "Trouble Up North" sponsored by Chiesi Other invited lectures for no cost [a]Will receive small honorarium for speaking at the Middlesbrough Neonatal meeting Honorarium as above from Chiesi	None	[a]European Resuscitation Council Board Member and ICC co-chair. Voluntary expenses only [a]Resuscitation Council (UK) Executive member and Chair of Newborn Life Support Working group. Voluntary expenses only [a]HEMS Clinical Governance Group. Voluntary expenses only [a]North east Ambulance Clinical Governance Group. Voluntary expenses only	[a]Occasional Case. Nothing recent but one pending report in the UK now
David Zideman	Imperial College NHS Trust: United Kingdom Healthcare provider – Consultant Anaesthetist; London Organising Committee of the Olympic Games: Lead Clinician for EMS	None	None	None	None	None	[a]Expert witness reviews for Her Majesty's Coroner for Surrey

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be 'modest' if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

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Part 4: Conflict of interest management before, during, and after the 2010 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations

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The International Liaison Committee on Resuscitation (ILCOR, www.ILCOR.org) established a conflict of interest (COI) policy in 2004 to manage actual or potential conflicts of interest in an open and effective manner.[1,2] This article describes the current ILCOR and American Heart Association (AHA) COI policies and their application throughout the 2010 evidence evaluation process. The purpose of the COI policies and procedures is to protect the integrity of the decision-making processes and ILCOR's Consensus on Science and Treatment Recommendations, as well as the integrity of the *2010 AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*. ILCOR and the AHA welcome readers' questions and feedback on this process.

The validity of the ILCOR evidence evaluation process depends on rigorous and objective expert review of published science. Therefore, it is essential that any potential professional conflict of interest be fully disclosed and managed transparently and effectively during the planning and conduct of the evidence evaluation process, especially when concerns are expressed or when issues arise. Many of the world's most qualified scientific experts have professional relationships that could pose a real or perceived conflict of interest. In order to benefit from their knowledge and expertise, it is necessary to manage the way these experts are involved in discussions of topics of potential conflict, and especially to minimise their influence over consensus statements or recommendations in such areas.

ILCOR COI procedures apply to all volunteers and staff working on any aspect of the evidence evaluation process. As host of the 2010 CPR Consensus Conference (C2010), the AHA required all participants to complete an AHA COI disclosure questionnaire and to comply with all current AHA COI policies, including new writing group authorship requirements noted below. Relationships that ended more than 12 months prior to COI submission were considered no longer relevant by current AHA policy.

Summary of COI procedures

Because the 2010 evidence evaluation process has taken place over the past 5 years, COI management for C2010 has been an ongoing, dynamic process; it was modeled on the COI model used successfully in the 2005 CPR Consensus Conference.[2–4] COI for the evidence evaluation process was overseen by co-chairs, who were appointed by the ILCOR Executive Committee in 2007. One of the identified co-chairs served that role for the C2005 process and provided continuity. Improvements in the COI process for C2010 (such as earlier identification of commercial relationships) were based on lessons learned from the C2005 process and were formally adopted by the ILCOR Steering Committee in March 2007 (see Table 1).

From 2005 onward, every participant joining the 2010 evidence evaluation process has completed and submitted an AHA COI disclosure form. Participants are required to update their disclosure form annually or whenever there is a substantive change from prior disclosure. AHA staff verified that a disclosure form was completed by each participant, C2010 attendee, and systematic review (worksheet) author, and they reviewed the disclosures.

ILCOR formed six task forces to review the evidence in key resuscitation areas: basic life support; advanced life support; paediatric life support; neonatal life support; acute coronary syndromes; and education, implementation, and teams. The ILCOR Executive Committee chose task force co-chairs to avoid any potential conflict of interest in the topics addressed by the task force. For one task force, both co-chairs had commercial conflicts in the same area relevant to the task force. ILCOR then created a separate "overlap" task force

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[1] Co-chairs and equal first co-authors.

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Table 1
ILCOR conflict of interest policy and procedures, including procedures for the 2005 Consensus Conference and evidence evaluation process.

This policy will ensure that ILCOR manages real and potential conflicts of interest in an open and effective manner in order to preserve public trust in the integrity of ILCOR's process and products. It is not always possible or prudent to avoid such situations, because experts in a clinical area often have relationships that could pose a real or potential conflict of interest in that area. It is essential that these potential conflicts be disclosed and managed effectively. Disclosure is the mainstay of effective management of potential conflicts of interest. COI procedures apply to all ILCOR delegates, C2005 participants, observers, editors, worksheet experts, worksheet authors, and others working on ILCOR projects.

Each ILCOR participant should follow the procedures listed below:

1. At each meeting in which resuscitation science is discussed, each ILCOR participant must disclose all relationships that could pose a direct or indirect conflict of interest. For most meetings this can be done at the time of introductions. ILCOR will keep written records of these disclosures through the minutes of the meeting. At large meetings speakers will also provide meeting organisers with a COI disclosure form before the meeting. A list of participants and their commercial relationships (commercial entity and type of relationship) will appear in the agenda/program for the meeting.
2. Each ILCOR participant will abstain from any vote in which he/she has a relationship that could pose a direct or indirect conflict of interest. The individual abstaining must leave the room during the vote. Abstentions will be recorded in the minutes.
3. Each ILCOR participant will bring COI concerns or issues to the ILCOR co-chairs for investigation and resolution. If the issue involves a co-chair, the issue will be raised with the other co-chair.
4. Whenever possible, an individual with a substantial relationship to a particular topic or area should not be selected to lead a group or serve as a reviewer (worksheet author) for that topic. ILCOR co-chairs will review disclosures by topic moderators and leaders of any subgroup to ensure that any commercial relationships are understood and that potential conflicts are limited and manageable. This shall not prevent an individual with a substantial relationship regarding a topic from contributing to discussions and deliberations on that topic, provided the individual has disclosed those relationships during that meeting.
5. At least annually each ILCOR participant must complete the COI disclosure form and update it if substantive changes occur. The ILCOR co-chairs will review the forms. Each co-chair will review the other co-chair's form. Difficult issues that cannot be resolved by the co-chairs will be brought to the entire group for discussion and resolution.

Special conflict of interest procedures for 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations (C2005).

1. The formal conference invitation packet for such conferences will include the following COI-related materials:
   a. AHA COI Annual Disclosure Form
   b. AHA ECC COI Policy and Procedures
   c. ILCOR COI Policy and Procedures
   d. ILCOR COI Annual Disclosure Form
2. When returning conference registration forms, worksheet authors, participants, speakers, moderators, and attendees will also submit to the AHA both the ILCOR COI Disclosure Form and the AHA ECC COI Disclosure Form. AHA staff will collate the forms and pursue any missing forms well before the conference. Task Force chairs (BLS, ACLS, and Paediatric Resuscitation) will review forms submitted by their speakers and Task Force members for potential conflicts. Questions or problems will be brought to the ILCOR COI co-chairs for resolution (David Zideman of the European Resuscitation Council and John Billi of the AHA). The AHA and ILCOR will keep written records of all disclosure forms and any actions taken. Failure to provide the disclosure form before the conference will result in the participant not being permitted to register for the conference and being removed from the program.
3. In those instances in which a worksheet author has substantive, relevant conflicts of interest, the Task Force chairs will name another reviewer to review the worksheet, including the draft scientific conclusion. In addition, a Task Force chair or designee without a conflict of interest will write the final summary statement, with attribution.
4. When worksheets are posted on the Internet before the conference, the author's conflict of interest will be listed even if the author's name is not. The website will have a hyperlink from each worksheet to this policy. Those who wish to comment on the worksheets must complete the same COI form so that the worksheet authors will have that information as context for the comment.
5. For the conference, the AHA will print a COI disclosure booklet that contains participants' names, institutions, assigned participant numbers, and the basic details of any relationship that might pose a conflict (name of company and nature of relationship). This booklet will be distributed at the conference, either as a separate booklet, part of the syllabus, or both. The disclosures will also be included on the conference website.
6. During presentations moderators will introduce themselves, state their participant numbers, and disclose potential conflicts of interest. Each presenter will use the standard slide listing his/her potential conflicts of interest at the beginning of the presentation. This procedure will be enforced by the moderators. Presenters will not orally state the relationships listed on the slide but must state their participant numbers so that the audience can refer to the disclosure list. If the presentation does not contain such a slide, the moderator will ask the presenter to state his/her disclosures at the beginning of the presentation (not optimal but necessary).
7. During discussions each moderator will ask the floor speakers to identify themselves, state their participant numbers and institutions/companies, and declare any potential conflicts. Each floor speaker must make this verbal disclosure once during each worksheet topic session.
8. If COI problems arise during the conference, moderators will handle them if possible. If necessary, moderators will refer any problems to the two ILCOR COI co-chairs for resolution. Either may call together the Ad Hoc COI Committee should it be needed. The Ad Hoc COI Committee will be composed of representatives from different councils and areas (paediatric, adult, basic, etc.) and outside experts in COI. If the session moderator believes the session should not continue until the COI issue is resolved, then the moderator should move to the next presentation to allow the ILCOR COI co-chairs to resolve the issue rapidly. After resolution, the topic discussion can resume.
9. A poster at the conference will display the ILCOR and AHA COI policies, including a copy of the booklet listing the disclosures of each conference participant by participant number. This will inform participants about the ILCOR and AHA COI process and allow the opportunity for questions and feedback. The poster will also include the multiple steps taken to manage potential conflicts, including policies, COI review steps, use of two independent worksheet reviewers, the disclosure process before and during the conference, and the Ad Hoc COI Committee.

Endorsed ILCOR Business Meeting.
September 13, 2004.

to handle the areas of conflict and appointed additional co-chairs who had no conflicts in the area of overlap.

Before assigning systematic review topics, ILCOR task force co-chairs reviewed the completed disclosure forms of proposed worksheet authors and avoided assigning worksheets to authors with potential conflicts. Although the COI process focused primarily on avoiding financial and commercial conflicts, worksheet topics were also assigned to minimise intellectual conflicts such as authorship of key studies relevant to the worksheet topic under review. As a second check, each evidence evaluation worksheet included a space for the author to disclose potential conflicts of interest relevant to that worksheet. Worksheets were not accepted unless the COI section was complete. The COI information submitted for each worksheet was reviewed by the task force co-chairs and by the evidence evaluation expert, and potential conflicts that had not been identified based on the pre-worksheet assignment COI disclosure were identified. When potential conflicts were discovered after initial assignment, the worksheet was reassigned to a non-conflicted author. In one case, a worksheet author was discovered to have a potential conflict of interest after completion of the worksheet first draft. A second worksheet author without any potential COI assumed responsibility for completing the worksheet, performed another literature search, and revised the consensus on science and the treatment recommendation sections.

If task force members or assigned worksheet authors received grants from industry within the past 12 months, the task force co-chairs attempted to reassign the author to avoid the conflict. In rare instances it was not feasible to replace a worksheet author who had industry grants. In these situations we investigated the conditions of the grant for details including investigator control of study design, control of data/analysis, lack of publication restrictions, and salary support. We initially planned not to scrutinise grants from non-profit foundations, but in view of the heavy support some foundations receive from industry, we applied these rules to foundation funding as well.

Completed worksheets were electronically posted for public comment from October 2009. Representatives of industry and industry employees were not permitted to participate in the evidence evaluation process. However, to be able to benefit from the expertise of industry representatives who had valuable input to contribute, ILCOR did permit them to provide 'public' comment to the posted worksheets. Those who commented were asked to declare potential COIs, but they were not required to identify themselves. Task Forces evaluated all posted comments on their merits, consistent with open peer review.

At all meetings for the 2010 evidence evaluation process, COI disclosure was required either orally or by simultaneous slide projection (described below), and monitors present in each room ensured compliance with COI procedures. Although potential COI was largely self-identified, all participants were encouraged to request clarification about affiliation or involvement during presentations and discussions. In several instances before and during the C2010 conference, participants raised issues regarding potential conflicts of interest that were clarified by the COI co-chairs, resulting in modification of disclosures and sometimes a change in role. Anyone with a potential conflict of interest was permitted to participate in debate but not in decisions or votes regarding wording of consensus on science and treatment recommendations.

In the 5 years leading up to the conference, COI co-chairs fielded many questions and issues regarding the nature of diverse relationships and whether they posed a potential conflict of interest. The co-chairs maintained written documentation of the issues raised and their resolution, for transparency and to aid consistency. Many participants asked co-chairs for feedback about their personal disclosures. In at least one instance, a participant ended his relationship with commercial entities to eliminate potential conflict, in order to participate fully in the process.

Management of potential COI during the C2010 conference

All participants who attended the C2010 conference were required to submit a COI disclosure within 12 months of the conference; late registrants and those with outdated or missing disclosures were required to complete the COI disclosure when they arrived on-site. Throughout the conference, participants were afforded the opportunity to revise or update their COI disclosure, as appropriate. Five computers dedicated to COI use allowed rapid completion or updating of the online COI form; COI information was then immediately entered into the COI database.

All C2010 participants were asked to bring a laptop computer and were given complimentary wireless Internet access throughout the conference. All conference materials including COI disclosures were available electronically. Participants were directed to access the C2010 COI website listing each attendee's name and institution and the basic details of any declared professional relationship that could pose a potential conflict of interest, categorised by type of relationship (grant, consultant, speakers bureau, stock ownership, intellectual property, other; see disclosures at the end of each part of the 2010 ILCOR Consensus for CPR and ECC Science with Treatment Recommendations. In addition, this information was available in a printed copy present at the moderator's table and the registration desk. As in C2005, each participant was assigned a participant number. Based on pilot testing at the 2009 Orlando ILCOR meeting, we assigned numbers in different ranges based on the participant's COI declaration, so all participants could easily determine whether the participant had declared commercial relationships. Participants were assigned numbers 1–399 if they declared no relationships and 400–599 if they declared relationships. Participants who had not completed COI disclosure in advance were assigned a temporary number (600–700), were required to complete the online COI form during on-site C2010 registration, and were required to make full oral disclosure prior to each comment until their COI slide was updated, usually within 4 h. COI information for each participant was listed numerically on the COI website, and the website was updated daily with new or revised COI disclosure information.

As in C2005, continuous COI disclosure for all speakers (scheduled or unscheduled) was provided without interruption or delay in the proceedings, throughout C2010. All participants who commented during the conference, whether they were a moderator, presenter, panelist, or conference attendee, were required to state their name and participant number prior to speaking. A slide listing the speaker's institution and COI disclosure information was projected on a designated screen for the duration of the speaker's presentation, question, response, or comment. This provided conference participants with immediate and continuous information about any relationships the speaker had that could pose a COI issue.

Questions from the audience, comments, and statements from all moderated sessions were audio recorded for future reference. Speakers' statement of participant number at the beginning of comments made the task of identifying recorded speakers easier and made it possible to assess the impact of potential conflicts of interest from the recordings.

A COI monitor was assigned to each session to ensure that policies were followed and to manage questions or issues arising during the session. The monitors' COI Attestation Forms were reviewed and retained as part of the AHA ECC COI documentation file. Conference participants were repeatedly reminded to raise COI issues with COI monitors, moderators, or COI co-chairs. COI co-chair mobile telephone numbers were printed at the bottom of all COI Attestation Forms. A confidential COI telephone "hotline" was announced and listed in the conference program to enable participants to report issues anonymously if they did not wish to make their comments in person. During the conference any new COI problems or questions that could not be resolved by the session moderators were referred to the COI co-chairs for rapid resolution. If a problem became apparent during a session, moderators were instructed to stop discussion if they could not resolve the problem immediately to enable the COI co-chairs time to resolve the issue. When the problem was resolved, the panel was then permitted to resume the earlier presentation and discussion. An Ad Hoc COI Committee composed of the 2010 Consensus Conference coordinator (William H. Montgomery), conference co-chairs (Robert W. Hickey and David Zideman), and COI co-chairs (John E. Billi and Michael Shuster) was available to deal with any issue the COI co-chairs deemed sufficiently challenging.

Potential COI management issues arising during the C2010 conference

COI co-chairs investigated and recommended resolution for a number of issues that arose during the conference. None of

the COI issues required interruption of discussion or convening of the Ad Hoc COI Committee (each of these events did occur once in C2005).[3,4] No anonymous calls were received on the COI hotline. Six participants voluntarily revised their COI disclosure forms once they observed the comprehensive level of disclosure of their peers or were reminded of relationships that might pose a potential conflict. In one instance, two participants provided information regarding a potentially conflicting, undisclosed relationship of another participant. A COI co-chair investigated each issue, and in both cases, the participant's disclosure form, online list, and slide were updated.

Minor difficulties with the planned COI procedures included difficulty experienced by the projectionist in hearing or understanding the speaker's number, the speaker forgetting to provide the number at the beginning of comments (this occurred less frequently as the conference progressed), occasional slow slide projection, the COI slide not yet being updated, or the speaker failing to highlight an intellectual or commercial relationship directly related to the issue under discussion. All of these minor difficulties occurred infrequently and were handled locally by the moderators or the COI monitor assigned to the session; no breaches of COI policy were observed or reported. One unintended benefit of the simultaneous projection of the COI slide was that the audience always knew who was speaking, something that can be difficult to discern in a large meeting room with floor microphones and a wide variety of native languages among participants.

Management of potential COI issues after the C2010 conference

Writing groups were formed to write the *Consensus on Science and Treatment Recommendations*. All writing group co-chairs and writing group members' COI forms were reviewed by AHA officers to ensure compliance with current AHA writing group COI policies. The chair of each writing group and more than 50% of the members of each writing group were required to be free of any relevant relationship with industry and of any significant intellectual bias or competing organizational relationship. Any chair of a writing group with any relevant COI was asked to step down as a chair.

Overall, the C2010 COI management process worked well. Nonetheless, ILCOR and the AHA continue to revise policies and procedures to enhance transparency and ensure scientific integrity throughout the evaluation of the evidence and development of consensus statements and treatment recommendations. Readers are welcome to provide feedback on any aspect of the ILCOR or AHA COI policies and implementation. Please contact the lead author.[2,3]

[2] ILCOR should be especially sensitive to potential COI issues among persons selected for a leadership role with oversight or responsibility for science review of a particular area or topic. These situations must be reviewed on a case-by-case basis. ILCOR may decide that the risk to the integrity of the process from the individual's relationship is not significant and that the individual still represents the best choice for ILCOR, taking into account the risks and benefits. If a person already playing a leadership role develops or is discovered to have a conflict that poses a significant risk to the integrity or credibility of the ILCOR process, another qualified individual without such a potential conflict should replace the first person. Such a substitution shall not imply any impropriety on the part of anyone but rather indicate a preventive step taken to avoid any perceived or real conflict from endangering the integrity of the process. A position of leadership can include the chair or vice chair of any committee, subcommittee, task force, working group, ad hoc group assigned to work on an issue, evidence panel, or evidence collection process. The fact that such perceived conflicts are usually without any improper intent does not protect the individual, ILCOR, or its work from the potential consequences of inadequate management of such a situation.

[3] In addition to financial relationships, other bases of potential conflicts of interest must be considered, such as in-kind support, intellectual collaboration or intellectual investment in one's own ideas, or a long-term research agenda in which an investigator has invested substantial time. Although these situations will be considered on an ad hoc basis, financial relationships are more likely to adversely affect the credibility of ILCOR and the integrity of its process and products.

Disclosures.

CoSTR Part 4: writing group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ Advisory board	Other
John E. Billi	University of Michigan: Medical School—Professor	None	None	None	None	None	None
Michael Shuster	Self-employed: emergency physician	None	None	None	None	None	None
Leo Bossaert	University of Antwerp; Professor	None	None	None	None	None	None
Alan R. de Caen	Self-employed	None	None	None	None	None	[a]Canadian Medical Protective Association-medical expert
Charles D. Deakin	South Hampton University Hospital	None	None	None	None	None	None
Brian Eigel	American Heart Association—Director of Science, ECC Programs	None	None	None	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing. Professor. I am a compensated consultant to the AHA. I am compensated to co-edit the international consensus on CPR and ECC Science with Treatment and to co-edit the AHA Guidelines on CPR and ECC	None	None	None	None	None	None
Robert W. Hickey	CHP—Attending Physician	[a]NIH—funded study investigating the role of cyclopentenone prostaglandins on hypoxic ischemic brain injury	None	None	None	None	[a]1–2 cases/year expert witness
Ian Jacobs	[b]Funds are received into the Discipline of Emergency Medicine—University of Western Australia from the Ambulance Service –Western Australia and Laerdal (Australia) to maintain the Cardiac Arrest Registry for Western Australia. Our role is to independently maintain, analyze and report outcomes of cardiac arrest in Western Australia. I oversee the operation of the registry and reporting of outcomes. These funds are not used in any way to provide any direct or indirect salary or other financial support	[b]Chief investigator on numerous grants awarded by: (a) National Health and Medical Research Council. (b) The Department of Health—Western Australia. (c) The National Heart Foundation of Australia. These funds are awarded to the University of Western Australia and none are used to provide any direct or indirect salary or other financial support	None	None	None	None	None
Monica E. Kleinman	Children's Hospital Anesthesia Foundation, Sr Associate in Critical Care Medicine	None	None	None	None	None	None
Rudolph W. Koster	Academic Medical Center; cardiologist, researcher	None	None	None	None	None	None
Mary E. Mancini	University of Texas at Arlington, Professor	None	None	None	None	None	None
William H. Montgomery	Self-employed anesthesiologist: staff anesthesiologist; AHA consultant—Conference: C2010 Conference Coordinator	None	None	None	None	None	None

Disclosures (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ Advisory board	Other
Peter T. Morley	Royal Melbourne Hospital: Director of Medical Education; University of Melbourne: Clinical Dean, Royal Melbourne Hospital; AHA—Evidence Evaluation Expert	None	None	None	None	None	None
Laurie J. Morrison	St. Michaels; clinician scientist	[a]Laerdal Foundation Centre Grant—infrastructure support without salary support	None	None	None	None	None
Henrietta Munoz	American Heart Association, Inc.—ECC Attorney	None	None	None	None	None	None
Vinay M. Nadkarni	University of Pennsylvania School of Medicine; attending physician	[b]Laerdal Foundation for Acute Care Medicine: Paediatric Cardiac Arrest Learning Laboratory PI [a]NIH: THAPCA (Therapeutic Hypothermia After Paediatric Cardiac Arrest) [a]NIH: NHTSA: Thoracic Compliance in Paediatric Cardiac Arrest and Car Crashes	None	None	None	None	[b]Cardiac arrest facts
Jerry P. Nolan	Royal United Hospital NHS Trust: Consultant in Anaesthesia and Critical Care	None	None	None	None	None	None
Robert E. O'Connor	University of Virginia Health System: Professor and Chair of Emergency Medicine	None	None	None	None	None	None
Jeffrey M. Perlman	Weill Cornell, Professor	[b]NIH—Improving antimicrobial prescribing practices in the NICU	None	None	None	None	None
Sam Richmond	City Hospital Sunderland	None	None	None	None	None	None
Michael R. Sayre	Self-employed, physician	None	None	None	None	None	None
Jasmeet Soar	North Bristol NHS Trust: Government Hospital in UK—Consultant in Anaesthetics & Intensive Care Medicine	None	None	None	None	None	None
Jonathan Wyllie	South Tees NHS Foundation Trust—Clinical Director of Neonatology, Consultant Neonatologist	None	None	[a]Spoke for "Trouble Up North" sponsored by Chiesi. Other invited lectures for no cost [a]Will receive small honorarium for speaking at the Middlesbrough Neonatal meeting Honorarium as above from Chiesi	None	[a]European Resuscitation Council Board Member and ICC co-chair. Voluntary expenses only [a]Resuscitation Council (UK) Executive member and Chair of Newborn Life Support Working group. Voluntary expenses only [a]HEMS Clinical Governance Group. Voluntary expenses only [a]North east Ambulance Clinical Governance Group. Voluntary expenses only	[a]Expert Witness—Occasional Case. Nothing recent but one pending report in the UK now

Disclosures (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ Advisory board	Other
David Zideman	Imperial College NHS Trust: United Kingdom Healthcare provider—Consultant Anaesthetist; London Organising Committee of the Olympic Games: Lead Clinician for EMS	None	None	None	None	None	[a]Expert witness reviews for Her Majesty's Coroner for Surrey

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

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Part 5: Adult basic life support
2010 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations☆

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The 2010 international evidence evaluation process addressed many questions related to the performance of basic life support. These have been grouped into the following categories: (1) epidemiology and recognition of cardiac arrest, (2) chest compressions, (3) airway and ventilation, (4) compression–ventilation sequence, (5) special circumstances, (6) emergency medical services (EMS) system, and (7) risks to the victim. Defibrillation is discussed separately in Part 6 because it is both a basic and an advanced life support skill. In the following summary, each question specific to the population, intervention, control group, and outcome (PICO Question) is listed with the consensus on science and treatment recommendation.

There have been several important advances in the science of resuscitation since the last ILCOR review in 2005. Not all topics reviewed in 2005 were reviewed in 2010. When evaluating the published science, evidence reviewers considered studies with adult and paediatric victims of cardiac arrest published or accepted for publication in peer-reviewed journals. However, the treatment recommendations in this chapter generally are limited to treatment of adult victims of cardiac arrest. Please see Part 10: "Paediatric Basic and Advanced Life Support" for information on basic life support for paediatric cardiac arrest victims. The following is a summary of the most important evidence-based recommendations for the performance of basic life support in adults:

- Rescuers should begin CPR if the victim is unresponsive and not breathing (ignoring occasional gasps). Gasping should not prevent initiation of CPR because gasping is not normal breathing, and gasping is a sign of cardiac arrest.
- Following initial assessment, rescuers may begin CPR with chest compressions rather than opening the airway and delivering rescue breathing.
- All rescuers, trained or not, should provide chest compressions to victims of cardiac arrest.
- A strong emphasis on delivering high-quality chest compressions remains essential: rescuers should push hard to a depth of at least 2 in. (or 5 cm) at a rate of at least 100 compressions per minute, allow full chest recoil, and minimise interruptions in chest compressions.
- Rescuers trained to provide ventilations use a compression–ventilation ratio of 30:2.
- For untrained rescuers, EMS dispatchers should provide telephone instruction in chest compression-only CPR.

Epidemiology and recognition of cardiac arrest

Many millions of people die prematurely every year from sudden cardiac arrest (SCA) worldwide, often associated with coronary heart disease. The following section summarises the burden, risk factors, and potential interventions to reduce the risk (Table 1).

Epidemiology

Incidence[BLS-014B]

What is the incidence, prevalence, and aetiology of cardiopulmonary arrest in-hospital and out-of-hospital?

Consensus on science

Measuring the global incidence of cardiac arrest is challenging, because there are many different definitions of patient populations. The Table lists the average crude incidence per 100,000 population reported for adult cases of cardiac arrest and cases of all ages (children and adults). The number of studies included is shown for each category.[1–22]

There are no significant differences in the incidence of out-of-hospital cardiac arrest (OHCA) or the incidence of patients in whom resuscitation was attempted with all causes of arrest when com-

☆ Note From the Writing Group: Throughout this article, the reader will notice combinations of superscripted letters and numbers (eg, "Initial Recognition BLS-003A, BLS-003B"). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.

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[1] On behalf of the Adult Basic Life Support Chapter Collaborators.

[2] Co-chairs and equal first co-authors.

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Table 1
Global incidence of cardiac arrest per 100,000 population.

Incidence definition (number of studies)	All ages included		Adult only	
	Mean	(SD)	Mean	(SD)
Incidence of out-of-hospital cardiac arrest ($n = 5$)	82.9	(21.4)	213.1	(177)
Incidence of patients considered for CPR ($n = 34$)	76.3	(35.7)	95.9	(30.5)
Incidence of arrest with CPR initiated ($n = 55$)	41.5	(18.4)	64.2	(19.9)
Incidence of arrest with CPR initiated, cardiac cause ($n = 87$)	40.5	(17.1)	61.8	(37.7)
Adjusted incidence of arrest with CPR initiated, cardiac cause ($n = 14$)	56.6	(13.7)	84.7	(58.8)
Percentage of cases with CPR initiated ($n = 43$)	72.3	(20.4)	68.9	(25.6)
Percentage of cases with cardiac aetiology ($n = 48$)	71.8	(12.4)	72.0	(11.8)

paring Europe, North America, Asia, and Australia. The incidence of patients with OHCA considered for resuscitation is lower in Asia (55 per year per 100,000 population) than in Europe (86), North America (94), and Australia (113). The incidence of patients in OHCA with presumed cardiac cause in whom resuscitation was attempted is higher in North America (58 per year per 100,000 population) than in the other three continents (35 in Europe, 32 in Asia, and 44 in Australia).

For in-hospital cardiac arrest, there are more limited incidence data.[23]

Recognition of cardiac arrest

Early recognition is a key step in the initiation of early treatment of cardiac arrest and relies on using the most accurate method of determining cardiac arrest.

Initial recognition[BLS-003A,BLS-003B]

In adults and children who are unresponsive (out-of-hospital and in-hospital), are there any specific factors (or clinical decision rules) as opposed to standard assessment that increase the likelihood of diagnosing cardiac arrest (as opposed to non-arrest conditions, such as post-seizure, hypoglycaemia or intoxication)?

Consensus on science

Pulse check. There are no studies assessing the accuracy of checking the pulse to detect human cardiac arrest. There have been 9 LOE D5 studies demonstrating that both lay rescuers[24–26] and healthcare providers[27–32] have difficulty mastering the pulse check and remembering how to perform it. Three LOE D5 studies support the ability of healthcare providers to perform the pulse check; two evaluated the direct ear-to-chest method in infants,[33,34] and the third supported an alternative technique for the carotid pulse check when tested by dental students on healthy volunteers.[35] In 1 LOE D5 study,[36] the technique of simultaneous pulse check and breathing check by professional rescuers increased the diagnostic accuracy.

Two LOE D5 studies[32,37] conducted in infants and children with nonpulsatile circulation during extracorporeal membrane oxygenation (ECMO) demonstrated that doctors and nurses in a paediatric tertiary care institution, when blinded to whether the child was receiving ECMO support or not, commonly assessed pulse status inaccurately and often took longer than 10 s. In these paediatric studies, healthcare professionals were able to accurately detect a pulse by palpation only 80% of the time. They mistakenly perceived a pulse when it was nonexistent 14–24% of the time and failed to detect a pulse when present in 21–36% of the assessments. Although some of the children in this study were pulseless, all children had circulation (i.e., none were in cardiac arrest), so other signs typically associated with pulseless arrest (delayed capillary refill, poor color) were absent in this population.

Breathing assessment

Several studies have shown that lay rescuers do not easily master the techniques of breathing assessment, and they are often unable to recognise agonal gasps (LOE D5[25,26,38,39]). There is a high incidence of agonal gasps after cardiac arrest (LOE D4[40–43]), and EMS dispatchers have difficulty in diagnosing agonal gasping.[40]

Several strategies for teaching students how to differentiate agonal gasps from normal breathing have been evaluated. In one LOE D5 study,[44] teaching recognition of agonal gasps using a video clip improved the accuracy of lay rescuers in recognizing cardiac arrest. Another study (LOE D5[45]) demonstrated that detection of true cardiac arrest cases improved after introduction of the question "Is he breathing regularly?" in a seizure complaint question sequence used by EMS dispatchers.

Signs of circulation

In the past, students were taught to recognise cardiac arrest by looking for the absence of signs of circulation, such as movement. No studies were found that measured the sensitivity and specificity of that approach for diagnosing cardiac arrest. An LOE D4 study[46] showed that CPR guidance by EMS dispatchers was impeded by callers mentioning "signs of life."

Treatment recommendation

It is reasonable that lay rescuers and healthcare professionals use the combination of unresponsiveness and absent or abnormal breathing to identify cardiac arrest. Palpation of the pulse as the sole indicator of the presence or absence of cardiac arrest is unreliable. Agonal gasps are common during cardiac arrest and should not be considered normal breathing. The general public and EMS dispatchers should be taught how to recognise agonal gasps as a sign of cardiac arrest.

Aetiology of cardiac arrest[BLS-050A,BLS-050B]

In adults and children with presumed cardiac arrest (out-of-hospital and in-hospital), are there any factors/characteristics that increase the likelihood of differentiating between an SCA (i.e., VF or pulseless ventricular tachycardia [VT]) and other etiologies (e.g., drowning, acute airway obstruction)?

Consensus on science

In 1 registry study (LOE 2[47]), cardiac arrest was more likely to be due to a cardiac cause in victims above the age of 35 years and to a noncardiac cause up to the age of 35 years. Two other registry studies (LOE 3[48,49]) do not demonstrate diagnostically useful cut-off ages. An additional registry study (LOE 2[50]) demonstrated that 83% of cardiac arrests under the age of 19 years are of noncardiac origin. One prospective study (LOE 2[51]) and 1 retrospective study (LOE 3[52]) showed that identification of the cause of cardiac arrest by healthcare providers can be inaccurate, leading to an underesti-

mation of noncardiac aetiology cardiac arrest, in particular, failure to diagnose exsanguination. Additional studies in children are summarised in Part 10: "Paediatric Basic and Advanced Life Support."

Treatment recommendation

For lay rescuers there is insufficient evidence to recommend any diagnostically reliable method to differentiate SCA of cardiac origin from one of noncardiac origin. Except in cases of obvious external causes of cardiac arrest (e.g., gunshot wound, drowning), professional rescuers should rely on rhythm analysis from cardiac monitors or AEDs and other diagnostic tests to determine the cause of cardiac arrest.

Check for circulation during BLS[BLS-008B]

In adults and children with cardiac arrest (out-of-hospital and in-hospital), does the interruption of CPR to check circulation, as opposed to no interruption of CPR, improve outcome (e.g., ROSC, survival)?

Consensus on science

A study in manikins (LOE D5[29]) confirmed a low ability (<50%) of EMS providers to correctly identify the presence of a carotid pulse as an indication to stop further chest compressions. A palpable pulse is usually absent immediately after defibrillation during OHCA (LOE 5[53,54]). AED algorithms that recommend that rescuers check for a pulse immediately after a shock delivery are not useful and will lead to delay in resumption of chest compressions following shock delivery (LOE 5[53–55]). Three LOE D5 studies show that measurement of thoracic impedance through the AED electrode pads may be an indicator of return of circulation.[56–58]

One LOE D5 study in adults[27] and two LOE D5 studies in children with nonpulsatile circulation[32,37] showed that blinded healthcare providers commonly made inaccurate assessments of the presence or absence of a pulse and often took much longer than 10 s. Another study (LOE D5[59]) showed that accurately determining the presence of a pulse took more than 10 s in 95% of cases.

Treatment recommendation

For lay rescuers, interrupting chest compressions to perform a pulse check is not recommended. For healthcare professionals, it is reasonable to check a pulse if an organised rhythm is visible on the monitor at the next rhythm check.

Epidemiology and recognition knowledge gaps

How accurately do rescuers identify cardiac arrest outside of the hospital? Is advanced technology useful to assist with diagnosing cardiac arrest? Which specific factors improve diagnostic accuracy? What is the accuracy of the pulse check performed by healthcare professionals in cardiac arrest patients? Is there an association between the time required to successfully detect a suspected cardiac arrest victim's pulse and resuscitation outcome? Is there a difference in outcome when the decision to start CPR is based on the absence of consciousness and normal breathing as opposed to absence of a pulse?

Chest compressions

Several components of chest compressions can alter effectiveness: hand position, position of the rescuer, position of the victim, and depth and rate of compression and decompression. Evidence for these techniques was reviewed in an attempt to define the optimal compression method.

Chest compression technique

Actual hand position during compressions[BLS-032A,BLS-032B]

In adults and children with cardiac arrest (out-of-hospital and in-hospital), does the use of any specific placement of hands for external chest compressions compared with standard care (e.g., "lower half of the victim's sternum") improve outcome (e.g., ROSC, survival)?

Method to locate hand position[BLS-033A]

In rescuers performing CPR on adults or children with cardiac arrest (out-of-hospital and in-hospital), does the use of any specific method for locating the recommended hand position compared with standard care (e.g., "placement of the rescuer's hands in the middle of the chest") improve outcome (e.g., time to commence CPR, decreased hands-off time, ROSC, survival)?

Consensus on science

No randomised controlled human trials support use of an alternative to the hand position recommended in 2005 ("The rescuer should compress the lower half of the victim's sternum.") when performing external chest compressions for adults or children in cardiac arrest.

In 1 study of CT scans, the internipple line was 3 cm superior to the lower third of the sternum (LOE 5[60]). One LOE 5 study[61] of adult surgical patients demonstrated that if the rescuer's hands are placed on the internipple line, hand deviation to or beyond the xiphisternum occurs in nearly half the cases, sometimes into the epigastrium.

During transesophageal echocardiography of humans receiving chest compressions with placement of the hands on the lower half of the sternum, the area of maximal compression was most often over the base of the left ventricle and the aortic root, a location that potentially impedes forward flow of blood (LOE 4).[62]

In four LOE 5 adult manikin studies,[63–66] locating the recommended hand position for chest compression using the instruction "place hands in centre of the chest" resulted in a significant reduction in hands-off time and no significant reduction in accuracy compared with locating the rib margins and xiphisternum. One LOE 5 adult manikin study[67] showed a similar reduction in hands-off time but also loss of hand-placement accuracy. In a single LOE 5 study using a template of an infant,[68] placing the fingers in the recommended position on the chest (below the internipple line) resulted in placement of fingers over the xiphisternum and abdomen.

Treatment recommendation

For adults receiving chest compressions, it is reasonable for rescuers to place their hands on the lower half of the sternum. It is reasonable to teach this location 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. Use of the internipple line as a landmark for hand placement is not reliable.

Chest compression rate[BLS-032A,BLS-032B]

In adults and children with cardiac arrest (out-of-hospital and in-hospital), does the use of any specific rate for external chest compressions compared with standard care (i.e., approximately 100/min) improve outcome (e.g., ROSC, survival)?

Consensus on science

The number of chest compressions during a certain period (e.g., 1 min) given to cardiac arrest patients is determined by two factors: the time interval between compressions (i.e., the compression rate) and the duration of any interruptions in compressions. One LOE 4 study of in-hospital cardiac arrest patients[69] showed that chest compression rates >80/min were associated with ROSC. An observational study of 506 patients with out-of-hospital cardiac arrest showed improved survival to hospital discharge with increasing chest compression fraction (CCF, i.e., proportion of total resuscitation time during which compressions are delivered), and best results when a CCF >0.60 was achieved. With compression rates between 100 and 127 per minute, this CCF corresponded with >60 chest compressions delivered in each minute. However, there was not an association between compression rate and survival (LOE 4[70]).

Treatment recommendation

It is reasonable for lay rescuers and healthcare providers to perform chest compressions for adults at a rate of at least 100 compressions per minute. There is insufficient evidence to recommend a specific upper limit for compression rate. Pauses should be minimised to maximise the number of compressions delivered per minute.

Chest compression depth[BLS-006A,BLS-006B]

In adults and children with cardiac arrest (out-of-hospital and in-hospital), does any specific compression depth, as opposed to standard care (i.e., depth specified in treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

Three adult human LOE 4 studies[71–73] showed that the measured compression depth during adult human resuscitation is often less than 4 cm (1.5 in.). No human studies directly compared the effectiveness of a compression depth of 4–5 cm (1.5–2 in.) with alternative compression depths.

One adult human LOE 4 case series,[74] two adult human studies with retrospective control groups (LOE 3[75,76]), and one LOE 5 study[77] suggest that compressions of 5 cm (2 in.) or more may improve the success of defibrillation and ROSC. These findings are supported by three swine studies (LOE 5[78–80]) showing improved survival with deeper compression depths and one adult human study (LOE 4[81]) showing that improved force on the chest produced a linear increase in systolic blood pressure. However, one swine study (LOE 5[82]) reported no improvement of myocardial blood flow with increased compression depth from 4 to 5 cm, although coronary perfusion pressure (CPP) improved from 7 to 14 mm Hg.

Treatment recommendation

It is reasonable to compress the sternum at least 2 in./5 cm for all adult cardiac arrest victims. There is insufficient evidence to recommend a specific upper limit for chest compression depth.

Chest decompression[BLS-045A]

In adults and children with cardiac arrest (out-of-hospital and in-hospital), does optimizing chest wall recoil during CPR, compared with standard care, improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no human studies specifically evaluating ROSC or survival to hospital discharge with or without complete chest wall recoil during CPR. One LOE 4 out-of-hospital case series[83] documented a 46% incidence of incomplete chest recoil by professional rescuers using the CPR technique recommended in 2000, and 2 in-hospital paediatric case series demonstrated a 23% incidence of incomplete recoil that was more common just after switching providers of chest compressions (LOE 4[84,85]). Another LOE 4 study[86] electronically recorded chest recoil during in-hospital paediatric cardiac arrests and found that leaning on the chest occurred in half of all chest compressions. Animal studies (LOE 5[87,88]) demonstrate significant reductions in mean arterial pressure, coronary perfusion pressure, cardiac output, and myocardial blood flow with only small amounts of incomplete chest recoil. Chest recoil can be increased significantly with simple techniques; for example, lifting the heel of the hand slightly but completely off the chest during CPR improved chest recoil in a manikin model. However, these alternative techniques may also reduce compression depth (LOE 5[83,89]).

Treatment recommendation

While allowing complete recoil of the chest after each compression may improve circulation, there is insufficient evidence to determine the optimal method to achieve the goal without compromising other aspects of chest compression technique.

Firm surface for chest compressions[BLS-035A,BLS-035B]

For adults or children in cardiac arrest on a bed (out-of-hospital and in-hospital), does the performance of CPR on a hard surface like a backboard or deflatable mattress, compared with performance of CPR on a regular mattress, improve outcome (e.g., ROSC, survival)?

Consensus on science

One case series (LOE 4[90]) and four manikin studies (LOE 5[91–94]) demonstrated that chest compressions performed on a bed are often too shallow. However, the case series (LOE 4[90]) and one of the manikin studies (LOE 5[94]) found that accelerometer-based CPR feedback devices failed to correct for compression of the underlying mattress, so it overestimated actual compression depth and may have contributed to delivery of shallow chest compressions. Two studies using manikins weighted to 70 kg (LOE 5[94,95]) suggested that adequate compressions can be performed on a bed if the immediate feedback mechanism measures actual chest compression, regardless of the presence or absence of a backboard. No studies have examined the risks or benefits of moving the patient from a bed to the floor to perform CPR.

No studies in humans have evaluated the risks or benefits of placing a backboard beneath a patient during CPR. Manikin studies (LOE 5[94,96,97]) suggested that placing a backboard may improve compression depth by a few millimeters. One manikin study (LOE 5[98]) showed that deflating a special mattress improved compression efficiency, but another manikin study (LOE 5[93]) failed to demonstrate any benefit from deflating an air-filled mattress.

Treatment recommendation

CPR should be performed on a firm surface when possible. Air-filled mattresses should be routinely deflated during CPR. There is insufficient evidence for or against the use of backboards during CPR. If a backboard is used, rescuers should minimise delay in initiation of chest compressions, minimise interruption in chest compressions, and take care to avoid dislodging catheters and tubes during backboard placement.

Feedback for chest compression quality[BLS-020A,BLS-020B]

In adults and children in cardiac arrest (out-of-hospital and in-hospital), does the use of feedback regarding the mechanics of CPR quality (e.g., rate and depth of compressions and/or ventilations), compared with no feedback, improve any outcomes (e.g., ROSC, survival)?

Consensus on science

Chest compression frequency, rate, and depth provided by lay responders (LOE 4[99]), hospital teams (LOE 4[71]), and EMS personnel (LOE 4[73,100]) were insufficient when compared with recommended methods. Ventilation rates higher than recommended during CPR will impede venous return (LOE 5).[101]

CPR feedback/prompt devices may improve several discrete measures (ventilation rate, end-tidal CO_2, and compression rate, depth, and chest recoil) that have been linked with CPR quality. Eleven studies investigated the effect of giving real-time CPR performance feedback to rescuers during actual cardiac arrest events in both in-hospital and out-of-hospital settings. Two studies in adults (LOE 2[102,103]) and one study in children (LOE 2[104]) showed improved end-tidal CO_2 measurements and consistent chest compression rates when feedback was provided from audio prompts (metronomes or sirens).

In four LOE 3 studies[75,86,105,106] and two LOE 4 studies,[76,107] real-time feedback from force transducers and accelerometer devices was useful in improving CPR quality metrics, including compression depth, rate, and complete chest recoil. Two manikin studies (LOE 5[90,94]) demonstrated the potential for overestimating compression depth when using an accelerometer chest compression feedback device if compressions are performed (with or without a backboard) on a soft surface. No studies to date have demonstrated a significant improvement in long-term survival related to the use of CPR feedback/prompt devices during actual cardiac arrest events (LOE 3[75]).

In one retrospective analysis of cardiac arrest records and one report of two cases (LOE 4[108,109]), changes in transthoracic impedance were potentially useful to measure ventilation rate and detect esophageal intubation. In a case series (LOE 4[110]), capnography and chest-wall impedance algorithms were inaccurate for determining ventilation rate.

Treatment recommendation

It is reasonable for providers and EMS agencies to monitor and improve the CPR quality, ensuring adherence to recommended compression rate and depth and ventilation rates. Real-time chest compression-sensing and feedback/prompt technology (i.e., visual and auditory prompting devices) may be useful adjuncts during resuscitation efforts. However, rescuers should be aware of the potential overestimation of compression depth when the victim is on a soft surface.

Alternative compression techniques

"Cough" CPR[BLS-017A,BLS-017B,BLS-017C]

In adult cardiac arrest (out-of-hospital and in-hospital), does the use of alternative methods of manual CPR (e.g., cough CPR, precordial thump, fist pacing), compared with standard CPR, improve any outcomes (e.g., ROSC, survival)?

Consensus on science

A few case reports (LOE 4[111–118]) documented limited benefit of cough CPR during the initial seconds to minutes of cardiac arrest in patients who remained conscious in a controlled, monitored setting of electrophysiology testing with patient instruction prior to the onset of anticipated cardiac arrest.

Treatment recommendation

Use of cough CPR may be considered only for patients maintaining consciousness during the initial seconds to minutes of VF or pulseless VT cardiac arrest in a witnessed, monitored, hospital setting (such as a cardiac catheterization laboratory).

Precordial thump[BLS-017A,BLS-017B,BLS-017C]

In adult cardiac arrest (out-of-hospital and in-hospital), does the use of alternative methods of manual CPR (e.g., cough CPR, precordial thump, fist pacing), compared with standard CPR, improve any outcomes (e.g., ROSC, survival)?

Consensus on science

In five prospective case series of out-of-hospital (LOE 4[119–123]) and two series (LOE 4[120,121]) of in-hospital VF cardiac arrest, healthcare provider administration of the precordial thump did not result in ROSC.

In three prospective case series of VT in the electrophysiology laboratory (LOE 4[120,124,125]), administration of the precordial thump by experienced cardiologists was of limited use (1.3% ROSC). When events occurred outside of the electrophysiology laboratory, in six case series of in- and out-of-hospital VT (LOE 4[121–123,126–128]), the precordial thump was followed by ROSC in 19% of patients. Rhythm deterioration following precordial thump occurred in 3% of patients and was observed predominantly in patients with prolonged ischaemia or digitalis-induced toxicity.

In three case series of asystolic arrest (LOE 4[119,122,129]), the precordial thump, but not fist pacing, was sometimes successful in promoting ROSC when administered by healthcare providers to patients with witnessed asystole (some clearly p-wave asystolic arrest) for OHCA and in-hospital cardiac arrest.

Two case series (LOE 4[123,130]) and a case report(LOE 5[131]) documented the potential for complications from use of the precordial thump, including sternal fracture, osteomyelitis, stroke, and rhythm deterioration in adults and children.

Treatment recommendation

The precordial thump is relatively ineffective for VF, and it should not be used for unwitnessed OHCA. The precordial thump may be considered for patients with monitored, unstable VT if a defibrillator is not immediately available. There is insufficient evidence to recommend for or against the use of the precordial thump for witnessed onset of asystole caused by atrioventricular conduction disturbance.

Fist pacing[BLS-017A,BLS-017B,BLS-017C]

In adult cardiac arrest (out-of-hospital and in-hospital), does the use of alternative methods of manual CPR (e.g., cough CPR, precordial thump, fist pacing), compared with standard CPR, improve any outcomes (e.g., ROSC, survival)?

Consensus on science

There is little evidence supporting fist or percussion pacing in cardiac arrest, particularly when the effect of the manoeuvre cannot be confirmed by continuous electrocardiographic (ECG) monitoring and assessment of a pulse. Evidence consists of six single-patient case reports (LOE 4[132–137]) and a moderate-sized case series (LOE 4[138]) with mixed asystole and bradycardia.

Treatment recommendation

For patients in cardiac arrest, percussion (fist) pacing is not recommended.

Chest compression technique knowledge gaps

What is the optimal hand position for maximizing cardiac output? How well is the simple method of teaching hand placement retained? Does a chest compression rate faster than 100/min increase long-term survival from cardiac arrest? What is the minimum number or count of chest compressions to be delivered each

minute to enhance survival? What is the relationship between chest compression rate and depth? Does a chest compression depth greater than 5 cm improve survival? What is the chest compression depth beyond which complications increase? What is the optimal technique to facilitate complete chest recoil and maximise survival? When does use of CPR feedback/prompt devices translate to improvements in survival?

Airway and ventilation

The best method of obtaining an open airway and the optimum frequency and volume of artificial ventilation were reviewed.

Airway

Opening the airway[BLS-011A,BLS-011B]

In adults and children with cardiac arrest (out-of-hospital and in-hospital), does the provision of airway manoeuvres by bystanders, as opposed to no such manoeuvres, improve outcome (e.g., ROSC, survival)?

Consensus on science

Evidence from a case series of drowning victims (LOE 4[139]) and 6 prospective clinical studies in patients under anaesthesia that evaluated clinical (LOE 5[140–142]) or radiological (LOE 5[143–145]) measures of airway patency indicates that the head tilt–chin lift manoeuvre is feasible, safe, and effective. Two prospective clinical studies evaluating clinical (LOE 5[146]) or radiological (LOE 5[147]) measures supported the chin lift manoeuvre in children under anaesthesia, while 3 other prospective clinical studies failed to prove the effect compared to neutral position (LOE 5[148–150]). Of five studies of the effectiveness of the jaw thrust manoeuvre to open the airway of patients who received general anaesthesia, three were supportive (LOE 5[148,151,152]), one was neutral (LOE 5[150]), and one opposed it (LOE 5[153]).

One LOE 5 study in anaesthetised children[154] recommended the jaw lift with the thumb in the mouth. However, three studies have reported harm to the victim (LOE 5[155,156]) or rescuer (LOE 4[139]) from inserting digits into the mouth in attempts to clear the airway.

Treatment recommendation

For unresponsive adults and children, it is reasonable to open the airway using the head tilt–chin lift manoeuvre when assessing breathing or giving ventilations.

Passive ventilation[BLS-009A]

In adults and children with cardiac arrest (out-of-hospital and in-hospital) and receiving chest compression-only CPR, does the addition of any passive ventilation technique (e.g., positioning the body, opening the airway, passive oxygen administration) as opposed to no addition improve outcome (e.g., ROSC, survival)?

Consensus on science

No study was identified that reported outcomes from lay rescuer techniques for airway maintenance and oxygen insufflation during chest compression-only CPR. Furthermore, no study was identified that compared outcomes of any passive airway or ventilation technique with no airway or ventilation technique during chest compression-only CPR. One LOE 2 study[157] failed to show a difference in neurologically intact survival when comparing EMS use of high-flow passive insufflation of oxygen through an oropharyngeal airway with bag-mask ventilation interposed during minimally interrupted chest compressions. Two other studies (LOE 5[158,159]) reported improved survival for OHCA patients receiving minimally interrupted chest compressions by EMS personnel. These studies evaluated variable, nonrandomised use of passive oxygen insufflation by nonrebreather mask or interposed bag-mask ventilation and did not include a control group (i.e., without any airway/ventilation intervention).

Treatment recommendation

For lay rescuers performing chest compression-only CPR, there is insufficient evidence to recommend the use of any specific passive airway manoeuvre or adjunct ventilation device.

Foreign-body airway obstruction[BLS-013A]

Like CPR, relief of foreign-body airway obstruction (FBAO) is an urgent procedure that should be taught to laypersons.[160] Evidence for the safest, most effective, and simplest methods were sought.

In adults and children with FBAO (out-of-hospital and in-hospital), does the provision of abdominal thrusts, and/or back slaps, and/or chest thrusts, compared with no action, improve outcome (e.g., clearance of obstruction, ROSC, survival)?

Consensus on science

Case series and case reports have documented successful relief of FBAO in conscious victims with the use of back blows (LOE 4[161,162]), abdominal thrusts (LOE 4[161–165]), and chest thrusts (LOE 4[161]; LOE 5[166]). More than one technique was occasionally required to relieve the obstruction.

Thirty-two case reports have documented life-threatening complications associated with the use of abdominal thrusts.[160,167] One randomised trial of manoeuvres to clear the airway in cadavers (LOE 5[168]) and two prospective studies in anaesthetised volunteers (LOE 5[166,169]) showed that higher airway pressures could be generated by using the chest thrust rather than the abdominal thrust. In a few case reports, a finger sweep was effective for relieving FBAO in unconscious adults and children aged >1 year (LOE 4[161,162,170]). Case reports documented harm to the victims or biting of the rescuer's finger with finger sweeps (LOE 4[145,171] and LOE 5[155,156,172,173]).

Treatment recommendation

Chest thrusts, back blows, or abdominal thrusts are effective for relieving FBAO in conscious adults and children >1 year of age. These techniques should be applied in rapid sequence until the obstruction is relieved. More than one technique may be needed; there is insufficient evidence to determine which should be used first. The finger sweep may be used in the unconscious patient with an obstructed airway if solid material is visible in the airway. At this time, there is insufficient evidence for a treatment recommendation specific for an obese or pregnant patient with FBAO.

Ventilation

Tidal volumes and ventilation rates[BLS-052B]

In adults in cardiac arrest (out-of-hospital and in-hospital) who are *not* intubated, does providing ventilation with a 1-s inspiratory time and tidal volume of about 600 mL compared with other inspiratory times and tidal volumes improve any outcomes (including ventilation, oxygenation)?

Consensus on science

In three human studies (LOE 5[174–176]), tidal volumes of 600 mL using room air were sufficient to maintain oxygenation and normocarbia in apnoeic patients. When tidal volumes less than 500 mL

were used, supplementary oxygen was needed to achieve satisfactory oxygenation. Three studies of mechanical models (LOE 5[177–179]) found no clinically important difference in tidal volumes when a 1- or 2-s inspiratory time was used. In one human study with eight subjects (LOE 4[180]), expired air resuscitation using tidal volumes of 500–600 mL led to hypoxia and hypercarbia.

Treatment recommendation

For mouth-to-mouth ventilation for adult victims using exhaled air or bag-mask ventilation with room air or oxygen, it is reasonable to give each breath within a 1-s inspiratory time and with an approximate volume of 600 mL to achieve chest rise. It is reasonable to use the same initial tidal volume and rate in patients regardless of the cause of the cardiac arrest.

Airway and ventilation knowledge gaps

What is the effectiveness of airway manoeuvres by bystanders during standard and chest compression-only CPR? What is the optimal ventilation tidal volume in cardiac arrest patients?

Compression–ventilation sequence

In the basic life support/CPR sequence for the lone rescuer, the choice is between starting with airway and breathing (ventilation) or starting with chest compressions. Because of the importance of initiating chest compressions as soon as possible, the need for initial breaths is questioned.

Starting CPR[BLS-026A,BLS-026B]

In adults and children in cardiac arrest (out-of-hospital and in-hospital), does the use of compressions first (30 compressions then 2 breaths) compared with standard care (2 breaths and then 30 compressions) improve outcome (e.g., ROSC, survival)?

Consensus on science

There is no published human or animal evidence to determine whether starting CPR in adults or children with 30 compressions rather than 2 ventilations leads to improved outcomes.

Evidence from one observational, adult manikin LOE 5 study[181] shows that starting with 30 compressions rather than two ventilations leads to a shorter delay to first compression.

Treatment recommendation

For treatment of adult victims of cardiac arrest, starting CPR with chest compressions rather than ventilations may be considered.

Effect of interruptions on delivery of chest compressions

Interruptions to chest compressions during CPR must be minimised. Legitimate reasons for the interruption of CPR include the need to ventilate, the need to assess the rhythm or to assess ROSC, and the need to defibrillate.

Interruption of compressions for post-shock rhythm analysis[BLS-022A,BLS-025A,BLS-025B]

- In patients with VF, will the resumption of chest compressions, compared with delayed initiation for rhythm analysis, result in better outcomes?[BLS-022A]
- In adults and children with cardiac arrest (out-of-hospital and in-hospital), does the minimization of hands-off time for rhythm analysis, including frequency and duration of checks, as opposed to standard care (according to treatment algorithm) improve outcome (e.g., ROSC, survival)?[BLS-025A,BLS-025B]

Consensus on science

In two observational studies (LOE 4[71,73]) and secondary analyses of two randomised trials (LOE 5[53,182]), interruptions of chest compressions were common. Interruption of CPR was associated with a decreased probability of conversion of VF to another rhythm (LOE 5[182]).

In two case series (LOE 4[53,54]), a palpable pulse was rarely present immediately after defibrillation, suggesting that a pulse check after a shock is not useful and delays the resumption of chest compressions. However, in one randomised study (LOE 1[183]), immediate resumption of chest compressions after defibrillation was associated with earlier VF recurrence when compared to a pulse check prior to resumption of CPR; there was no difference in cumulative incidence of VF 60 s after the shock.

Five animal studies (LOE 5[184–188]) and one human study (LOE 5[182]) confirmed that more interruption of chest compressions during CPR reduced ROSC and survival. In two adult out-of-hospital witnessed VF studies (LOE 3[21,55]) and three animal studies (LOE 5[185,188,189]), immediate resumption of chest compressions after defibrillation was associated with better survival rates and/or survival with favourable neurological outcome compared with immediate rhythm analysis and delayed resumption of chest compression. Another LOE 1 randomised study[190] of an AED protocol based on the 2005 Guidelines,[160,167] which included CPR during charging and immediate resumption of chest compressions after shock delivery, did not show significantly improved survival to admission or to discharge.

There is no evidence for or against immediate resumption of chest compressions in adults with VF of short duration.

Treatment recommendations

Rescuers should minimise interruptions of chest compressions during the entire resuscitation attempt.

Use of filtering devices for rhythm analysis during CPR[BLS-039]

In adults and children with cardiac arrest (out-of-hospital and in-hospital), does the analysis of cardiac rhythm during chest compressions compared with standard care (analysis of cardiac rhythm during pauses in chest compressions) optimise the time of appropriate chest compression by avoiding unnecessary interruptions and unnecessary prolongations?

Consensus on science

In six LOE 5 studies[191–196] using human-derived ECG recordings with actual or simulated CPR artifacts and one LOE 5 study in a swine model of VF,[197] the use of computerised algorithms that removed compression artifacts from the ECG during CPR reduced the accuracy of rhythm analysis relative to rhythm analysis during pauses. Sensitivity was between 90% and 98%, which would cause inappropriate prolongations in chest compression for shockable rhythms in up to one out of 10 patients. Specificity was between 80% and 89%, which could result in inappropriate interruptions in chest compression for shock delivery in victims who actually had nonshockable rhythms.

Treatment recommendations

There is insufficient evidence to support or refute the use of artifact-filtering algorithms for analysis of ECG rhythm during CPR.

Compression–ventilation ratio during CPR[BLS-023A,BLS-023B]

Any recommendation for a specific CPR compression–ventilation ratio represents a compromise between the need to generate blood flow and the need to supply oxygen to the lungs and remove carbon dioxide (CO_2) from the blood. At the same time, any such ratio must be taught to would-be rescuers, so the effect of the compression–ventilation ratio on skills acquisition and retention must be considered.

In adults and children in cardiac arrest (out-of-hospital and in-hospital), does the use of an alternative compression–ventilation ratio, compared with standard care (30:2 compression to ventilation ratio), improve outcome (e.g., ROSC, survival)?

Consensus on science

Evidence from six human studies (LOE 3[14,21,198,199]; LOE 4[70]; LOE 5[6]) in adults and 23 additional studies (LOE 5: animal, manikin, and computer models) provides conflicting information about the optimal compression–ventilation ratio to maximise ROSC and survival to hospital discharge when CPR is administered by lay rescuers or by professional rescuers to patients with cardiac arrest in any setting.

In 2005, a single compression–ventilation ratio of 30:2 for the lone rescuer of an infant, child, or adult victim was recommended.[200,201] After implementation of this new guideline, two studies (LOE 3[21,199]) showed improvement of survival compared to survival with use of the previous 15:2 compression–ventilation ratio. However, other studies (LOE 3[14,198,202]) failed to show any beneficial effect of the new guidelines on survival, although the potential contribution of each change in the guidelines could not be assessed.

Animal studies (LOE 5) showed improved survival with a compression–ventilation ratio above 30:2.[203,204] However, a compression–ventilation ratio of more than 100:2 was associated with a low ROSC rate and reduced arterial partial pressure of oxygen.[205] The mathematical studies (LOE 5) suggested that the optimal compression–ventilation ratio was near 30:2 for healthcare professionals and near 60:2 for lay rescuer[206] or was a function of body weight in children.[207] Other theoretical studies have recommended ratios of 15:2 or 50:5[208] or around 20:1.[209]

Many manikin studies (all LOE 5) showed that CPR performance, quality, and rescuer's fatigue were not significantly different with differing compression–ventilation ratios,[204,210–213] while others showed mixed results among various compression–ventilation ratios from 5:1 to 60:2.[64,214–219]

Treatment recommendation

A compression–ventilation ratio of 30:2 is reasonable for an adult victim of cardiac arrest whose airway is not secured.

Chest compression-only CPR[BLS-046A,BLS-046B,BLS-047A,BLS-047B,BLS-049A,BLS-049B]

Any recommendation regarding the use of compression-only CPR versus standard CPR is dependent not only on the skill level and ability of the provider (e.g., untrained layperson, trained layperson, professional rescuer) but also on the patient (e.g., age and aetiology of arrest) and the situation (e.g., number of providers, phases of prehospital care).

- In adults in cardiac arrest, does the calling of EMS and the provision of chest compressions (without ventilation) by trained laypersons or professionals compared with calling EMS only improve survival to hospital discharge?[BLS-046A,BLS-046B]
- In adults in cardiac arrest, does the provision of chest compressions (without ventilation) from bystanders, both trained and untrained, compared with chest compressions plus mouth-to-mouth breathing, improve survival to hospital discharge?[BLS-047A,BLS-047B]
- In adults in cardiac arrest, does provision of chest compressions (without ventilation) by EMS, compared to chest compressions plus ventilations, improve survival to hospital discharge?[BLS-049A,BLS-049B]

Consensus on science

There are no human studies that have compared compression-only CPR with standard CPR using a 30:2 ratio of compressions to ventilations. Multiple mathematical and educational studies (LOE 5[67,206,208,213,220–223]) show some supporting evidence favoring a high compression–ventilation ratio or compression-only CPR. Some animal models of sudden VF cardiac arrest (LOE 5[184,186,203,224]) demonstrate benefits of compression-only CPR compared with conventional CPR. Additional animal studies (LOE 5[225–231]) demonstrate neutral evidence, while other animal studies (LOE 5[184,232–236]) show advantages to adding ventilations to chest compressions.

Evidence from one interventional human trial (LOE 1[237A]) and 8 observational studies (LOE 2[8,15,99,238–241]; LOE 3[242]) document consistent improvement in survival to hospital discharge when compression-only CPR compared with no CPR is administered by untrained or trained bystanders to adults with an out-of-hospital witnessed cardiac arrest.

Four human studies (LOE 2[157,158]; LOE 3[159,243]) demonstrated that provision of continuous chest compressions by trained professional (EMS) providers led to an improvement in survival to hospital discharge compared to standard CPR. Lower methodological rigor limits the ability to determine whether those improvements in survival were attributable to the provision of continuous chest compressions without pauses for ventilation or to other factors.

However, three additional studies (LOE 1[244]; LOE 2[245]; LOE 5[246]) failed to consistently show improvement in survival to hospital discharge when compression-only CPR compared with conventional CPR was administered by professionals to adult patients with an OHCA.

Evidence from 1 LOE 2 large paediatric prospective observational investigation[247] showed that children in cardiac arrest of noncardiac aetiology (asphyxial arrest) had higher 30-day survival with more favorable neurological outcome if they received standard bystander CPR (chest compressions with rescue breathing) compared with chest compression-only CPR. Standard CPR and chest compression-only CPR were similarly effective and better than no bystander CPR for paediatric cardiac arrest from cardiac causes. Of note, the same study showed that more than 50% of children with OHCA did not receive any bystander CPR. Compression-only CPR was as ineffective as no CPR in the small number of infants and children with asphyxial arrest.

Treatment recommendation

All rescuers should perform chest compressions for all patients in cardiac arrest. Chest compressions alone are recommended for untrained laypersons responding to cardiac arrest victims. Performing chest compressions alone is reasonable for trained laypersons if they are incapable of delivering airway and breathing manoeuvres to cardiac arrest victims. The provision of chest compressions with ventilations is reasonable for trained laypersons who are capable of giving CPR with ventilations to cardiac arrest victims.

Professional rescuers should provide chest compressions with ventilations for cardiac arrest victims. There is insufficient evi-

dence to support or refute the provision of chest compressions plus airway opening and oxygen insufflation by professional rescuers during the first few minutes of resuscitation from cardiac arrest.

Chest compression knowledge gaps

What is the optimal duration of CPR following administration of a defibrillation shock prior to rechecking the patient? Can ECG rhythm analysis during chest compressions be incorporated into resuscitation algorithms? Should the compression to ventilation ratio vary according to the victim's age or arrest aetiology? What is the effect of compression-only CPR bystander training on the overall survival of OHCA in the community compared to standard CPR training? What is the effect of compression-only CPR training on the willingness of bystanders to perform CPR compared to standard CPR training? Does EMS provision of chest compressions plus airway opening and oxygen insufflation improve long-term survival of cardiac arrest when compared with high-quality CPR using a 30:2 compression:ventilation ratio?

Special circumstances

Cervical spine injury

For victims of suspected spinal injury, additional time may be needed for careful assessment of breathing and circulation, and it may be necessary to move the victim if he or she is found face-down.

Face-down victim[BLS-007B]

In adults and children with cardiac arrest (out-of-hospital and in-hospital) and suspected major injury, does any different strategy for positioning (e.g., leaving them in the position in which they are found), as opposed to standard care (i.e., positioning the victim on his or her back), improve outcome (e.g., ROSC, survival)?

Consensus on science

No human studies have evaluated the relative benefits of strategies for positioning adults and children with cardiac arrest and suspected major injury. Head position is an important factor affecting airway patency (LOE 5[248]), and it is more difficult to check for breathing with the victim in a face-down position. Checking for breathing by lay and professional rescuers is often inaccurate when done within the recommended 10 s (LOE 5[38,39]). A longer time to check for breathing will delay CPR and may compromise outcome.

Treatment recommendation

It is reasonable to roll a face-down, unresponsive victim into the supine position to assess breathing and initiate resuscitation. Concern for protecting the neck should not hinder the evaluation process or delay life-saving procedures.

Emergency medical services (EMS) systems

The call to EMS dispatchers for help is generally the first action when a collapsed victim is found. Recognition of cardiac arrest as the cause of the collapse is rarely simple and requires the dispatcher to elicit critical information from the caller. Failure to recognise the true cause of the collapse precludes the use of bystander CPR and telephone instructions and may also delay the arrival of appropriate help. Not recognizing a cardiac arrest occurs in up to 50% of cases and is associated with lower survival.[249]

Dispatcher recognition of cardiac arrest[BLS-044A,BLS-044B]

In adults and children with OHCA does the description of any specific symptoms to the dispatcher compared with the absence of any specific description improve accuracy of the diagnosis of cardiac arrest?

Consensus on science

One before-and-after trial (LOE D3[250]) demonstrated a significant increase from 15% to 50% in cardiac arrest recognition after the implementation of a protocol requiring that EMS dispatchers assess absence of consciousness and quality of breathing (normal/not normal). Many descriptive studies (LOE D4[46,251–259]) using a similar protocol to identify cardiac arrest report a sensitivity on the order of 70%, ranging from 38%[255] to 97%,[259] and a high specificity ranging from 95%[254] to 99%.[256]

One case–control trial (LOE D3[249]), one before-and-after trial (LOE D3[43]), and four observational studies (LOE D4[41,42,260,261]) describe agonal gasps or abnormal breathing as a significant barrier to cardiac arrest recognition by emergency medical dispatchers. Two before-and-after trials (LOE D3[262,263]) improved the recognition of abnormal breathing using education or counting of breaths. Information spontaneously provided by the caller about the quality of breathing and other information such as facial color or describing the victim as "dead" can aid in identifying cardiac arrest cases (LOE D3[249,262,263]).

One descriptive study (LOE D4[264]) suggests that in cases where the victim's problem is "unknown" to the EMS dispatcher, inquiring about the victim's level of activity (standing, sitting, moving, or talking) helps to identify cases who are not in cardiac arrest. Two descriptive studies (LOE D4[261,265]) suggest that confirming the absence of a past medical history of seizure may increase the likelihood of recognizing cardiac arrest among victims presenting with seizure activity. A case–control study (LOE D3[45]) suggests that asking about regularity of breathing may help to recognise cardiac arrest among callers reporting seizure activity.

Treatment recommendation

EMS dispatchers should inquire about a victim's absence of consciousness and quality of breathing (normal/not normal) when attempting to identify cardiac arrest victims. If the victim is unresponsive, it is reasonable to assume that the victim is in cardiac arrest when callers report that breathing is not normal. Dispatchers should be specifically educated about identification of abnormal breathing in order to improve cardiac arrest recognition. The correct identification of cardiac arrest may be increased by careful attention to the caller's spontaneous comments and by focused questions about seizures.

Dispatcher instruction in CPR[BLS-010A,BLS-010B]

In adults and children with cardiac arrest (out-of-hospital and in-hospital), does the provision of dispatch CPR instructions, as opposed to no instructions, improve outcome (e.g., ROSC, survival)?

Consensus on science

Three studies (LOE 2[249,258,266]) provide evidence that dispatcher telephone CPR instructions may improve survival from OHCA. In

three randomised trials (LOE 1[237A,237B,237C]), compression-only dispatcher telephone CPR instruction produced survival to discharge at least equivalent to compression plus ventilation dispatcher telephone CPR instruction. Five additional simulation studies (LOE 5[67,220,223,267,268]) demonstrated that simplified chest compression-only telephone instructions in CPR reduce barriers to achieving reasonable-quality bystander CPR.

In four simulation studies (LOE 5[269–272]), video-enabled cell phone delivery of visual CPR instructions enhanced performance of CPR. However, in another simulation study (LOE 5[273]), simplified CPR instructions did not improve performance of bystander CPR by elderly rescuers.

Treatment recommendation

Bystanders who call their local emergency response number should receive initial instructions on performing CPR. Dispatchers should assertively provide compression-only CPR instructions to untrained rescuers for adults with suspected OHCA without any delay. If dispatchers suspect asphyxial arrest, it is reasonable to provide instructions on rescue breathing followed by chest compressions. When performing quality improvement efforts, it is reasonable to assess the accuracy and timeliness of dispatcher recognition of cardiac arrest and the delivery of CPR instructions.

Risks to victim

Many rescuers are concerned that delivering chest compressions to a victim who is not in cardiac arrest will lead to serious complications, and thus, they do not initiate CPR for some victims of cardiac arrest.

Risks for the victim[BLS-051A,BLS-051B]

In adults and children who are *not* in cardiac arrest, how often does provision of chest compressions from lay rescuers lead to harm (e.g., rib fracture)?

Consensus on science

There are no data to suggest that the performance of CPR by bystanders leads to more complications than CPR performed by professional rescuers. One LOE 4 study[274] documented no difference in the incidence of injuries on chest radiograph for arrest victims with and without bystander CPR. One LOE 5[275] study documented a higher rate of complications among inpatient arrest victims treated by less-experienced (non-ICU) rescuers. Four LOE 5[276–279] reports document bystander CPR-related injuries in individual cases. Only one of these[276] was a patient who was not in cardiac arrest.

Two LOE 4 studies[237A,280] reported that serious complications among non-arrest patients receiving dispatch-assisted bystander CPR occurred infrequently. Of 247 non-arrest patients with complete follow-up who received chest compressions from a bystander, 12% experienced discomfort; only 5 (2%) suffered a fracture; and no patients suffered visceral organ injury.[280]

Treatment recommendation

In individuals with presumed cardiac arrest, bystander CPR rarely leads to serious harm in victims who are eventually found not to be in cardiac arrest; and therefore, bystander CPR should be assertively encouraged.

2005 topics not reviewed in 2010[160,167]

The following topics were included in 2005, but not in this document: devices for airway positioning, duty cycle, CPR in prone position, leg-foot chest compressions, mouth-to-nose ventilation, mouth-to-tracheal stoma ventilation, recovery position, airway opening, CPR for drowning victim in water, removing drowning victim from water, and improving EMS response interval. The reader is referred to the 2005 publication for the reviews.[160,167]

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Appendix A. Evidence-Based Worksheets for Part 5: Adult Basic Life Support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations

Task Force	WS ID	PICO Title	Short Title	Authors	URL
BLS	BLS-003A	In adult and paediatric patients with presumed cardiac arrest (prehospital or in-hospital) (P), are there any factors (eg. on clinical exam) (I) as opposed to standard care (C), that increase the likelihood of diagnosing cardiac arrest (as opposed to non-arrest conditions (eg post-seizure, hypoglycaemia, intoxication) (O)?	Differentiation of cardiac arrest from other causes of unresponsiveness	Koenraad Monsieurs	http://circ.ahajournals.org/site/C2010/BLS-003A.pdf
BLS	BLS-003B	In adult and paediatric patients with presumed cardiac arrest (prehospital or in-hospital) (P), are there any factors (eg. on clinical exam) (I) as opposed to standard care (C), that increase the likelihood of diagnosing cardiac arrest (as opposed to non-arrest conditions (eg post-seizure, hypoglycaemia, intoxication) (O)?	Differentiation of cardiac arrest from other causes of unresponsiveness	Tyler F. Vadebon-coeur	http://circ.ahajournals.org/site/C2010/BLS-003B.pdf
BLS	BLS-004B	In adult and paediatric patients with out-of-hospital cardiac arrest (including residential settings) (P), does implementation of a public access AED program (I) as opposed to traditional EMS response (C), improve successful outcomes (O) (eg. ROSC, survival)?	Public access AED programs	E. Brooke Lerner	http://circ.ahajournals.org/site/C2010/BLS-004B.pdf
BLS	BLS-006A	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (P), does any specific compression depth (I) as opposed to standard care (ie. depth specified in treatment algorithm) (C), improve outcome (O) (eg. ROSC, survival)?	Compression depth	Ahamed H. Idris	http://circ.ahajournals.org/site/C2010/BLS-006A.pdf
BLS	BLS-006B	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (P), does any specific compression depth (I) as opposed to standard care (ie. depth specified in treatment algorithm) (C), improve outcome (O) (eg. ROSC, survival)?	Compression depth	Koenraad Monsieurs	http://circ.ahajournals.org/site/C2010/BLS-006B.pdf
BLS	BLS-007B	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) and suspected major injury (P), does any different strategy regarding positioning (eg. leaving them in the position they are found) (I) as opposed to standard care (ie. positioning the victim on his or her back) (C), improve outcome (O) (eg. ROSC, survival)?	Positioning of victim with traumatic cardiac arrest	Keiichi Tanaka	http://circ.ahajournals.org/site/C2010/BLS-007B.pdf
BLS	BLS-008B	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (P), does the interruption of CPR to check circulation (I) as opposed to no interruption of CPR (C), improve outcome (O) (eg. ROSC, survival)?	Pulse check (risk benefit of interruption of CPR)	Peter Fenici, Ian Jacobs, Andrea Scapigliati	http://circ.ahajournals.org/site/C2010/BLS-008B.pdf
BLS	BLS-009A	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) and receiving chest compression-only CPR (P), does the addition of any passive ventilation technique (eg positioning the body, opening the airway, passive oxygen administration) (I) as opposed to no addition (C), improve outcome (O) (eg. ROSC, survival)?	Passive ventilation techniques	Douglas Kupas	http://circ.ahajournals.org/site/C2010/BLS-009A.pdf
BLS	BLS-010A	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (P), does the provision of dispatch CPR instructions (I) as opposed to no instructions (C), improve outcome (O) (eg. ROSC, survival)?	Dispatch CPR instructions	James V. Dunford	http://circ.ahajournals.org/site/C2010/BLS-010A.pdf
BLS	BLS-010B	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (P), does the provision of dispatch CPR instructions (I) as opposed to no instructions (C), improve outcome (O) (eg. ROSC, survival)?	Dispatch CPR instructions	Maaret Castrén	http://circ.ahajournals.org/site/C2010/BLS-010B.pdf

Appendix A (*Continued*)

Task Force	WS ID	PICO Title	Short Title	Authors	URL
BLS	BLS-011A	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (P), does the provision of airway manoeuvres by bystanders (I) as opposed to no such manoeuvres (C), improve outcome (O) (eg. ROSC, survival)?	Airway manoeuvres in bystander CPR	Robert A. Swor	http://circ.ahajournals.org/site/C2010/BLS-011A.pdf
BLS	BLS-011B	In adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (P), does the provision of airway manoeuvres by bystanders (I) as opposed to no such manoeuvres (C), improve outcome (O) (eg. ROSC, survival)?	Airway manoeuvres in bystander CPR	Sung Phil Chung	http://circ.ahajournals.org/site/C2010/BLS-011B.pdf
BLS	BLS-013A	In adult and paediatric patients with foreign-body airway obstruction (out-of-hospital and in-hospital) (P), does the provision of abdominal thrusts, and/or back slaps, and/or chest thrusts, compared with no action (C), improve outcome (O) (eg. clearance of obstruction, ROSC, survival)?	Choking treatment	Anthony J. Handley	http://circ.ahajournals.org/site/C2010/BLS-013A.pdf
BLS	BLS-014B	What is the incidence, prevalence, aetiology of cardiopulmonary arrest in-hospital and out-of-hospital?	Incidence and aetiology cardiac arrest	Jocelyn Berdowski	http://circ.ahajournals.org/site/C2010/BLS-014B.pdf
BLS	BLS-017A	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of alternative methods of manual CPR (eg. cough CPR, precordial thump, fist pacing) (I) compared with standard CPR (C), improve any outcomes (eg. ROSC, survival) (O)?	Alternative methods of CPR	Tom P. Aufderheide	http://circ.ahajournals.org/site/C2010/BLS-017A.pdf
BLS	BLS-017B	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of alternative methods of manual CPR (eg. cough CPR, precordial thump, fist pacing) (I) compared with standard CPR (C), improve any outcomes (eg. ROSC, survival) (O)?	Alternative methods of CPR	Jan Jensen	http://circ.ahajournals.org/site/C2010/BLS-017B.pdf
BLS	BLS-017C	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of alternative methods of manual CPR (eg. cough CPR, precordial thump, fist pacing) (I) compared with standard CPR (C), improve any outcomes (eg. ROSC, survival) (O)?"	Alternative methods of CPR	Peter Kohl, Tommaso Pellis	http://circ.ahajournals.org/site/C2010/BLS-017C.pdf
BLS	BLS-020A	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of feedback regarding the mechanics of CPR quality (e.g. rate and depth of compressions and/or ventilations) (I) compared with no feedback (C), improve any outcomes (eg. ROSC, survival) (O)?	Feedback for CPR quality	Diana Cave	http://circ.ahajournals.org/site/C2010/BLS-020A.pdf
BLS	BLS-020B	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of feedback regarding the mechanics of CPR quality (e.g. rate and depth of compressions and/or ventilations) (I) compared with no feedback (C), improve any outcomes (eg. ROSC, survival) (O)?	Feedback for CPR quality	Peter T. Morley	http://circ.ahajournals.org/site/C2010/BLS-020B.pdf
BLS	BLS-022A	In adult and paediatric patients with cardiac arrest (prehospital or in-hospital) (P), does the minimization of hands-off time after defibrillation for rhythm check (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (eg. ROSC, survival)?	Rhythm check (risk benefit of interruption of CPR)	Robert A. Berg	http://circ.ahajournals.org/site/C2010/BLS-022A.pdf
BLS	BLS-023A	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of another specific C:V ratio (I) compared with standard care (30:2) (C), improve outcome (eg. ROSC, survival) (O)?	Compression ventilation ratio	Sung Phil Chung	http://circ.ahajournals.org/site/C2010/BLS-023A.pdf
BLS	BLS-023B	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of another specific C:V ratio (I) compared with standard care (30:2) (C), improve outcome (eg. ROSC, survival) (O)?	Compression ventilation ratio	Michael Sayre	http://circ.ahajournals.org/site/C2010/BLS-023B.pdf
BLS	BLS-025A	In adult and paediatric patients with cardiac arrest (prehospital or in-hospital) (P), does the minimization of hands-off time for rhythm analysis including frequency and duration of checks (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (eg. ROSC, survival)?	Rhythm check (risk benefit of interruption of CPR)	Dana P. Edelson	http://circ.ahajournals.org/site/C2010/BLS-025A.pdf

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Task Force	WS ID	PICO Title	Short Title	Authors	URL
BLS	BLS-025B	In adult and paediatric patients with cardiac arrest (prehospital or in-hospital) (P), does the minimization of hands-off time for rhythm analysis including frequency and duration of checks (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (eg. ROSC, survival)?	Rhythm check (risk benefit of interruption of CPR)	David C. Cone	http://circ.ahajournals.org/site/C2010/BLS-025B.pdf
BLS	BLS-026A	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of compressions first (30:2) (I) compared with standard care (2:30) (C), improve outcome (eg. ROSC, survival) (O).	Compression first vs ventilation first	Anthony J. Handley	http://circ.ahajournals.org/site/C2010/BLS-026A.pdf
BLS	BLS-026B	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of compressions first (30:2) (I) compared with standard care (2:30) (C), improve outcome (eg. ROSC, survival) (O).	Compression first vs ventilation first	Diana Cave	http://circ.ahajournals.org/site/C2010/BLS-026B.pdf
BLS	BLS-032A	In adult and paediatric patients with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific placement of hands for external chest compressions (I) compared with standard care (eg. "placement of the rescuer's hands in the middle of the chest") (C), improve outcome (eg. ROSC, survival) (O)	Hand placement	Raina Merchant	http://circ.ahajournals.org/site/C2010/BLS-032A.pdf
BLS	BLS-032B	In adult and paediatric patients with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific placement of hands for external chest compressions (I) compared with standard care (eg. "placement of the rescuer's hands in the middle of the chest") (C), improve outcome (eg. ROSC, survival) (O)	Hand placement	Nigel M. Turner	http://circ.ahajournals.org/site/C2010/BLS-032B.pdf
BLS	BLS-033A	In rescuers performing CPR on adult or paediatric patients with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific method for locating recommended hand position (I) compared with standard care (eg. "placement of the rescuer's hands in the middle of the chest") (C), improve outcome (eg. time to commence CPR, decreased hands-off time, ROSC, survival) (O)	Hand placement	Anthony J. Handley	http://circ.ahajournals.org/site/C2010/BLS-033A.pdf
BLS	BLS-034A	In adult and paediatric patients with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific rate for external chest compressions (I) compared with standard care (ie. approximately 100/min) (C), improve outcome (eg. ROSC, survival) (O)?	Chest compression rate	Ahamed H. Idris	http://circ.ahajournals.org/site/C2010/BLS-034A.pdf
BLS	BLS-034B	In adult and paediatric patients with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific rate for external chest compressions (I) compared with standard care (ie. approximately 100/min) (C), improve outcome (eg. ROSC, survival) (O)?	Chest compression rate	Barbara Vantroyen	http://circ.ahajournals.org/site/C2010/BLS-034B.pdf
BLS	BLS-035A	In adult and paediatric patients with cardiac arrest while on a bed (prehospital [OHCA], in-hospital [IHCA]) (P), does the performance of CPR on a hard surface like backboard or deflatable mattress (I) compared with performance of CPR on a regular mattress (C), improve outcome (eg. ROSC, survival) (O)?	Soft vs hard surface for CPR	Gavin D. Perkins	http://circ.ahajournals.org/site/C2010/BLS-035A.pdf
BLS	BLS-035B	In adult and paediatric patients with cardiac arrest while on a bed (prehospital [OHCA], in-hospital [IHCA]) (P), does the performance of CPR on a hard surface like backboard or deflatable mattress (I) compared with performance of CPR on a regular mattress (C), improve outcome (eg. ROSC, survival) (O)?	Soft vs hard surface for CPR	Bo Løfgren	http://circ.ahajournals.org/site/C2010/BLS-035B.pdf
BLS	BLS-039	In adult and paediatric patients with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the analysis of cardiac rhythm during chest compressions (I) compared with standard care (analysis of cardiac rhythm during pauses in chest compressions) (C), optimise the time of appropriate chest compression by avoiding unnecessary interruptions and unnecessary prolongations (O)?	Analysis of rhythm during chest compression	Raúl J. Gazmuri, Michael A. Kuiper	http://circ.ahajournals.org/site/C2010/BLS-039.pdf

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Task Force	WS ID	PICO Title	Short Title	Authors	URL
BLS	BLS-044A	In adult and paediatric patients with cardiac arrest (prehospital [OHCA]) (P), does the description of any specific symptoms to the dispatcher (I) compared with the absence of any specific description (C), improve accuracy of the diagnosis of cardiac arrest (O)?	Rescuer communication with dispatcher for CPR	Manya Charette, Christian Vaillancourt	http://circ.ahajournals.org/site/C2010/BLS-044A.pdf
BLS	BLS-044B	In adult and paediatric patients with cardiac arrest (prehospital [OHCA]) (P), does the description of any specific symptoms to the dispatcher (I) compared with the absence of any specific description (C), improve accuracy of the diagnosis of cardiac arrest (O)?	Rescuer communication with dispatcher for CPR	Maaret Castrén	http://circ.ahajournals.org/site/C2010/BLS-044B.pdf
BLS	BLS-045A	In adult and paediatric patients with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does optimizing chest wall recoil (I) compared with standard care (C), improve outcome (eg. ROSC, survival) (O)? In patients with CA (P), does optimizing chest wall recoil (I), improve survival (O)?	Chest wall recoil	Tom P. Aufderheide	http://circ.ahajournals.org/site/C2010/BLS-045A.pdf
BLS	BLS-046A	In adult patients suffering from a cardiac arrest (P) does the calling of EMS and the provision of chest compressions (without ventilation) by untrained laypersons, trained laypersons, or professionals (I) compared with calling EMS only (C) improve survival to hospital discharge (O)?	Untrained lay rescuer CC Only vs call EMS	Tetsuo Hatanaka	http://circ.ahajournals.org/site/C2010/BLS-046A.pdf
BLS	BLS-046B	In adult patients suffering from a cardiac arrest (P) does the calling of EMS and the provision of chest compressions (without ventilation) by untrained laypersons, trained laypersons, or professionals (I) compared with calling EMS only (C) improve survival to hospital discharge (O)?	Untrained lay rescuer CC Only vs call EMS	Tom Rea	http://circ.ahajournals.org/site/C2010/BLS-046B.pdf
BLS	BLS-047A	In adult patients suffering from a cardiac arrest (P) does the provision of chest compressions (without ventilation) from bystanders, both trained and untrained, (I) compared with chest compressions plus mouth-to-mouth breathing (C) improve survival to hospital discharge (O)?	Chest compression-only CPR	Csaba Dioszeghy	http://circ.ahajournals.org/site/C2010/BLS-047A.pdf
BLS	BLS-047B	In adult patients suffering from a cardiac arrest (P) does the provision of chest compressions (without ventilation) from bystanders, both trained and untrained, (I) compared with chest compressions plus mouth-to-mouth breathing (C) improve survival to hospital discharge (O)?	Chest compression-only CPR	Andrew Travers	http://circ.ahajournals.org/site/C2010/BLS-047B.pdf
BLS	BLS-049A	In adult patients suffering from a cardiac arrest (P) does provision of chest compressions (without ventilation) by EMS (I) compared with chest compressions plus ventilations (C) improve survival to hospital discharge (O)?	EMS CC only vs standard CPR	Laura S. Gold, Peter J. Kudenchuk	http://circ.ahajournals.org/site/C2010/BLS-049A.pdf
BLS	BLS-049B	In adult patients suffering from a cardiac arrest (P) does provision of chest compressions (without ventilation) by EMS (I) compared with chest compressions plus ventilations (C) improve survival to hospital discharge (O)?	EMS CC only vs standard CPR	Andrew Travers	http://circ.ahajournals.org/site/C2010/BLS-049B.pdf
BLS	BLS-050A	In adult and paediatric patients with presumed cardiac arrest (prehospital or in-hospital) (P), are there any factors/characteristics (I) that increase the likelihood of differentiating between a sudden cardiac arrest (ie. VF) from other etiologies (eg drowning, acute airway obstruction) (O)?	Differentiating cardiac from noncardiac etiologies	Anthony J. Handley	http://circ.ahajournals.org/site/C2010/BLS-050A.pdf
BLS	BLS-050B	In adult and paediatric patients with presumed cardiac arrest (prehospital or in-hospital) (P), are there any factors/characteristics (I) that increase the likelihood of differentiating between a sudden cardiac arrest (ie. VF) from other etiologies (eg drowning, acute airway obstruction) (O)?	Differentiating cardiac from noncardiac etiologies	Michael Kuiper	http://circ.ahajournals.org/site/C2010/BLS-050B.pdf
BLS	BLS-051A	In adults and paediatric patients who are *not* in cardiac arrest (P), how often does provision of chest compressions from lay rescuers (I), lead to harm (eg rib fracture) (O)?	Harm from CPR to victims not in arrest	Anton P. M. Gorgels, Antonius M. W. van Stipdonk	http://circ.ahajournals.org/site/C2010/BLS-051A.pdf
BLS	BLS-051B	In adults and paediatric patients who are *not* in cardiac arrest (P), how often does provision of chest compressions from lay rescuers (I), lead to harm (eg rib fracture) (O)?	Harm from CPR to victims not in arrest	Daniel P. Davis	http://circ.ahajournals.org/site/C2010/BLS-051B.pdf

Appendix A (*Continued*)

Task Force	WS ID	PICO Title	Short Title	Authors	URL
BLS	BLS-052B	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) who are *not* endotracheally intubated (P), does providing ventilation with a 1 second inspiratory time and tidal volume of about 600 mL (I), compared with other inspiratory times and tidal volume (C), improve any outcomes (including ventilation, oxygenation) (O)?	Ventilation inspiratory time and volume	Colin A. Graham	http://circ.ahajournals.org/site/C2010/BLS-052B.pdf
BLS	BLS-053A	In adult patients in cardiac arrest (P), how frequently should chest compressions be paused to re-diagnose accurately the cardiac rhythm (I) to provide the best outcomes (eg ROSC, survival) (O)?	Timing of CPR cyles (2 min vs other)	Michael Cudnik	http://circ.ahajournals.org/site/C2010/BLS-053A.pdf

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CoSTR Part 5: writing group disclosures

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Rudolph W. Koster	Academic Medical Center—Staff cardiologist	†Physio-Control: Support to maintain infrastructure for cardiac arrest database. Grant covers salaries for PhD student, data managers, compensation for medical students, mobile phone & car for use by medical students to collect data. Employees are appointed by institution. Money goes directly to institution. I do not receive any direct or indirect benefit except scientific credit; *Physio-Control pays for defib pads to first responders (police, fire fighters) who participate in study	None	None	None	None	None
Martin Botha	Academy of Advanced Life Support: Education and training in emergency medicine and AHA courses—Training manager	None	None	None	None	None	None

Disclosures (*Continued*)

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Diana M. Cave	Legacy Health System, Emanuel Hospital: Emanuel Hospital is a Level 1 Trauma Center and is affiliated locally with the Oregon Health Sciences University for medical resident education—RN, MSN, Emergency Services; Portland Community College, Institute for Health Professionals: The Institute for Health Professionals provides CE courses for medical professional. Course offerings include AHA courses—Faculty, Instructor	None	None	None	None	*EMS Associates. Consultant. EMS Associates provides continuing education courses including all AHA, ENA and other courses. I provide direct education as an instructor or work with health care organizations to determine their educational needs.	None
Michael T. Cudnik	The Ohio State University Medical Center: Assistant Professor, Department of Emergency Medicine	†I am currently the PI on a 4 year Scientist Development Grant funded by the AHA. The grant is not in any financial or intellectual conflict with this writing group.	None	None	None	None	None
Anthony J. Handley	Colchester General University Hospital: Honorary Consultant Physician	None	None	None	None	*Consultant Medical Adviser (AED audit and Training) Virgin Atlantic Airways: direct payment *Consultant Medical Adviser (AED audit and training) British Airways: direct payment *Consultant Medical Adviser (AED audit and Training) DC Leisure Ltd (leisure centre management company): direct payment	†Expert witness for various legal firms on consultancy basis – no one particular firm. Preparation of medical reports; advice to solicitors and barristers; appearance in Court as required: direct payment
Tetsuo Hatanaka	Emergency Life Saving Technique Academy: Educational institution for municipal paramedics—Professor	†Research grant for "Cardiovascular Disease H18-Heart-01: A Study on Automated External Defibrillator Program and System Development for Improved Survival from Emergency Cardiovascular Disease" from the Ministry of Health, Labour and Welfare, Japan.	None	* Several kinds of honoraria for scientific meetings	None	None	None

Disclosures (*Continued*)

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Mary Fran Hazinski	Vanderbilt University School of Nursing: University and medical center—Professor AHA ECC Product Development: Senior Science Editor; *The significant compensation that I receive from the AHA ECC Product Development is to enable me to co-edit the 2010 CoSTR document and the 2010 AHA ECCGuidelines	None	None	None	None	None	None
Ian Jacobs	University of Western Australia: Discipline of Emergency Medicine Teaching / Research academic—Professor; American Heart Association: Evaluation of evidence worksheets for C2010—Work Sheet Expert	†Chief investigator on numerous grants awarded by: a) National Health and Medical Research Council b) The Department of Health – Western Australia c) The National Heart Foundation of Australia These funds are awarded to the University of Western Australia and none are used to provide any direct or indirect salary or other financial support.	†Funds are received into the Discipline of Emergency Medicine – University of Western Australia from the Ambulance Service -Western Australia and Laerdal (Australia) to maintain the Cardiac Arrest Registry for Western Australia. Our role is to independently maintain, analyze and report outcomes of cardiac arrest in Western Australia. I oversee the operation of the registry and reporting of outcomes. These funds are not used in any way to provide any direct or indirect salary or other financial support.	None	None	None	None
Koen Monsieurs	Ghent University Hospital and Ghent University: Specialist in Emergency Medicine, academic staff	None	* Laerdal (Norway), Zoll (US) and O-Two systems (Canada) for research projects in the field of resuscitation for technical support, training manikins and prototype devices. I have not received any payment of fees related to these studies.	None	None	None	None
Peter T. Morley	Royal Melbourne Hosp; Univ of Melbourne; Dir of Medical Education; AHA EEE	None	None	None	None	None	None
Jerry P. Nolan	Royal United Hospital NHS Trust ; National Health Service Hospital ; Consultant in Anaesthesia and Intensive Care	None	None	None	None	None	None

Disclosures (*Continued*)

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Andrew H. Travers	Emergency Health Services, Nova Scotia: Department of Health, Nova Scotia—Provincial Medical Director	†I was the lead Principal Investigator for the Public Access Defibrillation Trial for Edmonton, Alberta, Canada and received grant funding from NHLBI through contracts at the University of Washington which acted as the CRC.	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

* Modest.

† Significant.

Appendix C. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2010.08.005.

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Part 6: Defibrillation
2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations☆

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The 2010 Defibrillation Task Force considered many questions related to defibrillation. In general, the 2010 International Consensus on Science With Treatment Recommendations statement contains no major differences or dramatic changes from the 2005 International Consensus statement. The questions have been grouped into the following categories: (1) cardiopulmonary resuscitation (CPR) before defibrillation, (2) electrode–patient interface, (3) defibrillation strategy, (4) special circumstances, and (5) defibrillation-related topics.

Science and treatment recommendations dealing with the infant or child requiring defibrillation can be found in Part 10: Paediatric Basic and Advanced Life Support. The only treatment recommendations that differ for adult and children are defibrillation dose and automated external defibrillator (AED) use.

There are several knowledge gaps created by the lack of high-quality, large clinical studies. These include the minimal acceptable first-shock success rate; the characteristics of the optimal biphasic waveform; the optimal energy levels for specific waveforms; and the best shock strategy (fixed versus escalating).

Integration of CPR and defibrillation

Whether a period of CPR should be performed before defibrillation in ventricular fibrillation (VF), especially after a long response time, has recently been the subject of intense debate. The theoretical rationale for CPR before shock delivery is to improve coronary perfusion and thereby the chances of achieving sustained return of spontaneous circulation (ROSC).

CPR before defibrillation[BLS-024A,BLS-024B]

In adults and children with cardiac arrest due to VF (out-of-hospital or in-hospital) does the use of CPR before defibrillation, as opposed to standard care (according to treatment algorithm), improve outcomes (e.g., ROSC, survival)?

Consensus on science

In two randomised controlled trials (LOE 1)[1,2], a period of 1.5 to 3 min of CPR by emergency medical services (EMS) personnel before defibrillation did not improve ROSC or survival to hospital discharge in patients with out-of-hospital VF or pulseless ventricular tachycardia (VT), regardless of EMS response interval. One before-and-after study (LOE 3)[3] and another study (LOE 4)[4] failed to demonstrate significant improvements in ROSC or survival to hospital discharge when a strategy of CPR before defibrillation (CPR first) was compared to a shock-first strategy. In the Hayakawa study, the CPR-first group showed a higher rate of favorable neurological outcome 30 days and 1 year after cardiac arrest.[3]

One randomised controlled trial (LOE 1)[5] and one clinical trial with historic controls (LOE 3)[6] comparing CPR-first versus shock-first also found no overall difference in outcomes. However, in both studies, improvements in ROSC, survival to hospital discharge, neurological outcome, and 1-year survival were observed in a subgroup of patients who received CPR first where the EMS response interval was >4–5 min.

Treatment recommendation

There is inconsistent evidence to support or refute delay in defibrillation to provide a period of CPR (90 s to 3 min) for patients in non-EMS witnessed VF/pulseless VT cardiac arrest.

Electrode–patient interface

Studies on defibrillation for cardiac arrest and on cardioversion for atrial fibrillation (AF) are both included here. While few studies compared differences in outcome, many studies compared secondary end points such as effect on transthoracic impedance (TTI).

☆ *Note from the writing group*: Throughout this article, the reader will notice combinations of superscripted letters and numbers (e.g., "CPR Before Defibrillation[BLS-024A,BLS-024B]"). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.

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In ventricular arrhythmias, however, there is no direct evidence that TTI affects shock success.

Self-adhesive defibrillation pads compared with paddles[ALS-E-037A]

In adult cardiac arrest (out-of-hospital [OHCA], in-hospital [IHCA]) does the use of self-adhesive defibrillation pads, compared with paddles, improve outcomes (e.g., successful defibrillation, ROSC, survival)?

Consensus on science

Since 2005 there have been no new studies comparing self-adhesive defibrillation pads with paddles in cardiac arrest. Evidence from one small, good-quality controlled study (LOE 3)[7] in 1987 showed that self-adhesive pads were associated with a significantly improved rate of ROSC and hospital admission compared with hand-held paddles. Several studies have shown the practical benefits of pads over paddles for routine monitoring and defibrillation.[8–12]

One prospective study (LOE 3)[13] found lower TTI when paddles applied at an optimal force of 8 kg were compared with pads. In a cohort study in patients with atrial fibrillation (LOE 2)[14] the use of hand-held paddles placed in the anterior–posterior position increased the success rate of monophasic cardioversion compared with similarly placed self-adhesive electrodes for monophasic defibrillation. The overall cardioversion success rate for biphasic defibrillators was high (>95%) in all groups. In the majority of other studies, self-adhesive electrodes were associated with similarly high cardioversion success rates.

Treatment recommendation

For both defibrillation and AF cardioversion, when using biphasic defibrillators, self-adhesive defibrillation pads are safe and effective and are an acceptable alternative to standard defibrillation paddles. In AF cardioversion using monophasic defibrillators, hand-held paddles are preferable.

Treatment recommendation

For both defibrillation and AF cardioversion, when using biphasic defibrillators, self-adhesive defibrillation pads are safe and effective and are an acceptable alternative to standard defibrillation paddles. In AF cardioversion using monophasic defibrillators, hand-held paddles are preferable.

Placement of paddles/pads[ALS-E-030A]

In adult cardiac arrest (OHCA, IHCA) does the use of any specific paddle/pad size/orientation and position, compared with standard resuscitation (or other specific paddle/pad size/orientation and position), improve outcomes (e.g., successful defibrillation, ROSC, survival)?

Consensus on science

There are no studies in patients with VF/pulseless VT directly comparing the effects of various positions of paddle/pad placement on defibrillation success and ROSC. Most studies evaluate cardioversion (e.g., AF) or secondary end points (e.g., TTI). Eleven studies (LOE 5)[15–25] found all four positions (anterior–apex, anterior–posterior, anterior–left infrascapular, anterior–right infrascapular) to be equally effective in defibrillation (for VF/pulseless VT) or elective AF cardioversion success. Four studies support the anterior–posterior position (LOE 5),[26–30] one study supports the anterior–lateral position (LOE 5),[31] and one study supports the anterior–apex position (LOE 5).[32]

Five studies (LOE 5)[16,21–24] found no effect of electrode position on TTI. One study showed that paddles/pads should be placed under the breast tissue (LOE 5)[33] and two studies showed that hirsute males should be shaved before the application of pads (LOE 5).[34,35] Of the 36 studies reviewed, only four examined biphasic waveforms (LOE 5)[18,25,29,36] that have gained widespread use.

Treatment recommendation

It is reasonable to place paddles/pads on the exposed chest in an anterior–lateral position. Acceptable alternative positions are anterior–posterior (for paddles/pads) and apex–posterior (for pads). In large-breasted individuals it is reasonable to place the left electrode paddle/pad lateral to or underneath the left breast, avoiding breast tissue. Consideration should be given to the rapid removal of excessive chest hair before the application of paddles/pads but emphasis must be on minimizing delay in shock delivery.

Size of paddles/pads[ALS-E-030A]

In adult cardiac arrest (OHCA, IHCA) does the use of any specific paddle/pad size/orientation and position, compared with standard resuscitation (or other specific paddle/pad size/orientation and position), improve outcomes (e.g., successful defibrillation, ROSC, survival)?

Consensus on science

No new clinical study on this topic has been published since 2005. One study demonstrated that TTI decreased and shock success increased with increasing pad size (from 8 to 12 cm) (LOE 3).[37] Ten other studies showed that larger paddle/pad sizes (8- to 12-cm diameter) lowered TTI and that maximum paddle/pad size was limited by the chest wall size and anatomy (LOE 3[38]; LOE 5[23,36,39–45]). No data related to survival outcome was included in these studies.

Treatment recommendation

There is insufficient evidence to recommend a specific electrode size for optimal external defibrillation in adults. However, it is reasonable to use a paddle/pad size >8 cm.

Composition of conductive material[ALS-E-036]

In adult cardiac arrest (OHCA, IHCA) does the use of any specific composition of conductive material, compared with standard conductive material, improve TTI?

Consensus on science

Fourteen studies showed that the composition of the conductive material (e.g., saline, hypertonic sodium chloride [NaCl] solution, or silver-silver chloride) may alter TTI by more than 20% (LOE 2[39,46,47]; LOE 3[37]; LOE 4[48]; LOE 5[34,49–56]). Five studies (LOE 3[57,58]; LOE 5[59–61]) showed that TTI was not affected by electrode composition. The end point for all of these studies was TTI, and no studies involved outcomes following cardiac arrest.

Treatment recommendation

The composition of the conductive material of defibrillation electrodes influences TTI. In terms of cardiac arrest outcomes, there is insufficient evidence to recommend a specific composition of the defibrillation electrode conductive material.

Waveforms, energy levels, and strategies[ALS-E-033B]

All new defibrillators currently deliver shocks using biphasic waveforms. Although it has not been demonstrated conclusively in

randomised clinical studies that biphasic defibrillators save more lives than monophasic defibrillators, biphasic defibrillators achieve higher first-shock success rates. Shock success is usually defined as termination of VF 5 s after the shock.

In adult cardiac arrest due to VF or pulseless VT (OHCA, IHCA), does the use of any specific defibrillation strategy, compared with standard management (or other specific defibrillation strategy), improve outcomes (e.g., termination of VF 5 s after the shock)?

Biphasic compared with monophasic defibrillation waveform

Consensus on science

In three randomised trials (LOE 1)[62–64] and four other human studies (LOE 3)[65–68] biphasic waveforms had higher shock-success rates compared with monophasic defibrillation. One randomised study comparing transthoracic incremental monophasic with biphasic defibrillation for out-of-hospital pulseless VT/VF cardiac arrest failed to demonstrate any significant differences in any outcome (LOE 1).[69] A single-cohort study (LOE 3)[70] using the 2000 International Guidelines[71] demonstrated better hospital discharge and neurological survival with biphasic than with monophasic waveforms. However, there were confounding factors in that the intervals between the first and second shocks (of three-stacked shocks) were shorter with the biphasic defibrillators.

There is no clinical evidence for superiority of any specific biphasic waveform over another.

Treatment recommendation

Biphasic waveforms are more effective in terminating VF when compared with monophasic waveforms. There is insufficient evidence to recommend any specific biphasic waveform. In the absence of biphasic defibrillators, monophasic defibrillators are acceptable.

Multiphasic compared with biphasic defibrillation waveform

Consensus on science

There are no human studies to support the use of multiphasic waveforms over biphasic waveforms for defibrillation. Animal data suggests that multiphasic waveforms may defibrillate at lower energies and induce less postshock myocardial dysfunction.[72,73] These results are limited because in all studies duration of VF was very short (approximately 30 s) and results have not been validated in human studies.

Treatment recommendation

Currently, multiphasic defibrillators are not commercially available.

Waveforms, energy levels, and myocardial damage

Several different biphasic waveforms are used in commercially available defibrillators, but no human studies have directly compared these waveforms or compared them at different energy levels related to defibrillation success or survival.

For the different biphasic waveforms, studies of different size and quality have been performed and are presented separately. For all waveforms, insufficient evidence exists to make clear recommendations.

Consensus on science

Biphasic truncated exponential (BTE) waveform. Evidence from one well-conducted randomised trial (LOE 1)[74] and one other human study (LOE 2)[75] employing BTE waveforms suggested that higher energy levels are associated with higher shock-success rates. In the randomised trial, the first-shock success rate was similar with 150 J and 200 J.[74]

Pulsed biphasic waveform. In one study using pulsed biphasic waveforms at 130 J the first-shock success rate was 90% (LOE 4).[76]

Rectilinear biphasic waveform. When defibrillation success was defined as ROSC (this differs from the definition in other studies), one study using a rectilinear biphasic waveform showed that an organised rhythm was restored by the first shock (120 J) in 23% of cases (LOE 1).[62] Success rate for the termination of VF at 5 s was not published for this waveform.

Monophasic waveform (damped sinusoid or truncated exponential). Evidence from three studies of monophasic defibrillation suggested equivalent outcomes with lower and higher starting energies (LOE 1[77]; LOE 2[78,79]).

Myocardial damage associated with higher energy level shocks. Several animal studies have suggested the potential for myocardial damage with higher energy shocks using BTE or monophasic waveforms (LOE 5).[36,80–81] Human studies involving BTE waveforms[74,83] with energy levels up to 360 J have not shown harm as indicated by biomarker levels, ECG findings, and ejection fractions.

Treatment recommendation

It is reasonable to start at a selected energy level of 150–200 J for a BTE waveform for defibrillation of pulseless VT/VF cardiac arrest. There is insufficient evidence to determine the initial energy levels for any other biphasic waveform. Although evidence is limited, because of the lower total shock success for monophasic defibrillation, initial and subsequent shocks using this waveform should be at 360 J.

One-shock compared with three-stacked shock protocols

Consensus on science

One study showed no survival benefit from a protocol that included a single-shock protocol compared to a three-shock protocol (LOE 1).[84] Evidence from three pre–post design studies suggested significant survival benefit with a single-shock defibrillation protocol compared with three-stacked shock protocols (LOE 3).[85–87] However, these studies included confounders related to pre–post design and the multiple interventions that were included as part of the defibrillation protocol. Another pre–post study, with fewer confounding factors, showed a significantly lower hands-off ratio (i.e., percentage of total CPR time when no compressions were provided) with the one-shock protocol but no statistical difference in survival (LOE 3).[88]

One observational study of fixed-dose biphasic defibrillation suggested higher defibrillation success with three shocks (LOE 4).[89] The same study also suggested that chest compressions immediately following a shock did not result in recurrence of VF. In contrast another study showed earlier recurrence of VF when chest compressions were resumed immediately after the shock compared with delayed resumption of compressions (LOE 1).[90] There was no significant difference in total incidence of recurrent VF or outcome. A single study demonstrated that early termination of recurrent VF was associated with increased ROSC, but quality of CPR was poor and few patients achieved ROSC (LOE 4).[91] Another study showed decreased survival when defibrillation for recurrent VF was, for a variety of reasons, delayed (LOE 4).[92]

Treatment recommendation

When defibrillation is required, a single shock should be provided with immediate resumption of chest compressions after

the shock. Chest compressions should not be delayed for rhythm reanalysis or pulse check immediately after a shock. CPR should not be interrupted until rhythm reanalysis is undertaken.

Biphasic compared with monophasic defibrillation waveform

Consensus on science

In three randomised trials (LOE 1)[62–64] and four other human studies (LOE 3)[65–68] biphasic waveforms had higher shock-success rates compared with monophasic defibrillation. One randomised study comparing transthoracic incremental monophasic with biphasic defibrillation for out-of-hospital pulseless VT/VF cardiac arrest failed to demonstrate any significant differences in any outcome (LOE 1).[69] A single-cohort study (LOE 3)[70] using the 2000 International Guidelines[71] demonstrated better hospital discharge and neurological survival with biphasic than with monophasic waveforms. However, there were confounding factors in that the intervals between the first and second shocks (of three-stacked shocks) were shorter with the biphasic defibrillators.

There is no clinical evidence for superiority of any specific biphasic waveform over another.

Treatment recommendation

Biphasic waveforms are more effective in terminating VF when compared with monophasic waveforms. There is insufficient evidence to recommend any specific biphasic waveform. In the absence of biphasic defibrillators, monophasic defibrillators are acceptable.

Multiphasic compared with biphasic defibrillation waveform

Consensus on science

There are no human studies to support the use of multiphasic waveforms over biphasic waveforms for defibrillation. Animal data suggests that multiphasic waveforms may defibrillate at lower energies and induce less postshock myocardial dysfunction.[72,73] These results are limited because in all studies duration of VF was very short (approximately 30 s) and results have not been validated in human studies.

Treatment recommendation

Currently, multiphasic defibrillators are not commercially available.

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For the different biphasic waveforms, studies of different size and quality have been performed and are presented separately. For all waveforms, insufficient evidence exists to make clear recommendations.

Consensus on science

Biphasic truncated exponential (BTE) waveform. Evidence from one well-conducted randomised trial (LOE 1)[74] and one other human study (LOE 2)[75] employing BTE waveforms suggested that higher energy levels are associated with higher shock-success rates. In the randomised trial, the first-shock success rate was similar with 150 J and 200 J.[74]

Pulsed biphasic waveform. In one study using pulsed biphasic waveforms at 130 J the first-shock success rate was 90% (LOE 4).[76]

Rectilinear biphasic waveform. When defibrillation success was defined as ROSC (this differs from the definition in other studies), one study using a rectilinear biphasic waveform showed that an organised rhythm was restored by the first shock (120 J) in 23% of cases (LOE 1).[62] Success rate for the termination of VF at 5 s was not published for this waveform.

Monophasic waveform (damped sinusoid or truncated exponential). Evidence from three studies of monophasic defibrillation suggested equivalent outcomes with lower and higher starting energies (LOE 1[77]; LOE 2[78,79]).

Myocardial damage associated with higher energy level shocks. Several animal studies have suggested the potential for myocardial damage with higher energy shocks using BTE or monophasic waveforms (LOE 5).[36,80–82] Human studies involving BTE waveforms[74,83] with energy levels up to 360 J have not shown harm as indicated by biomarker levels, ECG findings, and ejection fractions.

Treatment recommendation

It is reasonable to start at a selected energy level of 150 J to 200 J for a BTE waveform for defibrillation of pulseless VT/VF cardiac arrest. There is insufficient evidence to determine the initial energy levels for any other biphasic waveform. Although evidence is limited, because of the lower total shock success for monophasic defibrillation, initial and subsequent shocks using this waveform should be at 360 J.

One-shock compared with three-stacked shock protocols

Consensus on science

One study showed no survival benefit from a protocol that included a single-shock protocol compared to a three-shock protocol (LOE 1).[84] Evidence from three pre–post design studies suggested significant survival benefit with a single-shock defibrillation protocol compared with three-stacked shock protocols (LOE 3).[85–87] However, these studies included confounders related to pre–post design and the multiple interventions that were included as part of the defibrillation protocol. Another pre–post study, with fewer confounding factors, showed a significantly lower hands-off ratio (i.e., percentage of total CPR time when no compressions were provided) with the one-shock protocol but no statistical difference in survival (LOE 3).[88]

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Treatment recommendation

When defibrillation is required, a single shock should be provided with immediate resumption of chest compressions after the shock. Chest compressions should not be delayed for rhythm reanalysis or pulse check immediately after a shock. CPR should not be interrupted until rhythm reanalysis is undertaken.

Fixed versus escalating defibrillation energy protocol[ALS-E-032B]

In adult cardiac arrest (OHCA, IHCA) does the use of an escalating defibrillation energy protocol, compared with a fixed-energy protocol, improve outcomes (e.g., ROSC)?

Consensus on science

One randomised trial (LOE 1)[74] of 150-J fixed versus 200-J to 300-J to 360-J shocks and one LOE 2 study[75] of 150-J fixed versus 100-J to 150-J to 200-J shocks supported the use of an escalating-energy biphasic defibrillation protocol compared with a fixed-dose defibrillation protocol. In one study (escalating 200-J to 200-J to 360-J shocks), the success rate of defibrillation for recurrent VF declined with the number of recurrences (LOE 4).[93] However, these studies were not designed to demonstrate an improvement in the rate of ROSC or survival to hospital discharge. One study of fixed-dose biphasic defibrillation suggested that defibrillation success improved with three shocks (LOE 4).[89] All of these studies were done with the three-shock protocol (before the change in Guidelines 2005).

Treatment recommendation

For second and subsequent biphasic shocks the same initial energy level is acceptable. It is reasonable to increase the energy level when possible.

Shock using manual versus semiautomatic mode[ALS-E-034B]

In adult cardiac arrest (OHCA, IHCA) does the use of an AED or a multifunctional defibrillator in automatic mode, compared with standard resuscitation (using manual defibrillation), improve outcomes (e.g., successful defibrillation, ROSC, survival)?

Consensus on science

Modern defibrillators can be operated in both manual and semiautomatic (AED-similar) modes. However, few studies compare these two options. One randomised controlled study showed no significant difference in survival-to-hospital-discharge rate but significant reduction in time to first shock in the AED group versus the manual group (1.1 min versus 2.0 min) (LOE 1).[94] One good concurrent controlled OHCA study in 36 rural communities showed no improvements in ROSC, survival, or neurological outcome but significantly shorter times to first shock and higher VF conversion rates when paramedics used AEDs in semiautomatic mode compared with manual mode (LOE 2).[95] One retrospective study demonstrated no improvement in survival to hospital discharge for adult IHCA when comparing AED with manual defibrillators (LOE 4).[96] In patients with initial asystole or pulseless electric activity (PEA), AEDs were associated with a significantly lower survival (15%) compared with manual defibrillators (23%, $P = 0.04$).[96]

In a study of three different EMS systems and one in-hospital centre, manual mode of defibrillation was associated with a lower total hands-off ratio (i.e., percentage of total CPR time when no compressions were provided) than AED mode (LOE 3).[97] However, more shocks were delivered inappropriately by rescuers using manual defibrillators (26% manual versus 6% AEDs). A randomised manikin study simulating cardiac arrest showed a lower hands-off ratio, mainly due to a shorter preshock pause, when trained paramedics used the defibrillator in manual mode compared with semiautomatic mode (LOE 5).[98] More inappropriate shocks (12% versus 0) were delivered in manual mode. All episodes of VF were detected and shocked appropriately.

A shorter preshock pause and lower total hands-off ratio increased vital organ perfusion and the probability of ROSC (LOE 5).[99–101]

Treatment recommendation

No significant survival differences have been demonstrated between defibrillation in semiautomatic and manual modes during out-of-hospital or in-hospital resuscitation; however, the semiautomatic mode is preferred because it is easier to use and may deliver fewer inappropriate shocks.

Trained personnel may deliver defibrillation in manual mode. Use of the manual mode enables chest compressions to be continued during charging, thereby minimizing the preshock pause. When using the defibrillator in manual mode, frequent team training and ECG recognition skills are essential.

The defibrillation mode that results in the best outcome will be influenced by the system of care and by provider skills, training, and ECG recognition.

Cardioversion strategy in atrial fibrillation[ALS-E-038]

In adult patients in a shockable nonarrest rhythm requiring cardioversion (in- or out-of-hospital) does any specific cardioversion strategy, compared with standard management (or other specific cardioversion strategy), improve outcomes (e.g., termination of rhythm)?

Consensus on science

Twenty-two studies have compared specific cardioversion strategies (e.g., monophasic versus biphasic defibrillators and different energy levels) administered by cardiologists in the hospital setting to patients with atrial fibrillation (both acute and chronic) (LOE 1[14,17,26,27,31,102–115]; LOE 2[116,117]). Most of these studies documented that biphasic shocks were more effective than monophasic shocks for cardioversion.

Studies with varying strategies (fixed and escalating) and energy levels all resulted in high cardioversion rates for a variety of biphasic waveforms, with no clear evidence of superiority. For monophasic defibrillation, higher initial energy levels (360 J) were associated with higher cardioversion rates and less total energy used than energy levels escalating from lower to higher. Body weight may affect cardioversion success, and one study suggested that initial shock should be 200 J for patients <90 kg and 360 J for patients >90 kg (LOE 1).[118] In general, increased total energy use was associated with more dermal injury and postprocedural pain (LOE 1).[103,112,119]

Treatment recommendation

Biphasic defibrillators are preferred for cardioversion of atrial fibrillation. There is no evidence to recommend a specific waveform, energy level, or strategy (fixed versus escalating) when using biphasic defibrillators. For monophasic defibrillators, a high initial energy (360 J) seems preferable.

Special circumstances

Some special circumstances, such as whether pacing is ever indicated during cardiac arrest or how to respond in cardiac arrest if the patient has a pacemaker or an internal defibrillator, are presented and discussed in this section.

Pacing (e.g., transcutaneous [TC], transvenous [TV], needle, and fist)[ALS-E-031]

In adult cardiac arrest (OHCA, IHCA) does the use of pacing (e.g., TC, TV, and needle), compared with standard resuscitation (or no pacing), improve outcomes (e.g., ROSC, survival)?

Consensus on science

Four studies addressed the efficacy of pacing in cardiac arrest (LOE 2[120–122]; LOE 3[123]). These studies found no benefit from routine pacing in cardiac arrest patients. Use of pacing (e.g., TC, TV, and needle) in cardiac arrest (in- or out-of-hospital) did not improve ROSC or survival. There was no apparent benefit related to the time at which pacing was initiated (early or delayed in established asystole), location of arrest (out-of-hospital or in-hospital), or primary cardiac rhythm (asystole or PEA). Five case series (LOE 4),[124–128] a review with two additional case reports,[129] and a moderate-sized case series (LOE 4)[130] support percussion pacing in p-wave asystolic cardiac arrest/complete heart block or haemodynamically unstable patients with bradycardia. In these reports, sinus rhythm with a pulse was restored using different pacing techniques.

Treatment recommendation

Electric pacing is not effective as routine treatment in patients with asystolic cardiac arrest. Percussion pacing is not recommended in cardiac arrest in general. However, fist pacing may be considered in haemodynamically unstable bradyarrhythmias until an electric pacemaker (TC or TV) is available. The use of epicardial wires to pace the myocardium following cardiac surgery is effective and is discussed elsewhere.

Implantable cardioverter defibrillator (ICD) or pacemaker[ALS-E-039B]

In adult patients with an ICD or pacemaker who are in a shockable rhythm requiring defibrillation/cardioversion (in- or out-of-hospital) does any unique or modified defibrillation/cardioversion strategy, compared with standard management, improve outcomes (e.g., termination of rhythm, ROSC)?

Consensus on science

Two case series reported pacemaker or ICD malfunction after external defibrillation when the pads were placed in close proximity to the device generator (LOE 4).[131,132] One small study on atrial cardioversion demonstrated that positioning the pads on the chest at least 8 cm from the device generator did not produce significant damage to pacing sensing and capturing (LOE 4).[131]

One case report suggested that pacemaker spikes generated by devices programmed to unipolar pacing may confuse AED software and emergency personnel and may prevent the detection of VF (LOE 4).[133]

Treatment recommendation

In patients with an ICD or a permanent pacemaker, the placement of paddles/pads should not delay defibrillation. When treating an adult with a permanent pacemaker or an ICD, the defibrillator paddle/pad should be placed on the chest wall ideally at least 8 cm from the generator position.

The anterior–posterior and anterior–lateral paddle/pad placements on the chest are acceptable in patients with a permanent pacemaker or ICD.

Defibrillation-related topics

Predicting success of defibrillation and outcome (VF waveform analysis)[ALS-D&P-015B]

VF waveform analysis has been shown to correlate with myocardial perfusion/coronary perfusion pressure. In theory waveform analysis could be a tool for predicting outcome of defibrillation and therefore indicate the optimal time for shock delivery.

In adult cardiac arrest (OHCA, IHCA) does the use of a technique for prediction of the likelihood of success of defibrillation (analysis of VF, etc.), compared with standard resuscitation (without such prediction), improve outcomes (e.g., termination of rhythm, ROSC)?

Consensus on science

Retrospective analysis of the VF waveform in multiple clinical (LOE 1[134,135]; LOE 4[136–154]; LOE 5[155,156]) and animal studies (LOE 5)[147,157–170] and theoretical models suggested that it is possible to predict the success of defibrillation from the fibrillation waveform with varying reliability. One animal study was neutral (LOE 5).[171] No human studies have specifically evaluated whether treatment altered by predicting success of defibrillation can improve successful defibrillation, ROSC, or survival from cardiac arrest. Multiple waveform parameters have been examined without consensus on the most important parameters to predict outcome.

Treatment recommendation

There is insufficient evidence to support routine use of VF waveform analysis to guide defibrillation management in adult cardiac arrest in- or out-of-hospital.

Defibrillation in the immediate vicinity of supplementary oxygen[ALS-E-035A,ALS-E-035B]

In adults and children in cardiac arrest (OHCA, IHCA) requiring defibrillation, does the presence of supplementary oxygen in the immediate vicinity, compared with no supplementary oxygen, increase the risk of fire with defibrillation attempts?

Consensus on science

Four case reports involving adults (LOE 4)[172–175] and one case report involving a neonate (LOE 4)[176] described fires caused by sparks generated during defibrillation attempts when paddles were used in the vicinity of high-flow (>10 L min^{-1}) oxygen. There are no case reports of fires caused by sparking when shocks were delivered using adhesive pads. In two manikin studies the oxygen concentration in the zone of defibrillation was not increased when ventilation devices (bag-valve device, self-inflating bag, and Hamilton Viola ventilator) were left attached to a tracheal tube or when the oxygen source was vented at least one metre behind the patient's mouth (LOE 5).[177,178] One study described higher oxygen concentrations and longer washout periods when oxygen was administered in confined spaces without adequate ventilation (LOE 5).[179]

Treatment recommendation

Rescuers should take precautions to minimise sparking (by paying attention to pad/paddle placement, contact, etc.) during attempted defibrillation. Rescuers should try to ensure that defibrillation is not attempted in an oxygen-enriched atmosphere (e.g., when high-flow oxygen is directed across the chest).

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Appendix A. Disclosures

Writing group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Ian Jacobs	University of Western Australia; Emergency Med. Teaching and Research-Professor; AHA-Evidence Eval. Expert	[a](a) National Health and Medical Research Council (b) The Department of Health – Western Australia (c) The National Heart Foundation of Australia	[a]Funds to the Discipline of Emergency Medicine – University of Western Australia from the Ambulance Service –Western Australia and Laerdal (Australia) to maintain the Cardiac Arrest Registry for Western Australia. Our role is to independently maintain, analyze and report outcomes of cardiac arrest in Western Australia. I oversee the operation of the registry and reporting of outcomes. These funds are not used to provide any direct or indirect salary or other financial support	None	None	None	None
Kjetil Sunde	Oslo University Hospital Ulleval	None	None	None	None	None	None
Charles D. Deakin	Southampton University Hospital NHS Trust–Doctor	None	None	None	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing – Professor; American Heart Association ECC Programs – Consultant, Senior Science Editor; [b]I receive significant compensation from the AHA for my work as a Senior Science Editor. Compensation is to ensure that I have the time to edit the Consensus on Science with Treatment Recommendations and the AHA Guidelines for CPR and ECC	None	None	None	None	None	None

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Richard E. Kerber	University of Iowa Hospital: Hospital-teaching – Physician	None	None	[b]Occasional lectures at Cardiology Grand Rounds at other institutions	[b]General Electric stock	[b]One-time consultant visit to Philips Defibrillator Division	[b]Occasional expert witness in malpractice litigation. These do not at present involve cardiac drugs or devices [b]DSMB member for "CIRC" clinical trial sponsored by Zoll medical
Rudolph W. Koster	Academic Medical Center: Academic hospital – clinical staff cardiologist	[a]Medtronic Physio-Control 2005–2009: salary support (€80.000-annually) for the maintenance of infrastructure of data collection and analysis of OOH cardiac arrest in the area of North-Holland. Money was donated to the hospital. I had no personal financial benefit from this support. Electrodes used by first responders during an embedded randomized trial (conducted under the umbrella of the infrastructural support) were replaced free of charge	[a]Medtronic Physio-Control 2005–2009: salary support (€80.000,-annually) for the maintenance of infrastructure of data collection and analysis of OOH cardiac arrest in the area of North-Holland. Money was donated to the hospital. I had no personal financial benefit from this support. Electrodes used by first responders during an embedded randomized trial (conducted under the umbrella of the infrastructural support) were replaced free of charge	None	None	None	None
Laurie J. Morrison	St. Michaels Hospital; Clinician	[b]Laerdal Foundation Centre Grant-infrastructure support no salary support	None	None	None	None	None
Jerry P. Nolan	Royal United Hospital NHS Trust – Consultant in Anesthesia and Critical care Medicine	None	None	None	None	None	None
Michael R. Sayre	The Ohio State University – Associate Professor	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Significant.

[b] Modest.

Worksheet collaborator disclosures

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Mark Angelos	The Ohio State University – Professor	None	None	None	None	None	None
Michael Baubin	General Hospital Innsbruck – senior anesthetist	[a]€99,000 from the Austrian National Bank for 2 years for the project "Satisfaction in the Emergency Medicine" for the institution for personal costs of other involved people [b]Personally – I receive no money from these grants	None	[b]Last year for an invited lecture on "Quality Management in Emergency Medicine"	None	None	None
M. Fernanda Bellolio	Mayo Clinic – Senior Research Fellow	None	None	None	None	None	None
Paul Berlin	Gig Harbor Fire & Medic One – Medical Division Chief	None	None	[b]Honoraria received for teaching at Physio-Control learning centre at EMS Today Conference 2009 on topics not directly related to Physio-Control products	None	None	None
Richard Bradley	The University of Texas Health Science Center at Houston – Associate Professor of Emergency Medicine; Texas Air National Guard – Residency Trained Flight Surgeon	None	None	None	None	None	[b]Volunteer member of the American Red Cross Council on First Aid, Safety and Preparedness, serve as the CPR sub-council chairman; also a volunteer board member of the Greater Houston Chapter of the Am.Red Cross
Steven Bradley	University of Washington – Cardiology Fellow	None	None	None	None	None	None
Saul Drajer	Clinica de la Esperanza: A 130 bed gen. hospital located in Buenos Aires, Argentina and affiliated to the Maimonides Univ. School of Medicine, with CPR teaching capabilities – Gen. Director	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Trygve Eftestol	Univ of Stavanger Acad. Institute, Prof I Tech	[a]I have been collaborator in a project receiving grant corresponding to the funding of one PostDoc position for 14.5 months from the Laerdal Foundation [a]I am collaborator in a project receiving grant corresponding to the funding of one PostDoc position for 30 months from the Norwegian Air Abulance Foundation	None	None	None	None	None
Chokoh Genka	Saiseika Kawaguchi General Hospital – Chief of Cardiology	None	None	None	None	None	None
Erik P. Hess	Mayo Clinic: Department of Emergency Medicine – Senior Associate Consultant	None	None	None	None	None	None
Toshihiko Mayumi	Nagoya University Hospital: Doctor – Assistant professor	None	None	None	None	None	None
Saman Nazarian	Johns Hopkins University – Assistant Professor of Medicine	None	None	None	None	None	None
Mark Peele	Department of Defense: Clinical cardiac eletrophysiologist – Cardiolo-gist/Electrphyiologist	None	None	None	None	None	None
Claudia Ranniger	George Washington University Medical Faculty Associates-staff emergency physician; George Washington University: Direct teaching activities in the GWU Medical Center Simulation Center – Medical Director, Simulation Center	[a]PI for AHA-sponsored grant to investigate (1) training modalities for ACLS; (2) ACLS skills retention; (3) ACLS checklist utilization. Total grant amount $300,000 to GWU. Grant concluded June 2009	None	None	None	None	None
Giuseppe Ristagno	Weil Institute of Critical Care Medicine – Assistant Professor	None	None	None	None	None	None
Comilla Sasson	University of Michigan – Clinical Lecturer Department of Emergency Medicine	[a]Robert Wood Johnson Clinical Scholars Program-3 year research fellowship	None	None	None	None	None
Shijie Sun	Weil Institute of Critical Care Medicine – Professor	None	None	None	None	None	None

This table represents the relationships of worksheet collaborators that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all worksheet collaborators are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Significant.

[b] Modest.

Appendix B. Evidence-based worksheets for part 6: defibrillation: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations

Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-D&P-015B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of a technique for prediction of the likelihood of success of defibrillation (analysis of VF, etc.) (I) compared with standard resuscitation (without such prediction) (C), improve outcomes (e.g., successful defibrillation, ROSC, survival) (O)	Waveform analysis for predicting successful defibrillation	Mark Angelos, Trygve Eftestol	http://circ.ahajournals.org/site/C2010/ALS-D-P-015B.pdf
ALS	ALS-E-030A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific paddle/pad size/orientation and position (I) compared with standard resuscitation or other specific paddle/pad size/orientation and position) (C), improve outcomes (e.g., successful defibrillation, ROSC, survival) (O)	Paddle size and placement for defibrillation	Michael Baubin, Comilla Sasson	http://circ.ahajournals.org/site/C2010/ALS-E-030A.pdf
ALS	ALS-E-031	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of pacing (e.g., TV, TC, needle) (I) compared with standard resuscitation (or no pacing) (C), improve outcomes (e.g., ROSC, survival) (O)	Pacing for cardiac arrest	M. Fernanda Bellolio, Paul Berlin, Erik Hess	http://circ.ahajournals.org/site/C2010/ALS-E-031.pdf
ALS	ALS-E-032B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of an escalating defibrillation energy protocol (I) when compared with a fixed energy protocol (C) increase outcome (e.g., return of spontaneous circulation) (O)?	Escalating versus fixed defibrillation energy	Steven Bradley	http://circ.ahajournals.org/site/C2010/ALS-E-032B.pdf
ALS	ALS-E-033B	In adult cardiac arrest due to VF or pulseless VT (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific defibrillation strategy (I) compared with standard management (or other defibrillation strategy) (C), improve outcomes (e.g., termination of rhythm, ROSC, survival) (O)?	Defibrillation strategies for VF or VT	Steven Bradley	http://circ.ahajournals.org/site/C2010/ALS-E-033B.pdf
ALS	ALS-E-034B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of an AED or a multifunctional defib in automatic mode (I) compared with standard resuscitation (using manual defibrillation) (C), improve outcomes (e.g., successful defibrillation, ROSC, survival) (O)?	AED versus manual defibrillator	Giuseppe Ristagno	http://circ.ahajournals.org/site/C2010/ALS-E-034B.pdf
ALS	ALS-E-035A	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P) requiring defibrillation, does the presence of supplementary oxygen in the immediate vicinity (I) compared with no supplementary oxygen (C), increase the risk of fire with defibrillation attempts (O)	Risk of fire with oxygen and defibrillation	Jerry Nolan	http://circ.ahajournals.org/site/C2010/ALS-E-035A.pdf
ALS	ALS-E-035B	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P) requiring defibrillation, does the presence of supplementary oxygen in the immediate vicinity (I) compared with no supplementary oxygen (C), increase the risk of fire with defibrillation attempts (O)	RIsk of fire with oxygen and defibrillation	Claudia Ranniger	http://circ.ahajournals.org/site/C2010/ALS-E-035B.pdf
ALS	ALS-E-036	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific composition of conductive material (I) compared with standard conductive material (C), improve transthoracic impedance (O)	Conductive materials for defibrillation	Saul Drajer, Richard Kerber	http://circ.ahajournals.org/site/C2010/ALS-E-036.pdf
ALS	ALS-E-037A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of self-adhesive defibrillation pads (I) compared with paddles (C), improve outcomes (e.g., successful defibrillation, ROSC, survival) (O)?	Adhesive pads versus paddles for defibrillation	Chokoh Genka, Toshihiko Mayumi	http://circ.ahajournals.org/site/C2010/ALS-E-037A.pdf
ALS	ALS-E-038	In adult patients in a shockable non-arrest rhythm requiring cardioversion (prehospital or in-hospital) (P), does the any specific cardioversion strategy (I) compared with standard management (or other cardioversion strategy) (C), improve outcomes (e.g., termination of rhythm) (O)	Cardioversion strategies	Richard Bradley, Shijie Sun	http://circ.ahajournals.org/site/C2010/ALS-E-038.pdf
ALS	ALS-E-039B	In adult patients with an ICD or pacemaker and who are in a shockable rhythm requiring defibrillation/cardioversion (prehospital or in-hospital) (P), does the any unique or modified cardioversion/defibrillation strategy (I) compared with standard management (C), improve outcomes (e.g., termination of rhythm, ROSC) (O)	Cardioversion strategies with ICD or pacemakers	Saman Nazarian, Mark Peele	http://circ.ahajournals.org/site/C2010/ALS-E-039B.pdf
BLS	BLS-024A	In adult and paediatric patients with cardiac arrest due to VF (prehospital or in-hospital) (P), does the use of CPR before defibrillation (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g., ROSC, survival)?	CPR prior to defibrillation	Ian Jacobs	http://circ.ahajournals.org/site/C2010/BLS-024A.pdf
BLS	BLS-024B	In adult and paediatric patients with cardiac arrest due to VF (prehospital or in-hospital) (P), does the use of CPR before defibrillation (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g., ROSC, survival)?	CPR prior to defibrillation	Rudolph W. Koster	http://circ.ahajournals.org/site/C2010/BLS-024B.pdf

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Part 7: CPR techniques and devices 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations☆

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The success of any cardiopulmonary resuscitation (CPR) technique or device depends on the education and training of the rescuers as well as on resources (including personnel). In the hands of some groups, novel techniques and adjuncts may produce better short- or long-term outcomes than standard CPR. However, a device or technique that provides good-quality CPR when used by a highly trained team or in a test setting may show poor quality and create frequent interruptions in CPR when used in an uncontrolled clinical setting.[1]

While no circulatory adjunct is currently recommended instead of manual CPR for routine use, some circulatory adjuncts *are* being routinely used in both out-of-hospital and in-hospital resuscitation. If a circulatory adjunct is used, rescuers should be well trained and a program of continuous surveillance should be in place to ensure that use of the adjunct does not adversely affect survival.

The following CPR techniques and devices were reviewed during the 2010 International Consensus Conference. It should be noted that interposed abdominal compression (IAC) has not been studied in humans since 1994 and active compression–decompression (ACD) has not been studied in humans since 2003. Therefore these techniques have not been evaluated against the international resuscitation guideline changes of 2000 and 2005 for IAC and 2005 for ACD.

Interposed abdominal compression (IAC)-CPR[ALS/BLS-CPR&A-082A]

Consensus on science

Two randomised controlled trials in in-hospital cardiac arrests, showed improved return of spontaneous circulation (ROSC) and survival to hospital discharge when IAC-CPR was compared with standard CPR (LOE 1[2]; LOE 2[3]). However, there were no differences in neurologically intact survival.

One randomised controlled trial in out-of-hospital cardiac arrest was unable to show any consistent benefits when IAC-CPR was compared with standard CPR (LOE 2).[4]

Evidence from LOE 3[5,6] and LOE 5[7] in-hospital studies suggested better or neutral[8,9] haemodynamics with IAC-CPR compared with standard CPR.

Treatment recommendation

There is insufficient evidence to support or refute the use of IAC-CPR.

Active compression–decompression (ACD)-CPR[ALS/BLS-CPR&A-084A]

Consensus on science

Five randomised controlled trials (LOE 1)[10–14] and three controlled trials (LOE 2)[15–17] failed to show a difference in ROSC or survival with use of ACD-CPR compared with standard CPR.

Six studies (LOE 2)[18–23] demonstrated improved ROSC or survival to hospital discharge although there were no statistically significant differences in neurologically intact survival.

A meta-analysis[14] of two trials (826 patients) comparing ACD-CPR with standard CPR after in-hospital cardiac arrest (IHCA) did not detect a significant increase in rates of immediate survival or survival to hospital discharge.

Treatment recommendation

There is insufficient evidence to support or refute the use of ACD-CPR.

Open-chest CPR[ALS-CPR&A-004A,ALS-CPR&A-004B]

Consensus on science

There are no published randomised controlled trials and very limited data in humans comparing open-chest CPR to standard CPR in cardiac arrest. One retrospective clinical trial (LOE 3)[24] demonstrated that ROSC was improved by open-chest CPR in out-of-hospital cardiac arrest. One case series in victims of out-of-hospital cardiac arrest who had failed standard CPR (LOE 4)[25]

☆ *Note from the Writing Group*: Throughout this article, the reader will notice combinations of superscripted letters and numbers (e.g. "Open-Chest CPR[ALS-CPR&A-004A,ALS-CPR&A-004B]"). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.

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reported ROSC in 13 of 33 highly selected patients; two survived to hospital discharge.

Multiple animal studies (LOE 5)[26–44] using a variety of endpoints demonstrated benefit with open-chest CPR.

Treatment recommendation

There is insufficient evidence to support or refute the routine use of open-chest CPR in cardiac arrest.

Load distributing band (LDB)-CPR[ALS/BLS-CPR&A-086A, ALS/BLS-CPR&A-086B]

Consensus on science

One multicentre RCT in over 1000 adults documented no improvement in 4-h survival and significantly worse neurological outcome when LDB-CPR administered by EMS providers was compared with traditional CPR for out-of-hospital cardiac arrest of presumed cardiac origin (LOE 1).[45] However, a post hoc analysis of this study revealed significant heterogeneity among study sites (LOE 1).[46]

In one LOE 3 study,[47] the use of LDB-CPR was associated with lower odds of 30-day survival (OR 0.4). However, when a smaller (77-patient) subgroup of LDB-CPR-treated patients was analyzed against concurrent controls, an increased rate of ROSC was noted.[47]

Other nonrandomised human series (LOE 3) have reported increased rates of sustained ROSC[48,49] and increased survival to discharge[49] following out-of-hospital cardiac arrest and improved haemodynamics following failed resuscitation from in-hospital cardiac arrest (LOE 4).[50] In a prospective before-and-after study (LOE 3),[51] the mean no-flow ratio with manual CPR was 0.28 in the first 5 min of CPR compared with 0.40 with LDB-CPR. However, between 5 and 10 min, no-flow time was 0.34 with manual CPR and 0.21 with LDB-CPR.

Evidence from both clinical (LOE 1)[45,46] and simulation (LOE 5)[52] studies suggested that site-specific factors may influence resuscitation quality and device efficacy.

A case report documented successful performance of a computed tomography (CT) scan while LDB-CPR was used (LOE 4).[53]

Treatment recommendation

There are insufficient data to support or refute the routine use of LDB-CPR instead of manual CPR. It may be reasonable to consider LDB to maintain continuous chest compression while undergoing CT scan or similar diagnostic studies, when provision of manual CPR would be difficult.

Mechanical (piston)-CPR[ALS/BLS-CPR&A-083A, ALS/BLS-CPR&A-083B]

Consensus on science

When a piston-CPR device was compared with manual CPR, one RCT documented no improvement in ROSC or survival among adults in cardiac arrest (LOE 1).[54]

Supportive data from one prospective, randomised crossover-design study (LOE 1)[55] and one paired-cohort study (LOE 2)[56] documented that the use of a piston-CPR device improved haemodynamics during CPR in adult cardiac arrest victims.

One prospective pseudorandomised trial documented improvement in haemodynamic variables during CPR in adult cardiac arrest victims but no improvement in ROSC or survival (LOE 2).[57]

Data from one prospective cohort study comparing the use of a piston-CPR device with manual CPR documented that the use of a piston-CPR device increased interruption in CPR because time was required to set up and remove the device from patients during transportation in adult OHCA (LOE 2).[58]

Treatment recommendation

There is insufficient evidence to support or refute the use of piston-CPR instead of manual CPR for adult victims of cardiac arrest.

Lund University cardiac arrest system (LUCAS) CPR[ALS/BLS-CPR&A-085A, ALS/BLS-CPR&A-085B]

Consensus on science

There are no RCTs evaluating the LUCAS device in human cardiac arrest.

One study using concurrent controls in witnessed out-of-hospital cardiac arrest was unable to show any benefit (ROSC, survival to hospital, or survival to hospital discharge) with the use of the LUCAS device over the use of standard CPR (LOE 2).[59]

One postmortem study showed similar injuries with LUCAS-CPR and standard CPR (LOE 2).[60]

Six case series involving approximately 200 patients have reported variable success in use of the LUCAS device when implemented after an unsuccessful period of manual CPR (LOE 4).[61-66]

Three adult human case reports (LOE 4),[62,67,68] three adult human case series (LOE 4),[63,66,69] and one animal study (LOE 5)[68] reported that the use of a mechanical chest-compression device in cardiac arrest during percutaneous coronary intervention (PCI) maintained circulation and enabled the procedure to be completed. A small number of patients in the case series survived.

Two case reports demonstrated that a CT scan could be performed during CPR with the LUCAS device (LOE 4).[53]

Treatment recommendation

There are insufficient data to support or refute the use of LUCAS-CPR instead of manual CPR. It may be reasonable to consider LUCAS-CPR to maintain continuous chest compression while undergoing CT scan or similar diagnostic studies, when provision of manual CPR would be difficult.

Impedance threshold device (ITD)[ALS/BLS-CPR&A-081A, ALS/BLS-CPR&A-081B]

Consensus on science

One meta-analysis that pooled the data from both conventional CPR and ACD-CPR RCTs demonstrated improved ROSC and short-term survival but no significant improvement in either survival to discharge or neurologically intact survival to discharge associated with the use of an ITD in the management of adult OHCA patients (LOE 1).[70]

One RCT suggested that the use of an ITD in combination with ACD-CPR improved 24-h survival and survival to intensive care unit (ICU) admission in adult out-of-hospital cardiac arrest patients, compared with ACD-CPR and a sham ITD (LOE 1).[71] This contrasts with another RCT that compared ITD plus ACD-CPR with ACD-CPR plus a sham ITD, which did not show significant improvement in ROSC or 24-h survival with use of the ITD (LOE 1).[72]

One RCT reported that the use of an ITD in combination with standard CPR did not significantly improve ROSC, 24-h survival, or survival to ICU admission in adult out-of-hospital cardiac arrest, compared with CPR and a sham ITD (LOE 1).[73]

One RCT comparing ACD-CPR plus ITD with CPR in adult out-of-hospital cardiac arrest showed improved ROSC and 24-h survival rates associated with ACD-CPR plus ITD, but no significant improvement in rates of hospital discharge or intact neurological survival to hospital discharge (LOE 1).[74]

One prospective cohort study (with historical control) of CPR plus ITD versus CPR without ITD in out-of-hospital cardiac arrest

reported improved survival to emergency department (ED) admission for patients presenting in any rhythm (LOE 3).[75]

Three cohort studies comparing CPR using the *2005 ECC Guidelines* plus ITD, with historic controls of CPR using the 2000 Guidelines, demonstrated improved survival to hospital discharge in out-of-hospital cardiac arrest (LOE 3).[76–78] It was not possible to determine the relative contribution of the ITD to the improved outcome.

In a porcine model of cardiac arrest, eight studies demonstrated improved haemodynamic variables during CPR with use of the ITD (LOE 5).[79–86] An additional three animal studies (LOE 5)[87–89] showed no difference in survival or in any haemodynamic variable, and two animal studies (LOE 5)[88,90] reported evidence of decreased ROSC, 20-min survival, and arterial oxygen saturation associated with the use of an ITD.

Treatment recommendation

There are insufficient data to support or refute the use of the ITD.

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Appendix A. Evidence-based worksheets for Part 7: CPR techniques and devices: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations

Task force	WS ID	PICO title	Short title	Authors	URL
ALS/BLS	ALS/BLS-CPR&A-081A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of a ITD (I) compared with no ITD (C), improve any outcomes (e.g. ROSC, survival) (O)?	Impedence threshold device	Suzanne R. Davies, Paul M. Middleton	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-081A.pdf
ALS/BLS	ALS/BLS-CPR&A-081B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of a ITD (I) compared with no ITD (C), improve any outcomes (e.g. ROSC, survival) (O)?	Impedence threshold device	Syed Sameer Ali	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-081B.pdf
ALS/BLS	ALS/BLS-CPR&A-082A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of interposed abdominal compressions-CPR (I) compared with standard CPR (C), improve any outcomes (e.g. ROSC, survival) (O)?	Interposed abdominal compression CPR	Michael Holzer, Kjetil Sunde	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-082A.pdf
ALS/BLS	ALS/BLS-CPR&A-083A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of a piston-CPR device (e.g. Thumper) (I) compared with manual CPR (C), improve any outcomes (e.g. ROSC, survival) (O)?	Piston (thumper) device CPR	Giuseppe Ristagno	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-083A.pdf
ALS/BLS	ALS/BLS-CPR&A-083B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of a piston-CPR device (e.g. thumper) (I) compared with manual CPR (C), improve any outcomes (e.g. ROSC, survival) (O)?	Piston (thumper) device CPR	Jim McKendry	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-083B.pdf
ALS/BLS	ALS/BLS-CPR&A-084A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of manual ACD-CPR (I) compared with standard CPR (C), improve any outcomes (e.g. ROSC, survival) (O)?	Active compression–decompression device (ACD) CPR	Pierre Carli	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-084A.pdf
ALS/BLS	ALS/BLS-CPR&A-085A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of mechanical compression full (e.g. Lucas) or partial decompression (e.g. US version) (I) compared with manual CPR (C), improve any outcomes (e.g. ROSC, survival) (O)?	Lucas device CPR	Peter T. Morley	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-085A.pdf
ALS/BLS	ALS/BLS-CPR&A-085B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of mechanical compression full (e.g. Lucas) or partial decompression (e.g. US version) (I) compared with manual CPR (C), improve any outcomes (e.g. ROSC, survival) (O)?	Lucas device CPR	Taku Iwami, Chika Nishiyama	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-085B.pdf
ALS/BLS	ALS/BLS-CPR&A-086A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of load distributing band (e.g. Autopulse) (I) compared with manual CPR (C), improve any outcomes (e.g. ROSC, survival) (O)?	Autopulse device CPR	Peter T. Morley	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-086A.pdf

Appendix A *Continued*

Task force	WS ID	PICO title	Short title	Authors	URL
ALS/BLS	ALS/BLS-CPR&A-086B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of load distributing band (e.g. Autopulse) (I) compared with manual CPR (C), improve any outcomes (e.g. ROSC, survival) (O)?	Autopulse device CPR	David Beiser	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-086B.pdf
ALS	ALS-CPR&A-004A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P) including traumatic arrest, does the use of open-chest CPR (I) compared with standard CPR (C), improve any outcomes (e.g. ROSC, survival) (O)	Open-chest CPR	Sten Rubertsson	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-004A.pdf
ALS	ALS-CPR&A-004B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P) including traumatic arrest, does the use of open-chest CPR (I) compared with standard CPR (C), improve any outcomes (e.g. ROSC, survival) (O).	Open-chest CPR	Mark S. Link	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-004B.pdf

Disclosures.

CoSTR Part 7: writing group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Michael Schuster	Self-employed—emergency physician	None	None	None	None	None	None
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Charles D. Deakin	Southampton University Hospital NHS Trust—Doctor	None	None	None	None	None	None
Monica E. Kleinman	Children's Hospital Anesthesia Foundation: Non-profit health care organization—Senior Associate in Critical Care Medicine	None	None	None	None	None	None
Rudolph W. Koster	Academic Medical Center—clinical staff cardiologist	[a]Zoll Medical for study of the safety of the Autopulse automated chest compression device. Funded to the hospital and limited to direct study costs without any personal financial consequence [a]Jolife for the study of the Lucas automated chest compression device. Money is funded to the hospital and limited to direct study costs without any personal financial consequence	[a]Zoll Medical: two Autopulse devices on loan to the hospital for safety study [a]Jolife: two Lucas devices on loan to the hospital for safety study Phillips: one MRX chest compression feedback device on loan to the hospital for safety study purposes	None	None	None	None
Laurie J. Morrison	St. Michael's Hospital; clinician scientist	[a]Laerdal Foundation Centre Grant—infrastructure support without salary support	None	None	None	None	None
Jerry P. Nolan	Royal United Hospital NHS Trust: Consultant in Anaesthesia and Critical Care	None	None	None	None	None	None
Michael R. Sayre	The Ohio State University—Associate Professor	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

CoSTR Part 7: worksheet collaborator disclosures

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Syed Sameer Ali	Penn State Hershey Medical Center—Critical Care/Resuscitation Fellow	None	None	None	None	None	None
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Michael Holzer	Department of Emergency Medicine, Medical University of Vienna—Specialist in Internal Medicine, Emergency Physician	None	None	None	None	None	None
Taku Iwami	Kyoto University Assistant professor	[b]Laerdal Foundation—Getting research grant [b]Sanofi Aventis Getting donation for clinical research on emergency care	None	None	None	None	None
Mark S. Link	Tufts Medical Center Hospital Physician	None	None	None	None	None	None
Jim McKendry	City of Winnipeg Training Inspector	None	None	None	None	None	None
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Peter T. Morley	Royal Melbourne Hosp; Univ of Melbourne; Director of Medical Education; AHA EEE	None	None	None	None	None	None
Chika Nishiyama	Postgraduate—RN, MPH	None	None	None	None	None	None
Giuseppe Ristagno	Weil Institute of Critical Care Medicine Assistant Professor Mario Negri Institute for Pharmacological Researches Researcher	None	None	None	None	None	None
Sten Rubertsson	Uppsala University—Professor at the Department of Surgical Sciences/Anesthesiology & Intensive Care	[a]I am receiving money from Jolife AB, Lund, Sweden as a consult dealing with their device LUCAS-mechanical chest compressions [a]I am also a PI for ïthe multi-center LINC trial which is a study of out-of-hospital CA victims allocated to either standard ACLS or ACLS including mechanical chest compressions	None	None	None	[a]Advisory board for Covidean regarding VAP	None
Kjetil Sunde	Oslo University Hospital Ulleval Professor and Senior Consultant	None	None	None	None	None	None

This table represents the relationships of worksheet collaborators that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all worksheet collaborators are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

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Part 8: Advanced life support
2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations☆,☆☆

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Introduction

The topics reviewed by the International Liaison Committee on Resuscitation (ILCOR) Advanced Life Support Task Force are grouped as follows: (1) airway and ventilation, (2) supporting the circulation during cardiac arrest, (3) periarrest arrhythmias, (4) cardiac arrest in special circumstances, (5) identifying reversible causes, (6) postresuscitation care, (7) prognostication, and (8) organ donation. Defibrillation topics are discussed in Part 6.

The most important developments and recommendations in advanced life support (ALS) since the 2005 ILCOR review are as follows:

The use of capnography to confirm and continually monitor tracheal tube placement and quality of cardiopulmonary resuscitation (CPR).

More precise guidance on the control of glucose in adults with sustained return of spontaneous circulation. Blood glucose values >10 mmol L^{-1} (>180 mg dL^{-1}) should be treated and hypoglycaemia, avoided.

Additional evidence, albeit lower level, for the benefit of therapeutic hypothermia in comatose survivors of cardiac arrest associated initially with nonshockable rhythms.

Recognition that many of the accepted predictors of poor outcome in comatose survivors of cardiac arrest are unreliable, especially if the patient has been treated with therapeutic hypothermia. There is inadequate evidence to recommend a specific approach to prognosticating poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia.

The recognition that adults who progress to brain death after resuscitation from out-of-hospital cardiac arrest should be considered for organ donation.

The recommendation that implementation of a comprehensive, structured treatment protocol may improve survival after cardiac arrest.

Airway and ventilation

Consensus conference topics related to the management of airway and ventilation are categorised as (1) basic airway devices, (2) cricoid pressure, (3) advanced airway devices, (4) confirmation of advanced airway placement, (5) oxygenation, and (6) strategies for ventilation.

Basic airway devices

Oropharyngeal and nasopharyngeal airways[ALS/BLS-CPR&A-080B]

Consensus on science

Despite frequent successful use of nasopharyngeal and oropharyngeal airways in the management of nonarrest patients, there are no published data on the use of these airway adjuncts during CPR in humans. When bag-mask ventilation was undertaken with an oral airway and compared with no oral airway, one study in anaesthetised patients demonstrated higher tidal volumes (LOE 5).[1]

One study of nasopharyngeal airways in anaesthetised patients showed that nurses inserting nasopharyngeal airways were no more likely than anaesthetists to cause nasopharyngeal trauma

☆ A Spanish translated version of the summary of this article appears as Appendix in the final online version at doi:10.1016/j.resuscitation.2010.08.027.

☆☆ *Note from the writing group*: Throughout this article, the reader will notice combinations of superscripted letters and numbers (e.g., "Cricoid Pressure[ALS-CPR&A-007B]"). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.

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(LOE 5).[2] One study showed that the traditional methods of sizing a nasopharyngeal airway (measurement against the patient's little finger or anterior nares) do not correlate with the airway anatomy and are unreliable (LOE 5).[3] In one report, insertion of a nasopharyngeal airway caused some airway bleeding in 30% of cases (LOE 5).[4] Two case reports reported inadvertent intracranial placement of a nasopharyngeal airway in patients with basal skull fractures (LOE 5).[5,6]

Treatment recommendation

Oropharyngeal and nasopharyngeal airways have long been used in cardiac arrest, despite never being studied in this clinical context. It is reasonable to continue to use oropharyngeal and nasopharyngeal airways when performing bag-mask ventilation in cardiac arrest, but in the presence of a known or suspected basal skull fracture an oral airway is preferred.

Cricoid pressure[ALS-CPR&A-007B]

In adults and children during ventilation and intubation, does the application and maintenance of cricoid pressure, compared to no cricoid pressure, reduce the incidence of aspiration?

Consensus on science

No studies addressing the use of cricoid pressure during cardiac arrest were identified. All the identified studies were conducted under anaesthesia or in awake volunteers, cadavers, or manikins. (All studies are therefore LOE 5 for cardiac arrest.) Cricoid pressure in nonarrest patients may, to some extent, protect the airway from aspiration, but it may also impede ventilation or interfere with insertion of an advanced airway.

The effect of cricoid presssure on gastric inflation during bag-mask ventilation was examined by two adult (LOE 1[7]; LOE 2[8]) and two paediatric studies (LOE 2).[9,10] All showed less gastric inflation with cricoid pressure than without, although all of the studies used ventilation volumes higher than those recommended in cardiac arrest.

Nine studies in nonarrest adult subjects undergoing anaesthesia showed that cricoid pressure impairs ventilation in many patients, increases peak inspiratory pressures, and causes complete obstruction in up to 50% of patients, depending on the amount of cricoid pressure (in the range of recommended effective pressure) that is applied (LOE 1[7,11–13]; LOE 2[14]; LOE 4[8,15–17]).

One study in anaesthetised patients determined that cricoid pressure prevents correct placement and ventilation with the laryngeal tube (LT) (LOE 1).[18] Eight studies in anaesthetised adults showed that when cricoid pressure was used before insertion of a laryngeal mask airway (LMA), there was a reduced proportion of LMAs correctly positioned, an increased incidence of failed insertion, and impaired ventilation once the LMA had been placed (LOE 1[19–23]; LOE 2[24–26]). No significant impairment to tracheal intubation was found by four LOE-1 studies performed in anaesthetised patients,[27–30] while seven LOE-1 studies[19,31–36] and one LOE-2 study[37] did show impairment of intubation with increased time to intubation and decreased intubation success rates. One cadaver study demonstrated a worse laryngoscopic view with the application of cricoid pressure (LOE 5).[38]

Twenty-one manikin studies demonstrated that many providers applied less cricoid pressure than has been shown to be effective (in cadaver studies) whereas many other providers applied more pressure than has been shown to be necessary (and far in excess of the amount of pressure shown to impede ventilation) (LOE 5).[39–59] Four of those studies determined that performance can be improved with training (although many cricoid pressure applications following training remain outside recommended effective pressures).[54–56,59] No study examined if cricoid pressure performance to the required standard could be maintained beyond the immediate post-training period.

Cricoid pressure prevented movement of liquid from the oesophagus into the pharynx in 5 cadaver studies (LOE 5)[60–64]; however, in 1 LOE-2 study[65] of 4891 obstetric patients undergoing anaesthesia, no significant difference was observed in regurgitation rates between patients who received cricoid pressure and those who did not. There are case reports where prevention of aspiration is ascribed to the application of cricoid pressure (LOE 4)[66–68] and other case reports documenting that aspiration occurs despite the application of cricoid pressure (LOE 4).[69–73]

Treatment recommendation

The routine use of cricoid pressure to prevent aspiration in cardiac arrest is not recommended. If cricoid pressure is used during cardiac arrest, the pressure should be adjusted, relaxed, or released if it impedes ventilation or placement of an advanced airway.

Knowledge gaps

Future research should address whether cricoid pressure prevents regurgitation and aspiration, the pressure required to be effective, and effectiveness trials evaluating if it can be done well by responders to a cardiac arrest.

Advanced airway devices

The tracheal tube was once considered the optimal method of managing the airway during cardiac arrest. There is considerable evidence that without adequate training or ongoing skills maintenance, the incidence of failed intubations and complications, such as unrecognised oesophageal intubation or unrecognised dislodgement, is unacceptably high.[74–79] Prolonged attempts at tracheal intubation are harmful if associated with interruption of chest compressions because this will compromise coronary and cerebral perfusion. Alternatives to the tracheal tube that have been studied during CPR include the bag-mask and supraglottic airway devices, such as the laryngeal mask airway, oesophageal–tracheal combitube and laryngeal tube, among others. Studies comparing supraglottic airway to tracheal intubation have generally compared insertion time and ventilation success rates. No study has shown an effect of the method of ventilation on survival. There are no data to support the routine use of any specific approach to airway management during cardiac arrest. The quality of CPR with various advanced airways was not included in the review for 2010. The best technique depends on the precise circumstances of the cardiac arrest, local guidelines, training facilities, and the competence of the rescuer.

Timing of advanced airway placement[ALS-SAM-062A]

In adult cardiac arrest (prehospital or in-hospital), does an alternate timing for advanced airway insertion (e.g., early or delayed), as opposed to standard care (standard position in algorithm), improve outcome (e.g., return of spontaneous circulation [ROSC], survival)?

Consensus on science

One registry study evaluated the impact of timing of advanced airway placement during 25,006 in-hospital cardiac arrests (LOE 2).[80] In this study, earlier time to invasive airway (<5 min) was associated with no improvement in ROSC but improved 24-h survival (NNT = 48). In an urban out-of-hospital setting, intubation in <12 min was associated with better survival than intubation ≥13 min.[81] In an out-of-hospital urban and rural setting, patients intubated during resuscitation had better survival than patients

not intubated;[82] whereas in an in-hospital setting, patients requiring intubation during CPR had worse survival.[83] A recent study found that delayed tracheal intubation bundled with passive oxygen delivery and minimally interrupted chest compressions was associated with improved neurologically intact survival after out-of-hospital cardiac arrest in patients with adult, witnessed, ventricular fibrillation (VF)/ventricular tachycardia (VT).[84] The independent contribution of the timing of the advanced airway was not available in the study.

Treatment recommendation

There is inadequate evidence to define the optimal timing of advanced airway placement during cardiac arrest.

Knowledge gaps

To advance the science in this area we need to define what is "early" and what is "delayed" placement of advanced airways, the superiority of advanced airways over simple bag-mask ventilation, and whether there is any significant difference between the advanced airway types.

Advanced airway versus ventilation with bag-mask[ALS/BLS-CPR&A-088A, ALS/BLS-CPR&A-088B]

In adult cardiac arrest (prehospital, out-of-hospital cardiac arrest [OHCA], in-hospital cardiac arrest [IHCA]), does the use of supraglottic devices, compared with bag-mask alone for airway management, improve any outcomes (e.g., increase ventilation, increase oxygenation, reduce hands-off time, allow for continuous compressions, and/or improve survival)?

Consensus on science

A retrospective case series (LOE 4) comparing a laryngeal mask airway with bag-mask ventilation in cardiac arrest patients demonstrated a regurgitation rate of 3.5% with use of a laryngeal mask airway and 12.4% with use of bag-mask ventilation.[85] When a variety of supraglottic airway devices were compared with bag-mask ventilation in manikin models, 6 studies showed improved ventilation and a decrease in gastric inflation (LOE 5).[86–91] One pseudorandomised and one nonrandomised clinical trial (LOE 2) found no difference in arterial blood gas values or survival rates when a variety of supraglottic airway devices were compared to bag-mask ventilation.[92,93] Three studies performed in manikin models of cardiac arrest (LOE 5)[94–96] found that, compared with a bag-mask, the use of a single-use, disposable laryngeal tube to provide ventilation may decrease no-flow times.

Treatment recommendation

A supraglottic airway device may be considered by healthcare professionals trained in its use as an alternative to bag-mask ventilation during cardiopulmonary resuscitation.

Knowledge gaps

Further data are needed on the adequacy of ventilation with the various supraglottic airway devices if chest compressions are not interrupted; also needed are comparisons of the various supraglottic airway devices with each other and with bag-mask ventilation when used clinically by inexperienced and by experienced providers.

Tracheal intubation versus the combitube/laryngeal mask airway[ALS/BLS-CPR&A-079A, ALS/BLS-CPR&A-079B]

Consensus on science

Nine studies compared a variety of supraglottic airway devices with the tracheal tube during cardiac arrest (LOE 1[97]; LOE 2[98–105]) and a further six studies compared a variety of supraglottic airway devices with the tracheal tube in patients undergoing anaesthesia (LOE 5).[106–111] Overall in these studies the supraglottic airway device performed as well as, or better than, the tracheal tube with respect to successful insertion and/or time to tube insertion or to ventilation. One study retrospectively compared outcomes in cardiac arrest patients treated with an oesophageal–tracheal–combitube or tracheal tube and found no difference in ROSC, survival to admission, or survival to discharge (LOE 2).[104] One study compared survival in cardiac arrests managed with a laryngeal mask airway with an historical control group of cardiac arrests managed with a tracheal tube and found that ROSC was significantly higher in the study period (61% versus 36%) (LOE 3).[105]

Eight manikin studies with simulated cardiac arrest (LOE 5)[89,90,96,112–116] and eight manikin studies without simulated cardiac arrest showed that successful insertion rates and/or time to insertion or to ventilation for a variety of supraglottic airway devices were as good, or better than, for the tracheal tube (LOE 5).[117–124]

Nine studies documented that when a supraglottic airway device is used as a rescue airway after failed tracheal intubation, most patients can be ventilated successfully with the supraglottic airway device (LOE 2[98,99,103]; LOE 3[125–128]; LOE 5[107,129]).

Two studies performed while wearing anti-chemical protective clothing, one randomised crossover trial on anaesthetised patients, and a pseudorandomised study on manikins found increased time to tracheal tube insertion but not to laryngeal mask airway insertion (LOE 5).[108,117]

Three manikin studies comparing a supraglottic airway device with the tracheal tube during ongoing chest compressions demonstrated decreased time to intubation with the supraglottic airway device, as well as reduced no flow time (LOE 5).[96,112,115] One nonrandomised manikin study found that chest compressions caused only a minor increase in time to tracheal intubation but not to supraglottic airway device insertion (LOE 5).[114]

Treatment recommendation

Healthcare professionals trained to use supraglottic airway devices may consider their use for airway management during cardiac arrest and as a backup or rescue airway in a difficult or failed tracheal intubation.

Knowledge gaps

The adequacy of ventilation with supraglottic airway devices during uninterrupted chest compressions is unknown. The performance of the various supraglottic airway devices should be compared with each other and with the tracheal tube when used in cardiac arrest. Use of the supraglottic airway devices by providers of differing experience should also be studied.

Confirming advanced airway placement

Exhaled carbon dioxide detection and oesophageal detection devices[ALS-CPR&A-008A, ALS-CPR&A-008B]

In adult cardiac arrest (out-of-hospital [OHCA], in-hospital [IHCA]), does the use of devices (e.g., CO_2 detection device, CO_2 analyser, or oesophageal detector device), compared with usual management, improve the accuracy of diagnosis of airway placement?

Consensus on science

Two studies of waveform capnography (LOE D2) to verify tracheal tube position in victims of cardiac arrest after intubation

demonstrated 100% sensitivity and 100% specificity in identifying correct tracheal tube placement.[130,131] One of these studies included 246 intubations in cardiac arrest with nine oesophageal intubations,[130] and the other included 51 cardiac arrests with an overall oesophageal intubation rate of 23%,[131] but it is not specified how many of these occurred in the cardiac arrest group. Three studies (LOE D1)[132–134] with a cumulative total of 194 tracheal and 22 oesophageal tube placements demonstrated an overall 64% sensitivity and 100% specificity in identifying correct tracheal tube placement when using the same model capnometer (no waveform capnography) on prehospital cardiac arrest victims. The sensitivity may have been adversely affected by the prolonged resuscitation times and very prolonged transport times of many of the cardiac arrest victims studied. Intubation was performed after arrival at hospital and time to intubation averaged more than 30 min.

Studies of colorimetric end-tidal CO_2 ($ETCO_2$) detectors (LOE D2[135,136]; LOE D4[137–139]; LOE D5[140,141]), the syringe aspiration oesophageal detector device (LOE D1[133]; LOE D4[142]), the self-inflating bulb oesophageal detector device (LOE D1),[132–134] and nonwaveform $ETCO_2$ capnometers (LOE D2[130,143]; LOE D4[137]; LOE D5[141]) showed that the accuracy of these devices is similar to the accuracy of clinical assessment (not uniformly defined across all studies) for confirming the tracheal position of a tracheal tube in victims of cardiac arrest.

Treatment recommendations

Waveform capnography is recommended to confirm and continuously monitor the position of a tracheal tube in victims of cardiac arrest, and it should be used in addition to clinical assessment (auscultation and direct visualization are suggested).

If waveform capnography is not available, a nonwaveform carbon dioxide detector or oesophageal detector device in addition to clinical assessment (auscultation and direct visualization are suggested) is an alternative.

Knowledge gaps

The relationships between $ETCO_2$, time from arrest, and the response time of emergency medical services (EMS) should be determined so that the meaning of a zero reading on waveform capnography can be understood.

Thoracic impedance[ALS-CPR&A-006A, ALS-CPR&A-006B]

In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]), does the use of thoracic impedance, compared with usual management, improve the accuracy of diagnosis of airway placement and adequacy of ventilation?

Consensus on science

Two studies in adults (LOE D5)[144,145] and one study in children (LOE D5)[146] in patients undergoing anaesthesia demonstrated high sensitivity (0.975–1.0) and specificity (0.925–1.0) of thoracic impedance in diagnosing tracheal and oesophageal intubations. One nonrandomised trial in immediately postmortem patients (LOE D2)[147] demonstrated smaller changes in thoracic impedance with oesophageal ventilations than with tracheal ventilations. One study (LOE D2)[148] tested impedance-based ventilation recognition during cardiac arrest with ongoing compressions and was able to detect 90.4% of ventilations with a 95.5% positive predictive value. Two case reports comprising a total of six cardiac arrest patients with ongoing CPR (LOE D3;[149] LOE 4[150]) demonstrated disappearance of ventilation-induced changes in thoracic impedance after oesophageal intubation.

The evidence evaluating the use of thoracic impedance in diagnosing adequacy of ventilation is scant. Supportive evidence from one animal study (LOE D5)[151] demonstrated that the intensity of the thoracic impedance signal was proportional to the observed tidal volumes. An exploratory study conducted in human cardiac arrest patients (LOE D2)[152] demonstrated a strong correlation between thoracic impedance changes and tidal volume changes in the absence of chest compressions, but large variations in measured impedance coefficients were observed.

Treatment recommendation

Thoracic impedance may be used as an adjunctive measure to diagnose airway placement in cardiac arrest patients; however, treatment decisions pertaining to the accuracy of airway placement should not be based solely on thoracic impedance measurements until further study has confirmed the utility and accuracy of such measurements in this population.

Knowledge gaps

More research is needed to clarify the usefulness of thoracic impedance to independently confirm placement of a tracheal tube and adequacy of ventilation during cardiopulmonary resuscitation.

Oxygen

Supplemental oxygen: 100% versus titration[ALS-CPR&A-011A]

In adult cardiac arrest (out-of-hospital [OHCA], in-hospital [IHCA]), does the use of titrated oxygen during cardiac arrest, compared with the use of 100% oxygen, improve outcome (e.g., ROSC, neurologically intact survival)?

Consensus on science

There were no adult (>8 years of age) human studies that addressed directly whether titrated oxygen compared with 100% oxygen during CPR affects outcome. Two animal studies (LOE 5)[153,154] that used a fibrillatory model of cardiac arrest suggested that use of 100% oxygen during CPR and for 15–60 min after ROSC results in worse neurological outcomes compared with normoxic (21% oxygen, room air) resuscitation, whereas one animal study (LOE 5)[155] using an asphyxial model documented that ventilation with either 100% oxygen or 21% oxygen during resuscitation did not affect outcome.

Treatment recommendation

There is insufficient evidence to support or refute the use of a titrated oxygen concentration or constant 21% oxygen (room air) when compared with 100% oxygen during adult cardiac arrest. In the absence of any other data there is no reason to change the current treatment algorithm, which includes use of 100% oxygen during adult cardiac arrest.

Knowledge gaps

Prospective clinical trials may be warranted to explore constant (including room air) versus titrated oxygen resuscitation approaches during human adult cardiac arrest.

Passive oxygen vs positive pressure oxygen during CPR[ALS-CPR&A-009A, ALS-CPR&A-009B]

In adults and children in cardiac arrest (out-of-hospital [OHCA], in-hospital [IHCA]), does the use of passive oxygen delivery during CPR, compared with oxygen delivery by positive pressure ventilation, improve outcome (e.g., ROSC, survival)?

Consensus on science

Two studies (LOE 1),[156,157] involving ALS providers in- and out-of-hospital settings, and two animal studies (LOE 5)[158,159] suggested that passive oxygen delivery through a Boussignac tube at

a flow of 15 L min^{-1} associated with continuous chest compressions (with or without active compression–decompression CPR) generated equal or improved gas exchange and haemodynamics, but without improved outcome (ROSC, hospital discharge survival, or neurological outcome), when compared with a standard tracheal tube and positive pressure ventilation.

Four animal models (LOE 5) using different devices or approaches (nasal cannula in the oropharynx,[160] pharyngeal-tracheal lumen airway,[161] and oxygen catheter tip at the level of the carina[162,163]) confirmed an equivalent or better gas exchange and/or haemodynamics, with continuous oxygen inflation compared with standard ventilation.

One swine model (LOE 5)[164] demonstrated equivalent gas exchange and 48-h survival following 4 min VF arrest with passive oxygen supplied via tracheal tube compared with oxygen supplied by positive pressure ventilation.

Two studies (LOE 3)[165,166] of a simplified minimally interrupted cardiac resuscitation (MICR) protocol (concept of cardiocerebral resuscitation), which included passive oxygen delivery via a standard oxygen mask with nonrebreather bag and continuous chest compressions, showed an improvement in neurologically intact survival in adults with bystander-witnessed cardiac arrest and an initially shockable rhythm when controlled with historical controls using standard CPR. Another study (LOE 3)[84] demonstrated better survival with passive oxygen delivery than with bag-mask ventilation. In this study the passive oxygen delivery was included as one intervention in a bundle of different treatment changes in patients with a bystander-witnessed cardiac arrest and an initially shockable rhythm. The relative effect of each component of the treatment bundle, including oxygenation, is unknown.

Treatment recommendation

There is insufficient evidence to support or refute the use of passive oxygen delivery during CPR to improved outcomes (ROSC, hospital discharge rate, and improve neurological survival) when compared with oxygen delivery by positive pressure ventilation.

Knowledge gaps

High-quality controlled clinical trials are required to evaluate the relationship between continuous positive airway pressure and important clinical outcomes and comparison with passive oxygen delivery during cardiopulmonary resuscitation.

Strategies for ventilation

Monitoring ventilatory parameters during CPR[ALS-CPR&A-005C]

In adult cardiac arrest (out-of-hospital and in-hospital) with either a protected or unprotected airway, does the monitoring and control of ventilatory parameters (e.g., minute ventilation and/or peak pressures), as opposed to standard care (without ventilatory monitoring), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no studies that directly addressed the relationship between monitoring of minute ventilation and peak pressure during CPR and changes in outcome (other than respiratory rate).

One animal study (LOE 5)[167] showed that hyperventilation was associated with decreased coronary perfusion pressure and decreased survival. The study also demonstrated that hyperventilation during cardiac arrest is common. One animal study (LOE 5)[168] showed that during CPR applying positive end–expiratory pressure (PEEP) up to 10 cm H_2O, in addition to intermittent positive pressure ventilation (IPPV), may improve oxygenation compared with IPPV alone. Another study demonstrated that continuous positive airway pressure (CPAP) with pressure support ventilation (CPAP PSV) during resuscitation also may improve oxygenation and outcome (LOE 5).[169] One study (LOE 3)[170] demonstrated that real-time feedback during CPR compared with no feedback resulted in a delivered ventilation rate closer to that indicated by current guidelines.

Treatment recommendation

There is insufficient evidence to support or refute the use of peak pressure and minute ventilation monitoring to improve outcome from cardiac arrest. There is indirect evidence that monitoring the respiratory rate with real-time feedback is effective in avoiding hyperventilation and achieving ventilation rates closer to recommended values, but there is no evidence that ROSC or survival is improved.

Knowledge gaps

Clinical trials evaluating ventilation monitoring during cardiac arrest resuscitation for all outcomes are needed. There is limited information on the accuracy of ventilation rate monitoring in the new defibrillator software that evaluates CPR process measures. This initial work would be helpful to enable controlled trials to determine the optimal ventilation rate associated with survival.

Monitoring physiological parameters during CPR[ALS-CPR&A-001A, ALS-CPR&A-001B]

In adult cardiac arrest (out-of-hospital [OHCA], in-hospital [IHCA]), does the use of physiological feedback about CPR quality (e.g., $ETCO_2$ monitoring), compared with no feedback, improve any outcomes (e.g., ROSC, survival)?

Consensus on science

None of the 17 studies that were reviewed evaluated physiological feedback ($ETCO_2$, coronary perfusion pressure, superior vena caval central venous oxygen saturation, bispectral index monitoring) specifically as a tool to guide resuscitation intervention in real time to improve outcomes from cardiac arrest. Eleven studies showed that physiological monitoring values ($ETCO_2$, coronary perfusion pressure, venous oxygen saturation) increased when ROSC was achieved (LOE 4)[135,171–180] and that they may be an indication of ROSC before it can be seen in vital signs.[181]

Five of the studies found that $ETCO_2$ was accurate for predicting patients who could not be resuscitated; some gave a time frame for that prediction of 20 min (LOE 4).[136,174,178,182,183] However, two studies documented patients who did not meet the $ETCO_2$ range but who survived (LOE 4).[174,184] Multiple studies by one group (LOE 4)[175–177] showed that when $ETCO_2$ exceeded 10 mm Hg, all patients achieved ROSC. In one of these studies all the survivors had an initial $ETCO_2$ higher than 10 mm Hg.[176] Similarly, two studies showed that if the $ETCO_2$ did not exceed 10 mm Hg, survival was zero (LOE 4).[182,183]

One study showed no correlation between bispectral index (BIS) values during cardiopulmonary resuscitation and ROSC and survival (LOE 4).[185]

Treatment recommendation

Continuous capnography or capnometry monitoring, if available, may be beneficial by providing feedback on the effectiveness of chest compressions. The prognostic value of end tidal CO_2 is further reviewed in the section on prognostication.[ALS-D&P-014]

Knowledge gaps

Animal and human studies evaluating the effects of modification of resuscitation based on physiological feedback would be helpful.

Automatic transport ventilators

Automatic ventilators versus manual ventilation during CPR[ALS-CPR&A-010A]

In adults and children in cardiac arrest (out-of-hospital [OHCA], in-hospital [IHCA]) and who have advanced airways in place, does the use of automatic ventilators, compared with manual ventilation, improve outcome (e.g., ventilation, oxygenation, reduce hands-off time, allow for continuous compressions and/or improves survival)?

Consensus on science

One pseudorandomised study suggested that the use of an automatic transport ventilator with intubated patients may enable the EMS team to perform more tasks while subjectively providing ventilation similar to that provided by hand with a resuscitation bag (LOE 2).[186] One study suggested that the use of an automatic transport ventilator with intubated patients provides oxygenation and ventilation similar to that achieved with a bag-valve device but with no difference in survival (LOE 2).[187]

Treatment recommendation

There is insufficient evidence to support or refute the use of an automatic transport ventilator over manual ventilation during resuscitation of the cardiac arrest victim with an advanced airway.

Knowledge gaps

Studies evaluating adequacy of oxygenation, difference between volume and pressure cycled ventilation, and survival and complication rates when comparing manual ventilation versus automatic transport ventilator in cardiopulmonary resuscitation with an advanced airway in place are needed to advance the science in this area.

Supporting the circulation during cardiac arrest

Questions related to circulatory support during cardiac arrest that were discussed during the 2010 Consensus Conference are categorised as (1) timing of drug delivery, (2) vasopressors during cardiac arrest, (3) other drugs during cardiac arrest, (4) intravenous (IV) fluids, and (5) extracorporeal support. It is recognised that the vast majority of studies assessing the effects of drugs on survival have been unable to control for the quality of cardiopulmonary resuscitation. Furthermore most drug evaluations to date have been conducted before recent advances in post-cardiac arrest care, including therapeutic hypothermia. Since most drug trials have, at most, demonstrated only short-term outcome advantage, it may be important to evaluate long-term outcome when these drugs are combined with optimised post-cardiac arrest care. One study (LOE 1)[188] compared the use of IV access and drugs (adrenaline [epinephrine], amiodarone, atropine, vasopressin, without isolating the effect of each individual drug alone), with no IV access and no drugs in adult out-of-hospital CPR without isolating the effect of each individual drug alone, with placebo in adult out-of-hospital CPR and demonstrated improvement in ROSC and survival to hospital and intensive care unit (ICU) admission, but no difference in survival to discharge or neurological outcomes at discharge and at 1-year follow-up; however, that study was not powered to detect clinically meaningful differences in long-term outcome. Similarly 1 study (LOE 3)[189] with a before-and-after design compared various outcomes after out-of-hospital cardiac arrest; it was unable to demonstrate any improvements after introduction of advanced life support (adrenaline, atropine, lidocaine). Neither of these studies was able to isolate outcomes specifically related to individual drug administration.

Timing of drug delivery[ALS-SAM-063A, ALS-SAM-063B]

In adult cardiac arrest (out-of-hospital or in-hospital), does an alternate timing for drug delivery (e.g., early or delayed), as opposed to standard care (standard position in algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no studies that addressed the order of drug administration. Subgroup analyses from two clinical studies reported decreased survival for every minute drug delivery was delayed, measured from call received at EMS dispatch (LOE 4).[190,191] This finding was likely to be biased by a concomitant delay in onset of ALS. In one study the interval from the first shock to the injection of the drug was a significant predictor of survival (LOE 4).[190] One animal study reported lower coronary perfusion pressure when delivery of vasopressor was delayed (LOE 5).[192] Time to drug administration was a predictor of ROSC in a retrospective analysis of cardiac arrest in swine (LOE 5).[193]

Treatment recommendation

There is inadequate evidence to define the optimal timing or order for drug administration. An incomplete review of animal studies suggests that timing of vasopressor administration may affect circulation, and further investigations are important to help guide the timing of drug administration.

Knowledge gaps

Advancing the science in the timing of drug administration is closely related to the need to conduct placebo-controlled trials to determine the efficacy of some drugs in CPR. The timing of drug administration and route of delivery are important data points to be captured in future studies. Animal models and clinical trials addressing efficacy can also be designed to provide substantial information on how timing and delivery can affect outcome. In the future, inclusion of studies on pharmacokinetics combined with dose response, as well as studies addressing the impact of timing of defibrillation on circulation and drug effect, might better address the question of optimal timing of drug delivery.

Vasopressors[ALS-D-023B]

Despite the continued widespread use of adrenaline and increased use of vasopressin during resuscitation in some countries, there is no placebo-controlled study that shows that the routine use of any vasopressor during human cardiac arrest increases survival to hospital discharge.

In adult patients in cardiac arrest (asystole, pulseless electric activity [PEA], pulseless VT, and VF) (out-of-hospital [OHCA], in-hospital [IHCA]), does the use of vasopressors (adrenaline, noradrenaline, others) or combination of vasopressors, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., ROSC, survival)?

Consensus on science

One study retrospectively compared adrenaline with no adrenaline for sustained VF and PEA/asystole and found improved ROSC with adrenaline for both rhythms but no difference in survival (LOE 2).[194] In a large retrospective registry-based study from Sweden (LOE 4) adrenaline was an independent predictor of poor outcome (LOE 4).[195]

Three studies (LOE 1)[196–198] and a meta-analysis (LOE 1)[199] demonstrated no difference in outcomes (ROSC, survival to dis-

charge, or neurological outcome) with vasopressin when compared with adrenaline as a first-line vasopressor in cardiac arrest.

Two studies (LOE 1)[200,201] demonstrated no difference in outcomes (ROSC, survival to discharge, or neurological) comparing adrenaline in combination with vasopressin with adrenaline alone in cardiac arrest.

No study demonstrated a survival benefit with high-dose versus standard-dose adrenaline in cardiac arrest. Two studies (LOE 1)[202,203] reported improvement in ROSC using high-dose adrenaline. One meta-analysis (LOE 1)[204] of pooled data from five studies[202,203,205–207] supported improvement in ROSC with high-dose adrenaline but no change in survival outcomes.

Treatment recommendation

Although there is evidence that vasopressors (adrenaline or vasopressin) may improve ROSC and short-term survival, there is insufficient evidence to suggest that vasopressors improve survival to discharge and neurological outcome. There is insufficient evidence to suggest the optimal dosage of any vasopressor in the treatment of adult cardiac arrest. Given the observed benefit in short-term outcomes, the use of adrenaline or vasopressin may be considered in adult cardiac arrest.

Knowledge gaps

Placebo-controlled trials to evaluate the use of any vasopressor in adult and paediatric cardiac arrest are needed.

Other drugs during cardiac arrest

There is no convincing evidence that the routine use of other drugs (atropine, amiodarone, lidocaine, procainamide, bretylium, magnesium, buffers, calcium, hormones, or fibrinolytics) during human CPR increases survival to hospital discharge.

Atropine[ALS-D-024B]

In adult patients in cardiac arrest (asystole, PEA, pulseless VT, and VF) (out-of-hospital, in-hospital), does the use of atropine or atropine in combination with other drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., ROSC, survival)?

Consensus on science

Three studies (LOE 4)[208–210] (total of 12 operating rooms, two catheterisation laboratories, two out-of-hospital cardiac arrest patients, and four in-hospital cardiac arrest patients) documented improvement in survival when atropine was given to patients in asystole in combination with adrenaline[208,210] and following induction with succinylcholine and fentanyl.[209] One study documented improvement in ROSC (14% versus 0%) when atropine was given to adults in asystolic out-of-hospital cardiac arrest in combination with adrenaline and sodium bicarbonate, but none survived to discharge (LOE 3).[211]

Three studies suggested that the use of atropine for treatment of cardiac arrest was not associated with any change in survival (LOE 2[212]; LOE 5[213,214]). Four human studies suggested that the use of atropine was associated with poor survival (LOE 4).[83,215–217]

Treatment recommendation

There is insufficient evidence to support or refute the use of atropine in cardiac arrest to improve survival to hospital discharge.

Knowledge gaps

Randomised placebo-controlled trials are required to define the role of atropine in PEA and asystolic cardiac arrest.

Lidocaine, procainamide, amiodarone, bretylium, magnesium[ALS-D-025A, ALS-D-025B]

In adult cardiac arrest (asystole, PEA, pulseless VT, and VF) (out-of-hospital, in-hospital), does the use of antiarrhythmic drugs (lidocaine, procainamide, amiodarone, bretylium, magnesium) or combination with other drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., ROSC, survival)?

Consensus on science

There was little evidence to suggest a survival-to-discharge advantage with any antiarrhythmic drug used during resuscitation from out-of-hospital or in-hospital cardiac arrest. Two randomised trials demonstrated the benefit of amiodarone over standard of care, which included lidocaine in 80% of cases,[191] or routine use of lidocaine[190] for shock refractory or recurrent VT/VF for the end point of survival to hospital admission, but not to survival to hospital discharge. A retrospective review demonstrated improved survival to admission with lidocaine (compared with standard treatment) for patients in VF out of hospital (LOE 4).[218]

A retrospective review found procainamide was associated with increased survival to 1-h postarrest in patients with VF in hospital (LOE 4).[214] Four randomised, controlled trials did not show any increase in ROSC or survival when magnesium was compared with placebo for patients in VF in out-of-hospital, ICU, and emergency department (ED) settings (LOE 1).[219–222]

Treatment recommendation

Amiodarone may be considered for those who have refractory VT/VF, defined as VT/VF not terminated by defibrillation, or VT/VF recurrence in out-of-hospital cardiac arrest or in-hospital cardiac arrest. There is inadequate evidence to support or refute the use of lidocaine in the same settings.

Knowledge gaps

All the studies to date were done with stacked shocks; it may be helpful to re-evaluate the efficacy of amiodarone in the setting of a single-shock defibrillation strategy.

Calcium[ALS-D-026A, ALS-D-026B]

In adult cardiac arrest (asystole, PEA, pulseless VT, and VF) (out-of-hospital, in-hospital), does the use of calcium alone or combination with other drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., ROSC, survival)?

Consensus on science

Three randomised control trials (LOE 1)[223–225] and three cohort studies (LOE 2)[214,217,226] and 1 case series (LOE 4)[227] demonstrated no effect on survival when calcium was given to in-hospital or out-of-hospital cardiac arrest patients. Two adult studies suggest that calcium administration during cardiac arrest was associated with decreased survival to hospital discharge (LOE 2).[217,228]

In VF, calcium did not restore a spontaneous circulation (LOE 4).[227] In one study of PEA arrests, calcium demonstrated improved ROSC, without reporting long-term survival, but only in a subgroup of patients with wide QRS (LOE 1).[224] Another study showed improved ROSC and survival to hospital arrival; however, there was no significant effect on survival (LOE 4).[227] Another study showed decreased rate of ROSC in the calcium group (LOE 2).[228] In two studies of asystole calcium administration failed to show any improvement in ROSC or survival to hospital discharge (LOE 1).[223,225] One study showed reduced ROSC in the calcium group (LOE 2).[228]

Treatment recommendation

Routine administration of calcium for treatment of in-hospital and out-of-hospital cardiac arrest is not recommended.

Knowledge gaps

More data are needed on the administration of calcium for specific circumstances, such as hyperkalaemia, documented hypocalcaemia, hypermagnesaemia, calcium channel blocker overdose, or wide QRS complexes.

Steroid and hormonal therapy[ALS-D-027]

During adult cardiac arrest (asystole, PEA, pulseless VT, and VF) (out-of-hospital, in-hospital), does the use of steroid or hormonal therapy (estrogen, progesterone, hydrocortisone, insulin, growth factor, etc.) alone or in combination with other drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., ROSC, survival)?

Consensus on science

There were no human or animal studies that directly addressed the use of the estrogen, progesterone, insulin, or insulin-like growth factor in cardiac arrest. Early observational studies of the use corticosteroids during cardiac arrest suggested possible benefit (LOE 4).[229,230] One complex randomised pilot study (LOE 1)[231] and one nonrandomised human study (LOE 2)[232] suggested benefit with corticosteroids, whereas one small, older, human prehospital controlled clinical trial suggested no benefit (LOE 1).[233] One animal study of corticosteroids suggested possible benefit (LOE 5).[234]

Treatment recommendation

There is insufficient evidence to support or refute the use of corticosteroids alone or in combination with other drugs during cardiac arrest.

Knowledge gaps

High-quality clinical trials are required to determine if there is a role in cardiopulmonary resuscitation for hormonal therapy with or without vasopressor while controlling for in-hospital use of hormonal therapy postarrest.

Buffers[ALS-D-029A, ALS-D-029C]

In adult cardiac arrest (asystole, PEA, pulseless VT, and VF) (out-of-hospital, in-hospital), does the use of buffering agents alone or combination with other drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., ROSC, survival)?

Consensus on science

Two studies evaluated buffering agents during CPR (LOE 1).[235,236] Both had limitations but showed no improvement in outcome. Two retrospective cohort studies also showed no benefit in the use of buffering agents during CPR (LOE 2).[237,238] Two studies demonstrated increased ROSC, hospital admission, and survival at hospital discharge with bicarbonate use (LOE 2[239]; LOE 3[240]). Four cohort studies reported that bicarbonate use was associated with poor short- and long-term outcome (LOE 2).[217,241–243]

Treatment recommendation

Routine administration of sodium bicarbonate for treatment of in-hospital and out-of-hospital cardiac arrest is not recommended.

Knowledge gaps

There are large differences in direction and effect between results from the laboratory and those derived from clinical trials; therefore, well-designed trials, using bicarbonate or non-CO_2 generating buffers, are necessary to clarify the role of buffers in the treatment of short or prolonged cardiac arrest.

Fibrinolytics[ALS-D-028A, ALS-D-028B]

In adult cardiac arrest, does the use of fibrinolytics alone or in combination with other drugs, compared with not using drugs, improve outcomes?

Consensus on science

Two studies failed to show any improvement in short- or long-term outcomes with the use of fibrinolytics (LOE 1).[244,245] One study showed an increased risk of intracranial bleeding associated with the routine use of fibrinolytics during cardiac arrest (LOE 1).[245] Seven studies showed benefit from fibrinolytic therapy in the treatment of victims of cardiopulmonary arrest unresponsive to standard therapy; however, those studies had significant limitations (LOE 1[246]; LOE 2[247–250]; LOE 3[251,252]).

Treatment recommendation

Routine administration of fibrinolytics for the treatment of in-hospital and out-of-hospital cardiac arrest is not recommended. (See "Cardiac Arrest Caused by Pulmonary Embolus" for the treatment of patients with ROSC following suspected pulmonary embolus.)

Knowledge gaps

The potential role of adjuvant antithrombotic and antiplatelet drugs needs exploration.

Intravenous fluids during cardiac arrest[ALS-D-016A, ALS-D-016B]

In adult cardiac arrest (out-of-hospital, in-hospital), does the use intravenous fluids, compared with not using fluids (or standard resuscitation), improve outcomes (e.g., ROSC, survival)?

Consensus on science

No published human study directly compared outcome of routine intravenous fluid administration with no fluid administration during CPR. Two animal studies reported that normothermic fluid infusion during CPR causes a decrease in coronary perfusion pressure (LOE 5),[253,254] and another animal study showed that the coronary perfusion pressure rise with adrenaline during CPR is not improved with the addition of a fluid infusion (LOE 5).[255] Most animal studies of fluid infusion during CPR lack a control group that receives no fluids; without a control group, it is difficult to assess of benefit or harm from fluid therapy (LOE 5).[256–267]

Hypertonic fluid

One small randomised clinical trial (RCT) in adults found no significant ROSC or survival benefit with hypertonic IV fluid infusion when compared to isotonic IV infusion during CPR (LOE 5).[256] One animal study showed that hypertonic saline improves cerebral blood flow during CPR (LOE 5).[262] Two animal studies found neither benefit nor harm with infusion of hypertonic saline (LOE 5).[260,267]

Chilled fluid versus room-temperature fluid

Two adult studies (LOE 5)[258,261] and two animal studies (LOE 5)[265,266] showed no improvement in ROSC when cold IV fluids (compared with room temperature intravenous fluids) were infused during CPR. One of the reported animal studies showed that the infusion of cold fluids during CPR caused a decrease

in coronary perfusion pressure when compared to no fluids (LOE 5).[268]

Treatment recommendation

There is insufficient evidence to recommend for or against the routine infusion of intravenous fluids during cardiac arrest resuscitation.

Knowledge gaps

Human studies are required that compare outcome with IV fluid administration versus no fluid administration during treatment of VF and non-VF cardiac arrest. Animal models evaluating the haemodynamic effects of intravenous fluids need to more closely approximate the human model of cardiac arrest with comorbidities and altered physiology.

Extracorporeal circulatory support during cardiac arrest[ALS-CPR&A-002A, ALS-CPR&A-002B]

In adult cardiac arrest (prehospital, IHCA, OHCA), does the use of rapid deployment extracorporeal membrane oxygenation (ECMO), aortic balloon pump, or emergency cardiopulmonary bypass, compared with standard treatment, increase survival to hospital discharge with favorable neurological outcomes?

Consensus on science

All the studies on this topic were small and there was a lack of consistency in the management before and after extracorporeal-CPR (ECPR). Three studies documented improvement in outcome in patients <70 years old, without significant comorbid conditions and with potential reversible/correctable conditions, when using ECMO compared with traditional CPR (LOE 2[269,270]; LOE 3[271]). One study demonstrated a three-month survival of 22.7% for ECPR during out-of-hospital cardiac arrest unresponsive to advance life support after 20 min, with 10.6% having a Cerebral Performance Category (CPC) of 1 (LOE 2).[270] However, the ECPR group was more likely to have had a witnessed arrest, received bystander CPR, and be younger (with a mean age of 52 years, compared with 70 years in the standard treatment group).

Treatment recommendation

There is insufficient evidence to support or refute the routine use of extracorporeal cardiopulmonary resuscitation in cardiac arrest.

Knowledge gaps

Future research should define the criteria for ECPR after out-of-hospital cardiac arrest and the criteria for ECPR as a bridge to left ventricular assist device (LVAD) or transplant. It is recommended that the intraaortic balloon pump (IABP) and LVADs be included in the list of questions to pursue in 2015.

Antiarrhythmics in the periarrest period

Narrow-complex tachycardia (excluding atrial fibrillation)[ALS-D-018]

There are four options for the treatment of narrow-complex tachycardia in the periarrest setting: electrical conversion, physical manoeuvres, pharmacological conversion, or rate control. The choice depends on the stability of the patient and the rhythm. In a haemodynamically unstable patient, narrow complex tachycardia is best treated with electrical cardioversion.

In adult patients with narrow-complex tachycardia (out-of-hospital and in-hospital), does the use of any drug or combination of drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., reversion rates)?

Consensus on science

Five trials supported the use of adenosine in the treatment of narrow-complex tachycardia (LOE 1).[272–276] Six trials demonstrated the effectiveness of verapamil in conversion to sinus rhythm (LOE 1).[272–275,277,278] The effectiveness of diltiazem in conversion to sinus rhythm is supported by four trials (LOE 1).[273,277,279,280] The evidence to support the use of other drugs for conversion to sinus rhythm is limited to a few trials for each drug, including sotalol (LOE 1),[281] amiodarone (LOE 4),[282] propafenone (LOE 1),[283]) and nadolol (LOE 1).[284] The study on nadolol suggested treatment effect on rate as well. There was no evidence of benefit with cibenzoline (LOE 1)[285] or magnesium (LOE 4)[286]; two studies reported that the response to magnesium is poor in patients with narrow-complex tachycardia.[287,288] Two studies demonstrated conversion effectiveness of vagal manoeuvres (carotid massage and Valsalva) (LOE 2[289]; LOE 4[290]).

Treatment recommendation

Vagal manoeuvres, IV adenosine, verapamil, and diltiazem are recommended as first-line treatment strategies in the termination of narrow-complex tachycardias. Nadolol, sotalol, propafenone, and amiodarone may be considered.

Knowledge gaps

Future studies should consider evaluating the safety of combining antiarrhythmic drugs and the efficacy of second-line therapies (some β-blockers, digoxin, amiodarone) for termination of narrow-complex tachycardia.

Atrial fibrillation[ALS-D-017]

In adult patients in atrial fibrillation (prehospital and in-hospital), does the use of any drug or combination of drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., reversion rates)?

Consensus on science

This topic has been comprehensively reviewed by the European Society of Cardiology, the American Heart Association, and the American College of Cardiology.[291]

Rate control in atrial fibrillation. A systematic review (LOE 1)[292] demonstrated superiority for β-blockers (esmolol, metroprolol, and propranolol) with 70% success in meeting target heart rate or verapamil and diltiazem with 54% success[293] as first-line therapy for rate control in atrial fibrillation without a known accessory pathway and amiodarone when an accessory pathway was known and amiodarone or digoxin when fast atrial fibrillation occurred with heart failure (LOE 1).[292]

Four studies showed benefit for diltiazem in controlling rate in hospital (LOE 1[294–296]; LOE 2[297]), and one study for out of hospital (LOE 3).[298] Two studies showed that verapamil is equally effective in rate control for atrial fibrillation (LOE 1).[299,300] Adverse event rates with calcium channel blockers were reported as 18%.[300]

Amiodarone may control rate and rhythm (LOE 1),[301] but significant complications were described in placebo-controlled trials: the risk of adverse events was 26.8% as a pooled estimate, and the most common side effects encountered were phlebitis, bradycardia, and hypotension (LOE 1).[301]

Digoxin is not effective for cardioversion (LOE 1),[302–304] but in some studies it has been shown to have moderate rate controlling properties (LOE 1).[297,303,304]

Rhythm control of atrial fibrillation. Ibutilide has consistently been more effective in converting atrial fibrillation to sinus rhythm when compared with placebo (LOE 1),[305–307] or other antiarrhythmic drugs (LOE 1: sotalol,[308] procainamide,[309] and amiodarone,[310]) and equal to other drugs (LOE 1: flecainide[311]).

Propafenone has been consistently more effective than placebo in converting AF to sinus rhythm (LOE 1),[312–314] but inferior to other drugs (LOE 1: amiodarone,[301] procainamide,[315] and flecainide[316]).

There are also data supporting flecainide (LOE 1)[317–320] and dofetilide (LOE 1)[321,322] for conversion in patients without coronary artery disease.

Data supporting amiodarone for cardioversion are relatively weak (LOE 1)[310,323–325]; however, amiodarone does have rate-controlling properties (LOE 1).[323,326]

Sotalol has consistently been shown to be inferior in conversion compared to other drugs (LOE 1: flecainide[318] and ibutalide[308]), but equal to amiodarone in one study (LOE 1).[325]

Most studies showed no conversion benefit for magnesium (LOE 1),[327,328] although 1 meta-analysis showed conversion benefit (LOE 1).[329] Most studies showed a benefit for magnesium in rate control (LOE 1),[295,329,330] although one study was neutral for magnesium for rate control (LOE 1).[328]

Quinidine has been shown to have greater conversion than sotalol in two studies (LOE 1),[331,332] although this was with greater toxicity. Clonidine has rate-controlling properties compared with placebo (LOE 1).[333,334]

Procainamide has shown increased efficacy in conversion of AF to sinus rhythm when compared with placebo[335] and to propafenone,[315] but appears to be as effective as amiodarone.[336]

Treatment recommendation

Patients who are haemodynamically unstable with atrial fibrillation should receive prompt electrical cardioversion.

Rate control in atrial fibrillation. Beta-blockers and diltiazem are the drugs of choice for acute rate control in most individuals with atrial fibrillation and rapid ventricular response. Digoxin and amiodarone may be used in patients with congestive heart failure, and amiodarone may also result in cardioversion to normal sinus rhythm. Magnesium and clonidine have rate-controlling effects, though there are fewer data supporting their use.

Rhythm control of atrial fibrillation. Chemical cardioversion can be achieved with ibutilide, dofetilide, and flecainide. Amiodarone can also be used for chemical cardioversion, but it is less effective. Quinidine or procainamide may be useful for cardioversion, but their use is less well established. Propafenone is more effective than placebo but not as effective as amiodarone, procainamide, or flecainide. There is no role for digoxin in chemical cardioversion.

Knowledge gaps

Future research should address unstable atrial fibrillation and the balance between rate control versus electrical cardioversion versus pharmacological cardioversion. Head-to-head comparisons to find the optimal drug with the best safety profile have not been done.

Wide-complex tachycardia

There are two options for the treatment of wide-complex tachycardia in the periarrest setting: electrical conversion and chemical conversion. The choice depends on the stability of the patient and the rhythm. In a haemodynamically unstable patient, wide complex tachycardia is best treated with electrical cardioversion.

Monomorphic VT[ALS-D-019-01A, ALS-D-019-01B]

In adult patients in haemodynamically stable monomorphic ventricular tachycardia (out-of-hospital, in-hospital), does the use of any drug or combination of drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., reversion rates)?

Consensus on science

Conversion of acute onset of monomorphic (wide-complex) haemodynamically stable VT.

Procainamide

One unblinded study comparing lidocaine with procainamide (LOE 1)[337] documented an improved reversion rate over lidocaine (1.5 mg kg^{-1}) when procainamide (10 mg kg^{-1}) was given to adult patients with haemodynamically stable monomorphic ventricular tachycardia (mVT), but without severe congestive heart failure or acute myocardial infarction in the hospital setting. Additional evidence from a case series suggested that procainamide was effective in terminating stable mVT in the hospital setting (LOE 4).[338]

Sotalol

A double-blind study comparing lidocaine with sotalol documented an improved reversion rate over lidocaine (100 mg) when sotalol (100 mg) was given to patients with spontaneous onset haemodynamically stable sustained mVT in the hospital setting (LOE 1).[339]

Amiodarone

The evidence on the effectiveness of amiodarone (150–300 mg) in terminating VT is conflicting with reported conversion rates between 20% and 40% based on one controlled trial (LOE 1)[340] and three case series (LOE 4)[341–343] in patients with coronary artery disease with a low left ventricular ejection fraction in the hospital setting. The use of amiodarone (300 mg) was associated with side effects (primarily hypotension),[341,343] but the effect of these on outcome remains unclear.

Lidocaine

Lidocaine was less effective than sotalol (LOE 1),[339] procainamide (LOE 2),[337] and amiodarone (LOE 2)[340] in terminating VT. Three retrospective analyses showed lidocaine was poorly effective when given to patients with or without a history of myocardial infarction with spontaneous sustained stable VT in the hospital setting (LOE 4).[344–346] One randomised controlled study (LOE 5)[347] and one case series (LOE 5)[348] suggested a variable termination of

the arrhythmia when lidocaine was injected by paramedics intramuscularly in patients with acute myocardial infarction and VT in the prehospital setting.

Cibenzoline

One case series suggested cibenzoline (70 ± 12 mg) may be effective in terminating VT (LOE 4).[349]

Magnesium

One study suggested magnesium was effective in terminating VT (LOE 5).[350]

Adenosine

Adenosine may aid in diagnosing VT, but it will not terminate it (LOE 4).[351,352]

Calcium channel blockers

The evidence for the use of calcium channel blockers in VT is conflicting, with most studies opposing their use (LOE 4),[353–355] but one study supported the use as long as coronary disease was not present (LOE 5).[356]

Nifekalant

Two retrospective control studies (LOE 3),[357,358] one case series (LOE 4),[359] and one other study (LOE 5)[360] suggested that nifekalant improved outcome in patients with shock refractory VF/VT, even though it did not seem to be effective in immediately terminating the arrhythmia.[359]

Preventing recurrence and late conversion in refractory ventricular tachyarrhythmias including mVT:

Amiodarone

Two RCTs (LOE 1) comparing amiodarone with lidocaine[340] or bretylium,[361] two double-blind randomised dose-range studies (LOE 4),[362,363] and five case series (LOE 4)[364–368] suggested that amiodarone reduced the number of life-threatening arrhythmias (event rate), required shocks, and episodes of symptomatic sustained VT that occurred in patients with recurrent refractory ventricular arrhythmias in hospital.

β-Blockers

A single prospective case series (LOE 4)[369] suggested that recurrent and refractory ventricular arrhythmias were reduced while long- and short-term survival were improved in patients treated with sympathetic blockade (including β-blockers) during electrical storm.

Electrical cardioversion

Electrical cardioversion at an early stage or as first-line treatment was reasonable based on a prospective case series (LOE 4).[370] Indirect evidence was also provided by 3 case studies (LOE 4).[344,371,372]

Treatment recommendation

Procainamide is recommended for patients with haemodynamically stable monomorphic ventricular tachycardia (mVT) who do not have severe congestive heart failure or acute myocardial infarction. Amiodarone is recommended for patients with haemodynamically stable mVT with or without either severe congestive heart failure or acute myocardial infarction. Nifekalant (not approved for use in all countries) may be useful in improving outcomes in shock refractory VF/VT even though it did not seem to be effective in immediately terminating the arrhythmia.

Sotalol may be considered for patients with haemodynamically stable sustained mVT, including patients with acute myocardial infarction.

Knowledge gaps

Overall the evidence for different drugs, in terms of both their efficacy and their side effects, is conflicting, and the evidence supporting the use of drugs such as sotalol and procainamide is limited to just one study each. There are no placebo-controlled trials comparing antiarrhythmics, nor are there studies comparing electrical with pharmacological strategy for sustained haemodynamically stable mVT. Future research should define a standard drug therapy to be used as the reference control for scientific advancement in this area.

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Adenosine may aid in diagnosing VT, but it will not terminate it (LOE 4).[351,352]

Calcium channel blockers

The evidence for the use of calcium channel blockers in VT is conflicting, with most studies opposing their use (LOE 4),[353–355] but one study supported the use as long as coronary disease was not present (LOE 5).[356]

Nifekalant

Two retrospective control studies (LOE 3),[357,358] one case series (LOE 4),[359] and one other study (LOE 5)[360] suggested that nifekalant improved outcome in patients with shock refractory VF/VT, even though it did not seem to be effective in immediately terminating the arrhythmia.[359]

Preventing recurrence and late conversion in refractory ventricular tachyarrhythmias including mVT.

Amiodarone

Two RCTs (LOE 1) comparing amiodarone with lidocaine[340] or bretylium,[361] two double-blind randomised dose-range studies (LOE 4),[362,363] and five case series (LOE 4)[364–368] suggested that amiodarone reduced the number of life-threatening arrhythmias (event rate), required shocks, and episodes of symptomatic sustained VT that occurred in patients with recurrent refractory ventricular arrhythmias in hospital.

β-Blockers

A single prospective case series (LOE 4)[369] suggested that recurrent and refractory ventricular arrhythmias were reduced while long- and short-term survival were improved in patients treated with sympathetic blockade (including β-blockers) during electrical storm.

Electric cardioversion

Electric cardioversion at an early stage or as first-line treatment was reasonable based on a prospective case series (LOE 4).[370] Indirect evidence was also provided by 3 case studies (LOE 4).[344,371,372]

Treatment recommendation

Procainamide is recommended for patients with haemodynamically stable monomorphic ventricular tachycardia (mVT) who do not have severe congestive heart failure or acute myocardial infarction. Amiodarone is recommended for patients with haemodynamically stable mVT with or without either severe congestive heart failure or acute myocardial infarction. Nifekalant (not approved for use in all countries) may be useful in improving outcomes in shock refractory VF/VT even though it did not seem to be effective in immediately terminating the arrhythmia.

Sotalol may be considered for patients with haemodynamically stable sustained mVT, including patients with acute myocardial infarction.

Knowledge gaps

Overall the evidence for different drugs, in terms of both their efficacy and their side effects, is conflicting, and the evidence supporting the use of drugs such as sotalol and procainamide is limited to just one study each. There are no placebo-controlled trials comparing antiarrhythmics, nor are there studies comparing electrical with pharmacological strategy for sustained haemodynamically stable mVT. Future research should define a standard drug therapy to be used as the reference control for scientific advancement in this area.

Undifferentiated regular stable wide-complex tachycardia[ALS-D-019-02]

In adult patients with undifferentiated regular stable wide-complex tachycardia (prehospital and in-hospital), does the use of adenosine or adenosine in combination with other drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., reversion rates)?

Consensus on science

Five studies involving more than 300 patients (LOE 4)[351,352,373–375] demonstrated that adenosine could safely be administered in regular wide-complex tachycardia: it converted wide-complex tachycardia secondary to supraventricular tachycardia to normal sinus rhythm, but rarely terminated VT. One small study showed poor rates of conversion to sinus rhythm in patients known to have VT (LOE 4).[344] No patient in these trials had serious adverse events; however, there are case reports in patients with irregular wide-complex tachycardia (generally pre-excited atrial fibrillation) in whom VF was precipitated by adenosine (LOE 4).[376–379]

Other studies that included lidocaine showed poor rates of conversion to sinus rhythm with lidocaine in patients known to have VT (LOE 4).[344] In one study, 11 of 25 patients known to have VT and treated with verapamil developed profound hypotension (LOE 4).[380]

Treatment recommendation

In undifferentiated regular stable wide-complex tachycardia, IV adenosine may be considered relatively safe, may convert the rhythm to sinus, and may help diagnose the underlying rhythm.

Knowledge gaps

The science in this area is limited: randomised trials have not been done.

Polymorphic wide-complex tachycardia[ALS-D-020B]

In adult patients in polymorphic wide-complex tachycardia (prehospital and in-hospital), does the use of any drug or combination of drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., reversion rates)?

Consensus on science

Evidence for benefit from these therapies is limited, mainly anecdotal, extrapolated, or from small, observational studies and based on the presumed mechanism for polymorphic wide-complex tachycardia, which may not always be clinically evident. There are three subtypes of polymorphic VT:

1. Polymorphic VT with delayed abnormal repolarization, usually known as torsades de pointes (twisting of the QRS complexes around the baseline), with long QT, as well as "pause-dependent" initiating sequence, and coexisting factors associated with delayed repolarization with 2 subtypes:
   a. Congenital long QT with torsades de pointes
   b. Acquired long QT with torsades de pointes
2. Polymorphic VT caused by ischaemia, which usually has a short QT; ischaemia often present by history, clinical picture, and ECG findings of ischaemia or infarction
3. Polymorphic VT of unknown cause, usually in the context of severe left ventricular dysfunction with or without congestive heart failure or severe structural heart disease

Familial (congenital) long QT (torsades de pointes). Recurrences of polymorphic wide-complex tachycardia associated with congenital long QT may be reduced with IV magnesium, based on extrapolation from a small case series of children (LOE 5)[381]; overdrive pacing (atrial or ventricular); or β-blockers derived from extrapolation from two registry case series of secondary prevention in patients with congenital long QT (LOE 5).[382,383] There is virtually no published experience regarding the acute use of these therapies in such patients.

Acquired long QT (torsades de pointes). Recurrences of polymorphic wide-complex tachycardia associated with acquired or drug-precipitated Long QT may be reduced with IV magnesium, based on five studies (LOE 3[384]; LOE 4[385]; LOE 5 (paediatrics)[381]; LOE 5 (animals)[386,387]); overdrive pacing (atrial or ventricular) based on seven studies (LOE 4[385,388–391]; LOE 5 [extrapolation from secondary prevention in patients with congenital LQTS][382,383]); and IV isoprenaline (when not contraindicated by presence of ischaemia or hypertension) is supported by four studies (LOE 4[385,388]; LOE 5 (animal)[387,392]) but opposed by one study (LOE 4).[389]

Preventing recurrences of polymorphic wide-complex tachycardia secondary to other mechanisms. The science on the management of polymorphic wide-complex tachycardia caused by short QT syndrome is limited to case reports involving amiodarone, β-blockers, and quinidine (LOE 4).[393,394]

Polymorphic wide-complex tachycardia associated with acute myocardial ischaemia responded to IV β-blockers in a modestly sized study (LOE 3)[369]; however, there was no benefit from IV magnesium in a small study (LOE 3).[384] A LOE-4 study[395] and extrapolation from a small case series suggested that isoprenaline attenuated the ST elevation associated with Brugada syndrome (LOE 5).[396] Extrapolation from one case series suggested worsened Brugada ST elevation with class IA antiarrhythmics (LOE 5).[396]

A paediatric case report (LOE 5)[397] and extrapolation from a small case series of secondary prevention using oral β-blockers alone (LOE 5)[398] or in combination with verapamil (LOE 5)[399,400] suggested IV propranolol successfully terminated catecholamine-induced polymorphic wide-complex tachycardia.

Haemodynamically unstable polymorphic VT of unspecified morphology and mechanism. Among patients with impaired ventricular function due to structural heart disease (ischaemic, valvular, or cardiomyopathy), in the absence of QT prolongation or drug provocation, treatment of haemodynamically unstable VT with intravenous amiodarone reduced the frequency of recurrent arrhythmias. This evidence rests on extrapolation from three prospective RCTs (LOE 5)[361–363] performed in the in-hospital setting but in which VT morphology was not addressed specifically.

Treatment recommendation

Polymorphic wide-complex tachycardia associated with familial long QT may be treated with IV magnesium, pacing and/or β-blockers; however, isoprenaline should be avoided. Polymorphic wide-complex tachycardia associated with acquired long QT may be treated with IV magnesium. Addition of pacing or IV isoprenaline may be considered when polymorphic wide-complex tachycardia is accompanied by bradycardia or appears to be precipitated by pauses in rhythm. Polymorphic wide-complex tachycardia without long QT may be responsive to IV β-blockers (ischaemic VT; catecholaminergic VT) or isoprenaline (Brugada).

Knowledge gaps

Since the occurrence of these unusual arrhythmogenic mechanisms is rare, randomised clinical trials are unlikely**;** therefore, future registries may contribute to associations that may guide treatment and advance care.

Bradycardia[ALS-D-022A]

In adult patients in significant bradycardia (out-of-hospital and in-hospital), does the use of any drug or combination of drugs, compared with not using drugs (or a standard drug regimen), improve outcomes (e.g., reversion rates)?

Consensus on science

Transcutaneous pacing. Four case series (LOE 4) demonstrated that in-hospital transcutaneous pacing had slightly higher success rates for rhythm capture[401] and survival to discharge (18–75%)[402–404] compared with survival-to-discharge rates (69%) when transcutaneous pacing was given for out-of-hospital bradycardia (LOE 1).[405] A systematic review supported this survival-to-discharge rate of 15–70% in the prehospital setting (LOE 3).[406]

Few studies have compared drugs with transcutaneous pacing for the treatment of bradycardia. A randomised trial of 45 patients (LOE 1)[407] comparing atropine, glycopyrrolate, and transcutaneous pacing in intraoperative patients showed no significant differences in long-term outcomes. Recurrent episodes of bradycardia were less common in the paced group. One feasibility study (LOE 1)[405] compared dopamine with transcutaneous pacing in patients with bradycardia refractory to atropine. There were no differences in outcomes of survival to discharge (70% versus 69%). Enrollment was slow in this feasibility trial because most patients got better with full-dose atropine in the out-of-hospital setting, making them ineligible for randomization.

One randomised clinical trial (LOE 1),[407] two retrospective cohort studies (LOE 4),[408,409] and two additional observational studies (LOE 4)[410,411] documented that IV atropine improved heart rate and symptoms and signs associated with bradycardia. An initial dose of 0.5–1 mg, repeated as needed to a total of 1.5–3 mg, was effective in both in-hospital and out-of-hospital treatment of symptomatic bradycardia. One study (LOE 4)[411] reported that a ≥0.8 mg dose increased the incidence of tachycardia. One other study in 10 healthy volunteers (LOE 5)[412] indicated that a 3-mg dose of atropine produces the maximum achievable increase in resting heart rate. Two studies indicated that atropine may paradoxically cause high-degree atrioventricular (AV) block in patients after cardiac transplantation (LOE 5[413]; LOE 4[414]).

Second-line drug therapy with dopamine (LOE 1)[405] and adrenaline for undifferentiated haemodynamically unstable bradycardia may be successful; it should be tailored according to potential causes in individual patients. For the treatment of bradycardia unresponsive to atropine after inferior myocardial infarction, cardiac transplant, or spinal cord injury, theophylline may be administered (LOE 2[415]; LOE 4[416,417]).

Treatment recommendation

First-line drug treatment for symptomatic bradycardia is atropine 0.5–1 mg IV repeated every 3–5 min as needed up to 1.5–3 mg total. If not effective, then consider adrenaline (2–10 $\mu g\,min^{-1}$) or dopamine (2–10 $\mu g\,kg^{-1}\,min^{-1}$). Transcutaneous pacing may be considered when full-dose atropine fails, although it may not be any more effective then second-line drug therapy.

Other second-line choices for symptomatic bradycardia should be tailored according to potential causes. After inferior myocardial infarction, cardiac transplant, or spinal cord injury, theophylline 100–200 mg slow injection IV (maximum 250 mg) may be given. Atropine should be used with caution in patients with bradycardia after heart transplant as it may cause paradoxical AV block.

Knowledge gaps

Randomised trials comparing transcutaneous pacing with pharmacotherapy in haemodynamically unstable bradycardia are required to advance the management. Based on the low incidence of bradycardia that is resistant to atropine these trials may not be pragmatic or possible.

Cardiac arrest in special circumstances

Environmental

Cardiac arrest caused by avalanche[ALS-SC-078B]

For avalanche victims in out-of-hospital cardiac arrest, what factors when present, compared with when absent, are associated with/predict an increased survival to hospital discharge?

Consensus on science

Time of burial and patent airway. Four studies (LOE P3)[418–421] demonstrated a progressive nonlinear reduction in survival as time of burial lengthened. In eight studies (LOE P3[419,420,422–425]; LOE P4[426,427]) victims who were buried beyond 35 min did not survive if they had an obstructed airway (defined as obstructed by avalanche debris or by other means) on uncovering the head. One study (LOE P5)[428] demonstrated that when breathing in simulated air pockets of different volumes, hypoxia and hypercapnia achieved a steady state after 10 min. This finding suggested that long-term survival was possible as long as an air pocket, even as small as 1 L, was present. One study (LOE P5)[429] indicated that deflection of expired air away from an air pocket may slow the development of hypoxia and hypercapnia.

Core temperature. Two relevant LOE P3-studies in the general hypothermia literature found that survival decreased with core temperatures less than 32 °C and reported the use of extracorporeal rewarming only when core temperatures were less than 32 °C.[430,431] One relevant LOE P3-study reported a maximum cooling rate of 8 °C/h in buried victims.[432] An avalanche case report described a maximum cooling rate of 9 °C/h (LOE P4).[426]) Those cooling rates suggested that, at 35 min of burial, the core temperature may drop as low as 32 °C. Three relevant studies (LOE P3)[423,432,433] and four case series or reports (LOE P4)[434,435,426,431] recorded ROSC in 22, and survival to hospital discharge in 7 of those 22, buried avalanche victims in cardiac arrest with a core temperature less than 32 °C with aggressive rewarming using extracorporeal circulation.

Serum potassium. A serum potassium of less than 8 mmol/L on hospital admission was found to be predictive of increased ROSC in avalanche burial victims in one study (LOE P3)[423] and for increased survival to hospital discharge in two studies (LOE P3).[422,432]

Five studies found an inverse correlation between admission potassium concentration and survival to discharge in all-cause hypothermic patients (LOE P3).[430,422,433,436,437] Four studies (LOE P3)[422,432,438,439] found that high potassium values were associated with asphyxia in all hypothermic patients. The highest reported serum potassium value in an avalanche survivor was 6.4 mmol/L,[432] although survival to hospital discharge from all-cause hypothermia with a potassium concentration as high as 11.8 mmol/L has been documented.[440]

Treatment recommendation

Avalanches occur in areas that are difficult for rescuers to access in a timely manner, and burials frequently involve multiple victims. The decision to initiate full resuscitative measures should be determined by the number of victims and the resources available, and it should be informed by the likelihood of survival.

Avalanche victims are not likely to survive when they are

Buried >35 min and in cardiac arrest with an obstructed airway on extrication.
Buried initially and in cardiac arrest with an obstructed airway on extrication, and an initial core temperature of <32 °C.
Buried initially and in cardiac arrest on extrication with an initial serum potassium of >8 mmol/L or more.

Full resuscitative measures, including extracorporeal rewarming, when available, are indicated for all other avalanche victims without evidence of an unsurvivable injury.

Knowledge gaps

Prospective validation studies of patent airway, core temperature, and serum potassium as prognostic factors among patients in cardiac arrest on extrication and prospective studies on effectiveness of prehospital treatment of nonarrested hypothermic avalanche victims would advance the science of avalanche resuscitation.

Pregnancy[ALS-SC-065]

In pregnant women with cardiac arrest (out-of-hospital or in-hospital), do any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs evaluating the effect of specialised obstetric resuscitation versus standard care in postarrest pregnant women. Many studies of women not in cardiac arrest document the important physiological changes that occur in pregnancy that may influence treatment recommendations and guidelines for resuscitation of cardiac arrest in pregnancy.

Aortocaval decompression to improve maternal haemodynamics and fetal well-being. In the nonarrest literature, left lateral tilt improved maternal blood pressure, cardiac output, and stroke volume (LOE 5)[441–443] and improved fetal parameters of oxygenation, nonstress test, and fetal heart rate.[444–446] While chest compressions in the left lateral tilt position were shown to be feasible in a manikin study,[447] they have been shown to result in less forceful chest compressions than in the supine position.[448] Two studies found no improvement in maternal haemodynamic or fetal parameters in nonarrest patients with 10–20° left lateral tilt.[449,450] One study found more aortic compression at 15° left lateral tilt when

compared to a full left lateral tilt.[442] In addition, aortic compression has been found to persist at over 30° of tilt;[451] however, the majority of these patients were in labor. Two nonarrest studies found that manual left uterine displacement (which is done with the patient supine) was as good as, or better than, left lateral tilt in relieving aortocaval compression, as assessed by the incidence of hypotension and ephedrine use.[452,453]

Respiratory considerations. One study documented that the upper airways in the third trimester of pregnancy are smaller (supine mean difference 0.20; 95% confidence interval [CI] 0.06–0.35) compared with their postpartum state and to nonpregnant controls (LOE 5).[454] One study found increased intrapulmonary shunting in normal pregnancy at 12.8–15.3% compared with the nonpregnant state normal value of 2–5% (LOE 5),[455] suggesting a change in the approach to oxygenation demands and in the size of the advanced airway may be physiologically justifiable in maternal cardiac arrest.

Perimortem Caesarean section. One retrospective cohort study of 55 maternal cardiac arrests evaluated the incidence of perimortem Caesarean section after the introduction of a targeted training course and compared it with a historical rate (LOE 4).[456] One systematic review of perimortem caesarean sections documented 38 cases, with 34 surviving infants and 13 maternal survivors at discharge, suggesting that perimortem caesarean section may have improved maternal and neonatal outcomes (LOE 4).[457] At older gestational ages (30–38 weeks), infant survival was possible even when delivery was after 5 min from the onset of maternal cardiac arrest (LOE 4).[457] One retrospective study concluded that for delivery of infants between 22 and 25 weeks gestational age, neonatal outcome is best at 25 weeks, and there was no infant survival when delivery occurred at 22 weeks (LOE 5).[458]

Changes in pharmacokinetics. One study documented an increase in glomerular filtration rate, cardiac output, and plasma volume early in the first trimester that starts to return to normal in the end of the third trimester, suggesting that known physiological vascular and fluid changes of pregnancy may respond to fluid resuscitation during maternal cardiac arrest (LOE 5).[459]

Defibrillation. One underpowered case control study reported no difference in transthoracic impedance during pregnancy compared with postpartum, suggesting current energy requirements for adult defibrillation were appropriate (LOE 4).[460]

Positioning. One study indicated that the human wedge technique can provide left lateral tilt and effective external chest compressions in a manikin (LOE 5).[447] However, another study found that the estimation of the degree of table tilt is unreliable and often overestimated, suggesting rescuers are more likely to employ an insufficient amount of tilt to achieve the required haemodynamic benefit (LOE 5).[461] A small study assessed the efficacy of resuscitation at various angles of inclination using a calibrated force transducer (LOE 5).[448] This study found that the maximum possible resuscitative force decreased as the angle of inclination of the plane increased, from 67% of body weight in the supine position to 36% in the full lateral position. Therefore at an inclination of 27° the maximum resuscitative force for chest compressions was only 80% of the force generated at 0° of inclination (supine). Also at an incline of >30° the patient/manikin tended to roll off the incline plane (LOE 5).[448]

Therapeutic hypothermia postarrest. A single case report suggested that post–cardiac arrest hypothermia was used safely and effectively in early pregnancy with fetal heart monitoring and resulted in favorable maternal and fetal outcome after a term delivery (LOE 4).[462]

Treatment recommendation

There is insufficient evidence to support or refute the use of specialised obstetric resuscitation techniques in maternal cardiac arrest and the use of therapeutic hypothermia in the postarrest period. Treatment may be guided by understanding the physiology of pregnancy, the importance of releasing aortocaval compression, the increased risk for hypovolaemia, the compression advantage through positioning, and the value of perimortem caesarean section early in maternal cardiac arrest.

Knowledge gaps

Research in the area of maternal resuscitation is lacking, and most of the science is extrapolated from nonpregnant women, manikin studies, or case reports. Epidemiological studies are needed to document the incidence of cardiac arrest in pregnancy as there as is a perception that it is increasing because of increased numbers of women with congenital heart conditions who are now having children.

Cardiac arrest in morbid obesity[ALS-SC-074A]

In morbidly obese adult patients with cardiac arrest (out-of-hospital or in-hospital), does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

Evidence from two studies did not find a survival difference associated with obesity following out-of-hospital cardiac arrest (LOE 2).[463–465]

Treatment recommendation

There is insufficient evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for obese patients.

Knowledge gaps

There is a paucity of research in this area, and studies looking at epidemiology, current variations from the standard protocol, and associated outcomes, as well as simple experimental studies, would be helpful.

Cardiac arrest caused by asthma[ALS-SC-067B]

In adult cardiac arrest due to asthma, does any modification of treatment, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs that specifically evaluate or compare adjuvant treatment with standard treatment for cardiac arrest in asthmatic patients. Most of the literature comprises case reports and case series.

Evidence from three non-cardiac arrest case series involving 35 patients suggests that asthmatic patients are at risk for gas trapping during cardiac arrest, especially if their lungs are ventilated with high tidal volumes and/or rapid rates (LOE 5).[466–468] One volunteer adult study demonstrated that increasing PEEP caused increased transthoracic impedance (LOE 5).[469]

Seven case series involving 37 patients suggested increased ease of ventilation and ROSC with lateral chest compressions at the base of the ribs (LOE 4).[470–476] In a single case report, lateral chest compressions were associated with cardiac arrest and poor cardiac

output (LOE 4).[477] Three single case reports (two intraoperative and one ED) involving cardiac arrest caused by asthma suggested improvement in ease of ventilation and ROSC with thoracotomy and manual lung compression (LOE 4).[471,475,476]

Treatment recommendation

There is insufficient evidence to suggest any routine change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by asthma.

Knowledge gaps

Several key areas for research include: the role of disconnecting from positive pressure ventilation and the ideal duration of this disconnection; the role of lateral external compression and the timing with respect to chest compressions; the comparison of these techniques and their cumulative advantage; and the role of magnesium infusions and ECMO in cardiac arrest caused by asthma.

Cardiac arrest caused by anaphylaxis[ALS-SC-066A,ALS-SC-066B]

In adult cardiac arrest caused by anaphylaxis, does any modification of treatment, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs evaluating conventional versus alternative therapies for the treatment of cardiac arrest caused by anaphylaxis. Evidence is limited to case reports, extrapolations from nonfatal cases, interpretation of pathophysiology, and animal studies.

One human study of a randomised venom immunotherapy trial where 19 of 21 patients became symptomatic and required emergency treatment suggests that carefully titrated continuous infusion of IV adrenaline in addition to volume infusion may be effective for the treatment of anaphylactic shock (not in cardiac arrest) (LOE 5).[478] One randomised controlled crossover study of animals preshock, but symptomatic with ragweed sensitivity, showed that a continuous IV infusion of 0.01 mg kg^{-1} adrenaline maintained a mean arterial pressure at 70% of preshock levels better than no treatment or bolus treatment (LOE 5).[479]

A small case series of patients with anaphylactic shock with or without cardiac arrest suggested that patients who did not respond to standard therapy may benefit from vasopressin (LOE 4).[480,481] A few small case series (LOE 4) have described promising initial findings with α-agonists such as noradrenaline,[482] methoxamine,[483] terlipressin,[484] and metaraminol.[485–487] A few small case reports (LOE 4) of cardiac arrest suggest cardiopulmonary bypass[488,489] or mechanical support of circulation[490] may be helpful in the setting of anaphylaxis.

Several case reports (LOE 4) document the use of a variety of interventions for cardiac arrest caused by anaphylaxis: six case reports support high dose $\alpha - 1$ receptor agonists: metaraminol,[485,486] methoxamine,[483,487] and noradrenaline.[482] Other case reports document the use of terlipressin,[484] vasopression,[481] steroids and antihistamines,[491] and cardiopulmonary bypass.[488,489]

Treatment recommendation

There is insufficient evidence to suggest any routine change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by anaphylaxis.

Knowledge gaps

Future research should consider a comparison between the different IV α-agonists and a comparison of infusion versus bolus doses for cardiac arrest caused by anaphylaxis. The value of secondary therapies such as glucagon, antihistamines, volume infusions, and steroids should be explored.

Drug overdose and poisoning

The majority of questions addressing cardiac arrest caused by drug toxicity remain unanswered. Epidemiological studies are required to document the incidence of cardiac arrests caused by drugs, current treatment strategies, and the safety and efficacy of existing treatments. Animal models, controlled clinical trials, and pharmacodynamic studies are needed to advance the treatment of cardiac arrest caused by drugs. Most of the evidence is limited to case reports, extrapolations from nonfatal cases (including severe cardiovascular toxicity cases), and animal studies.

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Cardiac arrest caused by local anaesthetic[ALS-SC-073-01A]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by local anaesthetic toxicity, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

Local anaesthetic toxicity typically occurs in the setting of regional anaesthesia, when a bolus of local anaesthetic inadvertently enters the arterial or venous system, leading to refractory seizures and/or rapid cardiovascular collapse. There are no RCTs evaluating conventional versus alternative therapies for the treatment of cardiac arrest caused by local anaesthetics (lidocaine). Evidence is limited to case reports involving cardiac arrest and severe cardiovascular toxicity and animal studies.

Five single-case reports describe patients in cardiac arrest attributed to local anaesthetic intoxication, who were refractory to advanced life support conventional treatment, but who obtained ROSC soon after treatment with IV lipid emulsion (LOE 4).[492–496] Five single-case reports (LOE 5) describe patients with acute, life-threatening cardiovascular toxicity from local anaesthetic intoxication, but who were not pulseless at the time of lipid administration. In three cases[497–499] severe cardiovascular toxicity resolved rapidly following IV lipid, but in two other cases[500,501] the patient's condition deteriorated to cardiac arrest after IV lipid, although the patients were resuscitated and survived to hospital discharge.

Five controlled animal studies demonstrated that a variety of dosages of IV lipid emulsion were more effective than placebo in models of local anaesthetic intoxication with ROSC as the primary outcome (LOE 5).[502–506]

Two controlled animal studies suggested that, in combination with basic life support (BLS), IV lipid emulsion improved the rate of ROSC when compared with vasopressor therapy (vasopressin and adrenaline) (LOE 5).[503,506] Contrasting results were published in one controlled animal study that demonstrated a survival advantage with vasopressin and adrenaline over lipid emulsion therapy in a model of asystole induced by low-dose bupivacaine and asphyxia

(LOE 5).[507] Two controlled animal studies reported no additional benefit from lipid emulsion infusions when combined with high-dose adrenaline 0.1 mg kg^{-1} (LOE 5)[508] and 0.01 and 0.025 mg kg^{-1} (LOE 5).[509] Lipid emulsion bolus doses and infusion rates vary across case reports and animal studies. Typical bolus doses were 1–3 mL kg^{-1}. When infusions were used the typical doses were 0.1–0.3 mL kg^{-1} h^{-1}. A 20% solution of long-chain fatty acid emulsion was used in almost all reports.

Two controlled animal studies showed a survival advantage when cardiac arrest from local anaesthetic toxicity was treated with high-dose insulin (1–2 U kg^{-1} IV bolus) accompanied by glucose and sometimes potassium, compared with basic life support resuscitation alone (LOE 5).[510,511] There were no animal studies comparing this intervention with advanced life support resuscitation.

The use of clonidine (150 μg boluses, repeated as needed) to treat cardiac arrest caused by local anaesthetic was described in one human case report (LOE 4)[512] while a second case report (LOE 4)[513] was neutral, and a third (LOE 5),[499] opposed. An animal study demonstrated partial improvement in bupivacaine-induced intracardiac conduction delays following clonidine administration (0.01 mg kg^{-1} IV), but nonperfusing rhythms were not studied (LOE 5).[514]

Treatment recommendation

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by local anaesthetics. Animal studies and case reports suggest severe cardiovascular toxicity or cardiac arrest attributable to local anaesthetic intoxication may respond to treatment with IV lipid emulsion.

Knowledge gaps

Controlled clinical trials and pharmacodynamic studies are needed to advance the treatment of cardiac arrest caused by local anaesthetics.

Benzodiazepine toxicity[ALS-SC-073-02A]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by benzodiazepine toxicity, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

No human studies or reports of any patients who had cardiac arrest solely resulting from benzodiazepine toxicity alone were identified.

Five reports of cardiac arrests resulting from exposure to combinations of medication that included one of the benzodiazepines were identified (LOE 4).[515–519] One case report indicated that standard care alone was sufficient to reverse the severe cardiovascular toxicity attributed to an anaphylactic reaction to a benzodiazepine (LOE 5).[520]

One case report described improved outcome when minor cardiovascular toxicity caused by benzodiazepines was treated with flumazenil (LOE 5).[521] Four studies indicated that flumazenil is unlikely to improve haemodynamic function in the setting of benzodiazepine overdose and may complicate other therapy (LOE 5).[518,522–524] Two studies described serious adverse effects such as seizure, arrhythmia, hypotension, and withdrawal syndrome after flumazenil was given to patients presenting with decreased level of consciousness attributed to either benzodiazepine toxicity or an unknown cause (LOE 5).[518,525] These side effects were more common with coingestants (such as tricyclic antidepressant and opioids), chronic benzodiazepine use or abuse, and known seizure disorder.

Treatment recommendation

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by benzodiazepines.

Knowledge gaps

Controlled clinical trials are needed to advance the treatment strategies for the management of cardiac arrest due to benzodiazepine toxicity.

β-Blocker toxicity[ALS-SC-073-03B]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by β-blocker toxicity, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs evaluating conventional versus alternative treatment of cardiac arrest caused by β-blockers. Evidence is limited to case reports, extrapolations from nonfatal cases, severe cardiovascular toxicity cases, and animal studies. The wide variety of β-blockers with differing pharmacological and physiochemical profiles makes it difficult to generalise from the limited data available.

In 13 case studies ($n = 16$) of human patients with severe cardiovascular toxicity caused by β-blockers refractory to standard treatment, including vasopressors, the administration of glucagon (50–150 μg kg^{-1}) was followed by haemodynamic improvement and survival (LOE 5).[526–538]

In two animal studies, high-dose insulin infusions (1 U kg^{-1} h^{-1}) given with glucose supplementation and electrolyte monitoring appeared effective (as measured by rates of improved haemodynamic stability and survival) in the setting of cardiovascular toxicity associated with β-blockers (LOE 5).[539,540] A single human case report documented that high-dose insulin (10 U kg^{-1} h^{-1} IV), given with glucose supplementation and electrolyte monitoring, was followed by improved haemodynamic stability and survival to hospital discharge in the setting of severe cardiovascular toxicity associated with β-blocker toxicity (LOE 5).[541]

Case reports described the use of phosphodiesterase inhibitors (LOE 5),[542,543] calcium salts (LOE 4),[544] extracorporeal support (LOE 5),[545] intraaortic balloon pumps (LOE 4),[546] and ECMO (LOE 4).[547] Animal studies supported the use of calcium salts (LOE 5)[548] and the phosphodiesterase inhibitor amrinone (LOE 5).[549] Animal studies suggested that dopamine (LOE 5),[550] a combination of dopamine and isoprenaline (LOE 5),[551] and milrinone (LOE 5)[552] may decrease the effectiveness of glucagon as an antidote for β-blocker toxicity.

Treatment recommendation

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by β-blockers. Animal studies and case reports suggest severe cardiovascular toxicity caused by β-blockers may respond to treatment with intravenous glucagon, high-dose insulin (with glucose supplementation and electrolyte monitoring), or IV calcium salts in addition to conventional treatment.

Knowledge gaps

Controlled clinical trials are needed to advance the treatment of cardiac arrest caused by β-blockers. While case reports focus on propranolol toxicity, the different properties of other β-blockers

may affect the response to the suggested treatment. Other special interest topics include the use of new and emerging therapies, namely IV lipid infusion and high-dose insulin and the safety and effectiveness of glucagon in combination with new therapies.

Calcium channel blocker toxicity[ALS-SC-073-04B]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by calcium channel blocker toxicity, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs evaluating conventional versus alternative therapies for the treatment of cardiac arrest caused by calcium channel blockers. Evidence is limited to extrapolations from nonfatal case reports of severe cardiovascular toxicity.

In 16 human case series ($n = 28$) high-dose insulin (bolus 0.5–2 U kg^{-1} followed by 0.5 U kg^{-1} h^{-1} infusion) given with glucose supplementation and electrolyte monitoring appeared effective (as measured by improved haemodynamic stability [25/28] and survival [26/28]) in the setting of severe cardiovascular toxicity associated with calcium channel blockers (LOE 5).[553–568]

Treatment recommendation

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by calcium channel blockers. Case reports suggest severe cardiovascular toxicity caused by calcium channel blockers may respond to treatment with high-dose insulin given with glucose supplementation and electrolyte monitoring in addition to conventional treatment.

Knowledge gaps

Controlled clinical trials are needed to advance the treatment of cardiac arrest caused by calcium channel blockers. While case reports focus on verapamil toxicity, the different properties of other calcium channel blockers may affect the response to the proposed treatment. Other special interest topics include the use of vasopressin to treat severe cardiovascular toxicity caused by dihyropyridines, the use of combination therapy, sequencing of interventions, and the evaluation of new and emerging therapies, namely IV lipid infusion and calcium sensitisers and nonpharmacological interventions.

Carbon monoxide toxicity[ALS-SC-073-05]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by carbon monoxide toxicity, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

Three studies suggested that most patients who develop cardiac arrest from carbon monoxide poisoning will not survive to hospital discharge, regardless of whether hyperbaric oxygen therapy is administered following ROSC (LOE 4).[569–571]

Two studies (LOE 5) suggested that neurological outcomes were improved in patients (all severity excluding cardiac arrest[572]; and mild-to-moderate, excluding loss of consciousness and cardiac instability[573]) who received hyperbaric oxygen therapy for carbon monoxide poisoning. However, two studies found no difference in neurologically intact survival (LOE 5).[574,575] Two systematic reviews concluded that improvement in neurologically intact survival following the administration of hyperbaric oxygen to carbon monoxide poisoning patients was possible but unproven (LOE 5).[576,577]

Two studies demonstrated that patients with carbon monoxide toxicity treated with hyperbaric oxygen who developed myocardial infarction have an increased risk of cardiovascular and all-cause mortality lasting at least seven years after the event (LOE 5).[578,579]

Treatment recommendation

Patients who develop cardiac arrest caused by carbon monoxide rarely survive to hospital discharge, even if ROSC is achieved; however, hyperbaric oxygen therapy may be considered in these patients because it may reduce the risk of developing persistent or delayed neurological injury. The risks inherent in transporting critically ill postarrest patients to a hyperbaric facility may be significant; it must be weighed against the possibility of benefit on a case-by-case basis. Patients who develop myocardial injury caused by carbon monoxide have an increased risk of cardiac and all-cause mortality lasting at least seven years after the event; it is reasonable to recommend cardiology follow-up for these patients.

Knowledge gaps

The epidemiology of cardiac arrest and severe cardiotoxicity caused by carbon monoxide needs further documentation. More precise estimates of the proportion of patients who survive to hospital discharge and who have full neurological recovery following severe carbon monoxide poisoning treated with various interventions are needed. Though challenging, further prospective treatment studies are important and necessary.

Cocaine toxicity[ALS-SC-073-06B]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by cocaine, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

Cardiac arrest (primary question). There are no RCTs evaluating conventional versus alternative therapies for the treatment of cardiac arrest caused by cocaine. Evidence is limited to a small case series that demonstrated excellent overall and neurologically intact survival (12/22, 55%) in patients with cocaine-associated cardiac arrest treated with standard therapy (LOE 4).[580]

Severe cardiotoxicity caused by cocaine (secondary question). No studies were found that addressed the treatment of severe cardiotoxicity caused by cocaine; however, human studies have evaluated the treatment of cocaine-associated wide-complex tachycardia and ischaemic acute coronary syndrome, as well as coronary artery vasospasm caused by cocaine. Thus the benefit or harm of specific agents in cocaine-associated peri-arrest states (defined as severe hypertension, tachycardia, cocaine-induced arrhythmias) is informed by LOE 5-studies (extrapolation for nonarrest patients and, in some cases, cocaine na[uml]ive patients).

α-Blockers. A single study demonstrated reversal of cocaine-induced coronary artery vasospasm in the coronary catheterisation laboratory with phentolamine (LOE 5).[581]

Benzodiazepines. A single study (LOE 5)[582] of patients with cocaine-associated chest pain demonstrated improved autonomic findings and resolution of chest pain when treated with diazepam. An additional study reported no additional benefit associated with benzodiazepine administration in patients already receiving nitroglycerin (LOE 5).[583]

β-Blockers. A retrospective case series of patients hospitalised for acute coronary syndrome associated with cocaine use suggested that there was a decrease in the incidence of death and nonfatal myocardial infarction with the use of β-blockers (LOE 5).[584] A prospective clinical trial in cocaine-na[uml]ve volunteers suggested that propanolol reduced cocaine-induced tachycardia (LOE 5).[585] A prospective clinical trial demonstrated worsening of cocaine-induced coronary artery vasoconstriction following the administration of propanolol to cocaine-na[uml]ive research subjects (LOE 5).[586] A retrospective case series of seven ED and hospitalised patients with cocaine-associated cardiovascular toxicity demonstrated no consistent improvement in hypertension or tachycardia following treatment with esmolol (LOE 5).[587] Three of seven patients developed apparent adverse effects (hypertension, hypotension, and CNS depression with vomiting).

β-Blockers with partial α-adrenergic antagonism. In a pair of double-blind, crossover studies (LOE 5) of volunteers with a history of crack cocaine use, pretreatment with oral carvedilol[588] or labetolol[589] attenuated the cocaine-induced increases in heart rate and blood pressure compared with placebo, without apparent adverse effect. A prospective clinical trial demonstrated no change in cocaine-induced coronary artery vasoconstriction following the administration of labetolol to cocaine-na[uml]ive research subjects (LOE 5).[590]

Calcium channel blockers. One study of cocaine-naüive human volunteers demonstrated resolution of cocaine-induced coronary artery vasospasm with verapamil (LOE 5).[591]

Lidocaine. A retrospective case series of 29 patients who received lidocaine in the setting of cocaine-associated myocardial infarction included eight patients with wide-complex tachycardia (two sustained, six nonsustained) (LOE 5).[592] No patient developed complications and all survived the event.

Morphine. One study of cocaine-na[uml]ive human volunteers demonstrated that morphine partially reversed cocaine-induced coronary artery vasospasm (LOE 5).[593]

Nitroglycerin. In a clinical trial of cocaine-na[uml]ive volunteers administration of nitroglycerin reversed cocaine-induced coronary artery vasospasm (LOE 5).[594] In a prospective observational study of patients presenting with cocaine-associated acute coronary syndrome, 37/83 (45%) of patients treated with nitroglycerin reported reduction in the severity of chest pain, while five patients had other forms of clinical improvement (resolution of ischaemia based on ECG, two; hypertension, two; or congestive heart failure, one) (LOE 5).[595]

Treatment recommendation

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest or cardiotoxicity caused by cocaine. In patients with severe cardiovascular toxicity (defined as severe hypertension, tachycardia, and/or cocaine-induced arrhythmias) it may be reasonable to try drugs known to be effective in acute coronary syndromes: α-blockers (phentolamine), benzodiazepines (lorazepam, diazepam), calcium channel blockers (verapamil), morphine, and sublingual nitroglycerin. The available data do not support the use of one drug over another.

Knowledge gaps

Controlled clinical trials are needed to advance the treatment of cardiac arrest and cardiotoxicity due to cocaine. Future studies should evaluate the role of sodium bicarbonate and lidocaine and the safety and effectiveness of other antiarrhythmic drugs, such as amiodarone, in the treatment of cocaine-associated VT.

Cyanide toxicity[ALS-SC-073-07]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by cyanide, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs evaluating conventional versus alternative therapies for the treatment of cardiac arrest caused by cyanide. The use of hydroxocobalamin (alone or with sodium thiosulfate) for cardiac arrest caused by cyanide was suggested by three LOE 4-studies.[596–598] The use of hydroxocobalamin (alone or with sodium thiosulfate) in life-threatening cardiovascular toxicity was supported by seven studies (LOE 5).[596–602]

The use of nitrites plus sodium thiosulfate was suggested by three studies, none of which enrolled cardiac arrest patients (LOE 5)[600,603,604]; however, one additional study found no benefit to this strategy (LOE 5).[605]

Treatment recommendation

Patients with severe cardiotoxicity (cardiac arrest, cardiovascular instability, metabolic acidosis, or altered mental status) caused by known or suspected cyanide poisoning should receive cyanide antidote therapy. In addition to standard resuscitation, initial therapy should include a cyanide scavenger (either IV hydroxocobalamin or a nitrite—i.e., IV sodium nitrite and/or inhaled amyl nitrite), followed as soon as possible by IV sodium thiosulfate. Hydroxocobalamin and nitrites are equally effective, but hydroxocobalamin may be safer because it does not cause methaemoglobin formation or hypotension.

Knowledge gaps

Controlled clinical trials are needed to advance the treatment of cardiac arrest and cardiotoxicity caused by cyanide. Comparative studies on antidote therapy and health outcomes including neurological outcomes are required to address the question of which combination of drugs is most effective.

Tricyclic antidepressant toxicity[ALS-SC-073-08B]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by tricyclic antidepressants, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs evaluating conventional versus alternative treatments for cardiac arrest caused by tricyclic antidepressant toxicity. Evidence was limited to one small case series of cardiac arrest patients; it demonstrated improvement with the use of sodium bicarbonate and adrenaline (LOE 4).[606] Notably in that case series the prearrest use of physostigmine was a significant potential confounder.

The evidence for the management of cardiotoxicity caused by tricyclic antidepressant was limited to case reports, case series, and animal studies. The use of sodium bicarbonate has been described in two case series (LOE 5)[607,608] and six animal studies (LOE 5).[609–614] The use of hyperventilation was described in one small case series (LOE 5)[615] and one animal study (LOE 5).[612] The evidence for the efficacy of specific antidysrhythmics (lidocaine, magnesium, amiodarone, and phenytoin) was limited to negative case reports (LOE 5).[612,616–622] Specific vasopressors that have been associated

with improvement in the treatment of tricyclic-induced hypotension include noradrenaline (LOE 5),[618,623–625] adrenaline (LOE 5),[611,618,626] dopamine (LOE 5),[625,627,628] and dobutamine (LOE 5).[627] Diazepam improved seizure control and survival in one animal study (LOE 5).[627] The use of physostigmine for tricyclic-induced anticholinergic symptoms was not supported by the current literature given the conflicting associations suggested by several case series (LOE 4[613]; LOE 5[608,629,630]). Limited animal research demonstrates a benefit for IV lipid infusions in models of tricyclic toxicity (LOE 5).[631,632] Antitricyclic Fab has been beneficial in animal models of varying degrees of tricyclic cardiotoxicity (LOE 5),[633–638] and one small human study (LOE 5)[639] provided evidence of safety and pharmacokinetic advantage; however, clinical benefit has yet to be demonstrated clearly.

Treatment recommendation

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest or cardiotoxicity caused by tricyclic antidepressants. Because sodium bicarbonate bolus is the mainstay of therapy in the setting of tricyclic-induced cardiac conduction abnormalities, and this treatment strategy should be applied to the postarrest period of care for patients surviving cardiac arrest caused by tricyclic antidepressant toxicity associated with wide QRS complexes. When mechanical ventilation is required, respiratory acidosis should be avoided.

Knowledge gaps

Controlled clinical trials are needed to advance the treatment of cardiac arrest and cardiotoxicity caused by tricyclic antidepressants. Future trials exploring novel therapies (Fab, IV lipid infusions) and the use of sodium bicarbonate for hypotension in the absence of cardiac conduction abnormalities would be helpful.

Digoxin toxicity[ALS-SC-073-09A]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by digoxin, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs evaluating conventional versus alternative treatments for cardiac arrest caused by digoxin. Evidence is limited to 14 studies demonstrating the usefulness of antidigoxin Fab fragments for severe cardiac glycoside toxicity (LOE 5).[640–653]

Treatment recommendation

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by digoxin. In adults and children with severe cardiovascular toxicity caused by digoxin and related cardiac glycosides, antidigoxin Fab fragment therapy should be administered.

Knowledge gaps

Animal models and controlled clinical trials are needed to advance the treatment of cardiac arrest caused by digoxin. Pharmacokinetic and clinical studies would help establish the dosing of antidigoxin Fab fragment for digoxin cardiotoxicity.

Opioid toxicity[ALS-SC-073-10]

In adult cardiac arrest (prehospital or in-hospital) caused by opioids, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

There are no RCTs evaluating conventional versus alternative treatments for cardiac arrest caused by opioids. Evidence is limited to studies of mild, moderate, and severe cardiovascular toxicity (LOE 5 for cardiac arrest). Evidence from studies assessing other endpoints (efficacy of naloxone), as well as animal studies, support the use of assisted ventilation before giving naloxone in opioid-poisoned patients with severe cardiopulmonary toxicity (LOE 1[654,655]; LOE 3[656]; LOE 4[657-659]; LOE 5[660]).

The use and safety of naloxone is supported by human studies (LOE 4),[657,661–664,658,659] as well as those assessing other endpoints (alternate routes of administration) (LOE 1[654]; LOE 3[656]; LOE 4[665,666]). Naloxone can be given intravenously (LOE 4),[657,658,662,665]intramuscularly (LOE 1[654]; LOE 4[657,658]), intranasally (LOE 1[654]; LOE 4[665]), and into the trachea (LOE 5).[667]

Treatment recommendation

There is insufficient clinical evidence to suggest any change to cardiac arrest resuscitation treatment algorithms for patients with cardiac arrest caused by opioids. In adults with severe cardiovascular toxicity caused by opioids, ventilation should be assisted using a bag-mask, followed by naloxone, and tracheal intubation if there is no response to naloxone. Naloxone should be given intravenously or intramuscularly. Intranasal or tracheal routes may be used if conditions preclude IV or intramuscular administration.

Knowledge gaps

Animal models and controlled clinical trials are needed to advance the treatment of cardiac arrest caused by opioids. In particular such studies should determine if naloxone has a role in the resuscitation of the cardiac arrest patient pre- or post-ROSC.

Cardiac arrest during coronary catheterisation[ALS-SC-068B, ALS-SC-068C]

In adult cardiac arrest during percutaneous coronary intervention, does use of any specific intervention, as opposed to standard care, improve outcome?

Consensus on science

There are no RCTs evaluating alternative treatment strategies as opposed to standard care for cardiac arrest during percutaneous coronary intervention (PCI). Evidence is limited to case studies for all interventions.

Mechanical CPR during PCI. Three adult human case reports (LOE 4),[668–670] two adult human case series (LOE 4),[671–673] and one animal study (LOE 5)[669] reported that the use of a mechanical chest compression device in cardiac arrest during PCI maintained circulation and enabled the procedure to be completed. Although a small proportion of patients in the case series (13/60) survived to hospital discharge, no randomised controlled or comparison study of this intervention has been performed.

Emergency cardiopulmonary bypass during PCI. One case study suggested that the use of emergency cardiopulmonary bypass to stabilise and facilitate emergency coronary angioplasty improved the survival of patients who had cardiac arrest during PCI that was unresponsive to advanced life support (LOE 4).[674]

Cough CPR during PCI. Five studies (LOE 4[675–677]; LOE 5[678,679]) supported the use of cough CPR as a temporary intervention

to maintain adequate blood pressure and level of consciousness in patients who developed ventricular arrhythmias during PCI[676,677,679] and PCI[678] while definite therapy for malignant arrhythmias was instituted.

Treatment recommendation

There are insufficient data to support or refute the use of mechanical chest compression, cough CPR, or emergency cardiopulmonary bypass to improve outcome of cardiac arrest during PCI.

Knowledge gaps

Clinical trials, perhaps initially with historical controls, are needed to advance the treatment of cardiac arrest during PCI.

Consensus on science

There are no RCTs evaluating alternative treatment strategies as opposed to standard care for cardiac arrest during percutaneous coronary intervention (PCI). Evidence is limited to case studies for all interventions.

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Treatment recommendation

There are insufficient data to support or refute the use of mechanical chest compression, cough CPR, or emergency cardiopulmonary bypass to improve outcome of cardiac arrest during PCI.

Knowledge gaps

Clinical trials, perhaps initially with historical controls, are needed to advance the treatment of cardiac arrest during PCI.

Cardiac arrest after open or closed heart surgery[ALS-SC-069A, ALS-SC-069B, ALS-SC-069C]

In adult cardiac arrest following open (including heart and lung transplantations) and closed heart surgery, does use of any specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

Resternotomy. Eleven studies documented improvement in outcome in patients with cardiac arrest following cardiac surgery who were treated with resternotomy and internal cardiac compression compared with standard protocol, when administered by experienced personnel in ICUs (LOE 2[680,681]; LOE 4[682–690]). Five studies neither supported nor opposed this finding (LOE 4[691–694]; LOE 5[695]). One study documented that the risk of infection was not significant after resternotomies conducted appropriately outside of the operating room (LOE 4)[689]; whereas three studies demonstrated very poor outcomes when resternotomy was performed outside an ICU (LOE 2[680]; LOE 4[686]; LOE 5[695]).

Mechanical circulatory support. Six studies supported the use of mechanical circulatory support devices during cardiac arrest following cardiac surgery (LOE 3[690]; LOE 4[696–698]; LOE 5[699,700]). Three studies reported equivocal findings (LOE 5).[701–703] No studies opposed use of mechanical circulatory support. Mechanical circulatory support devices in these studies included extra-corporeal membrane oxygenation or cardiopulmonary bypass.

Graft damage by chest compressions. Two case reports described damage to the heart caused possibly by external chest compressions before resternotomy (LOE 5).[704,705]

Adrenaline. One study reported two cases that responded to escalating doses of adrenaline (LOE 4).[706]

Antiarrhythmic therapy. One study reported 18 cases with VF/VT after cardiac surgery (LOE 4).[707]

Treatment recommendation

Resternotomy for patients with cardiac arrest following cardiac surgery should be considered in an appropriately staffed and equipped ICU. Resternotomy performed outside these specialised environments has poor results. Chest compressions should not be withheld while preparing for emergency resternotomy. Mechanical circulatory support may be considered in the setting of cardiac arrest following cardiac surgery. There is insufficient evidence to make any recommendations about adrenaline dose, antiarrhythmic use, or any other intervention separate from those recommended in standard protocols.

Knowledge gaps

Clinical trials are needed to determine the safety and efficacy of mechanical circulatory support devices, chest compressions, and pharmacological adjuncts for the treatment of cardiac arrest after cardiac surgery.

Cardiac arrest caused by cardiac tamponade[ALS-SC-070B]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by cardiac tamponade, does use of specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

Five studies indicate that echocardiographically guided pericardiocentesis is a safe and effective method of relieving tamponade, especially when used in conjunction with a pericardial drain, and it may obviate the need for subsequent treatment in the operating room (LOE 5).[708–712]

One study documented 39 patients who received prehospital emergency thoracotomy by physicians to treat cardiac arrest from penetrating trauma (LOE 4).[713] Eighteen patients had cardiac tamponade and four (22%) survived. Two additional studies indicated that ED thoracotomy may be beneficial in patients who have cardiac arrest associated with cardiac tamponade and may yield improved results over standard needle pericardiocentesis (LOE 4).[714,715] One study indicated that ED thoracotomy may be especially beneficial if gross blood causes clotting and blocking of a pericardiocentesis

needle (LOE 2).[716] Two studies indicated that emergency thoracotomy may also be beneficial in patients who have postprocedure complications (LOE 4).[682,717] One study indicated that a more definitive sternotomy or thoracotomy in an operating room may also be beneficial if transportation to the operating room does not introduce significant delay (LOE 5).[718]

Treatment recommendation

Pericardiocentesis guided by echocardiography should be considered for treatment of cardiac arrest associated with cardiac tamponade while nonimage-guided pericardiocentesis is an acceptable alternative if echocardiography is not available. Placement of a pericardial drain may be beneficial and may obviate the need for subsequent treatment in the operating room. ED thoracotomy and pericardiotomy should be considered as an acceptable alternative to operating room thoracotomy and pericardiotomy for treatment of traumatic cardiac arrest associated with cardiac tamponade, and they can be considered for use in the treatment of nontraumatic cardiac arrest when pericardiocentesis is unsuccessful in relieving cardiac tamponade.

Knowledge gaps

Clinical trials should include patients with pericardial tamponade secondary to nontraumatic arrest and compare safety and efficacy of needle drainage versus thoracotomy and prehospital versus emergency department versus operating room thoracotomy.

Cardiac arrest caused by pulmonary embolus[ALS-SC-071B]

In adult cardiac arrest (out-of-hospital or in-hospital) caused by pulmonary embolus, does use of aetiology-specific interventions, as opposed to standard care (according to treatment algorithm), improve outcome (e.g., ROSC, survival)?

Consensus on science

One double-blind RCT showed no improvement in survival to discharge with the use of tissue plasminogen activator following cardiac arrest with PEA (LOE 1).[244] One RCT of fibrinolytics showed no difference in short- or long-term (30 days) survival or bleeding in patients randomised to receive tenecteplase or placebo during CPR (LOE 1).[245] Patients with suspected pulmonary embolism were excluded from the study if open thrombolysis was possible in the prehospital setting. Thirty-seven cases with suspected pulmonary embolism were randomised in the trial. Of those, 2 of 15 patients survived when treated with tenecteplase compared with no survivors in the 22 patients of the placebo-treated group.[245]

One meta-analysis of eight retrospective cohort studies with a variety of causes of cardiac arrest (pulmonary embolism, two studies; myocardial infarctions, four studies; cardiology diseases, one study; and nontraumatic etiologies, one study) demonstrated an increased rate of ROSC, survival to discharge, and long-term neurological function with fibrinolytic, but it also showed an increased risk of severe bleeding (LOE 2).[719]

Nine studies of patients with presumed pulmonary embolism or all patients with cardiopulmonary arrests showed improvement with fibrinolysis in ROSC and admission to the hospital or ICU, but no improvement in survival to discharge (LOE 1[246]; LOE 2[248,250]; LOE 3[251]; LOE 4[247,720–723]). Three studies showed good neurological function in those who survived after successful fibrinolysis during CPR (LOE 2[719]; LOE 3[722]; LOE 4[721]).

Treatment recommendation

Fibrinolytic therapy may be considered when pulmonary embolism is suspected as the cause of the cardiac arrest. Routine use of fibrinolytics in undifferentiated cardiac arrest is addressed earlier in "Fibrinolytics."

Knowledge gaps

The true incidence of pulmonary embolus as a cause of cardiac arrest is not well documented. Surveillance studies of cardiac arrest noting contributing factors and pathological reports may help define the impact on public health of this cause of cardiac arrest.

Cardiac arrest caused by electrolyte disorders[ALS-SC-076A, ALS-SC-076B]

In adult cardiac arrest (out-of-hospital and in-hospital), does the treatment of electrolyte disturbances (e.g., hypokalaemia, hyperkalaemia, hypomagnesaemia, hypermagnesaemia, hypocalcaemia, or hypercalcaemia), as opposed to standard care (according to treatment algorithm, but without treatment of electrolyte disturbances), improve outcome (e.g., ROSC, survival)?

Consensus on science

Magnesium. No studies were identified that addressed specifically the correction of low magnesium concentrations. The presence of a low plasma magnesium concentration was associated with poor prognosis in cardiac arrest patients in three studies (LOE 5).[724–726] The use of magnesium in cardiac arrest was supported by five case series (LOE 4)[727–731]; however, five RCTs (LOE 1)[219–222,732] and a systematic review (LOE 1)[733] found no benefit from the use of magnesium in cardiac arrest.

Calcium. No studies were identified that specifically addressed the treatment of cardiac arrest caused by hypocalcaemia or hypercalcaemia.

Potassium. There are no randomised trials on the treatment of potassium abnormalities in the setting of cardiac arrest. The management of hypokalaemia and hyperkalaemia in the setting of cardiac arrest is based on case reports and animal studies. One case series of two patients reported the resolution of torsades de pointes with potassium replacement in patients with hypokalaemia (LOE 4).[734] Several clinical studies report an association between hypokalaemia and the development of VF (LOE 5),[724,735–737] and an animal study reported that hypokalaemia lowers the VF threshold (LOE 5).[738] In an animal model of cardiac arrest, it was reported that hyperkalemic animals had a higher rate of survival (LOE 5).[739]

Treatment recommendation

There are insufficient data to support or refute the routine treatment of electrolyte abnormalities during cardiac arrest resuscitation.

Knowledge gaps

Epidemiological studies are required to document the incidence of cardiac arrests secondary to electrolyte disturbance. Studies are needed to determine the safety and efficacy of current treatments and electrolyte replacement strategies during cardiac arrest.

Identifying reversible causes

Ultrasound during cardiac arrest[ALS-CPR&A-003B]

In adult cardiac arrest (out-of-hospital or in-hospital), does the use of ultrasound (including transthoracic and transoesophageal

echocardiography) during cardiac arrest, compared with standard CPR, improve any outcomes (e.g., ROSC, survival)?

Consensus on science

No studies examined the impact of ultrasound or echocardiography on patient outcomes in cardiac arrest specifically. Three studies examined the prognostic value of the presence or absence of sonographic cardiac motion in cardiac arrest (LOE 4).[184,740,741] One retrospective chart review (LOE 4)[742] and one prospective comparison (LOE 4)[743] documented the diagnostic accuracy of transoesophageal ultrasound in detecting the cause of circulatory collapse. One study documented the frequency of pulmonary embolism in PEA arrest as detected with transoesophageal ultrasound (LOE 4).[744] An additional two prospective observational studies examined the use of transthoracic ultrasound by "nonexpert" sonographers to detect pericardial effusion and other causes of PEA (LOE 4[745]; LOE 5[746]).

Three prospective studies examined ultrasound determination of cardiac standstill as a predictor of clinical outcomes and ROSC in patients in cardiac arrest (LOE 4).[184,740,741] Absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest was highly predictive of death: of the 341 patients from the three studies, 218 had no detectable cardiac activity and only two of those had ROSC (no data on survival to hospital discharge).

Treatment recommendation

There is insufficient evidence to support or refute the routine use of ultrasound or echocardiography to guide cardiac arrest resuscitation.

Knowledge gaps

Future research should address the role ultrasound (both transoesophageal and transtracheal) can perform as a targeted intervention (detection of potential causes, guidance of key procedures) during cardiac arrest resuscitation. With increasing emphasis on uninterrupted chest compressions, there is the potential for harm with the use of transthoracic ultrasound because it often requires interruption of compressions and ventilation to acquire adequate images. This is less of a concern with transoesophageal or intracardiac echocardiography.

Postresuscitation care

Postresuscitation treatment protocol[ALS-PA-047A, ALS-PA-047B]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of comprehensive treatment protocol, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

There are no RCTs addressing the use of comprehensive treatment protocols after sustained ROSC. Before-and-after studies report increase in survival of comatose patients with sustained ROSC after out-of-hospital cardiac arrest with implementation of a comprehensive treatment protocol (LOE 2[747]; LOE 3[748,749]). Protocols included multiple elements such as hypothermia, glucose control, goal-directed haemodynamic optimisation, ventilation, and PCI. The independent effect of each element of the bundle of care could not be established.

Treatment recommendation

A comprehensive treatment protocol that includes multiple interventions provided in a structured way may improve survival after cardiac arrest.

Knowledge gaps

Studies are needed to determine whether a comprehensive treatment protocol after cardiac arrest with a sustained ROSC improves short- and long-term outcomes. Future studies should define what interventions other than hypothermia are important inclusions in an effective comprehensive treatment protocol.

Treatment of precipitating causes of cardiac arrest

Pulmonary embolism[ALS-PA-046A, ALS-PA-046B]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital) diagnosed as pulmonary embolism, does the use of early fibrinolytic therapy with or without thrombectomy, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

Despite good theoretical reasons why fibrinolysis following cardiac arrest in patients with suspected pulmonary embolism might be beneficial, there is no direct evidence to that effect. Several studies (LOE 5)[251,750] and[247,248,751] and a case series (LOE 4)[752] showed no significant increase in survival to hospital discharge. There was an increase in bleeding complications following fibrinolysis in most of those studies. One study suggested that the risk of major haemorrhage was further increased in patients who have undergone CPR (LOE 5).[247]

Five retrospective reviews demonstrated that pulmonary embolectomy following cardiac arrest had a high mortality rate (LOE 4).[753–757] One case series reported outcomes of seven patients who had a cardiac arrest caused by pulmonary embolism and who were treated with percutaneous mechanical thrombectomy (LOE 4)[720]; three patients also received recombinant tissue plasminogen activator. Only one of the seven patients died and pulmonary perfusion was restored in the majority (85.7%).

Treatment recommendation

In patients with diagnosed or suspected pulmonary embolism after ROSC following cardiac arrest, there is inadequate evidence to recommend for or against the use of fibrinolytic therapy in addition to heparin. Because the mortality with surgical embolectomy for suspected or diagnosed pulmonary embolism is high if it follows cardiac arrest and it should be avoided in patients who have received CPR. There are few data on percutaneous mechanical thromboembolectomy, but it may be beneficial and may be considered in patients sustaining cardiac arrest from a pulmonary embolism who are not candidates for fibrinolytic therapy.

Knowledge gaps

Clinical studies directly comparing fibrinolysis, standard therapy, and percutaneous mechanical thromboembolectomy in patients with ROSC following cardiac arrest from confirmed or suspected pulmonary embolism are needed to further advance our knowledge on safety and efficacy.

Ventilation[ALS-PA-053B]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of a specific ventilation strategy (including specific CO_2 goal), as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

There were limited studies that addressed alternative ventilation strategies after cardiac arrest. A human study (LOE 2)[758] and studies in animals (LOE 5)[759–762] indicated that hyperventilation reduced cerebral blood flow after cardiac arrest. This cerebral blood

flow response to hyperventilation and to hypoventilation may be absent after prolonged cerebral ischaemia (LOE 5).[763,764] Avoiding hyperventilation, as part of a bundle of care, improved long-term outcome in humans (LOE 3)[749] and in dogs (LOE 5),[765] but the independent effect of ventilation could not be determined. A single animal study suggested that hyperventilation reduced degenerating neurons (LOE 5).[766,767]

Use of tidal volumes $\leq 9\,mL\,kg^{-1}$ in patients after cardiac arrest is associated with increased incidence of atelectasis (LOE 3).[768] Manipulation of tidal volume and PEEP are not associated independently with improved survival in cohorts, including cardiac arrest patients (LOE 2[769]; LOE 3[768]).

Treatment recommendation

After restoration of circulation, routine hyperventilation leading to hypocapnia should be avoided in order to prevent additional cerebral ischaemia.

Knowledge gaps

It is unclear if the changes in cerebral blood flow caused by hypercapnia or hypocapnia are important because there are no studies that relate ventilation strategies to patient-oriented outcomes in patients with sustained ROSC after resuscitation from cardiac arrest.

Controlled oxygenation[ALS-PA-061A, ALS-PA-061B]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of a controlled oxygenation strategy (including specific oxygenation goal), as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

One neutral randomised prospective clinical trial compared ventilation with 30% oxygen or 100% oxygen for the first 60 min after ROSC (LOE 1).[770] Mean partial pressure of oxygen in arterial blood (PaO_2) at 60 min after ROSC was 14.6 ± 3.3 kPa (110 ± 25 mm Hg) in the 30% oxygen group and 46.5 ± 23.2 kPa (343 ± 174 mm Hg) in the 100% oxygen group. No statistical difference was detected in serum biomarkers of acute brain injury, survival to hospital discharge, or the percent of patients with good neurological outcome (cerebral performance category of 1 or 2) at hospital discharge. However, this study was not adequately powered to detect important differences in survival and cerebral performance category at hospital discharge ($n = 14$ per group). A significant subset of patients in this study (30%) who were ventilated with 30% oxygen after ROSC required increased FiO_2 to maintain a pulse oximetry reading of >95%. The study was underpowered to determine efficacy or harm.

One supportive animal cardiac arrest study demonstrated that ventilation with 100% oxygen (generating $PaO_2 > 450$ mm Hg) during the first 15–60 min after ROSC caused neurodegeneration and worse-function neurological outcome when compared with FiO_2 titrated to an arterial pulse oximetry reading between 94% and 96% (LOE 5).[771]

Six supportive animal cardiac arrest studies demonstrated that ventilation with 100% oxygen (generating $PaO_2 > 250$–350 mm Hg) during the first 10–60 min after ROSC causes increased brain lipid peroxidation, increased metabolic dysfunction (glucose utilization and mitochondrial dysfunction), increased neurodegeneration, and worse-functional neurological outcome when compared to ventilation with room air (LOE 5).[153,154,772–775] These studies reported only short-term evaluation of outcomes ($\leq$24 h).

One animal study did not detect any difference in outcomes at 72 h when animals were ventilated with 100% oxygen or room air during CPR and for the first hour after ROSC (LOE 5).[155] Another animal study failed to show any difference in outcome when comparing 2 levels of hypoxic FiO_2 (0.085 and 0.12) with normoxic resuscitation when given for the intra- and early (15 min) period after ROSC (LOE 5).[776] The study did not demonstrate a significant difference in neurological assessment scores at 72 h or in survival. The study also failed to show a significant difference in the serum biomarkers of oxidant injury.

One supporting animal study reported that a PaO_2 of 250–350 mm Hg during the first 10 min of cardiopulmonary bypass reperfusion after cardiac arrest resulted in worse cardiac function compared to a PaO_2 40–90 mm Hg during the same time period (LOE 5).[777] A second animal study found no difference in myocardial function or injury when PaO_2 was gradually increased from 40 to 110 mm Hg over the first 15 min of cardiopulmonary bypass reperfusion after cardiac arrest compared to initiating reperfusion at 90 to 110 mm Hg (LOE 5).[778]

Treatment recommendations

There is insufficient clinical evidence to support or refute the use of inspired oxygen concentration titrated to arterial blood oxygen saturation in the early care of cardiac arrest patients following sustained ROSC.

Knowledge gaps

Prospective randomised controlled clinical trials are needed to compare ventilation with 100% oxygen versus ventilation with inspired oxygen titrated to an arterial blood oxygen saturation goal (possibly 94–96%) for the first hour after sustained ROSC. Studies evaluating combined myocardial infarction and cardiac arrest are needed to evaluate the impact of post-cardiac arrest arterial hyperoxaemia on cardiovascular outcomes.

Support of the circulation

Fluid therapy[ALS-PA-043A, ALS-PA-043C]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital) who have cardiovascular dysfunction, does the use of IV fluids, as opposed to standard care (or other IV fluids), improve outcome (e.g., survival)?

Consensus on science

There are no human studies that compare the use of IV fluids after sustained ROSC in patients with cardiac dysfunction compared with no IV fluids. One small human study used IV fluid (0.9% saline or lactated Ringer's) as part of early goal-directed therapy in post-cardiac arrest syndrome and found an improvement in survival that was not statistically significant (LOE 5).[748] In an additional before-and-after study (LOE 5), IV fluids (0.9% saline, lactated Ringer's, or colloids) were administered as part of a package of care (including PCI and therapeutic hypothermia) that improved survival with favorable neurological outcome in adult patients with sustained ROSC after cardiac arrest (prehospital or in-hospital).[749] The intervention period had a significantly increased positive fluid balance (345 mL versus 2300 mL). Six human studies showed that rapid infusion of fluids (500–3000 mL of 0.9% saline or lactated Ringer's) to induce therapeutic hypothermia after sustained ROSC produced little evidence of harm (LOE 5).[779–784] One human study showed that the deterioration in oxygenation that occurs after ROSC was not significantly affected by the infusion of cold 0.9% saline (3427 ± 210 mL) (LOE 5).[785] Three animal studies reported neurological and cardiac protection with the administration of hypertonic fluid compared to normal saline (LOE 5).[786–788] One animal study showed an increase in cerebral blood flow with fluid for haemodilution combined with induced hypertension (LOE 5).[789]

Treatment recommendation

There is insufficient evidence to support or refute the routine use of IV fluids following sustained ROSC after cardiac arrest. Rapid infusion of cold 0.9% saline or lactated Ringer's appears to be well tolerated when used to induce therapeutic hypothermia. Based on the pathophysiology of post-cardiac arrest syndrome, it is reasonable to use IV fluids as part of a package of post-cardiac arrest care.

Knowledge gaps

Larger studies are needed to assess optimal fluid strategy for haemodynamic optimisation in patients with sustained ROSC after adult cardiac arrest.

Haemodynamic optimisation[ALS-PA-056B]

In adult patients (out-of-hospital and in-hospital) with ROSC after cardiac arrest, does early haemodynamic optimisation, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

There are no published RCTs addressing early haemodynamic optimisation after cardiac arrest. Only one study suggested that the introduction of haemodynamic optimisation (fluids, inotropic agents, intra-aortic balloon pump, and reperfusion) as part of a bundle of interventions improved outcome in comparison with historical controls (LOE 3).[749] The independent effect of early haemodynamic optimisation was not assessed in this study. A recent study that included early haemodynamic optimisation as part of a post-cardiac arrest treatment bundle was not powered to measure a survival benefit (LOE 3).[748]

Treatment recommendation

Despite limited clinical data, the known pathophysiology of post-cardiac arrest syndrome provides a rationale for titrating haemodynamics to optimise organ perfusion.

Knowledge gaps

Clinical research is needed to define the optimal targets for haemodynamic optimisation and the best strategies to achieve these targets (fluids, vasopressors, inotropes, circulatory support, etc.)

Cardioactive drugs[ALS-PA-057A]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital) who have cardiovascular dysfunction, does the use of any specific cardioactive drugs, as opposed to standard care (or different cardioactive drugs), improve outcome (e.g., survival)?

Consensus on science

There are no clinical trials that have determined or compared the independent effect of vasopressor and/or inotrope use in the post-cardiac arrest period on cardiovascular dysfunction and/or survival to discharge. Four clinical trials have suggested improved survival to discharge with vasopressor or inotropes, but have been confounded by multiple simultaneous treatments and/or they are underpowered for survival (LOE 3[748,749,790]; LOE 4[791]). Six experimental studies showed improvement in postresuscitation cardiac dysfunction (left ventricular function) with the administration of cardioactive drugs, such as dobutamine or levosimendan, but none have shown that such improvement in function translates into improved survival (LOE 5).[792–797]

Treatment recommendation

There is insufficient evidence to support or refute the routine use of vasopressors and/or inotropes for improving survival in adult patients with cardiovascular dysfunction after resuscitation from cardiac arrest.

Knowledge gaps

Specific clinical research is required to investigate whether treatment of post-cardiac arrest cardiovascular dysfunction with vasopressors and/or inotropes will yield incremental beneficial impact on long-term outcomes beyond those achieved with therapeutic hypothermia alone.

Antiarrhythmic drugs[ALS-PA-058A, ALS-PA-058B]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of prophylactic antiarrhythmic drugs, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

No controlled studies addressed specifically and directly the use of amiodarone, lidocaine, or β-blockers early or immediately after resuscitation from cardiac arrest. One uncontrolled retrospective study did not demonstrate an improvement in six-month survival when amiodarone or lidocaine was given to patients resuscitated from VF or tachycardia during early (first 72 h) in-hospital postresuscitation care (LOE 4).[798] One single prospective nonrandomised study suggested that recurrent VF was reduced and long- and short-term survival were improved in patients treated with β-blockers during electrical storm (LOE 5).[369] One study reported an incidence of approximately 5% for VF or VT in hospitalised post-cardiac arrest patients (LOE 4).[799] Five RCTs documented consistent improvement in all-cause mortality and sudden death when implantable cardioverter defibrillators were inserted as late, secondary prophylaxis compared with amiodarone or β-blocker administration to patients that survived VF or VT cardiac arrest (LOE 5).[800–804]

Treatment recommendation

There is no evidence to support or refute continued administration of amiodarone or lidocaine in post-cardiac arrest patients after ROSC.

Knowledge gaps

The incidence of recurrent ventricular arrhythmias after hospital admission following survival of cardiac arrest and the effect of therapeutic hypothermia on their incidence during the early phase of the postresuscitation period should be further investigated. Studies that specifically address antiarrhythmic drugs during early post-cardiac arrest care are warranted. Furthermore, studies are needed to address the cohort of patients with VF or VT in the field and treated with amiodarone or lidocaine and whether or not and for how long this treatment should be continued after sustained ROSC.

Mechanical circulatory support[ALS-PA-060]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital) who have cardiovascular dysfunction, does the use of mechanical circulatory support, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

There are no studies directly addressing the use of mechanical circulatory support in patients with sustained ROSC but who have cardiovascular dysfunction. One human study showed that

patients with severe cardiovascular dysfunction who were non-responsive to standard care can be supported with mechanical chest compressions during PCI (LOE 4);[671] however, none of these patients survived. One swine study showed worse left ventricular function when an intra-aortic balloon pump was compared with standard treatment including dobutamine in the immediate post-cardiac arrest phase (LOE 5).[796] Five studies of nonarrested patients in cardiogenic shock or severe heart failure showed that left ventricular assist device or continuous aortic flow augmentation improved haemodynamics but not survival (LOE 5).[805–809] Two case series reported the use of the intraaortic balloon pump in patients with severe myocardial dysfunction after sustained ROSC, but the effect was impossible to isolate from other interventions (LOE 4).[749,810]

Treatment recommendation

There is insufficient evidence to support or refute the use of mechanical circulatory support in post-cardiac arrest patients who have cardiovascular dysfunction.

Knowledge gaps

RCTs are needed to explore different techniques for mechanical support in patients with severe cardiovascular dysfunction after sustained ROSC.

Temperature Control

Prevention and Treatment of HyperthermiaALS-PA-049A

In adult patients (out-of-hospital or in-hospital) who are comatose after cardiac arrest, does treatment of pyrexia, compared with no temperature intervention, improve outcome (eg, survival)?

Consensus on Science

There are no RCTs evaluating the effect of treatment of pyrexia (defined as 37.6C) compared with no temperature control in patients after cardiac arrest. Eleven studies suggested that there was an association between pyrexia and poor outcomes (LOE 4[811–815]; LOE 5[816–821]). For comparison, patients with cerebrovascular events who developed pyrexia had worsened short- and long-term outcomes (LOE 5).[zref]816-821[zrefx]

Treatment Recommendation

Patients who develop hyperthermia after cardiac arrest have a worse prognosis. Despite the lack of evidence, it is reasonable to treat hyperthermia if it occurs in the postresuscitation period.

Knowledge Gaps

Clinical trials are needed to determine whether the management of pyrexia after cardiac arrest improves outcomes and what strategy of care produces effective control in this patient population.

Therapeutic hypothermia[ALS-PA-044]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does therapeutic hypothermia, compared with usual care, improve morbidity or mortality?

Consensus on science

Who to cool?. All studies of post-cardiac arrest therapeutic hypothermia have included only patients in coma. One trial defined coma as "not responding to verbal commands" (LOE 1).[822] The other trials defined coma similarly, used the Glasgow Coma Score (GCS) ≤8, or did not provide a clear definition.

One randomised trial (LOE 1)[822] and a pseudorandomised trial (LOE 2)[823] demonstrated improved neurological outcome at hospital discharge or at 6 months after hospital discharge in comatose patients after out-of-hospital VF cardiac arrest. Cooling was initiated within minutes to hours after ROSC, and a temperature range of 32–34 °C was maintained for 12–24 h. Two studies with historical control groups (LOE 3) showed improvement in neurological outcome after therapeutic hypothermia for comatose survivors of VF cardiac arrest.[824,825] One systematic review demonstrated that conventional cooling methods were more likely to reach a best cerebral performance category score of 1 or 2 (5-point scale where 1 is good and 5 is brain death) with a relative risk of 1.55 (99.5% CI 1.22 to 1.96) and more likely to survive to hospital discharge (relative risk of 1.35 95% CI 1.1 to 1.65) compared with standard postresuscitation care (LOE 1).[826]

One small ($n = 30$) randomised trial showed reduced plasma lactate values and oxygen extraction ratios in a group ($n = 16$) of comatose survivors after cardiac arrest with asystole or PEA who were cooled with a cooling cap (LOE 1).[827]

Six studies with historical control groups showed benefit using therapeutic hypothermia in comatose survivors of out-of-hospital cardiac arrest after all-rhythm arrests (LOE 3).[749,828–832] One study with historical controls showed better neurological outcome after VF cardiac arrest but no difference after cardiac arrest from other rhythms (LOE 3).[833]

Two nonrandomised studies with concurrent controls indicated possible benefit of hypothermia following cardiac arrest from other initial rhythms in- and out-of-hospital (LOE 2).[834,835] One registry study, which included almost 1000 cooled comatose patients following cardiac arrest from all rhythms, showed that survival with good outcome at 6 months was 56% after initial VT/VF, 21% after initial asystole, and 23% after initial PEA (LOE 4).[836]

How to cool? (See also Implementing Therapeutic Hypothermia in Section 12). Nineteen studies indicated that cooling could be initiated safely with IV ice-cold fluids (30 mL kg^{-1} of saline 0.9% or Ringer's lactate) (LOE 3[748,749,825,831,833,837]; LOE 4[779,780,782–785,810,836,838–843]). Six studies indicated that cooling with IV cold saline can be initiated in the prehospital phase (LOE 1[781,844]; LOE 2[845]; LOE 3[261,846]). Thirteen studies documented the use of an intravascular heat exchanger to induce and maintain hypothermia (LOE 2[834,835]; LOE 3[748,749]; LOE 4[847,848,782,839,841,849–852]). Twelve studies documented the use of ice packs and either water- or air-circulating blankets to induce and maintain hypothermia (LOE 2[834]; LOE 3[749,825,829,832,833]; LOE 4[748,841,850,853–855]). Seven studies documented the use of ice packs (sometimes combined with wet towels) alone to induce and maintain hypothermia (LOE 2[823]; LOE 3[824,828,830]; LOE 4[847,849,856]). Four studies documented the use of ice packs alone to maintain hypothermia (LOE 3[837]; LOE 4[810,840,843]). Seven studies documented the use of cooling blankets or pads alone to induce and maintain hypothermia (LOE 2[857]; LOE 3[858]; LOE 4[841,859–861,862]). Eight studies documented the use of water-circulating, gel-coated pads to induce and maintain, or just maintain, hypothermia (LOE 3[749,831]; LOE 4[838,841,842,854,860,863]). One RCT (LOE 1) used a cold-air tent[822] and another used a cooling helmet[827] to induce and maintain hypothermia. In one registry study, cooling was maintained with ice packs (17%), air cooling (8%), circulating water blankets (63%), an intravascular cooling device (16%), and other methods (8%) (LOE 4).[836]

When to cool?. One registry-based case series of 986 comatose post-cardiac arrest patients suggested that time to initiation of cooling (median 90 min; interquartile range [IQR] 60 to 165 min) was not associated with improved neurological outcome post-

discharge (LOE 4).[836] A case series of 49 consecutive comatose post-cardiac arrest patients who were intravascularly cooled after out-of-hospital cardiac arrest also documented that time to target temperature (median 6.8 h; [IQR 4.5 to 9.2 h]) was not an independent predictor of neurological outcome (LOE 4).[852]

Safe with percutaneous coronary intervention?. Five studies indicated that the combination of therapeutic hypothermia and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction (LOE 3[749,837,864]; LOE 4[810,836]).

Treatment recommendation

Comatose adult patients (not responding in a meaningful way to verbal commands) with spontaneous circulation after out-of-hospital VF cardiac arrest should be cooled to 32–34 °C for 12–24 h. Induced hypothermia might also benefit comatose adult patients with spontaneous circulation after out-of-hospital cardiac arrest from a nonshockable rhythm, or cardiac arrest in hospital. Rapid infusion of ice-cold IV fluid 30 mL kg^{-1} or ice packs are feasible, safe, and simple methods for initially lowering core temperature up to 1.5 °C. When IV fluids are used to induce hypothermia, additional cooling strategies will be required to maintain hypothermia. Limited available evidence suggests that PCI during therapeutic hypothermia is feasible and safe and may be associated with improved outcome.

Knowledge gaps

Although the data support cooling to 32–34 °C, the optimal temperature has not been determined. Furthermore the optimal method, onset, duration and rewarming rate, and therapeutic window remain unknown. Further investigation is also needed to determine the benefit of post-cardiac arrest therapeutic hypothermia after nonshockable cardiac arrest, in-hospital cardiac arrest, and in children. Epidemiological and safety data would help describe the safety and adversity when cooling is interrupted across the system of care. Clinical and cost comparisons are required of the methods used for inducing and maintaining therapeutic hypothermia in- and out-of-hospital. The safety and efficacy of therapeutic hypothermia during cardiac arrest resuscitation needs to be explored through controlled clinical trials.

Seizure control[ALS-PA-050A, ALS-PA-050B]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of seizure prophylaxis or effective seizure control, as opposed to standard care (no prophylaxis or ineffective seizure control), improve outcome (e.g., survival)?

Consensus on science

No controlled clinical trials directly addressed prophylactic treatment for seizures after cardiac arrest. Five studies documented a 3–44% incidence of seizures after sustained ROSC (LOE 4).[749,814,865–867] Two studies reported no difference in neurological outcome after use of single-dose diazepam or magnesium or both; or thiopental given after sustained ROSC (LOE 5).[732,865] There are no studies addressing prompt and aggressive treatment after the first seizure occurring after circulation was restored. Seizures in the postarrest period may be refractory to multiple medications (LOE 4).[866,868] There was no reported difference in the occurrence of seizures after sustained ROSC in patients treated with therapeutic hypothermia or with normothermia care (LOE 5).[749,822]

Treatment recommendation

There are insufficient data to support or refute the use of specific antiseizure medication in the prevention or treatment of seizures after ROSC.

Knowledge gaps

Studies need to determine the true incidence of clinical and electrographic seizures in patients after cardiac arrest, particularly in those treated with therapeutic hypothermia.

Clinical trials are required to assess interventions and drugs for the prevention and treatment of seizures. Studies should evaluate whether continuous electroencephalograph (EEG) monitoring to diagnose and treat seizures after cardiac arrest is feasible, interpretable, of prognostic value, and beneficial for patients.

Other supportive therapies

Blood glucose control[ALS-PA-045A, ALS-PA-045B]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of a specific strategy to manage blood glucose (e.g., target range), as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

One human randomised interventional study that prospectively evaluated strict glucose control (4–6 mmol L^{-1}, [72–108 mg dL^{-1}]) compared with moderate glucose control (6–8 mmol L^{-1}, [108–144 mg dL^{-1}]) in patients resuscitated from prehospital cardiac arrest with VF found no survival benefit with strict glucose control (LOE 1).[869] Five retrospective studies in post-cardiac arrest patients suggested an association of higher glucose levels with increased mortality and worse neurological outcomes, but those findings may be related to other factors (LOE 4).[798,814,870–872] Based on those studies, the suggested target ranges for glucose values have been variable. A good randomised trial of intensive glucose control versus conventional glucose control in the largest number of ICU patients to date reported increased mortality in patients treated with intensive glucose control (LOE 5).[873] Two meta-analyses of studies of tight glucose control versus conventional glucose control in critically ill patients showed no significant difference in mortality but found tight glucose control was associated with a significantly increased risk of hypoglycaemia (LOE 5).[874,875]

Treatment recommendation

Strategies to treat hyperglycaemia >10 mmol L^{-1} (>180 mg dL^{-1}) should be considered in adult patients with sustained ROSC after cardiac arrest. Hypoglycaemia should be avoided.

Knowledge gaps

Adequately powered intervention trials of moderate ranges of glucose control in patients who survive cardiac arrest are required.

Steroid therapy[ALS-PA-048A]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does treatment with corticosteroids, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

Two observational studies (LOE 2)[876,877] and two animal studies (LOE 5)[878,879] failed to demonstrate any benefit or harm from the use of steroids after successful resuscitation from cardiac arrest. One small, single-centre randomised placebo-controlled trial showed benefit from the use of a package of care consisting of vasopressin and dexamethasone in addition to adrenaline during resuscitation, combined with the treatment of post-cardiac arrest shock with hydrocortisone in the study group (LOE 1).[231] The

complex design of this study makes it impossible to determine the independent effect of any interventions on outcome.

Treatment recommendation

There is insufficient evidence to support or refute the use of corticosteroids for patients with ROSC following cardiac arrest.

Knowledge gaps

It is important to determine the incidence of adrenal insufficiency after sustained ROSC following cardiac arrest. Clinical trials are needed to determine the effect of exogenous steroids administered after cardiac arrest.

Haemofiltration[ALS-PA-054A]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of haemofiltration as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

One RCT demonstrated no difference in survival or neurological outcome between groups treated with high-volume haemofiltration (200 mL kg^{-1} h^{-1} for 8 h) with or without mild hypothermia, and control group without haemofiltration (LOE 1).[880] The combined haemofiltration-only and haemofiltration-plus-hypothermia groups had increased survival at 6 months after cardiac arrest when compared to controls. One study suggested improved survival and neurological outcome in patients treated with high-volume haemofiltration after resuscitation from cardiac arrest (LOE 2).[881]

Treatment recommendation

There is insufficient evidence to support or refute the use of haemofiltration in patients with sustained ROSC after cardiac arrest.

Knowledge gaps

Randomised clinical trials are needed comparing haemofiltration to a control group that has similar management of temperature and other confounding protocols of care. It is unknown whether haemofiltration will have different effects in different subgroups of patients.

Neuroprotective therapy[ALS-PA-055A, ALS-PA-055C]

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of neuroprotective drugs, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

One small pilot study in witnessed, out-of-hospital cardiac arrests of presumed cardiac aetiology showed improved survival at 3 months when therapeutic hypothermia (35 °C) and the oral administration of coenzyme Q10 (250 mg followed by 150 mg TID for 5 days) was compared with therapeutic hypothermia alone; however, there was no difference in neurologically intact survival (LOE 1).[859]

Four RCTs (LOE 1) using nimodipine,[882,883] lidoflazine,[884] or diazepam[732] in out-of-hospital cardiac arrest showed no benefits from any of the drugs when compared with standard care. Two RCTs (LOE 1) using thiopental[884] or nimodipine[885] in out-of-hospital cardiac arrest were unable to show any benefits when compared with standard care. A retrospective analysis using glucocorticoids in out-of-hospital cardiac arrest was unable to show any benefits when compared with standard care (LOE 2).[877]

Treatment recommendation

The value of routine use of coenzyme Q10 in patients treated with hypothermia is not certain. There are insufficient data to recommend for or against the use of neuroprotective drugs (thiopental, glucocorticoids, nimodipine, lidoflazine, or diazepam) alone or as an adjunct to therapeutic hypothermia in comatose cardiac arrest after ROSC.

Knowledge gaps

Prospective, double-blind RCTs of promising neuroprotective agents alone, in combination, or in combination with therapeutic hypothermia are encouraged.

Specific research and larger clinical trials are required on the use of coenzyme Q10 in patients with therapeutic hypothermia of 33 °C on neurologically intact survival.

Prognostication

Prognostication during cardiac arrest

End-tidal CO_2 and prediction of outcome[ALS-D&P-014A]

In adult cardiac arrest (out-of-hospital or in-hospital), does the use of end-tidal CO_2 (e.g., absolute CO_2 values or changes in waveform), compared with not using end-tidal CO_2, accurately predict outcomes (e.g., ROSC, survival)?

Consensus on science

Thirteen studies (LOE P2[176–178,182,183,886,887]; LOE P3[888] (LOE P5[140,180,889–891]) indicated that higher maximal end-tidal CO_2 levels can predict ROSC. Seven studies demonstrate that end-tidal CO_2 values <1.33 kPa (10 mm Hg) obtained after intubation and during CPR efforts are associated with a low probability of survival from cardiac arrest (LOE P2).[176–178,182,183,886,887] Two prospective human studies demonstrated a significant increase in end-tidal CO_2 when ROSC occurs (LOE 5).[140,180]

Treatment recommendation

Quantitative measurement of end tidal CO_2 may be a safe and effective noninvasive indicator of cardiac output during CPR and may be an early indicator of ROSC in intubated patients. Although low values of end tidal CO_2 are associated with a low probability of survival, there are insufficient data to support or refute a specific cutoff of end tidal CO_2 at different time intervals as a prognostic indicator of outcome during adult cardiac arrest.

Knowledge gaps

More well-designed prognostic studies of end tidal CO_2 monitoring designed to measure long-term morbidity, mortality, and neurological survivability are recommended.

In future studies the cause of cardiac arrest should be documented. Use of vasopressors and ventilation rates may lower end-tidal CO_2; and this effect should be controlled in future studies. Evaluation of end-tidal CO_2 for prognosis should be repeated with supraglottic airway devices.

Prognostication after resuscitation

Clinical examination[ALS-PA-041]

In adult and paediatric patients who are comatose after cardiac arrest (out-of-hospital or in-hospital), does the use of the bedside neurological examination, as opposed to standard care, allow accurate prediction of outcome (e.g., survival)?

Consensus on science

In adult patients comatose after cardiac arrest who had not been treated with therapeutic hypothermia, the following parameters predicted poor outcome (CPC 3 or 4, or death) with a false-positive rate (FPR) of 0%: absent vestibulo-ocular reflexes at ≥24 h [(95% CI 0% to 14%)] (LOE P1) [892,893];absence of pupillary light *and* corneal reflex at 72 h [(95% CI 0% to 9%)](LOE P1)[894];GCS <5 at 48 h (95% CI 0% to 13%) (LOE P1)[895] and on day 3 (95% CI 0% to 6%) (LOE P2)[896] and a clinical examination score <15 on day 4 [(95% CI 0% to 18%) (LOE P1).[897] However, in one study an absent motor response (GCS motor = 1) at 72 h after cardiac arrest predicted poor outcome with a FPR of 5% [(95% CI 2% to 9%]](LOE P1).[894] The presence of myoclonus status in adults was strongly associated with poor outcome (LOE P1[866,894]; LOE P3[898];[868];LOE P4[899]), but rare cases of good neurological recovery have been described and accurate diagnosis was problematic.[900–904]

Treatment recommendation

There are no clinical neurological signs that reliably predict poor outcome <24 h after cardiac arrest. In adult patients who are comatose after cardiac arrest, have *not been treated with hypothermia* and have no confounding factors (e.g., hypotension, sedatives or neuromuscular blockers), the absence of both pupillary light and corneal reflex at ≥72 h reliably predicts poor outcome. Absence of vestibulo-ocular reflexes at ≥24 h and a GCS motor score of 2 or less at ≥72 h are less reliable. Other clinical signs, including myoclonus, are not recommended for predicting poor outcome.

Knowledge gaps

The reevaluation of prognostic indicators during therapeutic hypothermia and in the presence of other confounders needs to be completed to guide current post-cardiac arrest care.

Biochemical markers[ALS-PA-052A, ALS-PA-052B]

In adult patients who are comatose after cardiac arrest (out-of-hospital or in-hospital), does the use of biochemical markers, as opposed to standard care, allow accurate prediction of outcome (e.g., survival)?

Consensus on science

Serum neuronal-specific enolase (NSE) elevations are associated with poor outcome for comatose patients after cardiac arrest (LOE P1[905,906]; LOE P2[852,894,897,907–920]; LOE P3[921,922]). Although specific cutoff values with a FPR of 0% have been reported, clinical application is limited due to variability in the 0% FPR cutoff values reported among various studies.

Serum S100 elevations are associated with poor outcome for comatose patients after cardiac arrest (LOE P1[905,906]; LOE P2[894,897,907,913,915,917,918,923–928]; LOE P3[921]).

Many other serum markers measured after sustained ROSC have been associated with poor outcome after cardiac arrest, including brain natriuretic peptide (BNP) (LOE P3)[929]vWF (LOE P3)[930] ICAM-1 (LOE P3)[930]; procalcitonin (LOE P2)[924] IL-1ra, RANTES, sTNFRII, IL-6, IL-8 and IL-10 (LOE P3).[931] However, other studies found no relationship between outcome and serum IL-8 (LOE P1),[923] and procalcitonin and sTREM-1 (LOE P3).[932]

Worse outcomes for comatose survivors of cardiac arrest are also associated with increased levels of cerebrospinal fluid (CSF)-CK (LOE P2)[933,934] and cerebrospinal fluid-CKBB (LOE P1[905,906]; LOE P2[908,919,934,935]; LOE P3[936–938]). However, 1 study found no relationship between cerebrospinal fluid-CKBB and prognosis (LOE P2).[939]

Outcomes are also associated with increased cerebrospinal fluid levels of other markers including NSE (LOE P1[906]; LOE P2[915,919]); S100 (LOE P2)[915]LDH, GOT(LOE P2)[908,934]; neurofilament (LOE P3)[940]; and acid phosphatase and lactate (LOE P2).[934] Cerebrospinal fluid levels of β-D-N-acetylglucosaminidase and pyruvate were not associated with the prognosis of cardiac arrest (LOE P2).[934]

Treatment recommendation

Evidence does not support the use of serum or cerebrosphinal fluid biomarkers alone as predictors of poor outcomes in comatose patients after cardiac arrest with or without treatment with therapeutic hypothermia. Limitations included small numbers of patients and/or inconsistency in cutoff values for predicting poor outcome.

Knowledge gaps

Future studies should identify and resolve the heterogeneity of cutoff values used to predict poor outcome with a FPR of zero. Studies also must account for confounders that may alter levels or predictive performance of various markers (e.g., hypothermia, underlying disease, pregnancy, intra-aortic balloon pump, brain instrumentation, haemodialyisis, or other organ failure). Studies examining whether biomarkers can be used to monitor ongoing injury and response to therapy may be useful.

Electrophysiological studies[ALS-PA-051A]

In adult patients who are comatose after cardiac arrest (out-of-hospital or in-hospital), does the use of neurological electrophysiological studies, as opposed to standard care, allow accurate prediction of outcome (e.g., survival)?

Consensus on science

Somatosensory evoked potentials measured between 4 h and 2 weeks after cardiac arrest were associated with poor outcome in 14 studies (LOE P1[893,894,905,941–946]; LOE P2[897]; LOE P3[936,947–949]). In a meta-analysis of patients not treated with therapeutic hypothermia, the absence of cortical N20 response to median nerve stimulation at 24 to 72 h after cardiac arrest predicted poor outcome (CPC 3 or 4, or death) with a FPR of 0.7% (95% CI 0.1 to 3.7) (LOE P1).[905]

Abnormal brain stem auditory evoked potentials. Abnormal brain stem auditory evoked potentials recorded 1 to 56 days after cardiac arrest in patients not treated with hypothermia predicted poor outcome with a FPR of 0% (95% CI 0 to 14) in one LOE P1-study.[942] Abnormal brainstem auditory evoked potentials recorded 55 to 235 min after cardiac arrest before initiation of therapeutic hypothermia predicted poor outcome with a FPR of 0% (95% CI 0 to 32) (LOE P1).[950] One study found no predictive value with brainstem auditory evoked potentials (LOE P1).[946] In patients not treated with therapeutic hypothermia, medium-latency auditory evoked potentials predicted poor outcome after cardiac arrest in one LOE P1-study with a FPR of 0% (95% C I0 to14)[942] and in one LOE P3-study.[948] Auditory N100 and mismatch negativity was also associated with poor outcome in one LOE P1-study.[942]

Electroencephalography predicted poor outcome in comatose survivors of cardiac arrest within one week after cardiac arrest in 12 studies (LOE P1[893,894,905,941,951–953]; LOE P3[954,955]; LOE P4[956,957]; LOE P5[958]). In a meta-analysis, EEG showing generalised suppression to less than 20 μV, burst-suppression pattern associated with generalised epileptic activity, or diffuse periodic complexes on a flat background 12 to 72 h after sustained ROSC predicted a poor outcome (FPR of 3%, 95% CI 0.9% to 11%) in patients not receiving therapeutic hypothermia (LOE P1).[905]

Treatment recommendation

No electrophysiological study reliably predicts outcome of comatose patient after cardiac arrest in the first 24 h treated with-

out therapeutic hypothermia. After 24 h, bilateral absence of the N20 cortical response to median nerve stimulation predicts poor outcome in comatose cardiac arrest survivors not treated with therapeutic hypothermia. In the absence of confounding circumstances, such as sedatives, hypotension, hypothermia, or hypoxaemia, it is reasonable to use unprocessed electroencephalography interpretation (specifically identifying generalised suppression to less than 20 μV, burst suppression pattern with generalised epileptic activity, or diffuse periodic complexes on a flat background) observed between 24 and 72 h after sustained ROSC to assist the prediction of a poor outcome in comatose survivors of cardiac arrest not treated with hypothermia.

Knowledge gaps

More data are needed about the performance and timing of somatosensory evoked potentials and electroencephalography criteria for aiding prognostication in patients treated with induced hypothermia.

Imaging studies[ALS-PA-059]

In adult patients who are comatose after cardiac arrest (out-of-hospital or in-hospital), does the use of imaging studies, as opposed to standard care, allow accurate prediction of outcome (e.g., survival)?

Consensus on science

Magnetic resonance imaging. There are no LOE P1- or LOE P2-studies that support the use of magnetic resonance imaging (MRI) to predict outcome of comatose cardiac arrest survivors. Use of MRI to predict outcome is supported by 32 studies (LOE P3[959–963]; LOE P4[964–973];[974–976]; LOE P5[977–990]). The timing of MRI in these studies ranged from 1 day to 10 months after sustained ROSC. MRI parameters associated with poor outcome included lower gray matter volume, lower hippocampal volume, global cerebral atrophy, higher number of neuroradiological findings, extensive abnormalities on digital weight imaging, increased lactate on magnetic resonance spectroscopy, hyperintense lesions in basal ganglia, extensive digital weight imaging abnormalities, global apparent diffusion coefficient depression, extensive white matter abnormalities, and cortical laminar enhancement. Overall these studies were limited by small sample sizes, variable time of imaging (many very late in the course of the event), lack of comparison with a standardised method of prognostication, often nonmodern MRI techniques, and early withdrawal of care. One study found that MRI performed on comatose cardiac arrest survivors 1 to 47 days after sustained ROSC did not correlate with outcome (LOE P2).[991] MRI parameters used in this study were leukoaraiosis, cerebral infarcts, and edema. Modern MRI techniques (ie, diffusion-weighted imaging) were not used in this study.

Computed tomography. There are no LOE P1- or LOE P2-studies that support the use of computed tomography (CT) imaging to predict outcome of comatose cardiac arrest survivors. Use of CT imaging is supported by 22 studies (LOE P3[992]; LOE P4[969,984,993–1001]; LOE P5[980,981,985,1002–1006]). The timing of CT in those studies ranged from 1 hour to 20 days after sustained ROSC. CT parameters associated with poor outcome included gray matter to white matter Hounsfield unit ratio <1.22, cerebral atrophy (chronic), low cerebral blood flow, low acetazolamide reactivity, bicaudate ratio, low Hounsfield number in putamen and cortex, low density in basal ganglia and thalamus, diffuse mass effect, and global cortical gray matter density. Overall those studies were limited by small sample sizes, variable time of imaging (many very late in the course of the event), lack of comparison with a standardised method of prognostication, and early withdrawal of care. Two LOE P3-studies found that CT did not predict outcome,[954,1007] and one LOE P4-study was neutral in its findings [1008]. The timing of CT in those studies ranged from <72 h to 96 h after ROSC. CT parameters not associated with poor outcome included normal scans. Overall these studies were limited by small sample sizes, imaging performed too early in the clinical course, nonmodern CT imaging, and early withdrawal of care.

Single photon emission CT (SPECT) is supported by three LOE P5-studies[990,1006,1009] and is opposed by one LOE P2-study.[1010] The timing of SPECT in these studies ranged from 1 to 23 days after sustained ROSC. SPECT parameters associated with poor outcome included diminished cerebral blood flow, particularly frontal and temporal, particularly when persistent on repeated imaging. SPECT parameters not associated with outcome included the anterior-posterior perfusion ratio. These studies were limited by small sample sizes, variable imaging times, early withdrawal of care, and lack of comparison with a standardised method of prognostication.

Cerebral angiography has been reported by one case report (LOE P5).[980] The timing of cerebral angiography was 1 day after sustained ROSC. Cerebral angiography parameters associated with poor outcome included delayed cerebral circulation time.

Transcranial Doppler was evaluated in one study (LOE P4).[976] The timing of transcranial Doppler in this study ranged from 4 to 120 h after ROSC. Transcranial Doppler parameters associated with poor outcome included delayed hyperaemia. This study was limited by a small sample size, early withdrawal of care, and lack of comparison with a standardised method of prognostication.

Nuclear medicine. One case report was supportive of nuclear medicine studies (LOE P5),[985] but the timing of the images after sustained ROSC was not described. Nuclear medicine parameters associated with poor outcome included abnormal tracer uptake in the cerebral cortices. This case report included only a limited description of the findings; it was further limited by lack of comparison with a standardised method of prognostication.

Near-infrared spectroscopy. One study of near-infrared spectroscopy was not supportive (LOE P3).[1011] The timing of near-infrared spectroscopy in this study ranged from 6 to 24 h after sustained ROSC. This study was limited by a small sample size, early withdrawal of care, inclusion of non–cardiac arrest patients, and lack of comparison with a standardised method of prognostication.

Treatment recommendation

There is insufficient evidence to recommend for or against the routine use of neuroimaging to predict outcome of adult cardiac arrest survivors.

Knowledge gaps

Adequately powered prospective studies are required to evaluate the accuracy of CT, MRI, or both in prognosticating outcome of comatose cardiac arrest survivors. Prognostication studies should include calculation of FPR with 95% confidence intervals for predicting poor outcome. Outcome prediction should include a comparison with more conventional methods, including clinical examination and electrophysiology (e.g., somotosensory evoked potentials). All studies should allow for sufficient time to realise patient recovery, avoiding the bias of self-fulfilling prophecy and premature withdrawal of care. Specific brain structures responsible for coma and recovery after cardiac arrest (e.g., thalamus, rostral brainstem) should be a focus of future studies. The optimal timing of neuroimaging after cardiac arrest and the impact of hypothermia should be explored. Prognostic modalities have focused on predicting poor outcome, and the need to identify those with likely good outcome is becoming more important, especially because effective

therapies exist. Neuroimaging should be performed in a safe setting for critical patients or be done at the bedside.

Impact of therapeutic hypothermia on accuracy of post-cardiac arrest prognostication[ALS-PA-040A]

In post-cardiac arrest patients treated with hypothermia, can the same prognostication tools that are used in normothermic patients reliably predict outcome?

Consensus on science

Two studies (LOE P1)[898,946] provided evidence that status myoclonus (FPR 0%, 95% CI 0% to 40%), absence of corneal and pupillary reflexes at three days postsustained ROSC (FPR 0%, 95% CI 0% to 48%), and bilateral absence of N20 peak on somotosensory evoked potentials at 24 h postsustained ROSC (FPR 0%, 95% CI 0% to 69%) in patients treated with therapeutic hypothermia predict poor outcome. One study evaluated somatosensory evoked potential responses in 112 postarrest patients more then 24 h after cardiac arrest who were treated with hypothermia and found that 35 of 36 patients with bilateral absent N20 cortical response had a poor outcome (FPR 3%, 95% CI 0% to 14%).[1012] One patient with bilaterally absent N20 and another with a barely detectable N20 had a good recovery; both were evaluated at three days post-cardiac arrest (LOE P1).[1012] One LOE P1-study[898] provided evidence that a Glasgow Coma Motor Score of 2 or less at 3 days after sustained ROSC in patients treated with therapeutic hypothermia has a FPR of 14% (95% CI 3% to 44%) for poor outcome. Two studies provided evidence that status epilepticus in postarrest patients treated with hypothermia has a FPR of 7% (95% CI 1% to 25%) to 11.5% (95% CI 3% to 31%) for predicting poor outcome (LOE P2[1013]; LOE P3[955]). One study (LOE P3)[1014] suggested that glial fibrillary acidic protein level >1.0 ng dL^{-1} drawn 12 to 48 h after sustained ROSC predicts poor outcome (defined as CPC score 3 to 5 at 6 months) both in post-cardiac arrest patients treated with normothermia (FPR 0% 95% CI 0% to 27%) or hypothermia (FPR 0% 95% CI 0% to 48%). One study provided evidence that NSE and S-100b protein cutoff values that reliably predict poor outcome are significantly higher in post-cardiac arrest patients treated with hypothermia compared with those not treated with hypothermia (LOE P2).[917] Two studies prospectively measured NSE in cohorts of patients treated with post-cardiac arrest hypothermia and reported cutoff values for 0% FPR (LOE P2)[1015,1016]: one study[1015] reported that all patients with a 48-hour NSE value >33 $\mu g\,L^{-1}$ had a poor outcome (FPR 0%, 95% CI 0% to 23%); the other study[1016] reported that all patients with a 48-hour NSE >28 $\mu g\,L^{-1}$ had a poor outcome (FPR 0%, 95% CI 0% to 18%). Variability in 0% FPR cutoff values from these derivation cohorts potentially results from variability among assays and performance sites. Two studies examined the utility of bispectral index monitoring in prognosticating poor outcome in post-cardiac arrest patients treated with hypothermia who were under neuromuscular blockade (LOE P1).[953,1017] One study reported that an initial bispectral index monitoring score of ≥22 predicted poor outcome with a FPR of 6% (19 patients having a positive test), and a suppression ratio ≥48 predicted poor outcome with a FPR of 7% [(95% CI 1% to 26%)].[1018] The other study reported that a bispectral index monitoring level of 0 at any time in the first 72 h after cardiac arrest predicted poor outcome with a FPR of 0% [0% to 27%].[953] Finally, one study (LOE P1)[1019] of 111 post-cardiac arrest patients treated with therapeutic hypothermia attempted to validate prognostic criteria proposed by the American Academy of Neurology.[905] That study demonstrated that clinical examination findings at 36 to 72 h were unreliable predictors of poor neurological outcome [motor response less than flexion (FPR 16%, 95% CI 6% to 35%); ≥ one brainstem reflexes absent (FPR 8%, 95% CI 2% to 25%); early myoclonus (FPR 4%, 95% CI 1% to 19%), while bilaterally absent N20 peak on somatosensory evoked potentials (FPR 0%, 95% CI 0% to 13%) and unreactive electroencephalogram background (FPR 0%, 95% CI 0% to 13%) were the most reliable. A decision rule derived using that dataset demonstrated that the presence of two independent predictors of poor neurological outcome (incomplete recovery brainstem reflexes, early myoclonus, unreactive electroencephalogram, and bilaterally absent cortical somatosensory evoked potentials) predicted poor neurological outcome with a FPR of 0% (95% CI 0% to 14%).

Treatment recommendation

There is inadequate evidence to recommend a specific approach to prognosticating poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia. There are no clinical neurological signs, electrophysiological studies, biomarkers, or imaging modalities that can reliably predict neurological outcome in the first 24 h after cardiac arrest. Beyond 24 h, no single parameter for predicting poor neurological outcome in post-cardiac arrest patients treated with hypothermia is without reported false-positives. Based on limited available evidence, potentially reliable prognosticators of poor outcome in patients treated with therapeutic hypothermia after cardiac arrest include bilateral absence of N20 peak on somatosensory evoked potential ≥24 h after cardiac arrest or unreactive electroencephalogram background at 36 to 72 h; and the absence of both corneal and pupillary reflexes >72 h after cardiac arrest. Limited available evidence also suggests that a Glasgow Coma Motor Score of 2 or less at 3 days after sustained ROSC and the presence of status epilepticus are potentially unreliable prognosticators of poor outcome in post-cardiac arrest patients treated with therapeutic hypothermia. Serum biomarkers such as NSE are potentially valuable as adjunctive studies in prognostication of poor outcome in patients treated with hypothermia, but their reliability is limited by the relatively few patients who have been studied and lack of assay standardization. Given the limited available evidence, decisions to limit care should not be made based on the results of a single prognostication tool.

Knowledge gaps

Further research is needed to elucidate the impact of therapeutic hypothermia on the accuracy and timing of post-cardiac arrest prognostication tools. Prospective derivation and validation of a clinical decision rule for early prediction of poor outcome in post-cardiac arrest patients treated with or without hypothermia are urgently needed.

Organ donation[ALS-PA-042A, ALS-PA-042B]

In adult organ recipients, does the use of organs from donors brain dead after cardiac arrest (out-of-hospital or in-hospital), as opposed to the use of donors brain dead not due to cardiac arrest, improve outcome (e.g., transplant success)?

Consensus on science statements

Three studies suggested no difference in functional outcomes of organs transplanted from patients who were determined to be brain dead as a consequence of cardiac arrest when compared with donors who were brain dead from other causes (LOE 2). [1020–1022]

Treatment recommendation

Adult patients who progress to brain death after resuscitation from out-of-hospital cardiac arrest should be considered for organ donation.

Knowledge gaps

Further studies with larger populations and common definitions of outcomes are needed. There is no evidence regarding organ donation from children or adults who are brain dead after resuscitation from an in-hospital cardiac arrest.

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Appendix A. Evidence-Based Worksheets for Part 8: Advanced Life Support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations

Task force	WS ID	PICO title	Short title	Authors	URL
ALS/BLS	ALS/BLS-CPR&A-079A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of an a supraglottic airway device (I) vs an endotracheal tube (I), improve any outcomes (O).	Supraglottic devices vs intubation	Lauren Berkow	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-079A.pdf
ALS/BLS	ALS/BLS-CPR&A-079B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of an a supraglottic airway device (I) vs an endotracheal tube (I), improve any outcomes (O).	Supraglotic devices vs intubation	Michael Shuster	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-079B.pdf
ALS/BLS	ALS/BLS-CPR&A-080B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of oropharyngeal airway or nasopharyngeal airway adjuncts (I) compared with no airway adjuncts (C), improve any outcomes (e.g. ventilation, oxygenation) (O).	Oropharyngeal and nasopharyngeal adjuncts	Harinder Dhindsa, V. Ramana Feeser, Renee D. Reid	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-080B.pdf

Appendix A (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ALS/BLS	ALS/BLS-CPR&A-088A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of supraglottic devices (I) compared with bag-valve-mask alone for airway management (C), improve any outcomes (e.g. ventilation, oxygenation, reduce hands-off time, allow for continuous compressions and/or improves survival) (O).	Supraglottic devices vs BVM	Suzanne M. Davies, Paul M. Middleton	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-088A.pdf
ALS/BLS	ALS/BLS-CPR&A-088B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of supraglottic devices (I) compared with bag-valve-mask alone for airway management (C), improve any outcomes (e.g. ventilation, oxygenation, reduce hands-off time, allow for continuous compressions and/or improves survival) (O).	Supraglottic devices vs BVM	Lauren Berkow, Henry R. Halperin	http://circ.ahajournals.org/site/C2010/ALS-BLS-CPR-A-088B.pdf
ALS	ALS-CPR&A-001A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of physiological feedback regarding CPR quality (e.g. End-tidal CO2 monitoring) (I) compared with no feedback (C), improve any outcomes (e.g. ROSC, survival) (O)?	Physiological feedback (e.g. end tidal CO2) for CPR quality	Blair Bigham	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-001A.pdf

Appendix A (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-CPR&A-001B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of physiological feedback regarding CPR quality (e.g. End-tidal CO2 monitoring) (I) compared with no feedback (C), improve any outcomes (e.g. ROSC, survival) (O)?	Physiological feedback (e.g. end tidal CO2) for CPR quality	Marion Leary	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-001B.pdf
ALS	ALS-CPR&A-002A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P) – does the use of rapid deployment ECMO, Aortic Balloon Pump or emergency cardiopulmonary bypass (I), compared with standard treatment (C), increase survival to hospital discharge with favorable neurological outcomes (O)?	ECMO, balloon pump etc for CPR	Tetsuya Sakamoto	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-002A.pdf
ALS	ALS-CPR&A-002B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P) – does the use of rapid deployment ECMO, Aortic Balloon Pump or emergency cardiopulmonary bypass (I), compared with standard treatment (C), increase survival to hospital discharge with favorable neurological outcomes (O)?	ECMO, balloon pump etc for CPR	Michael S. Czekajlo	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-002B.pdf
ALS	ALS-CPR&A-003B	In adult in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of ultrasound (including transthoracic and transoesophageal echocardiography) during cardiac arrest (I) compared with standard CPR (C), improve any outcomes (e.g. ROSC, survival) (O).	Ultrasound during cardiac arrest	Amanda Hanson	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-003B.pdf
ALS	ALS-CPR&A-005C	In adult cardiac arrest (out-of-hospital and in-hospital) with either a protected and unprotected airway (P), does the monitoring and control of ventilatory parameters (e.g. minute ventilation and/or peak pressures) (I) as opposed to standard care (without ventilatory monitoring) (C), improve outcome (O) (e.g. ROSC, survival)?	Monitoring ventilatory parameters during CPR	Kate Crewdson	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-005C.pdf

Appendix A (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-CPR&A-006A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of thoracic impedence (I) compared with usual management (C), improve the accuracy of diagnosis of airway placement and adequacy of ventilation (O).	Thoracic impedence to confirm airway placement	F. Javier Garcia-Vega	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-006A.pdf
ALS	ALS-CPR&A-006B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of thoracic impedence (I) compared with usual management (C), improve the accuracy of diagnosis of airway placement and adequacy of ventilation (O).	Thoracic impedence to confirm airway placement	Heather Farley	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-006B.pdf
ALS	ALS-CPR&A-007B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) requiring ventilation and intubation (P), does the application and maintenance of cricoid pressure (I), compared to no cricoid pressure (C), reduce the incidence of aspiration (O)	Cricoid pressure	Michael Shuster	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-007B.pdf
ALS	ALS-CPR&A-008A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of devices (e.g. CO2 detection device, CO2 analyser or oesophageal detector device) (I) compared with usual management (C), improve the accuracy of diagnosis of airway placement (O)?	Devices to confirm airway placement	Douglas Kupas	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-008A.pdf
ALS	ALS-CPR&A-008B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of devices (e.g. CO2 detection device, CO2 analyser or oesophageal detector device) (I) compared with usual management (C), improve the accuracy of diagnosis of airway placement (O)?	Devices to confirm airway placement	Ian L. Cash	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-008B.pdf
ALS	ALS-CPR&A-009A	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of passive oxygen delivery during CPR (I) compared with oxygen delivery by positive pressure ventilation (C), improve outcome (e.g. ROSC, survival) (O).	Passive oxygen vs positive pressure oxygen during CPR	Csaba Dioszeghy	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-009A.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-CPR&A-009B	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of passive oxygen delivery during CPR (I) compared with oxygen delivery by positive pressure ventilation (C), improve outcome (e.g. ROSC, survival) (O).	Passive oxygen vs positive pressure oxygen during CPR	Peter Fenici, Andrea Scapigliati	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-009B.pdf
ALS	ALS-CPR&A-010A	In adult and paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) and who have advanced airways in place (P), does the use of automatic ventilators (I) compared with manual ventilation (C), improve outcome (e.g. ventilation, oxygenation, reduce hands-off time, allow for continuous compressions and/or improves survival) (O)?.	Automatic ventilators vs manual ventilation during CPR	Charles Otto	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-010A.pdf
ALS	ALS-CPR&A-011A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of an FIO2 titrated to oxygenation during cardiac arrest (I) compared with the use of 100% oxygen (C), improve outcome (e.g. ROSC, neurologically intact survival) (O)?	Supplemental oxygen: 100% versus titration	Colin A. Graham	http://circ.ahajournals.org/site/C2010/ALS-CPR-A-011A.pdf
ALS	ALS-D&P-014A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of end-tidal CO2 (e.g. absolute CO2 values or changes in waveform) (I) compared with not using ETCO2 (C), accurately predict outcomes (e.g. ROSC, survival) (O).	End-tidal CO2 to predict outcome of cardiac arrest	Sadiq S. Bhayani	http://circ.ahajournals.org/site/C2010/ALS-D-P-014A.pdf
ALS	ALS-D-016A	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use intravenous fluids (I) compared with not using fluids (or standard resucitation (C), improve outcomes (e.g. ROSC, survival) (O).	IV vluids during cardiac arrest	Jane A.H. Foster, Jasmeet Soar	http://circ.ahajournals.org/site/C2010/ALS-D-016A.pdf
ALS	ALS-D-016B	In adult cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use intravenous fluids (I) compared with not using fluids (or standard resucitation (C), improve outcomes (e.g. ROSC, survival) (O).	IV Fluids during cardiac arrest	Paul A. Jennings	http://circ.ahajournals.org/site/C2010/ALS-D-016B.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-D-017	In adult patients in atrial fibrillation (prehospital and in-hospital) (P), does the use of any drug or combination of drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. reversion rates) (O).	Drugs for atrial fibrillation	Steven Kronick, Mark S. Link, Rod S. Passman, Richard Schilling	http://circ.ahajournals.org/site/C2010/ALS-D-017.pdf
ALS	ALS-D-018	In adult patients in narrow complex tachycardia (prehospital and in-hospital) (P), does the use of any drug or combination of drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. reversion rates) (O).	Drugs for narrow complex tachycardia	Steven Kronick, Rod S. Passman, Volker Wenzel	http://circ.ahajournals.org/site/C2010/ALS-D-018.pdf
ALS	ALS-D-019-01A	In adult patients in monomorphic (wide complex) tachycardia (prehospital and in-hospital) (P), does the use of any drug or combination of drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. reversion rates) (O).	Drugs for monomorphic wide complex tachycardia	Tommaso Pellis	http://circ.ahajournals.org/site/C2010/ALS-D-019-01A.pdf
ALS	ALS-D-019-01B	In adult patients in monomorphic (wide complex) tachycardia (prehospital and in-hospital) (P), does the use of any drug or combination of drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. reversion rates) (O).	Drugs for monomorphic wide complex tachycardia	Markus Skrifvars	http://circ.ahajournals.org/site/C2010/ALS-D-019-01B.pdf
ALS	ALS-D-019-02	In adult patients with undifferentiated stable wide complex tachycardia (prehospital and in-hospital) (P), does the use of any drug or combination of drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. reversion rates) (O)?	Drugs for undifferenti-ated stable wide complex tachycardia	Steven Kronick	http://circ.ahajournals.org/site/C2010/ALS-D-019-02.pdf
ALS	ALS-D-020B	In adult patients in polymorphic (wide complex) tachycardia (prehospital and in-hospital) (P), does the use of any drug or combination of drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. reversion rates) (O).	Drugs for polymorphic wide complex tachycardia	Peter J. Kudenchuk	http://circ.ahajournals.org/site/C2010/ALS-D-020B.pdf

Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-D-021A	In adult patients in torsades de pointes (prehospital and in-hospital) (P), does the use of any drug or combination of drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. reversion rates) (O).	Drugs for torsades de pointes	Eliano Pio Navarese, Andrea Scapigliati	http://circ.ahajournals.org/site/C2010/ALS-D-021A.pdf
ALS	ALS-D-022A	In adult patients in significant bradycardia (prehospital and in-hospital) (P), does the use of any drug or combination of drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. reversion rates) (O).	Drugs for bradycardia	Thomas Nguyen	http://circ.ahajournals.org/site/C2010/ALS-D-022A.pdf
ALS	ALS-D-023B	In adult patients in cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of vasopressors (adrenaline, noradrenaline, others) or combination of vasopressors (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Vasopressors for cardiac arrest	Todd M. Larabee, Charles M. Little	http://circ.ahajournals.org/site/C2010/ALS-D-023B.pdf
ALS	ALS-D-024B	In adult patients in cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of atropine or atropine in combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Atropine for cardiac arrest	Swee Han Lim	http://circ.ahajournals.org/site/C2010/ALS-D-024B.pdf
ALS	ALS-D-025A	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of antiarrhythmic drugs (lidocaine, procainamide, amiodarone, bretylium, magnesium) or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Antiarrhytmic drugs for cardiac arrest	Marcus Ong	http://circ.ahajournals.org/site/C2010/ALS-D-025A.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-D-025B	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of antiarrhythmic drugs (lidocaine, procainamide, amiodarone, bretylium, magnesium) or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Antiarrhytmic drugs for cardiac arrest	Mark S. Link, Tommaso Pellis	http://circ.ahajournals.org/site/C2010/ALS-D-025B.pdf
ALS	ALS-D-026A	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of calcium alone or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Calcium for cardiac arrest	Fulvio Kette, Sara Tararan	http://circ.ahajournals.org/site/C2010/ALS-D-026A.pdf
ALS	ALS-D-026B	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of calcium alone or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Calcium for cardiac arrest	Jaspinder Ghuman	http://circ.ahajournals.org/site/C2010/ALS-D-026B.pdf
ALS	ALS-D-027	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of steroid or hormonal therapy (estrogen, progesterone, hydrocortisone, insulin, growth factor etc) alone or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Steroids and hormones for cardiac arrest	Michael Cocchi, Michael Donnino, Ian Seppelt	http://circ.ahajournals.org/site/C2010/ALS-D-027.pdf
ALS	ALS-D-028A	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of fibrinolytics alone or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Fibrinolytics for cardiac arrest	Michael Parr	http://circ.ahajournals.org/site/C2010/ALS-D-028A.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-D-028B	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of fibrinolytics alone or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Fibrinolytics for cardiac arrest	Steven Kronick	http://circ.ahajournals.org/site/C2010/ALS-D-028B.pdf
ALS	ALS-D-029A	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of buffering agents alone or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (!!e.g. ROSC, survival) (O).	Buffering agents for cardiac arrest	James J. McCarthy	http://circ.ahajournals.org/site/C2010/ALS-D-029A.pdf
ALS	ALS-D-029C	In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of buffering agents alone or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (e.g. ROSC, survival) (O).	Buffering agents for cardiac arrest	Edison Ferreira de Paiva	http://circ.ahajournals.org/site/C2010/ALS-D-029C.pdf
ALS	ALS-PA-040A	In post-cardiac arrest patients treated with hypothermia (P), can the same prognostication tools that are used in normothermic patients (I) reliably predict outcome (O)?	Hypothermia and prognosti-cation	Hans Friberg, Robert Neumar, Malin Rundgren	http://circ.ahajournals.org/site/C2010/ALS-PA-040A.pdf
ALS	ALS-PA-041	In adult and paediatric patients who are comatose after cardiac arrest (prehospital or in-hospital) (P), does the use of the bedside neurological exam (I) as opposed to standard care (C), allow accurate prediction of outcome (O) (e.g. survival)?	Bedside neuro exam for prog-nostication	Romergryko G. Geocadin, Giuseppe La Torre, Claudio Sandroni	http://circ.ahajournals.org/site/C2010/ALS-PA-041.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-PA-042A	In adult and paediatric organ recipients (P), does the use of organs from donors brain dead after cardiac arrest (prehospital or in-hospital) (I) as opposed to the use of donors brain dead not due to cardiac arrest (C), improve outcome (O) (e.g. transplant success?	Organ donation	Claudio Sandroni	http://circ.ahajournals.org/site/C2010/ALS-PA-042A.pdf
ALS	ALS-PA-042B	In adult and paediatric organ recipients (P), does the use of organs from donors brain dead after cardiac arrest (prehospital or in-hospital) (I) as opposed to the use of donors brain dead not due to cardiac arrest (C), improve outcome (O) (e.g. transplant success?	Organ donation	Christophe Adrie	http://circ.ahajournals.org/site/C2010/ALS-PA-042B.pdf
ALS	ALS-PA-043A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) who have cardiovascular dysfunction (P), does the use of intravenous fluids (I) as opposed to standard care (or other intravenous fluids) (C), improve outcome (O) (e.g. survival)?	IV vluids following cardiac arrest	Jane A.H. Foster, Jasmeet Soar	http://circ.ahajournals.org/site/C2010/ALS-PA-043A.pdf
ALS	ALS-PA-043C	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) who have cardiovascular dysfunction (P), does the use of intravenous fluids (I) as opposed to standard care (or other intravenous fluids) (C), improve outcome (O) (e.g. survival)?	IV vluids following cardiac arrest	Hitoshi Kano, Tomoyuki Sato	http://circ.ahajournals.org/site/C2010/ALS-PA-043C.pdf
ALS	ALS-PA-044	In adult patients with ROSC after cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does therapeutic hypothermia (I) compared with usual care (C), improve morbidity or mortality (O)?	Hypothermia following resuscitation	Jerry P. Nolan, Peter T. Morley	http://circ.ahajournals.org/site/C2010/ALS-PA-044.pdf
ALS	ALS-PA-045A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of a specific strategy to manage blood glucose (e.g. target range) (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Glucose control following resuscitation	Jon Rittenberger	http://circ.ahajournals.org/site/C2010/ALS-PA-045A.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-PA-045B	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of a specific strategy to manage blood glucose (e.g. target range) (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Glucose control following resuscitation	Janice L. Zimmerman	http://circ.ahajournals.org/site/C2010/ALS-PA-045B.pdf
ALS	ALS-PA-046A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P) diagnosed as pulmonary embolism, does the use of early fibrinolytic therapy (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Fibrinolytics for cardiac arrest	Markus Skrifvars	http://circ.ahajournals.org/site/C2010/ALS-PA-046A.pdf
ALS	ALS-PA-046B	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P) diagnosed as pulmonary embolism, does the use of early fibrinolytic therapy (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Fibrinolytics for cardiac arrest	Rachel Prout	http://circ.ahajournals.org/site/C2010/ALS-PA-046B.pdf
ALS	ALS-PA-047A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of comprehensive treatment protocol (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Treatment protocol post resuscitation	Maaret Castrén	http://circ.ahajournals.org/site/C2010/ALS-PA-047A.pdf
ALS	ALS-PA-047B	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of comprehensive treatment protocol (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Treatment protocol post resuscitation	Mary Ann Peberdy	http://circ.ahajournals.org/site/C2010/ALS-PA-047B.pdf
ALS	ALS-PA-048A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does treatment with corticosteroids (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Steroids post resuscitation	Andrew Padkin, Kjetil Sunde	http://circ.ahajournals.org/site/C2010/ALS-PA-048A.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-PA-049A	In adult patients (prehospital or in-hospital) who are comatose after cardiac arrest (P) does treatment of pyrexia (I) compared to no temperature intervention (C) improve outcome (e.g. survival).	Fever post resuscitation	Marios Georgiou, Marios Ioannides	http://circ.ahajournals.org/site/C2010/ALS-PA-049A.pdf
ALS	ALS-PA-050A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of seizure prophylaxis or effective seizure control (I) as opposed to standard care (no prophylaxis or ineffective seizure control) (C), improve outcome (O) (e.g. survival)?	Seizure prophylaxis post resuscitation	Nabil El Sanadi	http://circ.ahajournals.org/site/C2010/ALS-PA-050A.pdf
ALS	ALS-PA-050B	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of seizure prophylaxis or effective seizure control (I) as opposed to standard care (no prophylaxis or ineffective seizure control) (C), improve outcome (O) (e.g. survival)?	Seizure prophylaxis post resuscitation	Maaret Castrén	http://circ.ahajournals.org/site/C2010/ALS-PA-050B.pdf
ALS	ALS-PA-051A	In adult patients who are comatose after cardiac arrest (prehospital or in-hospital) (P), does the use of neurological electrophysiological studies (I) as opposed to standard care (C), allow accurate prediction of outcome (O) (e.g. survival)?	EEG post resusctitation	Tommaso Sanna	http://circ.ahajournals.org/site/C2010/ALS-PA-051A.pdf
ALS	ALS-PA-052A	In adult patients who are comatose after cardiac arrest (prehospital or in-hospital) (P), does the use of biochemical markers (I) as opposed to standard care (C), allow accurate prediction of outcome (O) (e.g. survival)?	Biomarkers	Tommaso Sanna	http://circ.ahajournals.org/site/C2010/ALS-PA-052A.pdf
ALS	ALS-PA-052B	In adult patients who are comatose after cardiac arrest (prehospital or in-hospital) (P), does the use of biochemical markers (I) as opposed to standard care (C), allow accurate prediction of outcome (O) (e.g. survival)?	Biomarkers	Michel Torbey	http://circ.ahajournals.org/site/C2010/ALS-PA-052B.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-PA-053B	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of a specific ventilation strategy (including specific CO2 goal) (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Ventilation strategy post resuscitation	Clifton Callaway	http://circ.ahajournals.org/site/C2010/ALS-PA-053B.pdf
ALS	ALS-PA-054A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of a haemofiltration (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Haemofiltration post resuscitation	Wilhelm Behringer	http://circ.ahajournals.org/site/C2010/ALS-PA-054A.pdf
ALS	ALS-PA-055A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of neuroprotective drugs (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Neuorprotective drugs	Michael Holzer	http://circ.ahajournals.org/site/C2010/ALS-PA-055A.pdf
ALS	ALS-PA-055C	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of neuroprotective drugs (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Neuorprotective drugs	Richard A. Bernstein	http://circ.ahajournals.org/site/C2010/ALS-PA-055C.pdf
ALS	ALS-PA-056B	In adult patients (prehospital and in-hospital) with ROSC after cardiac arrest (P), does early haemodynamic optimisation (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Haemodynamic support post resuscitation	Michael Fries	http://circ.ahajournals.org/site/C2010/ALS-PA-056B.pdf
ALS	ALS-PA-057A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) who have cardiovascular dysfunction (P), does the use of any specific cardioactive drugs (I) as opposed to standard care (or different cardioactive drugs) (C), improve outcome (O) (e.g. survival)?	Cardioactive drugs post resuscitation	Karl B. Kern, Sudhakar Sattur	http://circ.ahajournals.org/site/C2010/ALS-PA-057A.pdf
ALS	ALS-PA-058A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of prophylactic antiarrhythmic drugs (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Antiarrhytmic drugs post resuscitation	Tommaso Pellis	http://circ.ahajournals.org/site/C2010/ALS-PA-058A.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-PA-058B	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of prophylactic antiarrhythmic drugs (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Antiarrhytmic drugs post resuscitation	Mark S. Link	http://circ.ahajournals.org/site/C2010/ALS-PA-058B.pdf
ALS	ALS-PA-059	In adult patients who are comatose after cardiac arrest (prehospital or in-hospital) (P), does the use of imaging studies (I) as opposed to standard care (C), allow accurate prediction of outcome (O) (e.g. survival)?	Imaging studies post resuscitation	Romergryko G. Geocadin, David M. Greer	http://circ.ahajournals.org/site/C2010/ALS-PA-059.pdf
ALS	ALS-PA-060	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) who have cardiovascular dysfunction (P), does the use of mechanical circulatory support (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Mechanical circulatory support post resuscitation	Hitoshi Kano, Sten Rubertsson, Tomoyuki Sato	http://circ.ahajournals.org/site/C2010/ALS-PA-060.pdf
ALS	ALS-PA-061A	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of a controlled oxygenation strategy (including specific oxygenation goal) (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Supplemental oxygen: 100% versus titration	Robert Neumar	http://circ.ahajournals.org/site/C2010/ALS-PA-061A.pdf
ALS	ALS-PA-061B	In adult patients with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of a controlled oxygenation strategy (including specific oxygenation goal) (I) as opposed to standard care (C), improve outcome (O) (e.g. survival)?	Supplemental oxygen: 100% versus titration (duplicate with 11a?)	Gregory P. Comadira	http://circ.ahajournals.org/site/C2010/ALS-PA-061B.pdf
ALS	ALS-SAM-062A	In adult cardiac arrest (prehospital or in-hospital) (P), does an alternate timing for advanced airway insertion (e.g. early or delayed) (I) as opposed to standard care (standard position in algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Advanced airway placement (timing)	Sebastian G. Russo, Christoph H. Wiese, Daniel Wu	http://circ.ahajournals.org/site/C2010/ALS-SAM-062A.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-SAM-063A	In adult cardiac arrest (prehospital or in-hospital) (P), does an alternate timing for drug delivery (e.g. early or delayed) (I) as opposed to standard care (standard position in algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Drug delivery (timing)	James J. Menegazzi, Morten Pytte	http://circ.ahajournals.org/site/C2010/ALS-SAM-063A.pdf
ALS	ALS-SAM-063B	In adult cardiac arrest (prehospital or in-hospital) (P), does an alternate timing for drug delivery (e.g. early or delayed) (I) as opposed to standard care (standard position in algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Drug delivery (timing)	Elizabeth A. Hunt, Michael C. McCrory	http://circ.ahajournals.org/site/C2010/ALS-SAM-063B.pdf
ALS	ALS-SAM-064B	In adult cardiac arrest (prehospital or in-hospital) (P), initially with a non-shockable rhythm but who develop a shockable rhythm (prehospital or in-hospital) (P), does any specific alteration in treatment algorithm (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Algorithm for transition from shockable to non-shockable rhythm	Masami Ishikawa, Keiichi Tada, Wanchun Tang	http://circ.ahajournals.org/site/C2010/ALS-SAM-064B.pdf
ALS	ALS-SAM-064C	In adult cardiac arrest (prehospital or in-hospital) (P), initially with a non-shockable rhythm but who develop a shockable rhythm (prehospital or in-hospital) (P), does any specific alteration in treatment algorithm (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Algorithm for transition from shockable to non-shockable rhythm	Timothy J. Mader	http://circ.ahajournals.org/site/C2010/ALS-SAM-064C.pdf
ALS	ALS-SC-065	In pregnant patients with cardiac arrest (prehospital or in-hospital) (P), do any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Pregnancy and cardiac arrest	Farida M. Jeejeebhoy, Carolyn M. Zelop	http://circ.ahajournals.org/site/C2010/ALS-SC-065.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-SC-066A	In adult cardiac arrest due to anaphylaxis (P), does any modification of treatment (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Anaphylaxis and cardiac arrest	Eric Bruder	http://circ.ahajournals.org/site/C2010/ALS-SC-066A.pdf
ALS	ALS-SC-066B	In adult cardiac arrest due to anaphylaxis (P), does any modification of treatment (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Anaphylaxis and cardiac arrest	John Litell	http://circ.ahajournals.org/site/C2010/ALS-SC-066B.pdf
ALS	ALS-SC-067B	In adult cardiac arrest due to asthma (P), does any modification of treatment (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Asthma and cardiac arrest	Barry Brenner, Fred A. Severyn	http://circ.ahajournals.org/site/C2010/ALS-SC-067B.pdf
ALS	ALS-SC-068B	In adult cardiac arrest during PCI (P), does use of any specific intervention (I) as opposed to standard care (acc to treatment algorithm) (C), improve outcome.	Cardiac arrest during PCI	Pavan Battu	http://circ.ahajournals.org/site/C2010/ALS-SC-068B.pdf
ALS	ALS-SC-068C	In adult cardiac arrest during PCI (P), does use of any specific intervention (I) as opposed to standard care (acc to treatment algorithm) (C), improve outcome.	Cardiac arrest during PCI	Jonathan Weinstock	http://circ.ahajournals.org/site/C2010/ALS-SC-068C.pdf
ALS	ALS-SC-069A	In adult cardiac arrest following open (including heart and lung transplantations) and closed heart surgery (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Post op cardiaothoracic surgery cardiac arrest	Joel Dunning	http://circ.ahajournals.org/site/C2010/ALS-SC-069A.pdf
ALS	ALS-SC-069B	In adult cardiac arrest following open (including heart and lung transplantations) and closed heart surgery (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Post op cardiaothoracic surgery cardiac arrest	David Zideman	http://circ.ahajournals.org/site/C2010/ALS-SC-069B.pdf

Appendix A (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-SC-069C	In adult cardiac arrest following open (including heart and lung transplantations) and closed heart surgery (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Post op cardiaothoracic surgery cardiac arrest	Peter T. Morley, Will Ross	http://circ.ahajournals.org/site/C2010/ALS-SC-069C.pdf
ALS	ALS-SC-070B	In adult cardiac arrest (prehospital or in-hospital) due to a cardiac tamponade (P), does use of specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Cardiac tamponade	Henry Halperin	http://circ.ahajournals.org/site/C2010/ALS-SC-070B.pdf
ALS	ALS-SC-071B	In adult cardiac arrest (prehospital or in-hospital) (P) due to pulmonary embolus (P), does use of aetiology specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Pulmonary embolism cardiac arrest	C. Jessica Dine	http://circ.ahajournals.org/site/C2010/ALS-SC-071B.pdf
ALS	ALS-SC-072A	In adult cardiac arrest (prehospital or in-hospital) (P) due to non-cardiac aetiology (e.g. hemmorhagic shock, hypovolemic shock; septic shock; neurogenic shock) (P), does use of aetiology specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Non-cardiac aetiology cardiac arrest	Harinder Dhindsa, V. Ramana Feeser, Renee D. Reid	http://circ.ahajournals.org/site/C2010/ALS-SC-072A.pdf
ALS	ALS-SC-073-01A	In adult cardiac arrest (prehospital or in-hospital) due to local anaesthetic toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Local anaesthesia toxicity	Eric J. Lavonas, John J. Picard, Richard D. Shih	http://circ.ahajournals.org/site/C2010/ALS-SC-073-01A.pdf

Appendix A (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-SC-073-02A	In adult cardiac arrest (prehospital or in-hospital) due to Benzodiazepine toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Benzodiazepine toxicity	Mohammed Alhelail, Greene Shepherd	http://circ.ahajournals.org/site/C2010/ALS-SC-073-02A.pdf
ALS	ALS-SC-073-03B	In adult cardiac arrest (prehospital or in-hospital) due to Beta blockers toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Beta blocker toxicity	Melissa Givens, Greene Shepherd	http://circ.ahajournals.org/site/C2010/ALS-SC-073-03B.pdf
ALS	ALS-SC-073-04B	In adult cardiac arrest (prehospital or in-hospital) due to Calcium channel blockers toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Calcium channel blocker toxicity	Melissa Givens, Greene Shepherd	http://circ.ahajournals.org/site/C2010/ALS-SC-073-04B.pdf
ALS	ALS-SC-073-05	In adult cardiac arrest (prehospital or in-hospital) due to Carbon monoxide toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Carbon monoxide toxicity	Eric J. Lavonas, David Lobel	http://circ.ahajournals.org/site/C2010/ALS-SC-073-05.pdf
ALS	ALS-SC-073-06B	In adult cardiac arrest (prehospital or in-hospital) due to Cocaine toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Cocaine toxicity	Eric J. Lavonas	http://circ.ahajournals.org/site/C2010/ALS-SC-073-06B.pdf
ALS	ALS-SC-073-07	In adult cardiac arrest (prehospital or in-hospital) due to Cyanide toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Cyanide toxicity	Eric J. Lavonas, David Lobel	http://circ.ahajournals.org/site/C2010/ALS-SC-073-07.pdf

Appendix A (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-SC-073-08B	In adult cardiac arrest (prehospital or in-hospital) due to Cyclic antidepressants toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Tricyclic antidepressant toxicity	Allan R. Mottram	http://circ.ahajournals.org/site/C2010/ALS-SC-073-08B.pdf
ALS	ALS-SC-073-09A	In adult cardiac arrest (prehospital or in-hospital) due to Digoxin/etc toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Digoxin toxicity	Richard D. Shih	http://circ.ahajournals.org/site/C2010/ALS-SC-073-09A.pdf
ALS	ALS-SC-073-10	In adult cardiac arrest (prehospital or in-hospital) due to Opioids toxicity (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Opiod toxicity	Mohammed Alhelail, Allan R. Mottram	http://circ.ahajournals.org/site/C2010/ALS-SC-073-10.pdf
ALS	ALS-SC-074A	In morbidly obese adult patients with cardiac arrest (prehospital or in-hospital) (P), does use of any specific interventions (I) as opposed to standard care (according to treatment algorithm) (C), improve outcome (O) (e.g. ROSC, survival)?	Morbid obesity	Pavan Battu	http://circ.ahajournals.org/site/C2010/ALS-SC-074A.pdf
ALS	ALS-SC-076A	In adult cardiac arrest (out-of-hospital and in-hospital) (P), does the treatment of electrolyte disturbances (eg. hypo or hyper kalaemia, hypo or hyper magnesaemia, hypo and hyper calcaemia) (I) as opposed to standard care (according to treatment algorithm, but without treatment of electrolyte disturbances) (C), improve outcome (O) (eg. ROSC, survival)?	Electrolyte disturbances	William J. Meurer	http://circ.ahajournals.org/site/C2010/ALS-SC-076A.pdf
ALS	ALS-SC-076B	In adult cardiac arrest (out-of-hospital and in-hospital) (P), does the treatment of electrolyte disturbances (eg. hypo or hyper kalaemia, hypo or hyper magnesaemia, hypo and hyper calcaemia) (I) as opposed to standard care (according to treatment algorithm, but without treatment of electrolyte disturbances) (C), improve outcome (O) (eg. ROSC, survival)?	Electrolyte disturbances	Deborah Diercks	http://circ.ahajournals.org/site/C2010/ALS-SC-076B.pdf
ALS	ALS-SC-078B	For avalanche victims in out of hospital cardiac arrest (P), what factors when present (I), compared with when absent (C), are associated with/predict an increased survival to hospital discharge (O)?	Avalanche victims	Jeff Boyd, Hermann Brugger	http://circ.ahajournals.org/site/C2010/ALS-SC-078B.pdf

Disclosures

CoSTR Part 8: writing group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/newline advisory board	Other
Laurie J. Morrison	St. Michaels Hospital clinician scientist	None	None	None	None	None	None
Charles D. Deakin	Southampton University Hospital NHS Trust—Doctor	None	None	None	None	None	None
Bernd W. Bottiger	Uniklinik Köln—MD, DEAA	None	None	None	None	None	None
Clifton W. Callaway	University of Pittsburgh School of Medicine—Associate Professor; [a]AHA-Work Sheet Editor for 2010 Guidelines. My effort on this project is paid to University of Pittsburgh as a "contracted services agreement," and not paid to me	[b]Grants to University of Pittsburgh [b]NHLBI—Resuscitation Outcomes Consortium HRSA—Development and Dissemination of Program Tools for Uncontrolled Donation After Cardiac Death (UDCD)	[a]Loan of an Arctic Sun cooling device (without disposables) to human physiology laboratory for experiments on hypothermia by Medivance, Inc.	None	[b]Co-inventor on patent about ventricular fibrillation waveform analysis, licensed by University of Pittsburgh to Medtronic ERS, Inc.	None	None
Saul Drajer	Clinica de la Esperanza: General Director of a 130 bed hosp. (Clinica de la Esperanza) located in Buenos Aires, Argentina—General Director	None	None	None	None	None	None
Richard E. Kerber	University of Iowa Hospital—Professor of Medicine; Staff Physician	None	None	[a]Occasional (2–4 times/year) Grand Rounds speaker at other Universities. Usual honorarium is $1000 for each talk plus expenses. Money to me	[a]Stock owned in General Electric and Johnson and Johnson. GE makes echocardiographs, which are occasionally used as a diagnostic tool during resuscitation	[a]I performed a one-time consultation for Philips Defibrillator division earlier this year	[a]Occasional expert witness in legal cases, usually alleged malpractice. No cardiac drugs or devices at present; I consulted with a law firm defending a manufacturer of Phentermine about 10 years ago [a]I am presently serving on a DSMB for a clinical trial sponsored by Zoll Corp, which manufacture/sells defibrillators and resuscitation devices
Steven L. Kronick	University of Michigan: Healthcare—Assistant Professor	None	None	None	None	None	None

Appendix B (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Eric Lavonas	Denver Health Hospital Authority: A political division of the State of Colorado, DHHA operates a hospital and outpatient clinic system in Denver, CO. DHHA also operates the Rocky Mountain Poison and Drug Center (RMPDC), Denver Public Health, and Denver Emergency Medical Services—Associate Director, RMPDC	[a]List of all research grants (previous, current, and pending) germaine to the topics reviewed by ILCOR. All grants are awarded to the Denver Health Hospital Authority. Neither I nor any other DHHA employee derives personal financial benefit from these relationships. I don't get a bonus; My salary is supported by general institutional funds and an unrelated research endowment; my performance evaluation is not related to performance of any of these contracts Sponsor: Protherics-BTG Project: Study report for the safety and efficacy of CroFab for severe envenomations Related Product: None. However, Protherics-BTG manufactures DigiFab, which is used to treat digoxin poisoning My role: PI on one portion of the project, collaborator on the rest 2008 (ongoing)	None	None	None	None	None
Swee Han Lim	Singapore General Hospital: Public Tertiary Hospital-Senior Consultant, Emerg. Med.	None	None	None	None	None	None
Mark S. Link	Tufts Medical Center—Physician	None	None	None	None	None	None
Peter T. Morley	Royal Melbourne Hospital–Director of Medical Education; University of Melbourne–Clinical Dean, Royal Melbourne Hospital; AHA—Evidence Evaluation Expert	None	None	None	None	None	None

Appendix B (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Robert W. Neumar	University of Pennsylvania—Associate Professor of Emergency Medicine	[b]Funding source: NIH/NINDS Grant number: R21 NS054654 Funding period 06/01/07 to 06/31/2010 Role on project: PI Title: Optimizing Therapeutic Hypothermia After Cardiac Arrest Description: The goal is to evaluate the how the onset and duration of therapeutic hypothermia after cardiac arrest impacts survival and neuroprotection	None	None	None	None	None
Jerry P. Nolan	Royal United Hospital NHS Trust—Consultant in Anaesthesia and Intensive Care Medicine—Editor-in-Chief Resuscitation	None	None	None	None	None	None
Charles W. Otto	University of Arizona—Professor	None	None	None	None	None	None
Michael Parr	Liverpool Hospital, University of New South Wales—Director of Intensive Care—Editor: Resuscitation	None	None	None	None	None	None
Mary Ann Peberdy	Virginia Commonwealth University-Professor	None	None	None	None	None	None
Michael Shuster	Self-employed—emergency physician	None	None	None	None	None	None
Kjetil Sunde	Oslo University HospitalUlleval—Senior Consultant and post doctoral researcher	None	None	None	None	None	None
Wanchun Tang	Weil Institute of Critical Care Medicine: Non profit research institution—Professor and President	[b]NIH	None	None	None	None	None

Appendix B (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Terry L. Vanden Hoek	The University of Chicago—Associate Professor	[a]Vanden Hoek, Principal Investigator Department of Defense, Office of Naval Research "Proteomic Development of Molecular Vital Signs: Mapping a Mitochondrial Injury Severity Score to Triage and Guide Resuscitation of Hemorrhagic Shock" 9/6/04 to 4/31/10 $885,639 (this year) research grant awarded to University of Chicago	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

CoSTR Part 8: worksheet collaborator disclosures

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Christophe Adrie	Cochin Hosp. Assistance Publique des Hopitaux de Paris-Assist. Professor	None	None	None	None	None	None
Mohammed Alhaleil	King Abdulaziz Medical City—Emergency Medicine Consultant	None	None	None	None	None	None
Pavan Battu	Heart of England NHS Foundation trust Research Fellow	None	None	None	None	None	None
Wilhelm Behringer	Medical University of Vienna—Assoc. Prof	None	None	None	None	None	None
Lauren Berkow	Johns Hopkins School of Medicine Associate Professor	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Richard A. Bernstein	Northwestern University—Associate Professor	None	None	[b]Bristol Myers/Sanofi Partnership (Plavix) Boehringer Inelheim Pharmaceuticals (Aggrenox) [a]Medtronic (Loop recorder—I gave 2 lectures about syncope; a cardiologist who lectured with me discussed some monitoring device	None	[a]BMS/Sanofi Partnership (Plavix) Medtronic-Steering Committee for CRYSTAL-AF study	[b]I have served as an expert witness/medicolegal consultant in cases related to cardiac arrest; none have gone to trial
Sadiq Bhayani	Queen Medical Centre, Nottingham Specialist Trainee in Anaesthesia	None	None	None	None	None	None
Blair Bigham	York Region EMS Paramedic Paramedic	None	None	None	None	None	None
Jeff Boyd	Self-Emergency Physician	None	None	None	None	None	None
Barry Brenner	University Hospitals Case Medical Center—Professor of Emergency Med., Program Director	None	None	None	None	None	None
Eric Bruder	Kingston General Hospital/Queen's University—Assist. Prof Depart of Emergency Medicine	None	None	None	None	None	None
Hermann Brugger	National Health Service (Bolzano, Italy)—General Practitioner, Emergency physician, MD; Medical Univ. Innsbruck (Austria)—Assoc. Prof of Emergency. Med	None	None	None	None	None	None
Ian Cash	Knox Private Hospital Intensive Care Unit Associate Nurse Unit Manager Australasian SOS Oxygen & First Responder Training P/L Resuscitation training & Oxygen Equipment General Manager	None	None	None	None	None	None
Maaret Castren	Karolinska Inst. Prof in Emergency Med	[b]PI: Princess Study of mild hypothermia during CPR [a]Laerdal	[a]Equipment for multicenter hypothermia study Prince	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Michael Cocchi	Beth Israel Deaconess Medical Center Emergency Medicine Physician/Critical Care Fellow	[b]I have recently been awarded a grant from the American Heart Association's Clinical Research Program in the area of cardiac arrest research. I am the Principal Investigator for this project, which is funded for $110,000 over two years. [a]I am a co-investigator on an NIH-funded R21 grant (PI: Michael W. Donnino MD) and receive 5% salary support for my role in this study	None	None	None	None	None
Gregory Comadira	Queensland Health Department of IC, Gold Coast Hosp; Senior Staff Specialist	None	None	None	None	None	None
Kate Crewdson	Royal United Hospital Hospital Doctor	None	None	None	None	None	None
Michael Czekajlo	VCU Medical Center—Asst Professor of Critical Care Medicine	None	None	None	None	[a]Serve as course consultant for the Society of Critical Care Medicine's Fundamentals of Critical Care Support course. Received payments of $750 and $500 in the past 12 months. Course consultant monitors new courses being conducted	None
Suzanne Davies	Ambulance Service of New South Wales Paramedic Research Fellow Australian Resuscitation Council Research Officer	None	None	None	None	None	None
Harinder Dhindsa	Virginia Commonwealth Univ, Emergency physician	None	None	None	None	None	None
Deborah Diercks	University of California, Davis Medical Center—Professor of Emergency Medicine	None	None	[a]Sanofi Aventis [a]Bristol Myers Squibb	None	[a]Sanofi-Aventis [a]Scheuring Plough [a]Heartscape [a]Astellas [a]Beckman Coulter	None
C. Jessica Dine	University of Penn; Assist Prof	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Csaba Dioszeghy	Yeovil District Hospital HNS Foundation Trust: District General Hospital (NHS)—Consultant in Emergency Medicine	None	None	None	None	None	None
Michael Donnino	Harvard Medical School faculty	[b]NIH thiamine as metabolic resuscitation in septic shock [a]AHA corticosteroid in post arrest shock. [a]Clinical correlates of influenza genomic. Harv Med School-Statin in sepsis	None	None	None	None	None
Joel Dunning	James Cook University Hospital NHS Trust: NHS foundation Trust—Cardiothoracic surgical Registrar	None	None	None	None	None	[a]Set up and run a course called the Cardiac Surgery Advanced Life Support course (www.csu-als.com) which is a not-for-profit course designed to teach and promote the teaching of resuscitation after cardiac surgery; also published several papers in this area and I was the first author of the EACTS guidelines for resuscitation for patients who suffer cardiac arrest after cardiac surgery. I receive money for recovery of expenses incurred
Nabil El Sanadi	Self-employed, Chief of Emergency Medicine for Broward Health	None	None	None	None	None	None
Heather Farley	Doctors for Emergency Services (DFES)—Attending Emergency Physician	None	None	None	None	None	None
V. Ramana Feeser	Virginia Commonwealth Univ—Emergency Medicine Assist Professor	None	None	None	None	None	None
Peter Fenici	Bristol Myers Squibb Italy Pharma Company CV& Metabolics medical director	None	None	None	None	None	None
Jane Foster	Royal Devon & Exter NHS Foundation Trust-Core Med. Trainee Doctor	None	None	None	None	None	None
Hans Friberg	Region Skane Govt.agency Sweden, Emergency Med. Director	None	None	[a]Speaker Medivance	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Michael Fries	University Hospital RWTH Aachen - Academic University Hospital; Senior Consultant in Intensive Care	[b]GEMI fund Deutsche Forschungsgemeinschaft [a]IKARIA Inc. Deutsche Interdisziplinäre Vereinigung für Intensivmedizin	None	[a]BRAHMS AG [a]ZOLL Medical	None	None	None
Francisco Javier Garcia-Vega	Galician Health Service (SERGAS) Internal Medicine Service University Hospital of Vigo (CHUVI) MD, Internal Medicine specialist	None	None	None	None	None	None
Romergryko G. Geocadin	Johns Hopkins, Assoc Prof, Crit Care Med & Neurosurg	[b]NIH consequences of CArrest-brain injury; [a]NIH Cortical brain injury	None	[a]Academic Grand Rounds [a]American Academy Neurology	None	None	None
Marios Georgiou	Nicosia Gen Hosp-ministry of Health: Govt. Hosp. Republic of Cyprus-Resus officer	None	None	None	None	None	None
Jaspinder Ghuman	Hamilton Health Sciences—Emergency Physician	None	None	None	None	None	None
Melissa Givens	US Army—Emergency Med physician	None	None	None	None	None	None
Colin Graham	Chinese University of Hong Kong—Professor of Emergency Medicine	None	None	[a]I receive a modest honorarium from Wolters Kluwer Health (London) for the work I do as the Editor-in-Chief of the European Journal of Emergency Medicine	None	None	None
David M. Greer	Massachusetts General Hospital—Assistant in Neurology	[b]Boehringer Ingelheim Pharmaceuticals, Inc., sponsored an investigator initiated study of extended-release dipyridamole as administered via gastrostomy tubes, a pharmacokinetic study. The money went to my institution. [b]Boehringer Ingelheim Pharmaceuticals, Inc., manufacture the antiplatelet medication, Aggrenox, which contains extended-release dipyridamole	None	[b]Boehringer Ingelheim Pharmaceuticals, Inc, the money comes to me directly.	None	None	[a]Expert witness in a medical malpractice suit, the money came to me directly

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Henry R. Halperin	Johns Hopkins Prof	[b]Zoll Circulation	None	None	[b]Surgivision, [b]Lexmedone	[b]Zoll Circ [a]Cardiac Concepts	None
Amanda Hanson	Alberta Health and Wellness Emergency Physician Emergency Physician	None	None	None	None	None	None
Michael Holzer	Med Univ. of Vienna-Spec for Int. Med	None	None	None	None	None	None
Elizabeth A. Hunt	Johns Hopkins University School Med. Pediatric intensivist, researcher & Dir of Johns Hopkins Med Simulation Center-director, assist. Prof	[a]Co PI on AHA grant to study relationship between scripted debriefing & high fidelity simulation on learning during PALS course	None	None	None	None	None
Marios Ioannides	Nicosia Gen Hosp-Cyprus-Cardiologist	None	None	None	None	None	None
Masami Ishikawa	Kure Kyosai Hosp-MD	None	None	None	None	None	None
Farida Jeejeebhoy	Self employed cardiologist, have affiliation with Univ. HealthNetwork/Mt Sinai Hosp, and University of Toronto. I am paid fee for service	None	None	None	None	None	None
Paul Jennings	Ambulance Victoria—Intensive Care Paramedic	None	None	None	None	None	None
Hitoshi Kano	TBD	TBD	TBD	TBD	TBD	TBD	TBD
Karl B. Kern	Univ of Arizona Prof. of Medicine	[b]Laerdal Foundation'08-10	None	Medivance Inc (hypothermia device manuf)	None	[b]Zoll [a]PhysioControl	[b]State of Ariz attorney General
Fulvio Kette	Azienda per I Servizi Sanitari n.6 "Friuli Occidentale"-Dir Emergency department	None	None	None	None	None	None
Walter Kloeck	Academy of Advanced Life Support Basic and advanced life support training Medical Director	None	None	None	None	None	None
Peter J. Kudenchuk	University of Washington; Professor of Medicine	[b]NIH Resuscitation Outcomes Consortium	None	[a]Sanofi Aventis [a]Bristol Myers Squibb [a]A variety of CME organisations with topics related to arrhyth-mias/atrial fibrillation	[a]Sanofi Aventis (modest stock holding)	None	[a]Expert Witness: Levin Riback Dewsnup, King, Olson Treon, Aquirre, Newman, Norris

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Douglas Kupas	Geisinger Health System, Geisinger Clinic Employed as Associate Chief Academic Officer and emergency physician Associate Chief Academic Officer Commonwealth of Pennsylvania, Department of Health Serve as state EMS medical director for the Bureau of EMS Commonwealth EMS Medical Director	None	None	None	None	None	None
Giuseppe La Torre	Self-employed	None	None	None	None	None	None
Todd Larabee	Univ. Col, Denver School of Med. Assist Prof.	[a]NIH cardiac synch technique for pulseless elect.Activity grant held by Quest PD	None	[a]Bristol Myers Squibb	[a]Stock–Sanofi Aventis	None	Levin Riback Dewsnup, King, Olson Treon, Aquirre, newman, Norris
Marion Leary	Hospital of the University of Pennsylvania Center for Resuscitation Science. Nurse Researcher; Medical ICU Critical Care Nurse	[b]Philips Healthcare grant	[b]CPR feedback defibrilators were given to us by Philips Healthcare to record CPR quality metrics including ETCO2 and ventilation	[a]Philips Healthcare in 2008 to speak at NTI	None	[a]Velomedex in 2008 one time minor consultant fee	None
John Litell	Mayo Clinic Critical Care Fellow	None	None	None	None	None	None
Charles Little	Univ of Colorado, Denver, Assoc Prof Emergency Medicine	None	None	None	None	None	None
David Lobel	Maimonides Med. Center, Emergency Dept attending; Med. Director Prehospital (EMS)	None	None	None	None	None	None
Timothy Mader	Baystate Health; nonprofit healthcare system, Emergency physician	None	None	None	None	None	None
James McCarthy	University of Texas Health Science Center—Houston Assistant Professor	None	None	None	None	None	[a]Expert Witness—Brin and Brin
Michael McCrory	Johns Hopkins, Pediatric Intensive Care Fellow	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
James Menegazzi	University of Pittsburgh Research Professor of Emergency Medicine	[b]Received significant research grant support from the National Heart, Lung, and Blood Institute	[a]Received modest research support from Zoll, Medtronic, and Jolife, all in the form of the loan of medical equipment for use in laboratory	None	[b]Co-inventor of a patented method for analysing the electrocardiographic waveform during ventricular fibrillation. This method has been licensed by my University to Medtronic. I receive a significant payment from Medtronic, in the form of royalties, via this licensing agreement	None	[a]In 2009, and in 2010, I lectured at a medical conference in Anchorage, Alaska. While I did not receive an honorarium, my airfare, hotel, and per diem food costs were paid by the Loren Marshall Foundation
William Meurer	University of Michigan, Assistant Professor Departments of Emergency Medicine and Neurology	None	None	None	None	None	None
Paul Middleton	Ambulance Service of NSW—Medical Director/Director of Research	None	None	None	None	None	None
Allan Mottram	Univ. of Wisc. Emergency Med Division; Assist Prof	[b]NIH potential antidotal therapy for Ca Channel blocker OD	None	None	None	None	None
Eliano P. Navarese	Catholic University of Sacred Heart Cardiologist	None	None	None	None	None	None
Thomas Nguyen	Beth Israel medical center—Attending MD	None	None	None	None	None	None
Marcus Ong	Singapore General Hospital—Consultant	[b]Research grant from Zoll Medical Corporation for mechanical CPR trial	[a]Research support (in kind) from Medivance and Alsius for a hypothermia trial	[a]Honoraria for a lecture on Intraosseous Vascular Access from Vidacare Corp at the Asian Conference on Emergency Medicine 2009 Busan Korea	None	None	None
Andrew Padkin	Royal United Hosp. NHS Trust Bath UK; Healthcare Provider Consultant	None	None	None	None	None	[a]Unpaid editorial board member Resuscitation J
Edison Paiva	University of Sao Paulo School of Medicine—Professor	None	None	None	None	None	None
Rod S. Passman	NW Univ. Assoc Prof	None	None	[b]GSK [a]Medtronic	None	[a]Medtronic Steering for CRYSTAL AF	[a]Expert witness

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Tommaso Pellis	Santa Maria degli Angeli Hospital—Medical doctor, consultant in Anesthesia, Intensive Care & Emergency Med. Service	None	None	None	None	None	None
John Picard	Imperial College NHS Healthcare Trust, Univ. Teaching Hosp-group Consultant	None	None	None	None	None	None
Rachel Prout	University Hospitals Bristol NHS Foundation Trust—SpR	None	None	None	None	None	None
Morten Pytte	Oslo University Hospital, Ullevål-MD Attending anesthesiologit	None	None	None	None	None	None
Renee Reid	Virginia Commonwealth University—Emergency Physician	None	None	None	None	None	None
Jon Ritten-berger	UPMC,Assist Prof	[b]Zoll Med Fellowship;NIH Road map for Medical Research	None	[a]Sacramento Fire no honorarium; [a]Christopher Fanning Mem.Community Ed. 'The Big Chill'	None	[a]Advisor for Zoll Cool arrest study	None
Will Ross	Royal Melbourne Hosp. Medical intern	None	None	None	None	None	None
Sten Rubertsson	Uppsala University/Dept. of Surgical Sci-ences/Anesthesiology and Intensive Care—Professor	None	None	None	None	[a]Jolife AB, Lund, Sweden Manufacturer of LUCAS device-mechanical chest compressions Consult fees received not annually exceeding USD 10,000 [a]I am also a PI for the ongoing LINC trial-a multicenter trial with 2500pts comparing LUCAS concept with manual chest compressions in out of hospital CA. For this I receive no money	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Malin Rundgren	Region Skane, Lund University Hospital, Department of Intensive and Perioperative care—Consultant	[a]8 weeks per year in time from Region Skanes research and development foundation.	None	None	None	None	None
Sebastian Russo	University of Goettingen, Germany: Depart. of Anaesthesiology, Emergency and Intensive Care Medicine—Specialist	None	None	None	None	None	None
Tetsuya Sakamoto	Tokyo University—Professor	[b]Ministry of Health, Labor & Welfare, Jap.	None	None	None	None	None
Claudio Sandroni	Catholic University School of Medicine-Rome: Assistant Professor	None	None	None	None	None	None
Tommaso Sanna	Catholic University of the Sacred Heart—Researcher & cardiologist	None	None	None	None	None	None
Tomoyuki Sato	TBD	TBD	TBD	TBD	TBD	TBD	TBD
Sudhakar Sattur	University of Arizona—Assist Prof.	None	None	None	None	None	None
Andrea Scapigliati	Catholic University of the Sacred Heart—Assistant Professor	None	None	None	None	None	None
Richard Schilling	TBD	TBD	TBD	TBD	TBD	TBD	TBD
Ian Seppelt	Sydney West Area Health Service: Clinical Intensive Care Medicine—Senior Staff Specialist	None	None	None	None	[a]Sedation Advisory Board in Intensive Care' for Hospira Pharmaceuticals (manufacturers of dexmedetomidine)	None
Fred Severyn	University of Colorado—Emergency Physician	None	None	None	None	[a]I have been subpoenaed several times from Adams county Colorado to testify as expert witness in felony cases in which I provided medical care to a victim of injury/illness–not on my terms, but served as expert witness (or else get hit for contempt of court and go to jail!)	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Greene Shepherd	University of Georgia, College of Pharmacy—Professor	None	None	[a]CE talks about calcium channel blocker poisoning (one of my assigned topics) for professional societies. Amounts of honoraria never exceeded $500	None	None	None
Richard Shih	Emergency Medical Associates Emergency Medicine Physician Group Emergency Physician	None	None	None	None	None	None
Markus Skrifvars	Sydney South West Area Health Service—Senior Registrar ICU	[b]Research grant (15.000 American dollars) from the Laerdal Foundation for Acute Medicine in 2009 for a research project on outcome following intensive care of cardiac arrest patients	None	None	None	None	None
Jasmeet Soar	North Bristol NHS Trust—Consultant in Anaesthetics & Intensive Care Medicine	None	None	None	None	None	None
Keichi Tada	Hiroshima Hosp. MD	None	None	None	None	None	None
Sara Tararan	Azienda per i Servizi Sanitari n. 6 "Friuli Occidentale" RN	None	None	None	None	None	None
Michel Torbey	Medical College of Wisconsin—Associate Professor	None	None	None	None	None	None
Jonathan Weinstock	Tufts Medical Center—Staff Cardiologist	None	None	None	None	None	None
Volker Wenzel	Innsbruck Medical University—Associate Professor and Vice Chair	[b]Science Foundation Austrian National Bank grant 11448, Vienna, Austria (support for VITRIS.at trauma trial) [a]German Air Rescue, Filderstadt, Germany Swiss Air Rescue, Zürich, Switzerland Austrian Air Ambulance, Vienna, Austria	[b]AOP Orphand Drugs, Vienna, Austria, manufacturing of study drugs for VITRIS.at trauma trial Innsbruck Medical University, Department of Anesthesiology, employment of study nurse for VITRIS at trauma trial	None	None	None	None
Christoph Wiese	Univ of Regensberg Germany,Anesthesiology	None	None	None	None	None	None
Daniel Wu	Emory University School of Medicine; Assistant Professor	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ Honoraria	Ownership interest	Consultant/ advisory board	Other
Carolyn Zelop	SFH Dept of Ob/Gyn—Director of MFM/Associate Chair	None	None	None	None	None	[a]I have been an expert witness for VBAC cases.
David Zideman	Imperial College Healthcare NHS Trust–Consultant Anaesthetist; London Olympics 2012–Clinical Lead-EMS	None	None	None	None	None	[a]Her Majesty's Coroner – Surrey – Less than £1000 (UKP)
Janice Zimmerman	The Methodist Hospital Physician Organization–Head, Critical Care Division	None	None	None	None	None	None

This table represents the relationships of worksheet collaborators that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all worksheet collaborators are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

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Part 9: Acute coronary syndromes
2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations☆

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The International Liaison Committee on Resuscitation (ILCOR) ACS-MI Task Force included expert reviewers from Africa, Asia, Australia, Europe, North America, and South America. These experts reviewed 25 topics related to the acute initial management of acute coronary syndrome (ACS), which was further categorised as unstable angina, non-ST-elevation MI (UA/NSTEMI) and ST-elevation MI (STEMI). Topics were identified based on previous recommendations, emerging science, and clinical importance, using an iterative writing process involving all Task Force members. The Task Force reviewed the evidence specifically related to diagnosis and treatment of ACS in the out-of-hospital setting and the first hours of care in the in-hospital setting, typically in the emergency department (ED). The evidence review took place over several years, with ongoing refinement of recommendations being made as new evidence was published. The purpose of the review was to generate current, evidence-based treatment recommendations for healthcare providers who serve as the initial point of contact for patients with signs and symptoms suggestive of ACS.

The following is a summary of the most important changes in recommendations for diagnosis and treatment of ACS since the last ILCOR review in 2005.[1,2]

- The history and physical examination, initial ECG, and initial serum biomarkers, even when used in combination, cannot be used to reliably exclude ACS in the prehospital and ED settings.
- In contrast, chest pain observation protocols are useful in identifying patients with suspected ACS and patients who require admission or may be referred for provocative testing for coronary artery disease (CAD) to identify reversible ischaemia. Such strategies also reduce cost by reducing unnecessary hospital admissions and improve patient safety through more accurate identification of NSTEMI and STEMI.
- The acquisition of a prehospital 12-lead ECG is essential for identification of STEMI patients before hospital arrival and should be used in conjunction with pre-arrival hospital notification and concurrent activation of the catheter laboratory.
- Nonphysicians can be trained to independently interpret 12-lead ECGs for the purpose of identifying patients with STEMI, provided that appropriate and reliable STEMI criteria are used. This skill is of particular value in the prehospital setting where paramedics may independently identify STEMI, thus mitigating over-reliance on ECG transmission.
- Computer-assisted ECG interpretation can be used to increase diagnostic accuracy of STEMI diagnosis when used alone or in combination with ECG interpretation by a trained healthcare provider.
- STEMI systems of care can be implemented to improve the time to treatment. The following measures have been shown to reduce the time to primary percutaneous coronary intervention (PPCI): institutional commitment, use of a team-based approach, arranging single-call activation of the catheterisation laboratory by the emergency physician or prehospital provider, requiring the catheterisation laboratory to be ready in 20 min, having an experienced cardiologist always available, and providing real-time data feedback.
- Intravenous β-blockers should not be given routinely in the ED or prehospital setting, but may be useful in a subset of patients with hypertension or tachycardia in the setting of ACS.
- The routine use of high-flow supplemental oxygen in ACS is not recommended. Instead, oxygen administration should be guided by arterial oxygen saturation.
- Reinforce the need for time targets for reperfusion beginning from the time of first medical contact (FMC). The clinical circumstances that favor fibrinolysis and PCI are discussed, including the role of prehospital fibrinolytics.
- The prophylactic use of antiarrhythmics is discouraged.

☆ *Note from the writing group*: Throughout this article, the reader will notice combinations of superscripted letters and numbers (e.g., "Chest Pain Observation Units$^{ACS\text{-}005A''}$"). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.

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- Angiography and percutaneous coronary intervention (PCI) may be considered in patients with out-of-hospital cardiac arrest (OHCA) and return of spontaneous circulation (ROSC). It may also be acceptable to perform angiography in selected patients, despite the absence of ST-segment elevation on the ECG or prior clinical findings such as chest pain.

Despite progress in diagnostic and therapeutic strategies, numerous knowledge gaps have been identified during the discussions. These gaps include:

- Much of the research concerning the care of the patient with ACS has been conducted on in-hospital populations rather than specifically in the ED or out-of-hospital settings. By definition, extending the conclusions from such research to the early ED management or the out-of-hospital setting requires extrapolation.
- Strategies for improving layperson recognition of ACS and shortening time to diagnosis in vulnerable populations.
- The value of emergency dispatcher-initiated bystander administration of aspirin.
- Accurate decision rules for the early identification of patients with and without ACS in the prehospital and the ED settings.
- Feasibility of widespread paramedic interpretation of prehospital 12-lead ECGs versus reliance on transmission or computer interpretation.
- Impact on mortality of systems of care strategies designed to expedite reperfusion.
- The role of reperfusion including PCI in post-cardiac arrest care following either prehospital or in-hospital cardiac arrest, in the presence or absence of STEMI.
- The sensitivity and specificity of newer biomarkers for the detection of ACS.
- Is high-dose oxygen harmful in the setting of ACS?
- What is the role of analgesia and anxiolysis in patients with ACS?
- Optimal timing of platelet inhibition and anticoagulation in the prehospital and ED setting.
- While the time goals for reperfusion begin with first medical contact, time from symptom onset may be preferred, yet precise identification of this time point has been elusive.

The American Heart Association and the American College of Cardiology, the European Society of Cardiology, and others have developed comprehensive guidelines for the in-hospital management of patients with STEMI and UA/NSTEMI, and the reader is referred to these guidelines for more detailed recommendations regarding the care of patients with ACS.[3–6] The ILCOR CoSTR statements are intended to supplement these other resources by having a specific focus on the initial evaluation and treatment in the prehospital and ED phases of care. It is envisioned that these CoSTR documents will be used to develop treatment guidelines to assist providers during the initial acute phase of care.

The prognostic and diagnostic use of the signs and symptoms of ACS, cardiac markers, and 12-lead ECG can have enormous impact on the initial impression and management of patients with suspected ACS. As such, it is important to evaluate the sensitivity, specificity, and clinical impact of various diagnostic strategies in ACS through a comprehensive evidence-based process.

The 12-lead ECG in the ED and out-of-hospital settings is central to the initial triage of patients with possible ACS. Neither signs and symptoms nor cardiac markers alone are sufficiently sensitive to diagnose AMI or ischaemia in the prehospital setting or the first 4–6 h in the ED.

Diagnostic tests in ACS

Risk stratification

Demographic factors[ACS-002A,ACS-002B]

For patients with ACS, we evaluated whether any specific demographic factors (e.g., age, sex, race, weight) were associated with delayed treatment and classified these delays according to whether they occurred before or after hospital arrival.

Consensus on science

Prehospital treatment delay. Thirty-five studies (LOE P1[7,8]; LOE P3[9–40]) showed that demographic factors, such as older age,[8,11,16,19–25,28–31,35–39,41] female gender,[7,10–13,16,19,21,22,25,26,28–35,37,38,42] nonwhite race,[7,8,14,15,19–21,27,30,38–40] low socioeconomic status,[7–9,17,18,37,38,41] and living alone[19,25,7] are independent factors for prehospital treatment delay (symptom-to-door time).

Twenty studies indicated that old age, female gender, nonwhite race and/or living alone did not show any association with prehospital delay times (LOE P2[43]; LOE P3[13,17,20,24,25,36,40,41,44–54]).

As many studies analysed more than one demographic factor for prediction of treatment delay, and one factor may predict delay while another factor was not found to be independent for prediction of delay, eight studies were mixed in identifying factors associated with treatment delays (LOE P2).[13,17,20,24,25,36,40,41]

In-hospital treatment delay. Nineteen studies (LOE P2[8]; LOE P3[9,10,14,19,29,39,42,55–62]; LOE 5[63–65]) showed that demographic factors, such as older age,[8,19,29,39,55–58,60,61,63]), female gender,[8,10,19,29,39,42,55–58,60–64] nonwhite race,[8,14,19,39,55,58–60,63–65] low socioeconomic status,[8,9] and living alone[19] are independent factors for in-hospital treatment delay (door-to-balloon, door-to-needle, or door-to-reperfusion time).

Five studies indicated that older age, female gender, nonwhite race and/or living alone did not show any association with in hospital delay times (LOE P3).[48,49,54,62,66] Most data on the impact of demographic factors on delay to treatment for patients with ACS have been derived from studies in North America.

Treatment recommendation

Various patient-related factors impede seeking medical help rapidly, but also add to further in-hospital treatment delay; these factors include older age, racial and ethnic minorities, female gender, low socioeconomic status and residing alone. Providers should be trained to expeditiously identify patients with ACS irrespective of age, gender, socioeconomic status, or living arrangements.

Accuracy of history and physical examination for diagnosing ACS[ACS-011]

In patients with suspected ACS in various settings (e.g., prehospital, emergency or in-hospital), do specific historical factors, physical examination findings, and test results, compared with normal, increase the accuracy of diagnosis ACS and MI?

Consensus on science: diagnosis

Fourteen studies (LOE 2[67–70]; LOE 3[71–80];) did not support the use of any clinical signs and symptoms independent of ECG, cardiac biomarkers, or other diagnostic tests to rule in or rule out ACS in prehospital or ED settings. Although some signs are more sensitive and specific than others, no sign or symptom evaluated exceeded 92% sensitivity in the higher LOE studies (most reported sensitivity of 35–38%) or 91% specificity (range 28–91%).

Four LOE 1 studies[71,81–83] and 32 studies (LOE 3 to 5)[24,31,52,67–70,72–75,78,84–103] suggest that individual clinical signs and symptoms lack sufficient sensitivity and specificity to be used alone and independent of ECG, cardiac biomarkers, or other diagnostic tests to rule in or rule out ACS in prehospital or ED settings.

Consensus on science: prognosis and clinical impact

In 34 studies (LOE 1[71,83,92]; LOE 2[24,67–70,84,87,94,95,100]; LOE 3[31,52,72–74,76–79,82,85,86,89,90,93,96–99,101,104]) a variety of signs and symptoms assisted in the diagnosis of ACS and had clinical impact (defined as triage and some treatment and investigational decisions) on the prehospital emergency management and risk assessment for coronary atherosclerosis and unstable syndromes.

Three LOE 1 meta-analyses/systematic reviews[71,82,83] and 28 studies LOE 3–5 [24,31,52,67–70,72–75,84–87,89–95,97–101,103] suggest that some clinical signs (e.g., chest pain that radiates to the left arm, radiates to the right shoulder, or radiates to both arms, patients presenting with chest pain and sweating, S3 or hypotension, sweating, and/or vomiting, a history of risk factors [in addition to known coronary heart disease], and some demographic characteristics such as age) assisted in the diagnosis of ACS and had clinical impact (defined as influencing triage and some treatment and investigational decisions) on the out-of-hospital emergency management and risk assessment for ACS. One LOE 5 study[103] and extrapolations from 27 other studies LOE 3–5 studies[24,31,52,67–70,72–75,84–87,89–95,97–101] suggested that there are symptom clusters related to demographic factors such as age, race, and sex. These symptom clusters may have an impact on clinical decision-making (defined as influencing triage and some treatment and investigational decisions). One systematic review/meta-analysis (LOE 1)[81] found the sign of tenderness to chest wall palpation useful in ruling out a diagnosis of AMI.

Treatment recommendation

Signs and symptoms alone are neither sensitive nor specific and should not be used without other data for making the diagnosis of ACS. Signs and symptoms may be useful in combination with other important information (biomarkers, risk factors, ECG, and other diagnostic tests) in making triage and some treatment and investigational decisions for ACS in the out-of hospital and ED setting.

ACS and nitroglycerin[ACS-030A-1,ACS-030A-2]

In patients with suspected ACS/STEMI in the ED and prehospital settings, does the use of nitroglycerin, compared with no nitroglycerin, improve diagnosis of ACS/MI?

Consensus on science

Five studies (LOE D3[68,78,105]; LOE D4[106,107]) using reduction in pain after nitroglycerin administration as an end point, found that reduction of pain does not reliably identify presence of ACS.

Treatment recommendations

A reduction in chest pain following nitroglycerin administration may be unrelated to the presence or absence of ACS, and should not be used as a diagnostic test or strategy in the prehospital or ED setting.

ED interpretation of 12-lead ECG for STEMI

12-Lead ECG[ACS-014]

In patients with suspected ACS in various settings (e.g., prehospital or emergency), does the use of prehospital or emergency 12-lead ECG, compared with standard diagnostic techniques, increase sensitivity and specificity of diagnosis of ACS/MI?

Consensus on science

One study showed that prehospital or emergency ECGs had a sensitivity of 76% and a specificity of 88% for diagnosing acute cardiac ischaemia in patients with chest pain (LOE D1).[108] For diagnosing AMI, prehospital ECG had a sensitivity of 68% and a specificity of 97%. Two studies indicated that diagnostic accuracy of the prehospital ECG can be improved by repeating the ECG on arrival in the ED and by serial measurement of cardiac markers (LOE D2).[109,110] Two studies showed that computer-interpreted electrocardiograpy or field-transmitted electrocardiography can be applied if no adequate interpretation of the prehospital ECG is available on site (LOE D1).[111,112]

Treatment recommendation

In patients with suspected ACS, a 12-lead-ECG should be acquired and interpreted by prehospital or emergency providers as soon as possible after first patient contact. The interpretation should be used in conjunction with the clinical signs and presentation for diagnosis and triage, including destination decisions and activation of the cardiac catheterisation laboratory. If interpretation of the prehospital ECG is not available on site, field-transmission of the ECG for expert interpretation may be reasonable.

Diagnosis of STEMI by nonphysicians[ACS-007B]

In patients with suspected ACS in the prehospital, ED, or in-hospital settings, can nonphysicians (e.g., paramedics and nurses) accurately diagnose STEMI when compared to physicians?

Consensus on science

Eight observational studies reported paramedics can diagnose STEMI in the prehospital setting without transmission of a 12-lead ECG for physician consultation (LOE D3[113–115]; LOE D4[116–119]; LOE D5[120]). The limited evidence available about paramedic false-negative diagnostic decisions, including decisions not to obtain a 12-lead ECG, may affect paramedics' true overall diagnostic accuracy.

Eight observational studies reported that nurses can diagnose STEMI in the context of nurse-initiated fibrinolysis programs (LOE D3[121]; LOE D4[116,122–124]; LOE D5[125–127]). The literature largely describes the ability of nurses to avoid false-positive diagnosis in fibrinolysis programs without substantial evidence about false-negative decisions, which may affect true overall diagnostic accuracy.

Treatment recommendations

It is reasonable for paramedics and nurses to identify STEMI on a 12-lead ECG independently as long there is a mandatory program of initial training and ongoing concurrent medical oversight of all ECG interpretations.

Computer-assisted ECG interpretation[ACS-008A]

In patients with suspected ACS, does the use of computer-assisted ECG interpretation, compared with standard diagnostic techniques (emergency physicians), increase accuracy of diagnosis (e.g., of NSTEMI/STEMI)?

Consensus on science

Two studies found evidence of improved diagnostic accuracy with the use of computerised ECG interpretation (LOE D5).[128,129] Eight studies either found no effect or equivocal effect of the

use of computerised ECG interpretation on diagnostic accuracy (LOE 1[111,130–132]; LOE D5[133–136]). Two studies found evidence that the use of computerised ECG interpretation decreased diagnostic accuracy (LOE D1).[137,138] Three studies showed computer ECG interpretation relating to ACS to be reliable (LOE D1[137]; LOE 1[111,130]). The "gold standard" used most commonly was expert "electrocardiographer" review, although four studies used validated clinical diagnosis as the gold standard (LOE 1[130]; LOE D1[111]; LOE D1[131]; LOE D5[133]). Two studies reported a higher specificity for the computer-interpretation (identifying true negatives), while the physicians had a higher sensitivity (identifying true positives) (LOE 1[111]; LOE D1[131]). Three studies found that computer interpretation had a greater influence on nonexpert subject performance in interpreting ECGs than it did on more expert interpretation (LOE D1[137]; LOE D5[135]; LOE D5[133]).

Treatment recommendation

Prehospital ECG interpretation should be augmented with computer interpretation. Computer interpretation of the ECG may increase the specificity of diagnosis of STEMI, especially for clinicians less experienced in reading ECGs. The benefit of computer interpretation is dependent on accuracy, and therefore computer-assisted ECG interpretation should not replace, but may be used as an adjunct to, interpretation by an experienced clinician. The computer interpretation should be considered in the clinical context.

Diagnostic and prognostic test characteristics of cardiac biomarkers for ACS

Protein markers of coronary ischaemia[ACS-013B]

In patients with suspected ACS in various settings (e.g., prehospital, emergency, or in-hospital), do abnormal protein markers compared with normal levels allow the clinician to accurately diagnose acute coronary ischaemia?

Consensus on science

Eight studies supported cardiac troponin testing alone in the diagnosis of AMI, when serum testing was drawn at least 6 h from time of symptom onset or ED presentation, or drawn serially (LOE D2[139–141]; LOE D3[142]; LOE D4[143–146]).

No studies showed adequate sensitivity of cardiac troponin testing outside of the ED or short-stay cardiac unit (LOE 2[147]; LOE 4[148–150]) including the ICU (LOE 4).[151] Four studies showed increased sensitivity of new sensitive troponin assays compared with conventional troponin assays and supported their use to diagnose AMI (LOE D2[152,153]; LOE D3[154]; LOE D4[155]). Nine studies supported multimarker testing (CK-MB, ischaemia-modified albumin or myoglobin) in combination with cardiac troponin in the diagnosis of AMI (LOE D2[139,141,153,156–158]; LOE D4[145,156,159]).

There were heterogeneous data on the use of troponin point-of-care testing (POCT) in the diagnosis of ACS: five studies supported the use of troponin POCT (LOE D2[145]; LOE D4[145,160–163]), and five studies opposed the use of troponin POCT in the ED and cardiac short-stay units (LOE D3[164]; LOE D4[165–168]). Two studies opposed the use of troponin POCT in the prehospital setting (LOE D4),[148,149] and one opposed the use of troponin POCT in the outpatient clinic setting (LOE D2).[147]

Treatment recommendations

Clinicians should take into account the timing of symptom onset, the sensitivity, precision, and institutional norms of the assay, and the release kinetics and clearance of the measured biomarker.

All patients presenting to the ED with symptoms suspicious of cardiac ischaemia should have cardiac biomarker testing as part of an initial evaluation. A cardiac-specific troponin is the preferred biomarker. For patients who present within 6 h of symptom onset suggestive of cardiac ischaemia with negative cardiac troponin initially, it is recommended that a troponin level be remeasured between 6 and 12 h after symptom onset. It is reasonable to use highly sensitive cardiac troponin assays, defined as having a 10% coefficient of variation at the 99th percentile, to evaluate patients with symptoms suspicious of cardiac ischaemia. Multimarker evaluation with CK-MB or myoglobin in conjunction with troponin in patients with symptoms suspicious of cardiac ischaemia may be considered to improve the sensitivity of diagnosing AMI.

There is no evidence to support the use of troponin POCT in isolation as a primary test in the prehospital setting to evaluate patients with symptoms suspicious of cardiac ischaemia.

There is insufficient evidence to support the use of myoglobin, brain natriuretic peptide (BNP), NT-proBNP, D-dimer, C-reactive protein, ischaemia-modified albumin pregnancy-associated plasma protein A (PAPP-A), and/or interleukin-6 in isolation as primary tests to evaluate patients with symptoms suspicious for cardiac ischaemia.

Prognosis for discharge versus admission[ACS-004B]

In patients with suspected ACS, does the presence of any specific factors (e.g., history, examination, ECG, and /or biomarkers) or combination into a specific clinical decision rule compared with standard care increase accuracy of prediction of prognosis (e.g., decision rule for early discharge)?

Consensus on science statements

There are no randomised controlled studies addressing clinical decision rules for ACS in the prehospital or ED settings. Existing studies do not adequately address the question because they are heterogeneous (LOE P1).[169] There is not a single published clinical decision rule which is adequate and appropriate for identifying ED chest pain patients who can be safely discharged home from the ED (LOE P1).[169]

Younger patients with no history of previous ischaemic heart disease, atypical presentations, negative serial biomarkers, and a nondiagnostic 12-lead ECGs have a very low short-term rate of adverse events. Five studies demonstrated that younger patients with no history of previous ischaemic heart disease, atypical presentations, negative serial biomarkers, and nondiagnostic 12-lead ECGs have a very low short-term rate of adverse events (LOE P2).[88,170–175] One study demonstrated that older patients are evaluated less effectively and the subset of older patients who can be safely discharged from the ED are less easily identified than younger patients (LOE P2).[88]

Five studies demonstrated that the combined use of serial biomarkers and ECGs in selected patients (i.e., low risk, sensation-free, and clinically stable) can assist in the identification of a subset of patients who can be safely discharged from the ED (LOE P2).[88,170,171,174,175] This statement is not directly age-dependent, although older patients demonstrate higher rates of ACS diagnosis and adverse outcome.

Nine studies demonstrated that scoring systems derived from in-patient populations (e.g., TIMI Risk Score or Goldman Criteria) are not appropriate for ED use and do not assist in the identification of patients who can be safely discharged from the ED (LOE P1[176,177]; LOE P3[178–184]).

Treatment recommendations

None of the currently reported clinical decision rules should be used to select ED chest pain patients who can be safely discharged from the ED. Patients <40 years of age with non-classical presentations and lacking significant past medical history, who have normal

serial biomarkers and 12-lead ECGs, have a very low short-term event rate.

Chest pain observation units[ACS-005A]

In patients with suspected ACS, does the use of chest pain observation units (CPUs), compared with not using them, increase accuracy of diagnosis and safely identify patients who require admission or specific management of CAD?

CPUs have been developed to assess patients with chest pain and normal initial biomarkers and non-ischaemic ECG. The elements that define a CPU vary depending on the characteristics of the individual organisations and the clinical context in which the unit is sited (e.g., ED versus in-patient environment versus dedicated site).

Components of the CPU are typically a protocol or pathway based care strategy, dedicated physical space/infrastructure and staffing, use of an accelerated risk-stratification protocol comprising.

- Measurement of serial biomarkers of acute infarction (e.g., troponin or CK-MB)
- Serial ECG or continuous ECG monitoring
- A period of observation (6 h)
- Integrated with more advanced diagnostic testing (e.g., exercise stress test, myocardial perfusion scan)

Consensus on science

Eleven studies of patients with chest pain and normal initial biomarkers and nondiagnostic ECGs demonstrated that CPUs result in reduced length of stay, hospital admissions, quality of life measures, and healthcare costs (LOE 1).[185–195] One large case-control multicentre study showed that care in CPUs did not reduce the proportion of patients with chest pain admitted to hospital and may have increased ED attendances when implemented across a healthcare system (LOE 2).[196] Fifty-five studies from many healthcare settings demonstrate that CPUs enable evaluation of patients systematically, with a short length of stay, high diagnostic accuracy, and a low event rate at follow-up (LOE 4).[197–246]

Treatment recommendations

In patients with suspicion for ACS, normal initial biomarkers and non-ischaemic ECG, chest pain (observation) protocols may be recommended as a safe and effective strategy for evaluating patients in the ED.

Chest pain observation protocols should include a history and physical examination, a period of observation, serial electrocardiography, serial measurement of serum cardiac markers, and either an evaluation for anatomic coronary disease or for inducible myocardial ischaemia at some point after AMI is excluded. These protocols may be used to improve accuracy in identifying patients requiring in-patient admission or further diagnostic testing, and those who may be discharged.

Chest pain protocols may be recommended as a means to reduce length of stay, reduce hospital admissions, reduce healthcare costs, improve diagnostic accuracy, and improve quality of life. Since CPUs have not been shown to a reduce hospital admission rates, these protocols must be monitored so that they do not lead to overutilization of hospital resources. There is also no direct evidence demonstrating that CPUs or (observation protocols) reduce adverse cardiovascular outcomes, particularly mortality for patients presenting with possible ACS, normal serum cardiac biomarkers, and a nondiagnostic ECG.

Imaging techniques

Imaging techniques and diagnosis[ACS-006-1A,ACS-006-1B]

In patients with suspected ACS, does the use of specific imaging techniques (e.g., CT angiography, MRI, nuclear, echocardiography), compared with not using them, increase accuracy of diagnosis (e.g., of ACS).

Consensus on science

Data from one study (LOE D2)[247] documented a sensitivity of 89% and a specificity of 77% for detection of ACS when myocardial perfusion imaging was used in adults presenting to the ED with chest pain, a nondiagnostic ECG, and negative cardiac biomarkers. Supportive evidence was also provided by four other studies (LOE D4)[217,248–250] for adults presenting to the ED with chest pain.

Data from two studies showed high sensitivity (95%) and specificity (90%) for detection of ACS in adults who received multidetector CT angiography (MDCT, 64-slice scanner) after presentation to the ED with chest pain, a nondiagnostic ECG, and negative cardiac biomarkers (LOE D2).[251,252] This finding was also supported by four studies (LOE D4).[198,217,253,254]

Data from one study documented sensitivity 93% and specificity 66% when rest echocardiography is used for detection of ACS in low-risk patients who presented to the ED with chest pain, a nondiagnostic ECG, and negative cardiac biomarkers (LOE D2).[247] Supportive evidence was also provided by one prospective cohort study (LOE D4).[250] One prospective study provided similar estimates including specificity of 95% and positive predictive value of 81% for exercise stress echo in the same population (LOE D4).[248]

Data from two studies documented high sensitivity (85%), specificity (84%), and negative predictive value (95%) for the diagnosis of ACS in adult patients who received MRI within 24 h of presentation to the ED with chest pain after a nondiagnostic ECG and negative cardiac biomarkers (LOE D4).[255,256]

Treatment recommendations

A noninvasive test (CT angiography, cardiac MR, myocardial perfusion imaging, and echocardiography) may be considered in selected patients who present to the ED with chest pain and initial nondiagnostic conventional work-ups.

It is reasonable to consider both the exposure to radiation and iodinated contrast when utilizing MDCT and myocardial perfusion imaging.

Imaging techniques and outcome[ACS-006-2A,ACS-006-2B]

In patients with suspected ACS, does the use of specific imaging techniques (e.g., CT angiography, MRI, nuclear, or echocardiography), compared with not using them, improve outcome (survival, length of ED stay, hospital admission rate, cost?).

Consensus on science statements

Data from two studies of low-risk ED patients with an initial negative work-up of ACS with negative cardiac enzymes and non-diagnostic ECGs, who received SPECT perfusion imaging, demonstrated low rates of cardiac events, reduced costs, and reduced length of stay (LOE 4).[215,249]

Data from three studies of 64-slice MDCT utilised within 24 h in adult patients presenting to the ED with chest pain, showed that the procedure decreases time to diagnosis, reduces costs, reduces length of stay, is predictive of major adverse events, and can lead to safe discharge from the ED (LOE 1[257]; LOE 4[258,259]).

Data from five studies of echocardiography performed in adult ED patients presenting with chest pain, negative cardiac enzymes, and non-diagnostic ECGs demonstrated decreased mean length of

stay and reduced costs and predicted a low cardiovascular event rate (LOE 1[260]; LOE 4[261–264]).

Treatment recommendations

Based on studies which investigated limited numbers of selected individuals, patients presenting to the ED with suspected ACS and having a negative initial work-up, including a nondiagnostic ECG and negative cardiac biomarkers, an evaluation with a noninvasive test (CT angiography, myocardial perfusion imaging, or stress echocardiography) may be considered. In selected groups these noninvasive tests may decrease costs, length of stay, and time to diagnosis and may provide valuable short-term and long-term prognostic information of future major cardiac events. There are insufficient data to assess impact on mortality.

Initial therapeutic interventions

Few studies have been published that directly address out-of-hospital or ED interventions for ACS. In some situations, extrapolation from in-hospital evidence was needed to provide some guidance for out-of-hospital and early ED management.

Oxygen therapy

Supplemental oxygen[ACS-015]

In patients with suspected ACS in various settings (e.g., prehospital, emergency or in-hospital) and normal oxygen saturations, does the use of supplemental oxygen, compared with room air, improve outcomes (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

One study reported improvement in ST changes if oxygen was given to 17 patients with myocardial infarction (LOE 4).[265] One LOE 1 trial[266] conducted before the introduction of reperfusion therapy reported that the amount of aspartate aminotransferase released in the circulation was higher in patients who received oxygen therapy. Ventricular tachycardia (VT) and mortality was not significantly different in the two groups. Another LOE 1 study[267] involving myocardial infarction patients treated with streptokinase showed no impact of oxygen on the occurrence of VT. Severe hypoxaemia occurred less often in patients given oxygen therapy. One LOE 1 study[268] found that studies were small and lacked statistical power to detect a true influence on clinical outcomes. The review found no definite proof of a harmful effect of oxygen therapy; however, there is absolutely no evidence that oxygen was beneficial to patients with myocardial infarction unless complicated by hypoxaemia.

Treatment recommendations

There is insufficient evidence to support or refute the empirical use of high-flow oxygen therapy in patients with uncomplicated AMI without signs of hypoxaemia and/or heart failure. There are insufficient data to support or refute the fact that high-flow oxygen therapy might be harmful in this setting. In addition, there is lack of evidence to suggest that low flow oxygen is of any benefit in patients with normal oxygen saturation levels.

Oxygen therapy should be initiated if breathlessness, hypoxaemia, or signs of heart failure or shock are present. Noninvasive monitoring of oxygen saturation may be used to decide on the need for oxygen administration.

ACS and nitroglycerin[ACS-030A-1,ACS-030A-2]

In patients with suspected ACS/STEMI in the ED and prehospital settings, does the use of nitroglycerin, compared with no nitroglycerin, improve outcome (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

Despite multiple studies performed in the pre-reperfusion era that have shown a benefit of early nitroglycerin administration in patients with a myocardial infarction, no trial specifically evaluated patients in the ED or prehospital settings. The greatest reduction in infarct size was noted in those treated within 3 h of symptom onset in three studies of patients treated in the ICU (LOE 5).[269–271] In addition, two trials suggested that concomitant treatment of nitroglycerin and fibrinolytics may impair reperfusion (LOE 2).[272,273] One study of patients with NSTEMI showed a reduction in myocardial infarction size in those treated with diltiazem compared with intravenous glyceryl trinitrate (LOE 1).[274] There is insufficient evidence to determine the benefit or harm of initiating nitroglycerin treatment in the prehospital setting or ED.

Treatment recommendations

Although it is reasonable to consider the early administration of nitroglycerin in selected patients without contraindications, insufficient evidence exists to support or refute the routine administration of nitroglycerin in the ED or prehospital setting in patients with a suspected ACS. There may be some benefit if nitroglycerin administration results in pain relief.

Analgesics and sedation

The worksheet on the topic of Analgesics and Sedation was not completed for the 2010 International Consensus Conference, but the task force felt the topic was important to the care of patients with ACS. As a result, this topic was reviewed by the task force, and they developed the summary of science and treatment recommendations.

In patients with suspected ACS/STEMI in the ED and prehospital settings, does the use of analgesic and/or sedation (including NSAIDs, opiates, and benzodiazepines) compared with no analgesia or sedation, improve outcome (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

One study suggested increased mortality and myocardial infarction rates associated with the use of intravenous morphine in patients presenting with high-risk NSTEMI (LOE 4).[275] One study demonstrated that the early use of lorazepam with nitroglycerin was more effective than nitroglycerin alone and appears to be safe in relieving cocaine-associated chest pain (LOE 1).[276] One study was neutral when diazepam was compared with placebo on the end points of tachyarrhythmias, self-assessed anxiety, or other symptoms in undifferentiated patients with AMI (LOE 1).[277]

One analysis of case control and cohort studies studying patient exposure to NSAIDs (LOE 1)[278] and a large analysis of clinical trials randomizing patients to Cox inhibitors over placebo (LOE 1)[279] revealed an increased risk for developing myocardial infarction with use of NSAIDs. The risk appeared most consistent with rofecoxib, and was less consistently observed with celecoxib, naprosyn, ibuprofen, and diclofenac. One study suggested increased harm with the initiation or continuation of NSAID (except aspirin) in patients with suspected ACS (LOE 4).[280]

Treatment recommendations

Morphine should be administered intravenously and titrated to pain relief in patients with STEMI. Morphine may be considered for pain relief in subjects with suspected NSTEMI. Some form of analgesia should be considered for patients with active chest discomfort. While anxiolytics may be administered to patients with ACS to alleviate apprehension and anxiety, there is no evidence that anxiolytics facilitate ECG resolution, reduce infarct size, or decrease mortality in undifferentiated patients with suspected ACS. Lorazepam with nitroglycerin may be considered to alleviate pain in patients with cocaine-associated chest pain. NSAIDs should not be administered and may be harmful in subjects with suspected ACS. Patients with suspected ACS who are taking NSAIDs should have them discontinued when feasible.

Aspirin (acetylsalicylic acid)

Timing of aspirin administration[ACS-003B]

In patients with suspected ACS, does dispatcher guided administration of aspirin by bystanders before arrival of EMS, compared with later administration of aspirin by paramedic or ED staff, improve outcome?

Consensus on science

There was no clear evidence to support or refute the use of prehospital or EMS dispatch directed (versus hospital administered) aspirin. One study found that aspirin, given before fibrinolysis, increased long-term survival (LOE 1).[281] One study showed a benefit in STEMI patients with a decrease in in-hospital complications and 7- and 30-day mortality when given prehospital (LOE 4).[282] There was clear evidence that aspirin is associated with a reduction in long-term mortality, which is greatest when the aspirin is administered in the first 4 h of after an event. One study showed no benefit with administration within the first 4 h of symptoms, compared with later administration (LOE 1).[283] Two other studies showed that the potential benefit from early aspirin administration outweighs potential harm (LOE 1).[284,285]

Treatment recommendations

Despite limited direct evidence to support or refute the practice, it may be reasonable to consider EMS or dispatcher-guided bystander aspirin administration, provided an adequate history to exclude a true allergy or a bleeding disorder, can be obtained.

Clopidogrel and other platelet ADP-receptor antagonists

Clopidogrel (and similar drugs)[ACS-019A,ACS-019B]

In patients with non-ST-elevation ACS (NSTE-ACS), STEMI managed with fibrinolysis, and STEMI managed with P PCI, in prehospital and ED settings, does the use of clopidogrel or newer oral antiplatelet agents (prasugrel, ticagrelor) compared with standard management (e.g., no prehospital or ED use of clopidogrel or compared to clopidogrel or new tienopyridines), improve outcome (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 day mortality)?

Consensus on science

Clopidogrel. Seven studies (LOE 1[286–289]; LOE 2[290,291](LOE 3)[292]) documented consistent improvement, and one study (LOE 5)[293] was neutral in demonstrating benefit in the combined event rate of cardiovascular mortality, nonfatal infarction, nonfatal stroke, and overall mortality. There was a small increase in major bleeding when clopidogrel was administered by providers in the ED or hospital to patients with non-ST-elevation ACS.

Six studies documented consistent improvement in combined event rate of cardiovascular mortality, nonfatal infarction, and nonfatal stroke, with a resultant small increase in major bleeding when clopidogrel was administered by providers in the ED or prehospital to patients <75 years with STEMI managed with fibrinolysis (LOE 1[294–297]; LOE 3[298,299]).

Five studies documented improvement in combined event rate (cardiovascular mortality, nonfatal infarction, and nonfatal stroke) and mortality with a resultant small increase in major bleeding when clopidogrel was administered by ED, hospital and/or prehospital providers to patients with STEMI managed with PPCI (LOE 2[300,301]; LOE 3[298,299]; LOE 5[296]).

There was little evidence on the use of a loading dose of clopidogrel in patients ≥75 years of age treated by PPCI, and they were excluded from studies if treated with fibrinolysis.

Prasugrel. There was no direct evidence of use of prasugrel in the ED or prehospital setting for non-ST elevation ACS. Extrapolating evidence from an in-hospital setting, five studies (LOE 5)[302–306] documented improvement and one study (LOE 1)[307] documented no benefit in combined event rate (cardiovascular mortality, nonfatal infarction, and nonfatal stroke) or mortality, but with a resultant increase in major bleeding when prasugrel (compared to clopidogrel) was administered after angiography to patients with non-ST elevation ACS and stenoses suitable for PCI.

There was no direct or indirect evidence of benefit or risk of prasugrel administered by hospital, ED, or prehospital providers to patients with STEMI managed with fibrinolysis. There was no direct evidence of the use of prasugrel in the ED or prehospital setting for patients with STEMI ACS. There was no direct evidence of the use of prasugrel in the ED or prehospital setting for patients with STEMI ACS managed with PCI. Six studies demonstrated small improvements in combined event rate (cardiovascular mortality, nonfatal infarction, and nonfatal stroke) and/or mortality when prasugrel compared with clopidogrel was administered in the hospital setting before, during, or after angiography to patients with STEMI managed with PPCI (LOE 5).[302–306,308]

Post hoc exploratory analysis of a randomised control trial in STEMI and non-ST-elevation ACS patients treated by PCI identified risk factors associated with a higher rate of bleeding complications with prasugrel: patients ≥75 years of age, history of stroke or transient ischaemic attack (TIA), and body weight less than 60 kg (LOE 5).[302]

Ticagrelor. One study documented improvement in overall mortality and combined event rates (death from vascular causes, MI, or stroke) with a marginal increase in bleeding and an increase in dyspnea when ticagrelor, given by in-hospital providers to patients with high-risk non-ST elevation ACS, was compared with clopidogrel (LOE 1).[309] There was no direct or indirect evidence of benefit or risk of ticagrelor administered by hospital, ED, or prehospital providers to patients with STEMI managed with fibrinolysis. One study documented improvement in overall mortality and combined event rates (death from vascular causes, MI, or stroke) with a marginal increase in bleeding and an increase in dyspnea when ticagrelor was administered compared to clopidogrel by in-hospital providers to patients with STEMI managed by PPCI (LOE 1).[309]

Treatment recommendations

Clopidogrel. Administration of clopidogrel in addition to standard care (aspirin, anticoagulants, and/or reperfusion) for patients determined to have moderate to high-risk non-ST elevation ACS and STEMI is recommended. The ideal oral loading dose of clopidogrel in patients <75 years of age is dependent on the planned approach: 600 mg in a planned invasive strategy; or 300 mg in a planned noninvasive strategy or together with fibrinolysis. The

ideal dose in patients >75 years of age has not yet been delineated, but may range from 75 to 600 mg.

Prasugrel. Prasugrel may be administered after angiography to patients with NSTEMI presenting with stenoses amenable to PCI. ED or prehospital administration of clopidogrel should be withheld even in patients who are not at high risk for bleeding (age <75 years, no history of previous stroke or TIA, and body weight >60 kg), pending consideration of prasugrel administration following angiography. In patients who are not at high risk for bleeding with planned PCI, prasugrel (60 mg oral loading dose) may be substituted for clopidogrel for patients determined to have STEMI less than 12 h after the initial symptoms. Prasugrel is not recommended in STEMI patients receiving fibrinolysis.

Ticagrelor. Administration of ticagrelor (180-mg oral loading dose) in addition to standard care (aspirin, anticoagulants, and/or reperfusion) determined to have non-ST elevation ACS or STEMI managed with early invasive strategy by hospital personnel may be an option instead of clopidogrel. The risks and/or benefits of ticagrelor in STEMI patients managed with fibrinolysis is unknown.

Combination. The risks and/or benefits of combining these agents (clopidogrel, prasugrel, and/or ticagrelor) for loading and maintenance dosing has not been sufficiently determined.

Heparins

Anticoagulants and non-ST-elevation ACS[ACS-017-3]

In patients with suspected non-ST-elevation myocardial infarction in the prehospital and ED setting, does the use of new anticoagulants (i.e., pentasaccharide, enoxaparin, bivalirudin), compared with standard management (placebo, unfractionated heparin, other anticoagulant, or no anticoagulant), improve outcomes (e.g., mortality, reinfarction, revascularization, bleeding, stroke)?

Consensus on science

Twenty-two studies demonstrated improved combined end points (death, MI, revascularization) with an increase in the proportion of patients with bleeding complications when enoxaparin was administered in-hospital rather than UFH in patients with AMI (LOE 1[310–320]; LOE 2[321–326]; LOE 5[327–329]).

Four randomised controlled trials (RCTs) (LOE 1),[330–332,333] three meta-analyses (LOE 1),[334–336] six nonrandomised control trials (LOE 2–4),[337–344] and five additional studies (LOE 4–5)[345–349] did not demonstrate a difference for outcomes among in-hospital patients given enoxaparin compared with UFH.

One RCT (LOE 1),[350] three nonrandomised control studies (LOE 2),[351–353] and two additional studies (LOE 5)[354,355] demonstrated improved combined end points (death, MI, revascularization) without increased bleeding when fondaparinux, compared with UFH, was administered in-hospital in patients with AMI. Three studies did not demonstrate a difference in outcomes for fondaparinux compared with UFH when given in hospital (LOE 2[356,357]; LOE 5[358]). One RCT indicated fondaparinux may lead to excess catheter thrombosis when used as part of an invasive approach without the use of adjunctive medications (LOE 1).[350]

Twenty-eight studies (LOE 1[359–364]; LOE 2–4[365–375]; LOE 5[376–386]) did not demonstrate a difference in combined outcomes for major adverse cardiac events but did demonstrate less bleeding for bivalirudin administered in-hospital compared with UFH.

Treatment recommendations

For patients with non-ST-elevation ACS managed with a planned initial conservative approach, either fondaparinux or enoxaparin are reasonable alternatives to UFH. For patients with non-ST-elevation ACS managed with a planned invasive approach, either enoxaparin or UFH are reasonable choices. Bivalirudin may be considered as an alternative, but does not appear to offer an advantage over UFH. Fondaparinux may be used in the setting of PCI, but requires co-administration of UFH and does not appear to offer an advantage over UFH alone.

For patients with non-ST-elevation ACS and renal insufficiency, bivalirudin or UFH may be considered. For patients with non-ST-elevation ACS and increased bleeding risk, where anticoagulant therapy is not contraindicated, fondaparinux or bivalirudin are reasonable, and UFH may be considered. There is no specific evidence for or against anticoagulant use in non-ST-elevation ACS in the prehospital setting. There is currently insufficient evidence on other anticoagulants to make recommendations.

Anticoagulants and STEMI treated with fibrinolysis[ACS-017-1]

In patients with suspected STEMI in the prehospital and ED setting treated with fibrinolysis, does the use of new anticoagulants (i.e., pentasaccharide, enoxaparin, bivalirudin), compared with standard management (placebo, unfractionated heparin, other anticoagulant, or no anticoagulant), improve outcomes (e.g., mortality, reinfarction, revascularization, bleeding, or stroke)?

Consensus on science

Enoxaparin. For patients with STEMI to be treated with fibrinolysis, 17 studies supported enoxaparin over UFH (LOE1[336,387–393]; LOE 2[341,394,395,396]; LOE 4[397]; LOE 5[393,396,398–401]) Twelve other studies were neutral comparing enoxaparin and UFH.[402–411]

Reviparin. One study demonstrated improved clinical outcome with reviparin compared with UFH in STEMI patients treated with fibrinolysis (LOE 1).[412]

Other LMWH. There were two neutral meta-analyses of dalteparin, nadroparin, reviparin, parnaparin (LOE 5),[413,414] one dalteparin supporting study using a surrogate end point (LOE 1),[415] and three neutral studies of LOE 1[416] for nandroparin[416] and parniparin.[417,418]

Fondaparinux. One study demonstrated superiority in clinical outcomes when fondaparinux was compared with UFH in patients treated with fibrinolysis (LOE 1).[419] Two studies did not demonstrate a significant difference in outcomes (LOE 1[420]; LOE 2[421]).

Bivalirudin. Two studies did not demonstrate a significant difference in outcomes with bivalirudin (LOE 1[422]; LOE 2[423]).

Treatment recommendations

Enoxaparin: For patients with STEMI managed with fibrinolysis, it is reasonable to administer enoxaparin instead of UFH. For prehospital patients with STEMI managed with fibrinolysis, adjunctive enoxaparin instead of UFH may be considered. Patients initially treated with enoxaparin should not be switched to UFH and vice versa to avoid increased bleeding risk.

Fondaparinux: May be considered in the hospital for patients treated specifically with non-fibrin-specific thrombolytics (i.e., streptokinase), provided the creatinine level is <3 mg/dL.

Other LMWH or bivalirudin: There are insufficient data to recommend other LMWH or bivalirudin over UFH in patients treated with fibrinolysis in STEMI.

Anticoagulants and STEMI treated with PCI[ACS-017-2]

In patients with suspected STEMI in the prehospital and ED setting to be treated with PPCI, does the use of new anticoagulants (i.e., pentasaccharide, enoxaparin, bivalirudin), compared with standard management (placebo, unfractionated heparin, other anticoagulant, or no anticoagulant), improve outcomes (e.g., mortality, reinfarction, revascularization, bleeding, or stroke)?

Consensus on science

Bivalirudin. Two studies resulted in less bleeding and a short- and long-term reduction in cardiac events and overall mortality with bivalirudin compared with UFH plus a glycoprotein inhibitor in patients with STEMI and planned PCI (LOE 1).[424,425] Two case series also resulted in fewer cardiac events and less bleeding (LOE 4).[426,427] One study demonstrated better outcome of patients with cardiogenic shock if treated with or without a glycoprotein IIb/IIIa inhibitor, compared with UFH plus a glycoprotein IIb/IIIa inhibitor (LOE 4).[428] One study with prehospital initiation of bivalirudin versus UFH showed no difference (LOE 3).[429] One analysis showed no difference when bivalirudin and UFH were compared for PCI (LOE 5).[376] In two studies of bivalirudin versus UFH, outcomes were similar (LOE 2[430]; LOE 4[431]).

Enoxaparin. Three studies of PCI after fibrinolysis resulted in favorable outcome when enoxaparin was compared with UFH (LOE 4[397,432]; LOE 5[394]). Eight other studies showed no benefit using enoxaparin compared with UFH (LOE 2[342,433,434]; LOE 4[405,409,435,436]; LOE 5[345]).

Fondaparinux. One clinical trial comparing fondaparinux with UFH documented similar rates of cardiovascular events but a lower rate of bleeding (LOE 1).[419] One trial, which included patients with NSTEMI and patients undergoing elective PCI, was neutral in outcomes (LOE 5).[358] One analysis of NSTEMI patients documented fewer acute cardiac events and less bleeding using fondaparinux and PCI compared with other antithrombins (LOE 5).[353] Thrombus formation on catheter material in patients on fondaparinux required the addition of UFH during PCI.

Other LMWH. One nonrandomised study compared dalteparin with UFH in STEMI patients undergoing PCI and showed a neutral result (LOE 2).[437]

Treatment recommendations

For patients with STEMI undergoing contemporary PCI, enoxaparin may be considered a safe and effective alternative to UFH. To avoid increased bleeding risk, patients initially treated with enoxaparin should not be switched to UFH and vice versa.

In comparison with UFH, fondaparinux reduces the bleeding risk in STEMI patients undergoing PCI. There is an increased risk of catheter thrombi with fondaparinux alone; additional UFH (50–100 U kg^{-1} BW bolus) may help to avoid this complication, but using these two agents is not recommended over UFH alone. The dose of fondaparinux and enoxaparin requires adjustment in patients with renal impairment.

Bivalirudin may be superior to UFH plus glycoprotein IIb/IIIa inhibitors with respect to bleeding and reduces adverse cardiac events and mortality in STEMI patients undergoing PCI. An increased rate of stent thromboses has been observed with bivalirudin within the first 24 h after PCI.

There are insufficient data to recommend other LMWH than enoxaparin for antithrombin treatment in STEMI patients undergoing PCI.

Glycoprotein IIb/IIIa inhibitors[ACS-020]

In patients with suspected ACS/MI in prehospital and ED settings, does the use of glycoprotein IIb/IIIa inhibitors, compared with standard management, improve outcomes (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

Twelve larger randomised studies and metaanalyses (LOE 1)[438–449] and two smaller randomised studies[450,451] consistently reported better clinical outcome with use of glycoprotein IIb/IIIa inhibitors compared with placebo. This result was supported by many studies which consistently reported better outcomes with upstream or early use of glycoprotein IIb/IIIa inhibitor compared with deferred treatment or other strategies (LOE 1[452–467]; LOE 2[468–473]; LOE 3[474]; LOE 4[475–478]; LOE 5[479]).

There were 12 studies with neutral outcomes/evidence (LOE 1[373,480–487]; LOE 4[488,489]; LOE 5[490]). Seven LOE 1 studies[372,424,491–495] showed worse outcomes, or at least more bleeding and need for transfusion without clinical advantage, with glycoprotein IIb/IIIa inhibitors compared with standard /alternative procedures. In most of the supporting, as well as neutral and opposing, studies a higher rate of (major) bleedings has been observed.

Treatment recommendations

There were insufficient data to support the routine use of glycoprotein IIb/IIIa inhibitors in patients with suspected STEMI or NSTE-ACS in the prehospital or ED settings. For selected high-risk patients with NSTE-ACS, abciximab, eptifibatide, or tirofiban administration may be acceptable, provided PCI is planned. There is an increased bleeding risk with routine glycoprotein IIb/IIIa inhibitors when used with heparins. Alternatives for anticoagulation and antiplatelet treatment might be considered instead.

Reperfusion strategies

In the majority of patients, STEMI occurs as the result of a recent acute occlusion of a major epicardial coronary artery due to the disruption of atherosclerotic plaque and thrombus formation. Strategies aimed at restoring myocardial perfusion are an important part of the management of these patients. Restoring coronary blood flow and myocardial perfusion either by pharmacological (fibrinolytics) and/or mechanical therapy (PCI) has been demonstrated to improve outcomes in patients presenting within 12 h of symptom onset and later other patients group such as those with cardiogenic shock. There is evidence that prehospital fibrinolysis reduces delay to treatment, especially in rural areas with long transit times. In these settings prehospital fibrinolysis is a reasonable treatment strategy.

Out-of-hospital fibrinolytics for STEMI

Prehospital fibrinolytics for STEMI[ACS-018B]

In patients with STEMI in the prehospital setting, does the use of prehospital fibrinolytics, compared with in-hospital fibrinolytics, improve outcomes (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

Nineteen studies demonstrated significantly reduced time to treatment when fibrinolytics were given to patients with STEMI in the prehospital setting by either physicians, nurses, or paramedics (LOE 1[496,497,498,499–501] LOE 2[124,502–510]; LOE 3[511–513]).

Eleven studies showed that a greater proportion of the patients treated with prehospital fibrinolysis had shorter duration and increased frequency of total resolution of chest pain by the time of admission, ECG resolution, and decreased mortality (LOE 1[496,499,500,514–516]; LOE 2[505,506,508,511,513]).

Treatment recommendations

In patients with STEMI diagnosed in the prehospital setting, reperfusion may be achieved by administration of fibrinolytics by healthcare providers in the field. Alternately, fibrinolytic therapy may be administered on arrival at hospital. If fibrinolysis is chosen as the reperfusion strategy, it should be started as soon as possible, ideally in the prehospital setting, and should be administered by paramedics, nurses, or doctors using well-established protocols, competency training programs, and quality assurance programs, under medical oversight.

Choice of reperfusion strategy in the hospital

PPCI versus fibrinolytic therapy for STEMI[ACS-025B]

In patients with suspected STEMI in the ED setting, does the use of PPCI compared with fibrinolytic therapy, improve outcome (e.g., arrhythmias, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

PPCI is an effective reperfusion strategy. When provided in a timely manner, at a capable centre, by an experienced interventionalist, PPCI may be superior to fibrinolysis. Application of PPCI has been limited by access to catheter laboratory facilities and appropriately skilled clinicians. Fibrinolytic therapy is a widely available reperfusion strategy and may be used if delays to PPCI are anticipated. Both treatment strategies are well established and have been the subject of large randomised multicentre trials been over the last two decades.

Consensus on science

For patients admitted to hospital with PCI facilities, evidence from two studies demonstrated that PPCI conferred clinical benefit compared with fibrinolysis both in terms of mortality and morbidity (reinfarction/stroke) for the majority of patients (LOE 1).[517,518] The evidence from two studies was scant for additional benefit of PCI over fibrinolysis for specific subgroups such as post CABG patients or patients with renal failure (LOE 1[519]; LOE 3[520]).

For patients admitted to hospital without PCI facilities, two studies showed benefit associated with transferring patients for PPCI versus on-site fibrinolysis in terms of reinfarction and stroke and a trend to a lower mortality in the PPCI group (LOE 2).[521,522] The average time from randomization to PCI varied among the separate trials in this meta-analysis and ranged between 82 and 122 min. The benefit was correlated directly to risk status of the patient, with those at high risk benefiting more from transfer.

For patients with cardiogenic shock, evidence from one randomised trial demonstrated that early revascularization improves survival at 6 months (LOE 1).[523] The survival benefit was seen mainly in patients less than 75 years of age.

Data from registries[524] and a meta-analysis from previous published studies[525] highlight the variability in PCI-related time delay (between 40 and 179 min), that mitigated the benefit of mechanical intervention over fibrinolysis (LOE 3[524,525]). This variability was influenced by several factors including age, symptom duration, and location of infarction. Similarly one study showed that the benefit of PCI over fibrinolytic therapy is offset when PCI is carried out in low-volume PCI centres (LOE 1).[526]

Treatment recommendations

Programs should be implemented to reduce the time to PCI. Shorter intervals to reperfusion increase myocardial salvage, whereas delays to reperfusion increase morbidity and mortality. The precise threshold of PPCI-related delays that should trigger the decision for fibrinolysis has not been definitively established, but time to PCI should be as short as possible. Individual Councils will determine the acceptable limit or target interval from first medical contact to PCI in light of likely patient factors and available healthcare system resources, and the reader is referred to those Council-specific guidelines for more detailed information.

For patients presenting within 12 h of symptom onset and with ECG findings consistent with STEMI, reperfusion should be initiated as soon as possible independently of the method chosen. The benefit of mechanical intervention over fibrinolysis varies considerably depending on the patient's condition and the duration of PPCI-related delays. For those patients with a contraindication to fibrinolysis, PCI should still be pursued despite the delay, rather than offering no reperfusion therapy.

For those STEMI patients presenting in shock, PCI (or coronary artery bypass surgery) is the preferred reperfusion treatment. Fibrinolysis should only be considered if there is a substantial delay to PCI.

Combined PCI and fibrinolysis

Fibrinolytics and immediate PCI (facilitated PCI) versus immediate PCI[ACS-028A,ACS-028B]

In patients with suspected STEMI in the ED and prehospital settings, does the use of fibrinolytics and immediate PCI, compared with immediate PCI, improve outcome (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Fibrinolytics and PCI may be used in a variety of combinations to restore coronary blood flow and myocardial perfusion. There are several ways in which the two therapies can be combined. There is some lack of uniformity in the nomenclature used to describe these regimes. In this analysis, facilitated PCI is used to describe PCI performed immediately after fibrinolysis, a pharmaco-invasive strategy refers to PCI performed routinely 2–6 h after fibrinolysis, and rescue PCI is defined as PCI performed for a failed reperfusion (as evidenced by <50% resolution of ST-segment elevation at 60–90 min post-lytic). These strategies are distinct from a routine PCI approach where the angiography and intervention is performed more than 12 h after successful fibrinolysis.

Consensus on science

Twelve studies demonstrated poorer outcome with routine PCI shortly after fibrinolysis (LOE 1[481,527–532]; LOE 2[533]; LOE 5[534–537]). Most of these studies have been performed in recent years. Eleven studies supported a facilitated PCI strategy (LOE 1[538]; LOE 2;[464,539–541] LOE 3[542–544]; LOE 5[545–547]). Thirty studies show no benefit of PPCI over fibrinolysis (LOE 1[405,491,548–554]; LOE 2[555–560]; LOE 5[451,561–566,567–574]).

Treatment recommendations

The routine use of fibrinolysis-facilitated PPCI, compared with PPCI, is not recommended in patients with suspected STEMI. It is reasonable to perform angiography and possible PCI in patients with failed fibrinolysis according to clinical signs and/or insufficient ST-segment resolution.

Additional medical therapy

Several additional medical therapies have been proposed for ACS patients with the goal of reducing complications from myocardial ischaemia, major adverse cardiac events, and ultimately long-term survival. Therapeutic options include antiarrhythmics, β-blockers, angiotensin-converting enzyme (ACE) inhibitors, and HMG-CoA reductase inhibitors (statins). The bulk of data available to determine the usefulness of these therapies has not been derived from patients in the prehospital or ED settings. Traditional preventive interventions usually start with the first admission with a confirmed diagnosis of ACS. The current evidence indicates that none play a significant role in the out-of-hospital and ED management of ACS.

Antiarrhythmics

Prophylactic antiarrhythmics[ACS-021B]

In patients with suspected ACS/MI in prehospital and ED settings, does the use of prophylactic antiarrhythmics, compared with standard management (i.e., no prophylactic antiarrhythmics), improve outcome (e.g., arrhythmias, survival to discharge, 30/60 days mortality)?

Consensus on science

Evidence from three studies suggested a reduction in ventricular fibrillation (VF), which was not statistically significant; however, there was no improvement in survival to hospital discharge (LOE 1[575–577]; LOE 4[578]). The studies had heterogeneous clinical protocols, and most were underpowered. Twelve studies showed no improvement in suppression of ventricular arrhythmias (LOE 1[579–588]; LOE 2[589]; LOE 4[590]). The studies showed no improvement in survival to hospital discharge. Four studies showed worsening of arrhythmias and the potential for harm (LOE 1[584,591,592]; LOE 2[593]).

Lidocaine is the antiarrhythmic drug that has been studied most extensively in this clinical setting. The majority of the evidence suggests lidocaine is not associated with improved clinical outcomes. There were three studies supporting arrhythmia suppression with lidocaine; however, no clinical benefit was shown (LOE 1[575–577]; LOE 4[578]). There were eight studies that were neutral for demonstrating arrhythmia suppression with lidocaine (LOE 1[581,583,586–588]; LOE 2[589,593]; LOE 4.[590] There were two studies that showed harm (LOE 1).[580,592]

One trial showed a statistically significant benefit in decreasing the incidence of VT using sotalol (LOE 1).[594] Three studies were neutral with respect to tocainide and disopyramide (LOE 1),[582] mexilitine (LOE 1),[579] and tocainamide (LOE 1).[585] One study showed harm with amiodarone (LOE 1)[584] and one trial (LOE 1)[591] showed harm with a variety of drugs, including β-blockers.

Treatment recommendations

Prophylactic antiarrhythmics are not recommended for patients with suspected ACS or myocardial infarction.

β-Blockers [ACS-023A]

In patients with suspected ACS/MI in prehospital and ED settings, does the use of β-blockers, compared with standard management (i.e., no prehospital and ED use of β-blockers), improve outcome (e.g., arrhythmias, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Studies of β-blockers are heterogenous with respect to the time of β-blocker administration. There is a paucity of data on the administration of β-blockers in the prehospital or early ED settings (i.e., within the first hour of a suspected ACS).

Eight studies showed no advantage for IV β-blockers on mortality, infarct size, prevention of arrhythmias, or reinfarction (LOE 1).[595–602] None of the papers reviewed showed that β-blockers caused irreversible harm when given early in the development of suspected ACS. One study showed a statistically significant reduction in 6-week mortality in a subgroup of low-risk (i.e., Killip Class I) patients (LOE 1).[596] Other studies (LOE 1) have shown reduced mortality[603,604] and decreased infarct size[605,606,607] with early IV β-blocker use.

Four studies showed that early β-blocker administration helped prevent dangerous arrhythmias, (LOE 1)[604,606,608,609] while two studies showed a prevention of reinfarction but increased incidence of cardiogenic shock (LOE 1).[604,608] Many of the β-blocker trials in the early 1980 s were small and had wide confidence intervals. One study suggested that the earlier IV β-blockers were administered, the greater the reduction in infarct size and mortality (LOE 3).[610]

Treatment recommendations

For patients with ACS, there is no evidence to support the routine administration of IV β-blockers in the prehospital setting or during initial assessment in the ED. It may be reasonable to administer IV β-blockers in specific situations, such as severe hypertension or tachycardia, in patients without contraindications. Starting oral β-blockers at low doses is recommended once the patient's condition has been stabilised.

Angiotensin converting enzyme inhibitors

Angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs)[ACS-022A]

In patients with suspected ACS/MI in prehospital and ED settings, does the use of ACE inhibitors or ARBs, compared with standard management (i.e., no prehospital and ED use of ACE inhibitors), improve outcome (e.g., infarct size, survival to discharge, 30/60 days mortality)?

Consensus on science

Despite multiple studies that have shown a benefit for ACE inhibitors and ARBs in patients with a myocardial infarction, no trial specifically evaluated patients in the ED or prehospital settings. One randomised trial showed a reduction in mortality for patients treated with ACE inhibitors soon after presentation, despite causing some hypotension (LOE 1).[611] Three randomised trials showed a reduction in the rate of heart failure and mortality in patients treated soon after fibrinolysis (LOE 1).[612–614] One study (LOE 1)[613] failed to show a benefit with the use of ACE inhibitors within 1 h of reperfusion and two meta-analyses (LOE 1)[615,616] documented no benefit with ACE inhibitor administration.

Treatment recommendations

ACE inhibitors and ARBs reduce mortality in patients with AMI; however, there is insufficient evidence to support the routine initiation of ACE inhibitors and ARBs in the prehospital or ED setting in patients with a myocardial infarction.

HMG CoA reductase inhibitors (statins)

A and B statins[ACS-024B]

In patients with suspected ACS/MI in prehospital and ED settings, does the use of statins, compared with standard management (i.e., no prehospital and ED use of statins), improve outcome (e.g.,

infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

Nineteen studies documented a reduction in short- and long-term major adverse cardiovascular events after intensive treatment with statins within the first 24 h after hospital admission for patients with ACS (LOE 1[617–622]; LOE 2[623–635]). Multiple studies reported consistently reduced short-term mortality and reduced incidence of death and nonfatal myocardial infarction during the 30-day follow-up with continued statin treatment or early initiation of this treatment, compared with discontinuation of statins at hospital admission of ACS patients (LOE 3[636]; LOE 4[637–645]). Some of the studies also report the reduction in markers of myocardial necrosis or inflammation in statin treatment in patient groups undergoing PCI. One meta-analysis (LOE 1)[646] and two other studies (LOE 4)[647,648] were neutral with regard to death and nonfatal myocardial infarction during the 30-day follow-up. There were no reports on the risk or safety considerations of early initiation of statin treatment in ACS.

Treatment recommendations

Intensive statin treatment should be considered early after onset of an ACS event (e.g., immediately after hospital admission) in patients presenting with ACS unless contraindicated (e.g., by proven intolerance). Pre-existing statin therapy should be continued in patients presenting with an ACS.

Healthcare system interventions for ACS

Several systems-related strategies have been developed to improve quality of care for patients with ACS and reduce reperfusion delays for patients with STEMI. Strategies exist for patients identified in the prehospital setting and in the ED. These strategies focus on the use of prehospital 12-lead ECG and time-saving strategies to facilitate early diagnosis and rapid treatment for patients with STEMI.

12-Lead out-of-hospital ECG and advance ED notification

Prehospital ECGs[ACS-026B]

In patients with suspected ACS/MI in prehospital setting, does the use of prehospital ECG and advance ED notification, compared with no prehospital ECG, improve outcome (e.g., arrhythmias, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

Eight studies demonstrated a reduction in the door-to-needle time interval ranging from 20 to 60 min when physician- or paramedic-interpreted prehospital 12-lead ECG was used to evaluate patients with suspected AMI who are then treated with a fibrinolytic (LOE 1[649–652]; LOE 2[116,117,653,654]).

Eight studies demonstrated a reduction in the reperfusion delay (with varied time interval definitions) ranging from 15 to 65 min in patients treated with PCI (LOE 2[655–658]; LOE 3[112,659,660]; LOE 4[661]).

Two studies suggested that the time saved by using prehospital ECGs was dependent on advanced hospital notification of an incoming STEMI patient and activation of the catheterisation team before the patient's arrival (LOE 2).[655,660] When comparing the door-to-reperfusion time for patients with a prehospital ECG and prehospital activation to patients with no prehospital ECG, the mean door-to-reperfusion interval was reduced by more than 30 min.[660]

Two nonrandomised trials reported no significant reductions in mortality with the use of prehospital ECGs (LOE 2).[117,657] In one of these studies in-hospital all-cause mortality was 15.6% in a group of STEMI patients brought by EMS to the ED without prehospital ECGs, and 8.4% for patients who had a prehospital ECG and were brought directly to the critical care unit for fibrinolysis.[117] The study was not powered to detect a mortality difference. The second study reported an 11% in-hospital mortality for STEMI patients brought by EMS without a prehospital ECG versus 5% in those with a prehospital ECG.[657]

Treatment recommendations

Prehospital 12-lead ECGs facilitate earlier diagnosis of STEMI and provide the opportunity for rapid prehospital reperfusion or for rapid triage of patients to awaiting institutions able to provide such reperfusion. EMS personnel should acquire a prehospital 12-lead ECG on all patients exhibiting signs and symptoms of ACS and provide advance notification to receiving institutions for patients diagnosed with STEMI. Advance notification may be achieved with direct transmission of the ECG or with interpretation of the ECG by prehospital personnel. Advance notification should prompt preparations at the receiving institution for rapid reperfusion of the arriving STEMI patient.

Improving systems of care for ACS[ACS-009A]

In patients with suspected STEMI, do any specific techniques improve STEMI system or process of care, compared with standard management, to improve time to treatment and clinical outcome?

Consensus on science

Emergency physician or prehospital activation of the catheterisation laboratory team. Two studies suggested an association between the ability of emergency physicians to activate the catheterisation laboratory team and decreased door-to-ballon time interval (LOE 5).[662,663] Twelve studies demonstrated that emergency physician activation of the catheterisation laboratory was associated with significant reductions in door-to-balloon time intervals (20–68 min) (LOE 2[664–666]; LOE 3[667–673]; LOE 5[663,674]). False-positive activation rate in these studies ranged from 0% to 15%.[674,663–673]

Prehospital activation of the catheterisation laboratory. Seven studies demonstrated the effectiveness of prehospital activation on reducing door-to-balloon time intervals (22–69 min) (LOE 2[656,675]; LOE 3[676,677]; LOE 4[660,678]). The studies were variable in their implementation and all had significant limitations. False-positive activation of the catheterisation laboratory was not assessed by any of the studies.

Single call to a central page operator. One qualitative survey suggested an association between single call to a central page operator and reduced reperfusion delay (LOE 5).[679] There were no studies that investigated the effect of this specific technique in isolation.

Real-time data feedback. Four studies demonstrated a positive impact of feedback on reducing the door-to-balloon interval (10–54 min) (LOE 3[667,671]; LOE 5[679,680]). These studies were heterogeneous and had significant limitations.

Institutional commitment. Two qualitative studies suggested that senior management commitment and leadership was crucial to improving treatment of STEMI. However, no other studies proved this relationship (LOE 5).[681,682]

Team based approach. One qualitative study suggested a team-based approach led to improvements in STEMI systems of care (LOE 5).[681] However, no other studies proved this relationship empirically.

Expecting the catheterisation laboratory staff to arrive in 20 min. One study established an association between hospitals that expect the catheterisation team to arrive in 20 min and having decreased door-to-balloon time (LOE 5).[679] However, no studies have investigated the impact of implementing this specific technique in isolation. One study used this specific expectation of arrival of catheterisation laboratory staff along with other methods as part of a quality improvement initiative (LOE 3).[667] Another study evaluated the outcomes of patients that presented during peak hours compared with off-peak hours and found decreased door-to-balloon time intervals among patients who presented when the catheterisation laboratory team was in house (LOE 5).[683]

Having an interventional cardiologist immediately available at the hospital. One study demonstrated an association between having an interventional cardiologist always at the hospital and decreased door to balloon times of 8.2 min (LOE 5).[679] No studies have investigated the impact of implementing this specific technique on reperfusion delay. No studies demonstrated direct effect on mortality or other outcomes data.

Treatment recommendations

Hospitals should implement prehospital activation of the catheterisation laboratory for patients with suspected STEMI who arrive by EMS and should implement first-physician-contact activation of the catheterisation laboratory for patients suspected of having STEMI arriving by other means. Hospitals may implement additional institution-specific techniques to improve STEMI systems of care; however there is little evidence to support their widespread implementation. These techniques include:

- Arranging single-call activation of the catheterisation laboratory
- Requiring the catheterisation laboratory to be ready in 20 min
- Having the interventional cardiologist immediately available at the hospital
- Providing real-time data feedback
- Fostering the commitment of senior management
- Encouraging a team-based approach

Out-of-hospital triage for PCI[ACS-027A,ACS-027B]

In patients with ST-elevation identified on prehospital ECG, does the use of direct transport to PPCI, compared with transport to the closest hospital, improve outcomes (mortality, left ventricular function, re-infarction, or stroke) as compared with other standard strategies?

Consensus on science

Two studies suggested that transportation of STEMI patients diagnosed by paramedics directly to PCI centres for PPCI as part of a coordinated regional response to STEMI reduced in-hospital mortality when compared with historical controls with a strategy of transportation to the closest hospital for fibrinolysis (LOE 3[684]; LOE 5[685]).

Four studies failed to show that a strategy of prehospital diagnosis and direct transportation for PCI was any better than prehospital fibrinolysis followed by early PCI in patients with STEMI (in systems involving the presence of physicians in mobile intensive care units) in reducing the composite outcome of death, nonfatal reinfarction, and nonfatal stroke at 30 days (LOE 1[562,686,687]; LOE 4[555]).

Three studies suggested a benefit of prehospital fibrinolysis (when coupled with an early invasive strategy) over that of PCI for patients presenting early after the onset of chest pain (less than 2 h) and in certain clinical subsets (<65 years-of-age, anterior STEMI) in reduction of mortality (LOE 1; [688]LOE 4[525,689]).

Six studies comparing interfacility transfer for PPCI with on-site ED fibrinolysis in STEMI patients diagnosed in the ED demonstrated improved outcomes, including the triple end point of death, reinfarction, and stroke at 30 days; and outcomes for 30-day survival alone and reinfarction alone supported the strategy of direct transport for PPCI over fibrinolysis (LOE 5).[521,530,690–693]

Eleven studies demonstrated improved outcomes for patients diagnosed with STEMI in the prehospital setting and brought directly to PCI centres for PPCI compared with STEMI patients diagnosed in the ED of a non-PCI hospital who were transferred for PPCI (LOE 4[115,676,678,694–700]; LOE 5[685]). Clinical outcomes that were reported to improve with diversion for PPCI in this group included left ventricular function, in-hospital mortality, long-term mortality, and a triple end point of death, reinfarction, or stroke at 30 days.

Thirteen studies suggested equivalent outcomes between a strategy of transfer for PPCI and of fibrinolysis in the prehospital or hospital setting, particularly in patients presenting early after the onset of chest pain (<2 h) and in certain clinical subsets (<65 years-of-age, anterior STEMI) (LOE 2[657,677]; LOE 4[405,450,456,487,701–707]; LOE 5[525]).

Treatment recommendations

It is reasonable to consider direct transport to PCI capable facilities for PPCI for patients diagnosed with STEMI by EMS in the prehospital setting, bypassing closer EDs as necessary, in systems where time intervals between first medical contact and balloon time are brief. In patients presenting early after the onset of chest pain (<2 h) and in certain clinical subsets (<65 years-of-age, anterior STEMI), prehospital fibrinolysis may offer similar outcomes compared to PPCI.

PCI following ROSC[ACS-010A,ACS-010B]

In patients with ROSC after cardiac arrest, does the routine use of PCI, compared with standard management (without PCI), improve outcomes (e.g., survival, rearrest, etc)?

There is evidence of underlying ischaemic heart disease in the majority of patients who have an out-of-hospital cardiac arrest (OHCA). Acute coronary artery occlusion is known to be the precipitating factor in many of these patients. While coronary artery occlusion after cardiac arrest is associated with ECG ST-elevation or left bundle branch block (LBBB), it can also occur in the absence of these findings. Fibrinolysis in setting of OHCA is addressed in Part 8: "Advanced Life Support."

Consensus on science

One study suggested that cardiac angiography and PCI, when used as part of a standardised advanced post-cardiac arrest protocol, may be associated with improved survival to hospital discharge when compared with no standardised protocol (LOE 3).[708] Sixteen studies suggested that percutaneous intervention (PCI) was feasible following ROSC (LOE 3[708]; LOE 4[709–724]). These studies demonstrated that successful PCI versus no PCI may be associated with improved cardiac ejection fraction and survival,[724] and coronary angiography may be favorably associated with neurologically intact survival.[723] In most of the patients in these studies, immediate angiography and PPCI were performed.

Evidence from two studies suggested that outcomes after angiography and PCI vary considerably depending on patient-related factors (LOE 4).[709,711] The survival in patients who had

witnessed VF-arrests of short durations, STEMI, and recovery of consciousness was as high as 95% to 100%. One study showed that therapeutic hypothermia in combination with PPCI was feasible and safe in patients resuscitated after cardiac arrest (LOE 4).[725] One study compared PCI with fibrinolysis and demonstrated no difference in functional neurological recovery or survival at 6 months in patients with ROSC after cardiac arrest (LOE 4).[726]

Two additional retrospective case series (LOE 4[726,727]) compared outcomes of PCI in patients with and without cardiac arrest. One study compared 20 post-cardiac arrest patients who underwent PCI and mild hypothermia with a control group of 70 patients who underwent mild hypothermia without PCI. There was no difference in the rate of arrhythmias (the primary end point) or other adverse events between the two groups).[727] In the other retrospective study[728] of 948 STEMI patients without cardiogenic shock treated by PPCI, 20 were post-cardiac arrest. There was no difference in 1-month mortality between the non-arrest (cardiogenic shock) group and the post-cardiac arrest group, but non-cardiac mortality was higher in the post-cardiac arrest group.[728]

Recent publications provide additional information about the survival and functional outcome of patients who have PCI following ROSC after cardiac arrest. One retrospective series (LOE 4[729]) of 98 post-cardiac arrest patients who had ECG evidence of STEMI and underwent emergent angiography included 59 patients who were unresponsive. The survival rate to discharge (and proportion of these with full neurological recovery) was 64% (92%) overall and 44% (88%) among the initially unresponsive patients.[729] In a prospective observational registry (LOE 3[729]) of out-of-hospital cardiac arrest patients, (the Parisean Regional Out of hospital Cardiac Arrest Trial [PROCAT]), 435 patients had no obvious extracardiac cause and all underwent immediate coronary angiography, followed by PCI if indicated. At least one significant coronary artery lesion was found in 128 (96%) of 134 patients with STEMI on the ECG and in 176 (58%) of 301 patients without STEMI. In patients with a significant coronary lesion, PCI was successful in 99 of the 128 STEMI patients and in 78 of the 176 patients with other ECG patterns. Hospital survival was 40%. Multivariate analysis showed successful PCI to be an independent predictor of survival, regardless of the post-resuscitation ECG (odds ratio 2.06; 95% CI 1.16-3.66).[730]

Treatment recommendations

In OHCA patients with STEMI or new LBBB on ECG following ROSC, early angiography and PPCI should be considered. It is reasonable to perform early angiography and PPCI in selected patients despite the absence of ST-segment elevation on the ECG or prior clinical findings, such as chest pain, if coronary ischaemia is considered the likely cause on clinical grounds. Out-of-hospital cardiac arrest patient are often initially comatose but this should not be a contraindication to consider immediate angiography and PCI. It may be reasonable to include cardiac catheterisation in a standardised post-cardiac-arrest protocol as part of an overall strategy to improve neurologically intact survival in this patient group. Therapeutic hypothermia is recommended in combination with primary PCI, and should be started as early as possible, preferably before initiation of PCI.

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Appendix A. Writing Group Disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Robert E. O'Connor	University of Virginia Health System: Professor and Chair of Emergency Medicine	None	None	None	None	None	None
Leo Bossaert	University of Antwerp—Professor	None	None	None	None	None	None
Steven C. Brooks	University of Toronto—Assistant Professor; St. Michael's Hospital—Clinician-Scientist; Sunnybrook Health Sciences Centre—Emergency Physician and Clinician-Scientist	[a]$5000 CDN one time grant for the completion of a systematic review comparing direct transportation to a PCI centre versus transportation to the closest hospital for patients with STEMI diagnosed by EMS personnel in pre-hospital setting. Peer-reviewed grant awarded by the Canadian Association of Emergency Physicians	None	None	None	None	None
Gilson Feitosa-Filho	Hospital Aliança-Cardiologist; Escola Bahiana de Medicina e Saúde Pública-Professor; Hospital Santa Izabel—Santa Casa de Misericórdia da Bahia—Cardiologist	None	None	None	None	None	None
Jerry P. Nolan	Royal United Hospital NHS Trust: Consultant in Anaesthesia and Intensive Care Medicine; Editor-in-Chief *Resuscitation*	None	None	None	None	None	None
Michelle Welsford	Hamilton Health Sciences Medical Director, CPER	None	None	None	None	None	None
Karen Woolfrey	University of Toronto/Sunnybrook Health Science Centre Director, Royal College Emergency Medicine Program and Postgraduate Program Coordinator	None	None	None	None	None	None
Aaron Wong	National Heart Centre-Senior Consultant	None	None	None	None	None	None
Hans-Richard Arntz	Charite Medical University, Berlin, Germany—Consultant	[a]Sanofi-Aventis; [a]Boehringer	None	[a]Sanofi Aventis; [a]Boehringer; [a]Daiichi-Sankyo	None	[a]Boehringer	None
Deborah Diercks	University of California, Davis Medical Center—Professor	None	None	[a]Sanofi-Aventis; [a]Bristol Myers Squibb	None	[a]Sanofi-Aventis; [a]Bristol Myers Squibb; [a]Heartscape; [a]Schering Plough; [a]Beckman Coulter; [a]Nanosphere; [a]Astellas	None
Terry L. Vanden Hoek	The University of Chicago—Associate Professor	[a]Vanden Hoek, PII Depart.of Defense, Office of Naval Research "Proteomic Development of Molecular Vital Signs: Mapping a Mitochondrial Injury Severity Score to Triage and Guide Resuscitation of Haemorrhagic Shock" 9/6/04–4/31/10 $885,639 (current year) Research grant awarded to the University of Chicago	None	None	None	None	None

Appendix A. Writing Group Disclosures (*Continued*)

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Darren Walters	Queensland Health—Director of Cardiology	[a]Merck Sharp and Dohme; [a]CSL; [a]Medtronic	[a]Medtronic Biosensors; [a]Merck; [a]GSK; [a]AstraZeneca; [a]Johnson & Johnson; [a]Abbott Vascular; [a]Boston Scientific	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.
[†]Significant.
[a] Modest.

Appendix A. Worksheet Collaborator Disclosures

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
William J. Brady	University of Virginia, Charlottesville, VA—Professor & Vice Chair of Emerg. Med.	None	None	None	None	None	None
Teresa R. Camp-Rogers	VCU Healthsystem Emergency Medicine Residency Resident	None	None	None	None	None	None
Marc J. Claeys	University hospital—Professor-Physician	[a]National coordinator of PLATO trial (AstraZeneca)	None	None	None	[a]Member of advisory board Eli Lilly Benelux: advising marketing of prasugrel	None
Alan M. Craig	Toronto Emergency Medical Services: Municipal agency providing EMS Deputy EMS Chief	None	None	None	None	None	None
Russell Denman	Q Heatlh; Cardiologist	None	None	[a]Asia Pacific Heart Rhythm meeting Beijing 2009—$1000 plus travel expenses	None	None	None
Judith Finn	University of Western Australia—Professor	[b]Multiple National Health and Medical Research Grants (NH&MRC), National Heart Foundation Australia and State Government grants of >$10,000 since 1999. No money came to me—all came to my University to employ research staff and meet research expenses. No grants were directly related to any topic on which I am undertaking a Worksheet	None	[a]<$1000 from the Japanese Resuscitation Council to speak at the JRC Conference in Osaka in 2009	None	None	None
Chris Ghaemmaghami	University of Virginia—Associate Professor	None	None	None	None	None	None

Appendix A. Worksheet Collaborator Disclosures (*Continued*)

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Ian Jacobs	University of Western Australia: Academic (Teaching/ Research)—Professor; AHA: Evaluation of evidence worksheets for C2010—Work Sheet Expert	[b]Chief investigator on numerous grants awarded by: (a) National Health and Medical Research Council, (b) The Department of Health—Western Australia, (c) The National Heart Foundation of Australia. These funds are awarded to the University of Western Australia and none are used to provide any direct or indirect salary or other financial support.	[b]Funds are received into the Discipline of Emergency Medicine—University of Western Australia from the Ambulance Service—Western Australia and Laerdal (Australia) to maintain the Cardiac Arrest Registry for Western Australia. Our role is to independently maintain, analyse and report outcomes of CA in Western Australia. I oversee the operation of the registry and reporting of outcomes. These funds are not used in any way to provide any direct or indirect salary or other financial support	None	None	None	None
Michael C. Kurz	Virginia Commonwealth University Health System: Academic Hospital—Assistant Professor	[b]Co-Investigator for NIH RCI grant Health Information Prescriptions for ED/Observation Patients (HIP-HOP). ($20,000). Money comes to my institution. Grant is pending, slated for decision in September 2009. [a]Co-Investigator for NETT Hub at VCUHS ($3800). Money comes to institution.	None	[a]Zoll Medical Corporation, Honorarium for speaking at Zoll Data Systems 2009 National Summit. ($1000). Money came to me directly	None	None	[a]Expert for legal case involving AED usage. ($1400) Expert for legal case involving cardiac bio-markers ($200) Expert for legal case involving missed ED diagnosis of aortic dissection ($9000)
Dawn Yin Lim	Toronto Hospitals' Postgraduate Payroll Association Emergency Medicine Resident	None	None	None	None	None	None
Steve Lin	University of Toronto—Resident Physician	None	None	None	None	None	None
Venu Menon	Cleveland Clinic Hospital Director CCU	None	None	None	None	None	None
Patrick Meybohm	University Hospital Schleswig-Holstein, Campus Kiel, Germany: Medical doctor; Dept of Anesthesiology—Anesthetist	None	None	None	None	None	None

Appendix A. Worksheet Collaborator Disclosures (*Continued*)

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
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Dirk Mueller	Charité University Hospital Physician	None	None	None	None	None	None
Hiroshi Nonogi	National CV Center the Government Hosp. Japan; Director Division of Cardiology	1. Research grant (H19Shinkin-003) from the Ministry of Health, Labor and Welfare in Japan, to me directly. 2. Research grant for the Cardiovascular Diseases (19C-4) from the Ministry of Health, Labor and Welfare in Japan	None	None	None	None	None
Brian J. O'Neil	Self employed	[a]SanofiAventis, Bristol Myers Squibb	None	[b]Bristol Myers Squibb; [a]SanofiAventis, GlaxoSmithKline	None	None	None
Joseph P. Ornato	Virginia Commonwealth University: Academic health center-Prof/Chair of Emergency Medicine	None	None	[a]Grand Rounds hospital presentations—funded by educational grant from Bristol-Myers-Squibb Sanofi	None	None	None
Julian J. Owen	Hamilton Health Sciences—Emergency Medicine Resident Physician	None	None	None	None	None	None
Valeria Rac	St Michael's Hospital, University of Toronto Rescu, Keenan Research Centre, Li Ka Shing Knowledge Institute Postdoctoral Fellow	None	None	None	None	None	None
Hiromi Seo	Kochi Medical School Hospital—Professor	None	None	None	None	None	None
Kimberly A. Skelding	Geisinger Med. Center, Interventional Cardiologist	None	None	[a]Medtronics; [a]Society for Cardiovascular Angio & Interventions; [a]HMG Communications	None	None	None

Appendix A. Worksheet Collaborator Disclosures (*Continued*)

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Christian Spaulding	Assistance Publique Hôpitaux de Paris—Director, Cardiac Catheterisation Laboratory; Paris-Descartes University—Professor of Cardiology	None	None	[a]Cordis, Johnson & Johnson: participation to 7 workshops or sponsored symposiums in 2008 and 2009. Total amount paid: 6000 euros. Topic: drug eluting stents, no relationship with the guidelines Abbot Vascular: participation to four workshops or sponsored symposiums in 2008 and 2009. Total amount paid: 4000 euros. Topics: drug eluting stents, primary angioplasty, no relationship with the guidelines. The topic of my talk was on the safety of drug eluting stents. In 2009, I received 4224 euros from Lilly for 2 symposiums on acute MI and for a board on IIB IIIA inhibitors. The aim of this board was the future of reopro[a] in management of ACS. My talks were on the declining rate of AMI and the increasing rate of primary angioplasty compared to thrombolytic therapy in France.	None	[a]Cordis, Johnson & Johnson: advisory board on drug eluting stents. 3500 euros in 2008 and 3000 euros in 2009	[a]Member of an advisory board for the French government on coronary angioplasty and drug eluting stents, focused on the financing by the French Ministry of Health. Received 2500 euros in 2008 and 1500 euros in 2009
Nico R. Van de Veire	Leiden University Medical Center—Cardiologist	None	None	[a]Boston Scientific; -Medtronic; -GE Cardiac Ultrasound; -Philips Cardiac Ultrasound	None	[a]Biotronik advisory board	None
Hiroyuki Yokoyama	National Cardiovascular Center Cardiology	None	None	None	None	None	None

This table represents the relationships of worksheet collaborators that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all worksheet collaborators are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

Appendix B. Evidence-Based Worksheets for Part 9: Acute Coronary Syndromes: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations

Task force	WS ID	PICO title	Short title	Authors	URL
ACS	ACS-002	In patients with ACS (P) does the presence of any specific demographic factors (e.g. age, sex, race, weight) (I), compared with their absence (C), increase accuracy of prediction of delayed treatment (O)?	Demographic factors	Patrick Meybohm, Aaron Wong	http://circ.ahajournals.org/site/C2010/ACS-002.pdf
ACS	ACS-003B	In patients with suspected ACS (P), does dispatcher guided administration of aspirin by bystanders before arrival of EMS (I), compared with later administration of aspirin by paramedic or emergency department staff (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Timing of aspirin administration	Brian J. O'Neil	http://circ.ahajournals.org/site/C2010/ACS-003B.pdf
ACS	ACS-004B	In patients with suspected ACS (P), does the presence of any specific factors (e.g. history, examination, ECG, and/or biomarkers) or combination into a specific clinical decision rule (I), compared with standard care (C), increase accuracy of prediction of prognosis (e.g. decision rule for early discharge) (O)?	Prognosis for discharge vs. admission	William J. Brady, Dirk Mueller	http://circ.ahajournals.org/site/C2010/ACS-004B.pdf
ACS	ACS-005A	In patients with suspected ACS (P), does the use of chest pain observation units (I), compared with not using them (C), increase accuracy of to safely identify patients who require admission or specific management of CAD (O)?	Chest pain observation units	Chris Ghaemmaghami, Darren L. Walters	http://circ.ahajournals.org/site/C2010/ACS-005A.pdf
ACS	ACS-006-1A	In patients with suspected ACS (P), does the use of specific imaging techniques (e.g. CT angio/MRI/nuclear testing/ECHO) (I), compared with not using them (C), increase accuracy of diagnosis (e.g. of ACS) (O)?	Imaging techniques and diagnosis	Julian J. Owen, Karen Woolfrey	http://circ.ahajournals.org/site/C2010/ACS-006-1A.pdf
ACS	ACS-006-1B	In patients with suspected ACS (P), does the use of specific imaging techniques (e.g. CT angio/MRI/nuclear testing/ECHO) (I), compared with not using them (C), increase accuracy of diagnosis (e.g. of ACS) (O)?	Imaging techniques and diagnosis	Hiroshi Nonogi	http://circ.ahajournals.org/site/C2010/ACS-006-1B.pdf
ACS	ACS-006-2A	In patients with suspected ACS (P), does the use of specific imaging techniques (e.g. CT angio/MRI/nuclear testing/ECHO) (I), compared with not using them (C), improve outcome (e.g. size of infarct, LV function, survival) (O)?	Imaging techniques and outcome	Julian Owen, Karen Woolfrey	http://circ.ahajournals.org/site/C2010/ACS-006-2A.pdf
ACS	ACS-006-2B	In patients with suspected ACS (P), does the use of specific imaging techniques (e.g. CT angio/MRI/nuclear testing/ECHO) (I), compared with not using them (C), improve outcome (e.g. size of infarct, LV function, survival) (O)?	Imaging techniques and outcome	Hiroshi Nonogi	http://circ.ahajournals.org/site/C2010/ACS-006-2B.pdf
ACS	ACS-007B	In patients with suspected ACS in the prehospital, emergency department or in-hospital settings (P), can non-physicians (e.g. paramedics and nurses) (I) accurately diagnose STEMI (O), when compared to physicians (C)?	Diagnosis of STEMI by non-physicians	Alan Craig	http://circ.ahajournals.org/site/C2010/ACS-007B.pdf
ACS	ACS-008A	In patients with suspected ACS (P), does the use of computer-assisted ECG interpretation (I), compared with standard diagnostic techniques (emergency physicians) (C), increase accuracy of diagnosis (e.g. of NSTEMI/STEMI) (O)?	Computer-assisted ECG interpretation	Judith Finn	http://circ.ahajournals.org/site/C2010/ACS-008A.pdf

Appendix B (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ACS	ACS-009A	In patients with suspected ACS (P), do any specific techniques (I), improve ACS/MI system or process of care compared with standard management (C), to improve time to treatment and clinical outcome (O)?	Improving systems of care for ACS	Teresa R. Camp-Rogers, Michael C. Kurz	http://circ.ahajournals.org/site/C2010/ACS-009A.pdf
ACS	ACS-010A	In patients with ROSC after cardiac arrest (P), does the routine use of PCI (I), compared with standard management (without PCI) (C), improve outcomes (e.g. TBD survival/re-arrest/etc) (O)?	PCI following ROSC	Terry Vanden Hoek	http://circ.ahajournals.org/site/C2010/ACS-010A.pdf
ACS	ACS-010B	In patients with ROSC after cardiac arrest (P), does the routine use of PCI (I), compared with standard management (without PCI) (C), improve outcomes (e.g. TBD survival/re-arrest/etc) (O)?	PCI following ROSC	Darren L. Walters	http://circ.ahajournals.org/site/C2010/ACS-010B.pdf
ACS	ACS-011	In patients with suspected ACS in various settings (e.g. prehospital, emergency or in-hospital) (P), do specific historical factors, physical examination findings and test results (I), compared with normal (C), increase the accuracy of diagnosis ACS and MI (O)?	Accuracy history and PE for diagnosing ACS and MI	Hans-Richard Arntz, Peter Morley, Darren L. Walters	http://circ.ahajournals.org/site/C2010/ACS-011.pdf
ACS	ACS-013B	In patients with suspected ACS in various settings (e.g. prehospital, emergency or in-hospital) (P), do abnormal protein markers, compared with normal levels (C) allow the clinician to accurately diagnose acute coronary ischaemia? (O)?	Protein makers of coronary ischaemia	Steve Lin, Hiroyuki Yokoyama	http://circ.ahajournals.org/site/C2010/ACS-013B.pdf
ACS	ACS-014	In patients with suspected ACS in various settings (e.g. prehospital or emergency) (P), does the use of prehospital or emergency 12 lead ECG (I), compared with other diagnostic techniques (C), increase sensitivity and specificity of diagnosis of ACS/MI (O)?	12 lead ECG	Marc J. Claeys, Dirk Mueller	http://circ.ahajournals.org/site/C2010/ACS-014.pdf
ACS	ACS-015	In patients with suspected ACS in various settings (e.g. prehospital, emergency or in-hospital) and normal oxygen saturations (P), does the use of supplemental oxygen (I), compared with room air (C), improve outcomes (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Supplemental oxygen	Kimberly A. Skelding, Nico R. Van de Veire	http://circ.ahajournals.org/site/C2010/ACS-015.pdf
ACS	ACS-017-1	In patients with suspected St-elevation myocardial infarction in the prehospital and emergency department setting (P) treated with fibrinolysis, does the use of new anticoagulants, i.e. pentasaccharide, enoxaparin, bivalirudin (I), compared with standard management (unfractionated heparin) (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Anticoagulants and STEMI	Hans-Richard Arntz, Michelle Welsford	http://circ.ahajournals.org/site/C2010/ACS-017-1.pdf
ACS	ACS-017-2	In patients with suspected St-elevation myocardial infarction in the prehospital and emergency department setting (P) to be treated with primary PCI, does the use of new anticoagulants, i.e. pentasaccharide, enoxaparin, bivalirudin (I), compared with standard management (unfractionated heparin) (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Anticoagulants plus PCI	Hans-Richard Arntz, Michelle Welsford	http://circ.ahajournals.org/site/C2010/ACS-017-2.pdf

Appendix B (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ACS	ACS-017-3	In patients with suspected non-St-elevation ACS in prehospital and emergency department settings (P), does the use of new anticoagulants, i.e. pentasaccharide, enoxaparin, bivalirudin (I), compared with standard management (unfractionated heparin or other anticoagulant) (C), improve outcome (e.g. mortality, reinfarction, bleeding) (O)?	Anticoagulants and non-ST-elevation ACS	Hans-Richard Arntz, Michelle Welsford	http://circ.ahajournals.org/site/C2010/ACS-017-3.pdf
ACS	ACS-018B	In patients with STEMI in the prehospital setting (P), does the use of prehospital fibrinolytics (I), compared with inhospital fibrinolytics (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Prehospital fibrinolytics for STEMI	Dirk Mueller, Valeria Rac	http://circ.ahajournals.org/site/C2010/ACS-018B.pdf
ACS	ACS-019A	In patients with non-ST elevation ACS/STEMI and fibrinolysis/suspected STEMI and PCI in prehospital and emergency department settings (P), does the use of clopidogrel (I) compared with standard management (i.e. no prehospital or ED use of clopidogrel) (C) or new tienopyridines, prasugrel) (I) compared to clopidogrel (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Clopidogrel (and similar drugs) and non-ST elevation ACS	Michelle Welsford	http://circ.ahajournals.org/site/C2010/ACS-019A.pdf
ACS	ACS-019B	In patients with non-ST elevation ACS/STEMI and fibrinolysis/suspected STEMI and PCI in prehospital and emergency department settings (P), does the use of clopidogrel (I) compared with standard management (i.e. no prehospital or ED use of clopidogrel) (C) or new tienopyridines, prasugrel) (I) compared to clopidogrel (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Clopidogrel (and similar drugs) and non-ST elevation ACS	Ian Jacobs, Christian Spaulding	http://circ.ahajournals.org/site/C2010/ACS-019B.pdf
ACS	ACS-020	In patients with suspected ACS/MI in prehospital and emergency department settings (P), does the use of IIB IIIA Inhibitors (I), compared with standard management (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	IIB IIIA inhibitors	Hans-Richard Arntz, Venu Menon	http://circ.ahajournals.org/site/C2010/ACS-020.pdf
ACS	ACS-021A	In patients with suspected ACS/MI in prehospital and emergency department settings (P), does the use of Prophylactic Antiarrhythmics (I), compared with standard management (i.e. no Prophylactic Antiarrhythmics) (C), improve outcome (e.g. arrhythmias, survival to discharge, 30/60 d mortality) (O)?	Prophylactic Antiarrhythmics	Joseph P. Ornato, Peter T. Morley	http://circ.ahajournals.org/site/C2010/ACS-021A.pdf
ACS	ACS-021B	In patients with suspected ACS/MI in prehospital and emergency department settings (P), does the use of Prophylactic Antiarrhythmics (I), compared with standard management (i.e. no Prophylactic Antiarrhythmics) (C), improve outcome (e.g. arrhythmias, survival to discharge, 30/60 d mortality) (O)?	Prophylactic Antiarrhythmics	Russell Denman	http://circ.ahajournals.org/site/C2010/ACS-021B.pdf

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Task force	WS ID	PICO title	Short title	Authors	URL
ACS	ACS-022A	In patients with suspected ACS/MI in prehospital and emergency department settings (P), does the use of ACE inhibitors (I), compared with standard management (i.e. no prehospital and emergency department use of ACE inhibitors) (C), improve outcome (e.g. infarct size, survival to discharge, 30/60 d mortality) (O)?	ACE inhibitors	Deborah Diercks	http://circ.ahajournals.org/site/C2010/ACS-022A.pdf
ACS	ACS-023A	In patients with suspected ACS/MI in prehospital and emergency department settings (P), does the use of beta-blockers (I), compared with standard management (i.e. no prehospital and emergency department use of beta-blockers) (C), improve outcome (e.g. arrhythmias, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Beta-blockers	Gilson Feitosa Filho, Dawn Yin Lim	http://circ.ahajournals.org/site/C2010/ACS-023A.pdf
ACS	ACS-024B	In patients with suspected ACS/MI in prehospital and emergency department settings (P), does the use of statins (I), compared with standard management (i.e. no prehospital and emergency department use of statins) (C), improve outcome (e.g. infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Statins	Hans-Richard Arntz, Gilson Feitosa Filho	http://circ.ahajournals.org/site/C2010/ACS-024B.pdf
ACS	ACS-025B	In patients with suspected STEMI in the emergency department setting (P), does the use of PTCA (I), compared with fibrinolytic therapy (C), improve outcome (e.g. arrhythmias, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	PTCA vs. fibrinolytic therapy for STEMI	Marc J. Claeys, Michael C. Kurz	http://circ.ahajournals.org/site/C2010/ACS-025B.pdf
ACS	ACS-026B	In patients with suspected ACS/MI in prehospital setting (P), does the use of prehospital ECG and advance ED notification (I), compared with no prehospital ECG (C), improve outcome (e.g. arrhythmias, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Prehospital ECGs	Steven C. Brooks, Michael C. Kurz	http://circ.ahajournals.org/site/C2010/ACS-026B.pdf
ACS	ACS-027A	In patients with suspected STEMI in the prehospital setting (P), does the use of direct transport to a centre for PTCA (I), compared with transportation to the closest hospital with any other reperfusion strategy (prehospital fibrinolysis, inhospital fibrinolysis, interhospital transfer for PTCA) (C) improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 mortality) (O)?	PTCA centres closest hospital	Steven C. Brooks	http://circ.ahajournals.org/site/C2010/ACS-027A.pdf
ACS	ACS-027B	In patients with suspected STEMI in the prehospital setting (P), does the use of direct transport to a centre for PTCA (I), compared with transportation to the closest hospital with any other reperfusion strategy (prehospital fibrinolysis, inhospital fibrinolysis, interhospital transfer for PTCA) (C) improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 mortality) (O)?	PTCA centres closest hospital	Darren L. Walters	http://circ.ahajournals.org/site/C2010/ACS-027B.pdf

Appendix B (*Continued*)

Task force	WS ID	PICO title	Short title	Authors	URL
ACS	ACS-028A	In patients with suspected STEMI in the ED and prehospital settings (P), does the use of fibrinolytics and immediate PTCA (I), compared with immediate PTCA (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Fibrinolytics and immediate PTCA vs. immediate PTCA	Hans-Richard Arntz	http://circ.ahajournals.org/site/C2010/ACS-028A.pdf
ACS	ACS-028B	In patients with suspected STEMI in the ED and prehospital settings (P), does the use of fibrinolytics and immediate PTCA (I), compared with immediate PTCA (C), improve outcome (e.g. chest pain resolution, infarct size, ekg resolution, survival to discharge, 30/60 d mortality) (O)?	Fibrinolytics and immediate PTCA vs. immediate PTCA	Hiromi Seo	http://circ.ahajournals.org/site/C2010/ACS-028B.pdf
ACS	ACS-030A-1	In patients with suspected ACS/STEMI in the ED and prehospital settings (P), does the use of nitroglycerin (I), compared with no nitroglycerin (C), improve diagnosis of ACS/MI (O)? (diagnosis)	ACS and nitroglycerin (diagnosis)	Deborah Diercks	http://circ.ahajournals.org/site/C2010/ACS-030A-1.pdf
ACS	ACS-030A-2	In patients with suspected ACS/STEMI in the ED and prehospital settings (P), does the use of nitroglycerin (I), compared with no nitroglycerin (C), improve diagnosis of ACS/MI (O)? (treatment)	ACS and nitroglycerin (treatment)	Deborah Diercks	http://circ.ahajournals.org/site/C2010/ACS-030A-2.pdf

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Part 10: Paediatric basic and advanced life support 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations☆

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The 2010 ILCOR Paediatric Task Force experts developed 55 questions related to paediatric resuscitation. Topics were selected based on the 2005 Consensus on Science and Treatment Recommendations (CoSTR) document,[1,2] emerging science, and newly identified issues. Not every topic reviewed for the 2005 International Consensus on Science was reviewed in the 2010 evidence evaluation process. In general, evidence-based worksheets were assigned to at least two authors for each topic. The literature search strategy was first reviewed by a "worksheet expert" for completeness. The expert also approved the final worksheet to ensure that the levels of evidence were correctly assigned according to the established criteria. Worksheet authors were requested to draft CoSTR statements (see Part 3: Evidence Evaluation Process). Each worksheet author or pair of authors presented their topic to the Task Force in person or via a webinar conference, and Task Force members discussed the available science and revised the CoSTR draft accordingly. These draft CoSTR summaries were recirculated to the International Liaison Committee on Resuscitation (ILCOR) Paediatric Task Force for further refinement until consensus was reached. Selected controversial and critical topics were presented at the 2010 ILCOR International Evidence Evaluation conference in Dallas, Texas, for further discussion to obtain additional input and feedback. This document presents the 2010 international consensus on the science, treatment, and knowledge gaps for each paediatric question.

The most important changes or points of emphasis in the recommendations for paediatric resuscitation since the publication of the *2005 ILCOR International Consensus on CPR and ECC Science with Treatment Recommendations*[1,2] are summarised in the following list. The scientific evidence supporting these changes is detailed in this document.

- Additional evidence shows that healthcare providers do not reliably determine the presence or absence of a pulse in infants or children.
- New evidence documents the important role of ventilations in CPR for infants and children. However, rescuers who are unable or unwilling to provide ventilations should be encouraged to perform compression-only CPR.
- To achieve effective chest compressions, rescuers should compress at least one-third the anterior–posterior dimension of the chest. This corresponds to approximately 4 cm (1.5 in.) in most infants and 5 cm (2 in.) in most children.
- When shocks are indicated for ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) in infants and children, an initial energy dose of 2–4 J kg^{-1} is reasonable; doses higher than 4 J kg^{-1}, especially if delivered with a biphasic defibrillator, may be safe and effective.
- More data support the safety and effectiveness of cuffed tracheal tubes in infants and young children, and the formula for selecting the appropriately sized cuffed tube was updated.
- The safety and value of using cricoid pressure during emergency intubation are not clear. Therefore, the application of cricoid pressure should be modified or discontinued if it impedes ventilation or the speed or ease of intubation.
- Monitoring capnography/capnometry is recommended to confirm proper tracheal tube position.
- Monitoring capnography/capnometry may be helpful during CPR to help assess and optimise quality of chest compressions.
- On the basis of increasing evidence of potential harm from exposure to high-concentration oxygen after cardiac arrest, once

☆ *Note from the writing group*: Throughout this article, the reader will notice combinations of superscripted letters and numbers (e.g., "Family Presence During Resuscitation[Peds-003*]"). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.

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spontaneous circulation is restored, inspired oxygen concentration should be titrated to limit the risk of hyperoxaemia.
- Use of a rapid response system in a paediatric inpatient setting may be beneficial to reduce rates of cardiac and respiratory arrest and in-hospital mortality.
- Use of a bundled approach to management of paediatric septic shock is recommended.
- The young victim of a sudden, unexpected cardiac arrest should have an unrestricted, complete autopsy, if possible, with special attention to the possibility of an underlying condition that predisposes to a fatal arrhythmia. Appropriate preservation and genetic analysis of tissue should be considered; detailed testing may reveal an inherited "channelopathy" that may also be present in surviving family members.

Systems

Medical emergency teams (METs) or rapid response teams (RRTs) have been shown to be effective in preventing respiratory and cardiac arrests in selected paediatric inpatient settings.

Family presence during resuscitations has been shown to be beneficial for the grieving process and in general was not found to be disruptive. Thus, family presence is supported if it does not interfere with the resuscitative effort.

Medical emergency or rapid response team[Peds-025A, Peds-025B]

Consensus on science

The introduction of METs or RRTs was associated with a decrease in paediatric hospital mortality in one LOE 3 meta-analysis[3] and three paediatric LOE 3 studies with historical controls.[4–6] The introduction of a MET or RRT was associated with

- a decrease in respiratory but not cardiac arrest in one LOE 3[7] study with historical controls;
- a decrease in preventable total number of arrests in one LOE 3 study compared with a retrospective chart review[8];
- a decrease in total number of arrests in two LOE 3[4,8] studies;
- a decrease in preventable cardiac arrests in one LOE 3[6] study and
- a decrease in cardiac arrest and non-paediatric intensive care unit (PICU) mortality in one LOE 3[9] paediatric cohort study using historical controls.

Treatment recommendations

Paediatric RRT or MET systems may be beneficial to reduce the risk of respiratory and/or cardiac arrest in hospitalised paediatric patients outside an intensively monitored environment.

Knowledge gaps

Is it the team or the staff education associated with MET or RRT implementation that leads to improved patient outcomes? Is the team effectiveness due to validated team activation criteria or specific team composition? Do the benefits attributed to these teams extend to children in a community hospital setting?

Family presence during resuscitation[Peds-003]

Consensus on science

Ten studies (LOE 2[10]; LOE 3[11]; LOE 4[12–19]) documented that parents wish to be given the option of being present during the resuscitation of their children. One LOE 2,[10] one LOE 3,[11] two LOE 4,[13,19] and one LOE 5[20] studies confirmed that most parents would recommend parent presence during resuscitation to others.

One LOE 2,[10] one LOE 3,[11] six LOE 4,[12,14,19,21–23] and two LOE 5[20,24] studies of relatives present during the resuscitation of a family member reported that they believed their presence was beneficial to the patient.

One LOE 2,[10] one LOE 3,[11] six LOE 4,[12,13,16–19] and one LOE 5[24] studies reported that most relatives present during the resuscitation of a family member benefited from the experience. One LOE 3,[11] four LOE 4,[12,13,20,21] and two LOE 5[24,25] studies reported that being present during the resuscitation helped their adjustment to the family member's death.

One LOE 2[10] and two LOE 4[12,13] studies observed that allowing family members to be present during a resuscitation in a hospital setting did them no harm, whereas one LOE 4[26] study suggested that some relatives present for the resuscitation of a family member experienced short-term emotional difficulty.

One LOE 2,[10] one LOE 3,[27] three LOE 4,[12,23,28] and three LOE 5[20,24,29] studies showed that family presence during resuscitation was not perceived as being stressful to staff or to have negatively affected staff performance. However, one survey (LOE 4[30]) found that 39–66% of emergency medical services (EMS) providers reported feeling threatened by family members during an out-of-hospital resuscitation and that family presence interfered with their ability to perform resuscitations.

Treatment recommendations

In general, family members should be offered the opportunity to be present during the resuscitation of an infant or child. When deciding whether to allow family members to be present during an out-of-hospital resuscitation, the potential negative impact on EMS provider performance must be considered.

Knowledge gaps

How does the presence of a dedicated support person help family members and, potentially, healthcare providers during the resuscitation of an infant or child? What training is appropriate for staff who may serve as support persons for family members during resuscitation of an infant or child? Why is family presence during resuscitation perceived more negatively by out-of-hospital care providers than by in-hospital staff?

Assessment

Many healthcare providers find it difficult to rapidly and accurately determine the presence or absence of a pulse. On the basis of available evidence, the Task Force decided to deemphasise but not eliminate the pulse check as part of the healthcare provider assessment. The Task Force members recognised that healthcare providers who work in specialised settings may have enhanced skills in accurate and rapid pulse checks, although this has not been studied.

There are considerable data regarding use of end-tidal carbon dioxide ($P_{ET}CO_2$) measurement, capnography and capnometry, during cardiopulmonary resuscitation (CPR) as an indicator of CPR quality and as a predictive measure of outcome. Although capnography/capnometry may reflect the quality of CPR, there is insufficient evidence of its reliability in predicting resuscitation success in infants and children.

Pulse check versus check for signs of life[Peds-002A]

Consensus on science

Thirteen LOE 5 studies[31–43] observed that neither laypersons nor healthcare providers are able to perform an accurate pulse check in healthy adults or infants within 10 s. In two LOE 5 studies in adults[44,45] and two LOE 3 studies in children with nonpulsatile circulation,[46,47] blinded healthcare providers commonly assessed pulse status inaccurately and their assessment often took >10 s.

In the paediatric studies, healthcare professionals were able to accurately detect a pulse by palpation only 80% of the time. They mistakenly perceived a pulse when it was nonexistent 14–24% of the time and failed to detect a pulse when present in 21–36% of the assessments. The average time to detect an actual pulse was approximately 15 s, whereas the average time to confirm the absence of a pulse was 30 s. Because the pulseless patients were receiving extracorporeal membrane oxygenation (ECMO) support, one must be cautious in extrapolating these data to the arrest situation; all pulseless patients did have perfusion and therefore had signs of circulation as evidenced by warm skin temperature with brisk capillary refill. All patients evaluated were in an intensive care unit (ICU) setting without ongoing CPR.

Treatment recommendations

Palpation of a pulse (or its absence) is not reliable as the sole determinant of cardiac arrest and need for chest compressions. If the victim is unresponsive, not breathing normally, and there are no signs of life, lay rescuers should begin CPR. In infants and children with no signs of life, healthcare providers should begin CPR unless they can definitely palpate a pulse within 10 s.

Knowledge gaps

Is there an association between the time required to successfully detect a suspected cardiac arrest victim's pulse and resuscitation outcome? Is there a difference in outcome when the decision to start chest compressions is based on the absence of signs of life as opposed to absence of a pulse?

Focused echocardiogram to detect reversible causes of cardiac arrest[Peds-006B]

Consensus on science

In one small LOE 4 paediatric case series[48] cardiac activity was rapidly visualised by echocardiography without prolonged interruption of chest compressions, and this cardiac activity correlated with the presence or absence of a central pulse. In one paediatric LOE 4 case report,[49] echocardiography was useful for diagnosing pericardial tamponade as the cause of cardiac arrest and was useful in guiding treatment.

In eight LOE 5 adult case series,[50–57] echocardiographic findings correlated well with the presence or absence of cardiac activity in cardiac arrest. These reports also suggested that echocardiography may be useful in identifying patients with potentially reversible causes for the arrest.

Treatment recommendations

There is insufficient evidence to recommend for or against the routine use of echocardiography during paediatric cardiac arrest. Echocardiography may be considered to identify potentially treatable causes of an arrest when appropriately skilled personnel are available, but the benefits must be carefully weighed against the known deleterious consequences of interrupting chest compressions.

Knowledge gaps

Can echocardiography be performed during cardiac arrest in infants and children without significant interruptions in chest compressions? How often does echocardiography during cardiac arrest provide information that can affect treatment and outcome?

End-tidal CO_2 (P_{ETCO_2}) and quality of CPR[Peds-005A, Peds-005B]

Consensus on science

Three LOE 5 animal studies,[58–60] four LOE 5 adult,[61–64] and one LOE 5 paediatric series[65] showed a strong correlation between P_{ETCO_2} and interventions that increase cardiac output during resuscitation from shock or cardiac arrest. Similarly three LOE 5 animal models[66–68] showed that measures that markedly reduce cardiac output result in a fall in P_{ETCO_2}.

Two LOE 5 adult out-of-hospital studies[69,70] supported continuous P_{ETCO_2} monitoring during CPR as a way of determining return of spontaneous circulation (ROSC), particularly if the readings during CPR are ≥15 mm Hg (2.0 kPa). In one LOE 4[71] and two LOE 5 adult case series,[72,73] an abrupt and sustained rise in P_{ETCO_2} often preceded identification of ROSC.

Two LOE 4 paediatric cases series,[65,74] eight LOE 5 adult,[70,75–81] and one LOE 5 animal study[59] showed that a low P_{ETCO_2} (≤10 mm Hg [1.33 kPa] to ≤15 mm Hg [2.0 kPa]) despite 15–20 min of advanced life support (ALS) is strongly associated with failure to achieve ROSC. On the basis of two LOE 5 animal studies[71,82] and two adult LOE 5 case series,[70,78] P_{ETCO_2} after at least 1 min of CPR may be more predictive of outcome than the initial value because the initial P_{ETCO_2} is often increased in patients with asphyxial cardiac arrest.

The wide variation for initial P_{ETCO_2} during resuscitation limits its reliability in predicting outcome of resuscitation and its value as a guide to limiting resuscitation efforts. Two LOE 5 animal studies[71,82] and two large LOE 5 adult trials[70,78] suggested that the initial P_{ETCO_2} concentration is higher if the aetiology of the cardiac arrest is asphyxial rather than if it is a primary cardiac arrest.

Interpretation of the end-tidal CO_2 concentration during resuscitation is affected by the quality of the measurement, the minute ventilation delivered during resuscitation, the presence of lung disease that increases anatomic dead space, and the presence of right-to-left shunting.[83–85]

In one LOE 5 adult study,[86] sodium bicarbonate transiently increased end-tidal CO_2, and in three LOE 5 adult[87–89] and two LOE 5 animal[90,91] studies, adrenaline (and other systemic vasoconstrictive agents) transiently decreased P_{ETCO_2}.

Treatment recommendations

Continuous capnography or capnometry monitoring, if available, may be beneficial by providing feedback on the effectiveness of chest compressions. Whereas a specific target number cannot be identified, if the P_{ETCO_2} is consistently ≤15 mm Hg, it is reasonable to focus efforts on improving the quality of chest compressions and avoiding excessive ventilation.

Although a threshold P_{ETCO_2} may predict a poor outcome from resuscitation and might be useful as a guide to termination of CPR, there are insufficient data to establish the threshold and the appropriate duration of ALS needed before such evaluation in children. The P_{ETCO_2} must be interpreted with caution for 1–2 min after administration of adrenaline or other vasoconstrictive medications because these medications may decrease the P_{ETCO_2}.

Knowledge gaps

Does P_{ETCO_2} monitoring during CPR improve quality of chest compressions and/or outcome of paediatric resuscitation? During CPR, can P_{ETCO_2} be reliably measured via a laryngeal mask airway (LMA)? Is there a threshold concentration of P_{ETCO_2} that predicts ROSC or low likelihood of ROSC during resuscitation from paediatric cardiac arrest? Can the initial P_{ETCO_2} distinguish asphyxial from cardiac aetiology of paediatric cardiac arrest? Is detection of ROSC using P_{ETCO_2} monitoring more accurate than palpation of a pulse? Are P_{ETCO_2} targets during CPR different for subgroups of infants and children with alterations in pulmonary blood flow or high airway resistance?

Airway and ventilation

Opening and maintaining a patent airway and providing ventilations are fundamental elements of paediatric CPR, especially

because cardiac arrest often results from, or is complicated by, asphyxia. There are no new data to change the 2005 ILCOR recommendation to use manual airway maneuvers (with or without an oropharyngeal airway) and bag-mask ventilation (BMV) for children requiring airway control or positive-pressure ventilation for short periods in the out-of-hospital setting. When airway control or BMV is not effective, supraglottic airways may be helpful when used by properly trained personnel.

When performing tracheal intubation, data suggest that the routine use of cricoid pressure may not protect against aspiration and may make intubation more difficult.

Routine confirmation of tracheal tube position with capnography/capnometry is recommended with the caveat that the P_{ETCO_2} in infants and children in cardiac arrest may be below detection limits for colorimetric devices.

Following ROSC, toxic oxygen byproducts (reactive oxygen species, free radicals) are produced that may damage cell membranes, proteins, and DNA (reperfusion injury). Although there are no clinical studies in children (outside the newborn period) comparing different concentrations of inspired oxygen during and immediately after resuscitation, animal data and data from newborn resuscitation studies suggest that it is prudent to titrate inspired oxygen after return of a perfusing rhythm to prevent hyperoxaemia.

Supplementary oxygen[Peds-015]

Consensus on science

There are no studies comparing ventilation of infants and children in cardiac arrest with different inspired oxygen concentrations. Two LOE 5 meta-analyses of several randomised controlled trials comparing neonatal resuscitation initiated with room air versus 100% oxygen[92,93] showed increased survival when resuscitation was initiated with room air.

Seven LOE 5 animal studies[94–100] suggested that ventilation with room air or an F_{IO_2} of $\leq$1.0 during cardiac arrest may be associated with less neurological deficit than ventilation with an F_{IO_2} of 1.0, whereas one LOE 5 animal study[101] showed no difference in outcome. In five LOE 5 animal studies[95,97–99,102] ventilation with 100% oxygen during and following resuscitation contributed to free radical-mediated reperfusion injury to the brain.

Treatment recommendations

There is insufficient evidence to recommend any specific inspired oxygen concentration for ventilation *during* resuscitation from cardiac arrest in infants and children. Once circulation is restored, it is reasonable to titrate inspired oxygen to limit hyperoxaemia.

Knowledge gaps

Does the use of any specific concentration of supplementary oxygen during resuscitation from cardiac arrest in infants and children improve or worsen outcome? What is the appropriate target oxygen saturation for the paediatric patient after achieving ROSC?

Cuffed versus uncuffed tracheal tube[Peds-007]

Consensus on science

There are no studies that compare the safety and efficacy of cuffed versus uncuffed tubes in infants and children who require emergency intubation.

Two LOE 5 randomised controlled studies[103,104] and one LOE 5 cohort-controlled study[105] in a paediatric anaesthesia setting showed that the use of cuffed tracheal tubes was associated with a higher likelihood of selecting the correct tracheal tube size (and hence a lower reintubation rate) with no increased risk of perioperative or airway complications. Cuff pressures in these three studies were maintained at <20–25 cm H_2O. Two perioperative LOE 5 cohort-controlled paediatric studies[105,106] similarly showed that cuffed tubes were not associated with an increased risk of perioperative airway complications.

One LOE 5 paediatric case series[107] observed that the use of cuffed tracheal tubes was not a risk factor for developing subglottic stenosis in patients having corrective surgery for congenital cardiac defects. In the intensive care setting, two LOE 5 prospective cohort-controlled studies[108,109] and one LOE 5 retrospective cohort-controlled study[110] documented no greater risk of complications for children <8 years of age who were intubated with cuffed compared with uncuffed tracheal tubes.

One small LOE 5 case-controlled study[111] showed that cuffed tracheal tubes decreased the incidence of aspiration in the PICU, and one LOE 5 case series[105] of children with burns undergoing general anaesthesia showed a significantly higher rate of excessive air leak requiring immediate reintubation in patients initially intubated with an uncuffed tracheal tube.

Treatment recommendations

Both cuffed and uncuffed tracheal tubes are acceptable for infants and children undergoing emergency intubation. If cuffed tracheal tubes are used, avoid excessive cuff pressures.

Knowledge gaps

What is the best technique to determine cuff pressure and/or the presence of an air leak when using cuffed tracheal tubes in infants and children? What is the optimal cuff or leak pressure for children of different ages? Does optimal cuff pressure vary based on the type of cuffed tube (e.g., Microcuff®) used?

Are the data generated in elective operating room studies applicable to emergency resuscitation scenarios? Are there select populations of children whose outcomes are improved by the use of cuffed tracheal tubes during resuscitation?

Tracheal tube size[Peds-057A, Peds-057B]

Consensus on science

Evidence from one LOE 2 prospective randomised trial of elective intubation in a paediatric operating room[103] was used to support the existing formula for estimation of appropriate cuffed tracheal tube internal diameter (ID): ID (mm) = (age in years/4) + 3, also known as the Khine formula. Detailed analysis of this paper, however, reveals that the aggressive rounding up of age employed by the authors in their calculations commonly resulted in selection of a tube with an ID 0.5 mm larger than the size derived from the formula.

Evidence from one LOE 2 prospective randomised multicentre study,[104] one LOE 2,[112] and three LOE 4 prospective observational studies of elective intubation in the paediatric operating room[113–115] supported use of 3.0-mm ID cuffed tracheal tubes for newborns and infants (3.5 kg to 1 year of age) and 3.5-mm ID cuffed tracheal tubes for patients 1–2 years of age.

One LOE 2 prospective randomised multicentre study[104] and three LOE 4 prospective observational studies of elective intubation in the paediatric operating room[113–115] using Microcuff® tracheal tubes support the use of the following formula for cuffed tracheal tubes in children: ID (mm) = (age/4) + 3.5. One LOE 2 prospective observational study of elective intubation in the paediatric operating room[112] found that formula acceptable but associated with a marginally greater reintubation rate than with the Khine formula (ID [mm] = [age in years/4] + 3.0).

Treatment recommendations

If a cuffed tracheal tube is used in infants ≥3.5 kg and <1 year of age, it is reasonable to use a tube with an ID of 3.0 mm. If a cuffed tracheal tube is used in children between 1 and 2 years of age, it is reasonable to use a tube with an ID of 3.5 mm.

After the age of 2, it is reasonable to estimate the cuffed tracheal tube size with the formula ID (mm) = (age in years/4) + 3.5. If the tracheal tube meets resistance during insertion, a tube with an ID 0.5 mm smaller should be used. If there is no leak around the tube with the cuff deflated, reintubation with a tube ID 0.5 mm smaller may be beneficial when the patient is stable.

Knowledge gaps

Are the formulas for estimation of tracheal tube size that are used for elective intubation in the operating room setting applicable during resuscitation? Is there an upper age limit for the validity of the formula to estimate tube size? Are length-based formulas more accurate compared with age- or weight-based formulas for estimating tracheal tube size in infants and children?

Bag-mask ventilation versus intubation[Peds-008]

Consensus on science

One LOE 1 study[116] compared paramedic out-of-hospital BMV with intubation for children with cardiac arrest, respiratory arrest, or respiratory failure in an EMS system with short transport intervals and found equivalent rates of survival to hospital discharge and neurological outcome. One LOE 1 systematic review that included this study[117] also reached the same conclusion.

One LOE 2 study of paediatric trauma patients[118] observed that out-of-hospital intubation is associated with a higher risk of mortality and postdischarge neurological impairment compared with in-hospital intubation. These findings persisted even after stratification for severity of trauma and head trauma.

In one LOE 2 (nonrandomised) prehospital paediatric study,[119] if paramedics provided BMV while awaiting the arrival of a physician to intubate the patient, the risk of cardiac arrest and overall mortality was lower than if the patient was intubated by the paramedics. These findings persisted even after adjusting for Glasgow Coma Scale score.

Four LOE 4 studies[120–123] showed a significantly greater rate of failed intubations and complications in children compared with adults in the out-of-hospital and emergency department settings. Conversely one LOE 3 out-of-hospital study[124] and one LOE 4 out-of-hospital study[125] failed to demonstrate any difference in intubation failure rates between adults and children.

Treatment recommendations

BMV is recommended over tracheal intubation in infants and children who require ventilatory support in the out-of-hospital setting when transport time is short.

Knowledge gaps

For the experienced airway specialist, does tracheal intubation improve outcomes in comparison with BMV for paediatric resuscitation? Does the use of neuromuscular blocking drugs improve the outcome of children undergoing intubation during resuscitation? What is the minimal initial training and ongoing experience needed to improve success rate and reduce complications of emergent intubation of infants and children?

Bag-mask ventilation versus supraglottic airway[Peds-009]

Consensus on science

No studies have directly compared BMV to the use of supraglottic airway devices during paediatric resuscitation other than for the newly born in the delivery room. Nine LOE 5 case reports[126–134] demonstrated the effectiveness of supraglottic airway devices, primarily the LMA, for airway rescue of children with supraglottic airway abnormalities.

One LOE 5 out-of-hospital adult study[135] supports the use of LMAs by first responders during CPR, but another LOE 5 out-of-hospital adult cardiac arrest study[136] of EMS personnel providing assisted ventilation by either bag-mask device or LMA failed to show any significant difference in ventilation ($Paco_2$). Six LOE 5 studies during anaesthesia[137–142] demonstrated that complication rates with LMAs increase with decreasing patient age and size.

In two LOE 5 manikin studies[143,144] trained nonexpert providers successfully delivered positive-pressure ventilation using the LMA. Tracheal intubations resulted in a significant incidence of tube misplacement (oesophageal or right mainstem bronchus), a problem not present with the LMA, but time to effective ventilation was shorter and tidal volumes were greater with BMV.

In two LOE 5 studies of anaesthetised children[145,146] suitably trained ICU and ward nurses placed LMAs with a high success rate, although time to first breath was shorter in the BMV group. In a small number of cases ventilation was achieved with an LMA when it proved impossible with BMV.

Treatment recommendations

BMV remains the preferred technique for emergency ventilation during the initial steps of paediatric resuscitation. In infants and children for whom BMV is unsuccessful, use of the LMA by appropriately trained providers may be considered for either airway rescue or support of ventilation.

Knowledge gaps

Are the data regarding use of supraglottic airways for elective intubation in the operating room applicable to emergency resuscitation scenarios? With an LMA in place, is it necessary to pause chest compressions to provide effective ventilations? Is the combination of an oropharyngeal airway with BMV more or less effective than supraglottic airways?

Minute ventilation[Peds-013A]

Consensus on science

There are no data to identify the optimal minute ventilation (tidal volume or respiratory rate) for infants or children with an advanced airway during CPR, regardless of arrest aetiology.

Three LOE 5 animal studies[147–149] showed that ventilation during CPR after VF or asphyxial arrest resulted in improved ROSC, survival, and/or neurological outcome compared with no positive-pressure breaths.

Evidence from four LOE 5 adult studies[150–153] showed that hyperventilation is common during resuscitation from cardiac arrest. In one LOE 5 animal study[150] hyperventilation during resuscitation from cardiac arrest decreased cerebral perfusion pressure, ROSC, and survival compared with lower ventilation rates. One good LOE 5 animal study[149] found that increasing respiratory rate during conditions of reduced cardiac output improved alveolar ventilation but not oxygenation, and it reduced coronary perfusion pressure.

In one LOE 5 prospective, randomised adult study[154] constant-flow insufflation with oxygen compared with conventional mechanical ventilation during CPR did not change outcome (ROSC, survival to admission, and survival to ICU discharge). In another LOE 5 adult study,[155] adults with witnessed VF arrest had improved neurologically intact survival with passive oxygen insufflation compared with BMV, whereas there was no difference in survival if the VF arrest was unwitnessed.

Two LOE 5 animal studies showed that ventilation or continuous positive airway pressure (CPAP) with oxygen compared with no ventilation improved arterial blood gases[156] but did not change neurologically intact survival.[157] One good-quality LOE 5 animal study[158] showed that reducing tidal volume by 50% during CPR resulted in less hyperventilation without affecting ROSC.

Treatment recommendations

Following placement of a secure airway, avoid hyperventilation of infants and children during resuscitation from cardiac arrest, whether asphyxial or due to VF. A reduction in minute ventilation to less than baseline for age is reasonable to provide sufficient ventilation to maintain adequate ventilation-to-perfusion ratio during CPR while avoiding the harmful effects of hyperventilation. There are insufficient data to identify the optimal tidal volume or respiratory rate.

Knowledge gaps

What is the optimal minute ventilation to achieve ventilation–perfusion matching during paediatric CPR? Is it preferable to reduce tidal volume or respiratory rate to achieve optimal minute ventilation during paediatric CPR? Does hypoventilation (i.e., hypercarbia) during resuscitation affect outcome from paediatric cardiac arrest? Does passive oxygen insufflation or CPAP during cardiac arrest in infants and children provide adequate gas exchange or improve outcome from resuscitation?

Devices to verify advanced airway placement[Peds-004]

Consensus on science

No single assessment method accurately and consistently confirms tracheal tube position. Three LOE 4 studies[71,159,160] showed that when a perfusing cardiac rhythm is present in infants (≥2 kg) and children, detection of exhaled CO_2 using a colorimetric detector or capnometer has a high sensitivity and specificity for confirming tracheal tube placement. One of these studies[71] included infants and children in cardiac arrest. In the cardiac arrest population the sensitivity of exhaled CO_2 detection was only 85% (i.e., false-negatives occurred), whereas the specificity remained at 100%.

One neonatal LOE 5 study[161] of delivery room intubation demonstrated that detection of exhaled CO_2 by capnography was 100% sensitive and specific for detecting oesophageal intubation and took less time than clinical assessment to identify oesophageal intubation. Two additional neonatal LOE 5 studies[162,163] showed that confirmation of tracheal tube position is faster with capnography than with clinical assessment.

Two paediatric LOE 4 studies[164,165] showed that in the presence of a perfusing rhythm, exhaled CO_2 detection or measurement can confirm tracheal tube position accurately during transport, while two LOE 5 animal studies[166,167] showed that tracheal tube displacement can be detected more rapidly by CO_2 detection than by pulse oximetry.

One LOE 2 operating room study[168] showed that the oesophageal detector device (EDD) is highly sensitive and specific for correct tracheal tube placement in children ≥20 kg with a perfusing cardiac rhythm; there have been no studies of EDD use in children during cardiac arrest. An LOE 4 operating room (i.e., nonarrest) study[169] showed that the EDD performed well but was less accurate in children ≤20 kg.

Treatment recommendations

Confirmation of tracheal tube position using exhaled CO_2 detection (colorimetric detector or capnography) should be used for intubated infants and children with a perfusing cardiac rhythm in all settings (e.g., out-of-hospital, emergency department, ICU, inpatient, operating room).

In infants and children with a perfusing rhythm, it may be beneficial to monitor continuous capnography or frequent intermittent detection of exhaled CO_2 during out-of-hospital and intra-/interhospital transport.

The EDD may be considered for confirmation of tracheal tube placement in children weighing ≥20 kg when a perfusing rhythm is present.

Knowledge gaps

Which technique for CO_2 detection (colorimetric versus capnography) is more accurate during paediatric resuscitation? For infants and children in cardiac arrest, what is the most reliable way to achieve confirmation of tracheal tube position?

Cricoid pressure[Peds-039A, Peds-039B]

Consensus on science

There are no data to show that cricoid pressure prevents aspiration during rapid sequence or emergency tracheal intubation in infants or children. Two LOE 5 studies[170,171] showed that cricoid pressure may reduce gastric inflation in children. One LOE 5 study in children[172] and one LOE 5 study in adult cadavers[173] demonstrated that oesophageal reflux is reduced with cricoid pressure.

In one LOE 5 adult systematic review[174] laryngeal manipulation enhanced BMV or intubation in some patients while impeding it in others. One LOE 5 study in anaesthetised children[175] showed that cricoid pressure can distort the airway with a force of as low as 5 N.

Treatment recommendations

If cricoid pressure is used during emergency intubations in infants and children it should be discontinued if it impedes ventilation or interferes with the speed or ease of intubation.

Knowledge gaps

Can cricoid pressure reduce the incidence of aspiration during emergent intubation of infants or children? How much cricoid pressure should be applied, and what is the best technique to reduce gastric inflation during BMV?

Chest compressions

The concept of chest compression-only CPR is appealing because it is easier to teach than conventional CPR, and immediate chest compressions may be beneficial for resuscitation from sudden cardiac arrest caused by VF or pulseless VT. Animal studies showed that conventional CPR, including ventilations and chest compressions, is best for resuscitation from asphyxial cardiac arrest. In a large study of out-of-hospital paediatric cardiac arrest,[176] few children with asphyxial arrest received compression-only CPR and their survival was no better than in children who received no CPR.

To be effective, chest compressions must be deep, but it is difficult to determine the optimal depth in infants and children; should recommended depth be expressed as a fraction of the depth of the chest or an absolute measurement? How can this be made practical and teachable?

Compression-only CPR[Peds-012A]

Consensus on science

Evidence from one LOE 2 large out-of-hospital paediatric prospective observational investigation[176] showed that children with cardiac arrest of noncardiac aetiology (asphyxial arrest) had a higher 30-day survival with more favorable neurological outcome if they received standard bystander CPR (chest compressions with rescue breathing) compared with chest compression-only

CPR. Standard CPR and chest compression-only CPR were similarly effective and better than no bystander CPR for paediatric cardiac arrest from cardiac causes. Of note, the same study showed that more than 50% of children with out-of-hospital cardiac arrest did not receive any bystander CPR. Compression-only CPR was as ineffective as no CPR in the small number of infants and children with asphyxial arrest who did not receive ventilations.

Two LOE 5 animal studies[148,177] demonstrated improved survival rates and favorable neurological outcome with standard CPR compared with no CPR. One LOE 5 animal study[178] showed that blood gases deteriorated with compression-only CPR compared with standard CPR in asphyxial arrests.

Data from one LOE 5 animal study[177] indicated that compression-only CPR is better than no CPR for asphyxial arrest but not as effective as standard CPR, and seven LOE 5 clinical observational studies in adults[179–184] showed that compression-only CPR can result in successful resuscitation from an asphyxial arrest. Moreover, in 10 LOE 5 animal studies[185–194] and seven LOE 5 adult clinical observational studies[179–184,195] compression-only bystander CPR was generally as effective as standard one-rescuer bystander CPR for arrests from presumed cardiac causes.

Treatment recommendations

Rescuers should provide conventional CPR (rescue breathing and chest compressions) for in-hospital and out-of-hospital paediatric cardiac arrests. Lay rescuers who cannot provide rescue breathing should at least perform chest compressions for infants and children in cardiac arrest.

Knowledge gaps

Does teaching compression-only CPR to lay rescuers increase the likelihood that CPR will be performed during out-of-hospital paediatric cardiac arrest?

One- versus two-hand chest compression in children[Peds-033]

Consensus on science

There are no outcome studies comparing one- versus two-hand chest compressions for children in cardiac arrest. Evidence from one LOE 5 randomised crossover child manikin study[196] showed that higher chest-compression pressures are generated by healthcare professionals using the two-hand technique. Two LOE 5 studies[197,198] report no increase in rescuer fatigue comparing one-hand with two-hand chest compressions delivered by healthcare providers to a child-sized manikin.

Treatment recommendations

Either a one- or two-hand technique can be used for performing chest compressions in children.

Knowledge gaps

Does the use of one-hand compared with two-hand chest compressions during paediatric cardiac arrest affect quality of CPR or outcome?

Circumferential chest squeeze in infants[Peds-034]

Consensus on science

There are no studies that compare the two-thumb chest compression technique with and without a "circumferential squeeze" in infants.

Treatment recommendations

There are insufficient data for or against the need for a circumferential squeeze of the chest when performing the two-thumb technique of external chest compression for infants.

Knowledge gaps

Does the addition of a circumferential squeeze to two-thumb compression in infants provide more effective chest compressions or improve resuscitation outcome?

Chest compression depth[Peds-040A, Peds-040B]

Consensus on science

Evidence from anthropometric measurements in three good-quality LOE 5 case series[199–201] showed that in children the chest can be compressed to one-third of the anterior–posterior chest diameter without causing damage to intrathoracic organs. One LOE 5 mathematical model based on neonatal chest computed tomography scans[202] suggests that one-third anterior–posterior chest compression depth is more effective than one-fourth compression depth and safer than one-half anterior–posterior compression depth.

A good-quality LOE 5[152] adult study found that chest compressions are often inadequate, and a good-quality LOE 4 paediatric study[200] showed that during resuscitation of patients ≥8 years of age, compressions are often too shallow, especially following rescuer changeover. Evidence from one paediatric LOE 4 systematic review of the literature[203] showed that rib fractures are rarely associated with chest compressions.

Treatment recommendations

In infants, rescuers should be taught to compress the chest by *at least* one-third the anterior–posterior dimension or approximately 4 cm (1.5 in.). In children, rescuers should be taught to compress the chest by *at least* one-third the anterior–posterior dimension or approximately 5 cm (2 in.).

Knowledge gaps

Can lay rescuers or healthcare providers reliably perform compressions to the recommended depth during paediatric cardiac arrest? Is there harm from compressions that are "too deep" in infants?

Compression–ventilation ratio

The ILCOR Neonatal Task Force continues to recommend a compression–ventilation ratio of 3:1 for resuscitation of the newly born in the delivery room, with a pause for ventilation whether or not the infant has an advanced airway. The Paediatric Task Force reaffirmed its recommendation for a 15:2 compression–ventilation ratio for two-rescuer infant CPR, with a pause for ventilation in infants without an advanced airway, and continuous compressions without a pause for ventilation for infants with an advanced airway.

No previous recommendations were made for hospitalised newborns cared for in areas other than the delivery area or with primary cardiac rather than asphyxial arrest aetiology. For example, consider the case of a three-week-old infant who has a cardiac arrest following cardiac surgery. In the neonatal intensive care unit such an infant would be resuscitated according to the protocol for the newly born, but if the same infant is in the PICU, resuscitation would be performed according to the infant guidelines. A resolution to this dilemma is suggested on the basis of the arrest aetiology and ease of training.

Optimal compression–ventilation ratio for infants and children[Peds-011B]

Consensus on science

There are insufficient data to identify an optimal compression–ventilation ratio for CPR in infants and children. In four LOE 5 manikin studies[204–207] examining the feasibility of compression–ventilation ratios of 15:2 and 5:1, lone rescuers could not deliver the desired number of chest compressions per minute at a ratio of 5:1. In five LOE 5 studies[208–212] using a variety of manikin sizes comparing compression–ventilation ratios of 15:2 with 30:2, a ratio of 30:2 yielded more chest compressions with no, or minimal, increase in rescuer fatigue. One LOE 5 study[213] of volunteers recruited in an airport to perform one-rescuer layperson CPR on an adult-sized manikin observed less "no flow time" with the use of a 30:2 ratio compared with a 15:2 ratio.

One LOE 5 observational human study[214] comparing resuscitations by firefighters before and after the change from a recommended 15:2–30:2 compression–ventilation ratio reported more chest compressions per minute with the 30:2 ratio, but the rate of ROSC was unchanged. Three LOE 5 animal studies[192,215,216] showed that coronary perfusion pressure, a major determinant of success in resuscitation, rapidly declines when chest compressions are interrupted; once compressions are resumed, several chest compressions are needed to restore coronary perfusion pressure to preinterruption levels. Thus, frequent interruptions of chest compressions prolong the duration of low coronary perfusion pressure and flow and reduce the mean coronary perfusion pressure. Three LOE 5 manikin studies[213,217,218] and three LOE 5[151,152,219] in- and out-of-hospital adult human studies documented long interruptions in chest compressions during simulated or actual resuscitations. Three LOE 5 adult studies[220–222] demonstrated that these interruptions reduced ROSC.

In five LOE 5 animal studies[191,192,194,215,216] chest compressions without ventilations were sufficient to resuscitate animals with VF-induced cardiac arrest. Conversely in two LOE 5 animal studies[223,224] decreasing the frequency of ventilation was detrimental in the first 5–10 min of resuscitation of VF-induced cardiac arrest.

One LOE 5 mathematical model[225] suggested that the compression–ventilation ratio in children should be lower (more ventilations to compressions) than in adults and should decrease with decreasing weight. Two LOE 5 studies of asphyxial arrest in pigs[148,177] showed that ventilations added to chest compressions improved outcome compared with compressions alone. Thus, ventilations are more important during resuscitation from asphyxia-induced arrest than during resuscitation from VF. But even in asphyxial arrest, fewer ventilations are needed to maintain an adequate ventilation–perfusion ratio in the presence of the low cardiac output (and consequently low pulmonary blood flow) produced by chest compressions.

Treatment recommendations

For ease of teaching and retention, a compression–ventilation ratio of 30:2 is recommended for the lone rescuer performing CPR in infants and children, as is used for adults. For healthcare providers performing two-rescuer CPR in infants and children, a compression–ventilation ratio of 15:2 is recommended. When a tracheal tube is in place, compressions should not be interrupted for ventilations.

Knowledge gaps

What is the optimal compression–ventilation ratio to improve outcome for neonates, infants, and children in cardiac arrest?

Newborns (out of the delivery area) without a tracheal tube[Peds-027A]

Consensus on science

There are insufficient data to identify an optimal compression–ventilation ratio for all infants in the first month of life. One LOE 5 animal study[192] showed that coronary perfusion pressure declined with interruptions in chest compressions; after each interruption, several chest compressions were required to restore coronary perfusion pressure to preinterruption levels. One LOE 5 adult human study[221] and two LOE 5 animal studies[215,222] showed that interruptions in chest compression reduced the likelihood of ROSC in VF cardiac arrest.

One LOE 5 one-rescuer manikin study[207] showed that more effective ventilation was achieved with a 3:1 ratio than with a 5:1, 10:2, or 15:2 ratio. One LOE 5 mathematical study of cardiovascular physiology[226] suggested that blood flow rates in neonates are best at compression rates of $\geq 120\ \text{min}^{-1}$.

Treatment recommendations

There are insufficient data to recommend an optimal compression–ventilation ratio during CPR for all infants in the first month of life (beyond the delivery room). The limited data available suggest that if the aetiology of the arrest is cardiac, a 15:2 ratio (two rescuers) may be more effective than a 3:1 ratio.

Knowledge gaps

Do healthcare providers perform better CPR if they learn one rather than two compression–ventilation ratios based on aetiology of the arrest (cardiac or asphyxial)?

Newborns (out of delivery area) with a tracheal tube[Peds-026A]

Consensus on science

There is insufficient evidence to determine if an intubated neonate has a better outcome from cardiac arrest using a 3:1 compression–ventilation ratio and interposed ventilations compared with continuous chest compressions without pause for ventilations (asynchronous compressions and ventilations).

Two LOE 5 adult[220,222] and two LOE 5 animal[191,192] studies demonstrated that interruptions in chest compressions reduced coronary perfusion pressure, a key determinant of successful resuscitation in adults, and decreased ROSC. There are no equivalent studies evaluating the impact of interrupted chest compressions in asphyxiated neonates or neonatal animal models.

In one LOE 5 piglet study[227] of VF arrest, myocardial blood flow increased using simultaneous chest compressions and high airway pressure ventilations in a 1:1 ratio as compared with conventional CPR at a 5:1 ratio. Another LOE 5 VF piglet study[228] demonstrated equivalent cardiac output but worsened gas exchange using a 1:1 compression–ventilation ratio (i.e., simultaneous compressions and ventilations) with high airway pressures compared with conventional CPR at a 5:1 ratio.

One LOE 5[148] study in nonintubated asphyxiated piglets resuscitated with a 5:1 compression–ventilation ratio showed that ventilations are important for successful resuscitation. One LOE 5 study in intubated asphyxiated piglets[178] showed that the addition of ventilations resulted in lower arterial CO_2 tension ($Paco_2$) without compromising haemodynamics when compared with compressions alone. One LOE 5 manikin study[229] found that healthcare providers were unable to achieve the recommended rate of ventilations during infant CPR at a 3:1 compression–ventilation ratio, with <20% delivering a net rate of 40 breaths/min after 5 min of resuscitation. There are no studies that evaluate the impact of continuous compressions on minute ventilation, gas exchange, or the outcome of resuscitation during CPR for intubated neonates.

Treatment recommendations

For ease of training, providers should use the compression–ventilation ratio and resuscitation approach that is most commonly used in their practice environment for intubated term or near-term newborns within the first month of life. Intubated newborns (i.e., those with an advanced airway) who require CPR in non-neonatal settings (e.g., prehospital, emergency department, PICU) or those with a cardiac aetiology of cardiac arrest, regardless of location, should receive CPR according to infant guidelines (continuous chest compressions without pause for ventilations).

Knowledge gaps

In intubated infants in cardiac arrest, can effective ventilations be performed during continuous chest compressions with asynchronous ventilations? Do pauses for ventilations during CPR affect the outcome from cardiac arrest in intubated infants?

Vascular access and drug delivery

There is no new evidence to change the 2005 ILCOR recommendations on vascular access, including the early use of intraosseous (IO) access and de-emphasis of the tracheal route of drug delivery. Epidemiological data, largely from the National Registry of CPR (NRCPR), reported an association between vasopressin, calcium, or sodium bicarbonate administration and an increased likelihood of death. These data, however, cannot be interpreted as a cause-and-effect relationship. The association may be due to more frequent use of these drugs in children who fail to respond to standard basic and advanced life support interventions. These and other data in adults question the benefit of intravenous (IV) medications during resuscitation and reemphasise the importance of high-quality CPR.

Intraosseous access[Peds-035]

Consensus on science

There are no studies comparing IO with IV access in children with cardiac arrest. In one LOE 5 study of children in shock[230] IO access was frequently more successful and achieved more rapidly than IV access. Eight LOE 4 case series[231–238] showed that providers with many levels of training could rapidly establish IO access with minimal complications for children with cardiac arrest.

Treatment recommendations

IO cannulation is an acceptable route of vascular access in infants and children with cardiac arrest. It should be considered early in the care of critically ill children whenever venous access is not readily attainable.

Knowledge gaps

Does the use of IO compared with IV vascular access improve outcome of paediatric cardiac arrest? Does the use of newer IO devices (e.g., bone injection guns and drills) compared with conventional IO needles affect outcome in paediatric cardiac arrest?

Tracheal drug delivery[Peds-036]

Consensus on science

One LOE 3 study of children with in-hospital cardiac arrest[239] demonstrated similar ROSC and survival rates, whereas two LOE 5 studies of adults in cardiac arrest[240,241] demonstrated reduced ROSC and survival to hospital discharge rates when tracheal instead of IV adrenaline was given. One LOE 5 case series of neonatal asphyxial bradycardia[242] demonstrated similar rates of ROSC whether IV or tracheal adrenaline was administered, whereas another LOE 5 study[243] demonstrated a lower rate of ROSC in neonates given tracheal as opposed to IV adrenaline. Many of the human studies used tracheal adrenaline doses of $\leq 0.1\ mg\ kg^{-1}$.

In some animal studies[244–249] lower doses of tracheal adrenaline ($0.01–0.05\ mg\ kg^{-1}$) produced transient deleterious β-adrenergic vascular effects resulting in lower coronary artery perfusion. One LOE 5 study[250] of animals in VF cardiac arrest demonstrated a higher rate of ROSC in those treated with tracheal vasopressin compared with IV placebo.

Four LOE 5 studies of animals in cardiac arrest[251–254] demonstrated similar ROSC and survival rates when either tracheal or IV routes were used to deliver adrenaline. These studies also demonstrated that to reach an equivalent biological effect, the tracheal dose must be up to 10 times the IV dose.

Treatment recommendations

The preferred routes of drug delivery for infants and children in cardiac arrest are IV and IO. If adrenaline is administered via a tracheal tube to infants and children (not including the newly born) in cardiac arrest, the recommended dose is $0.1\ mg\ kg^{-1}$ ($100\ \mu g\ kg^{-1}$).

Knowledge gaps

What is the optimal dose of tracheal adrenaline during paediatric cardiac arrest?

Defibrillation

The Paediatric Task Force evaluated several issues related to defibrillation, including safe and effective energy dosing, stacked versus single shocks, use of automated external defibrillators (AEDs) in infants ≤1 year of age and paddle/pad type, size, and position. There were a few new human and animal studies on these topics, and the level of evidence (LOE) was generally 3–5. No new data are available to support a change in drug treatment of recurrent or refractory VF/pulseless VT. There were several human and animal publications on defibrillation energy dose, but the data are contradictory and the optimal safe and effective energy dose remains unknown.

The new recommendation of an initial dose of $2–4\ J\ kg^{-1}$ is based on cohort studies showing low success in termination of VF in children with $2\ J\ kg^{-1}$. However, these studies do not provide data on success or safety of higher energy doses. The reaffirmation of the recommendation for a single initial shock rather than stacked shocks (first made in 2005) is extrapolated from the ever-increasing adult data showing that long pauses in chest compressions required for stacked shocks are associated with worse resuscitation outcomes and that the initial shock success rate is relatively high with biphasic defibrillation.

No changes are recommended in pad/paddle size or position. Although the safety of AEDs in infants ≤1 year is unknown, case reports have documented successful defibrillation using AEDs in infants. A manual defibrillator or an AED with paediatric attenuation capabilities is preferred for use in infants and small children.

Paddle size and orientation[Peds-029]

Consensus on science

One LOE 5 study in adults[255] demonstrated that shock success increased from 31% to 82% when pad size was increased from 8×8 cm to 12×12 cm. Three paediatric LOE 4,[256–258] three adult LOE 5,[255,259,260] and three LOE 5 animal[261–263] studies demonstrated that transthoracic impedance decreases with increasing pad size. Decreased transthoracic impedance increases transthoracic current and, thus, presumably, transmyocardial current.

Pad position

Consensus on science

One paediatric LOE 4 study[264] observed no difference in the rate of ROSC between antero-lateral and anterior–posterior electrode positions for shock delivery. One paediatric LOE 2 study,[256] two adult LOE 5 studies,[265,266] and one LOE 5 animal study[263] demonstrated that transthoracic impedance is not dependent on pad position. Transthoracic impedance was increased in one adult LOE 5[267] study by placing the pads too close together and in one LOE 5[260] study when the pads were placed over the female breast. Additionally, one adult LOE 5[268] study showed that placing the apical pad in a horizontal position lowers transthoracic impedance.

Treatment recommendation

There is insufficient evidence to alter the current recommendations to use the largest size paddles/pads that fit on the infant or child's chest without touching each other or to recommend one paddle/pad position or type over another.

Pad position

Consensus on science

One paediatric LOE 4 study[264] observed no difference in the rate of ROSC between antero-lateral and anterior–posterior electrode positions for shock delivery. One paediatric LOE 2 study,[256] two adult LOE 5 studies,[265,266] and one LOE 5 animal study[263] demonstrated that transthoracic impedance is not dependent on pad position. Transthoracic impedance was increased in one adult LOE 5[267] study by placing the pads too close together and in one LOE 5[260] study when the pads were placed over the female breast. Additionally, one adult LOE 5[268] study showed that placing the apical pad in a horizontal position lowers transthoracic impedance.

Treatment recommendation

There is insufficient evidence to alter the current recommendations to use the largest size paddles/pads that fit on the infant or child's chest without touching each other or to recommend one paddle/pad position or type over another.

Self-adhesive pads versus paddles[Peds-043A, Peds-043B]

Consensus on science

There are limited studies comparing self-adhesive defibrillation pads (SADPs) with paddles in paediatric cardiac arrest. One paediatric LOE 4[264] study demonstrated equivalent ROSC rates when paddles or SADPs were used. One LOE 5[269] adult out-of-hospital cardiac arrest study suggested improved survival to hospital admission when SADPs rather than paddles were used.

One adult LOE 5[270] study showed a lower rate of rhythm conversion, and one small adult LOE 5[271] study showed at least equivalent success with the use of SADPs in comparison with paddles in patients undergoing cardioversion for atrial fibrillation. Two adult LOE 5[272,273] studies showed equivalent transthoracic impedance with SADPs or paddles. One adult LOE 5[266] and two LOE 5 animal[274,275] studies showed that SADPs had a higher transthoracic impedance than paddles.

One LOE 4[276] study described difficulty with fitting self-adhesive pads onto the thorax of a premature infant without the pads touching. One LOE 5[277] study demonstrated the improved accuracy of cardiac rhythm monitoring following defibrillation using SADPs compared with the combination of paddles and gel pads.

Using standard resuscitation protocols in simulated clinical environments, one LOE 5[278] study found no significant difference in the time required to deliver shocks using either SADPs or paddles, and one LOE 5[279] study found no significant difference in time without compressions when SADPs or paddles were used.

Treatment recommendations

Either self-adhesive defibrillation pads or paddles may be used in infants and children in cardiac arrest.

Knowledge gaps

Is the use of hands-on defibrillation safe for rescuers and does it improve outcome for infants and children in cardiac arrest (e.g., by presumably reducing interruptions in chest compressions)?

Number of shocks[Peds-022A]

Consensus on science

There are no randomised controlled studies examining a single versus sequential (stacked) shock strategy in children with VF/pulseless VT. Evidence from seven LOE 5 studies in adults with VF[221,280–285] supported a single-shock strategy over stacked or sequential shocks because the relative efficacy of a single biphasic shock is high and the delivery of a single shock reduces duration of interruptions in chest compressions.

Treatment recommendations

A single-shock strategy followed by immediate CPR (beginning with chest compressions) is recommended for children with out-of-hospital or in-hospital VF/pulseless VT.

Knowledge gaps

Are there circumstances during which the use of stacked or multiple shocks can improve outcome from paediatric cardiac arrest?

Energy dose[Peds-023A, Peds-023B]

Consensus on science

Two LOE 4[264,286] studies reported no relationship between defibrillation dose and survival to hospital discharge or neurological outcome from VF/pulseless VT. Evidence from three LOE 4 studies in children in out-of-hospital and in-hospital settings[264,287,288] observed that an initial dose of 2 J kg^{-1} was effective in terminating VF 18–50% of the time. Two LOE 4 studies[286,289] reported that children often received more than 2 J kg^{-1} during out-of hospital cardiac arrest, with many (69%) requiring ≥3 shocks of escalating energy doses. One in-hospital cardiac arrest LOE 4 study[264] reported that the need for multiple shocks with biphasic energy doses of 2.5–3.2 J kg^{-1} was associated with lack of ROSC.

Evidence from two LOE 5 animal studies[290,291] observed that 0–8% of episodes of long-duration VF were terminated by a 2 J kg^{-1} monophasic shock and up to 32% were terminated by biphasic shocks. Animals in these studies received both fixed and escalated doses, and most required two or more shocks to terminate VF. In one LOE 5 animal study[263] the defibrillation threshold for short-duration VF was 2.4 J kg^{-1}, whereas in another[291] it was 3.3 J kg^{-1}.

In four LOE 5 animal studies[290,292–294] of AED shocks delivered using a paediatric attenuator, 50 J and 50 → 76 → 86 J (2.5–4 J kg^{-1}) escalating doses were effective at terminating long-duration VF but required multiple shocks. In one LOE 5 animal study[295] 10 J kg^{-1} shocks were more effective at terminating long-duration VF (6 min) with one shock than 4 J kg^{-1} shocks.

In two LOE 4 paediatric studies[264,286] and four LOE 5 animal studies,[290,292–294] energy doses of 2–10 J kg^{-1} for short- or long-duration VF resulted in equivalent rates of survival. Myocardial damage, as assessed by haemodynamic or biochemical measurements, was less when a paediatric attenuator was used with an adult energy dose compared with a full adult AED dose, but the

degree of myocardial damage was not associated with any difference in 4- or 72-h survival. An LOE 5 animal study[295] found no difference in haemodynamic parameters or biochemical measurements of myocardial damage comparing biphasic 150 J (4 J kg^{-1}) with monophasic 360 J (10 J kg^{-1}) shocks.

In two LOE 5 animal studies[290,291] biphasic waveforms were more effective than monophasic waveforms for treatment of VF/pulseless VT. There are no human data that directly compare monophasic to biphasic waveforms for paediatric defibrillation.

Treatment recommendations

An initial dose of 2–4 J kg^{-1} is reasonable for paediatric defibrillation. Higher subsequent energy doses may be safe and effective.

Knowledge gaps

What is the minimum effective and maximum safe defibrillation energy dose for paediatric VF/pulseless VT? What is the optimal parameter (e.g., weight or length) on which to base defibrillation energy doses for infants and children? Should the energy dose for defibrillation be escalated for shock-refractory VF?

Does the use of biphasic waveforms when compared to monophasic waveforms improve outcome from paediatric cardiac arrest?

Amiodarone versus lidocaine for refractory VF/pulseless VT[Peds-019]

Consensus on science

In two LOE 5 prospective out-of-hospital adult trials IV amiodarone improved ROSC and survival to hospital admission but not hospital discharge when compared with placebo[296] or lidocaine[297] for treatment of shock-refractory VF/pulseless VT. Evidence from two LOE 5 case series in children[298,299] supported the effectiveness of amiodarone for the treatment and acute conversion of life-threatening (nonarrest) ventricular arrhythmias. There are no paediatric data investigating the efficacy of lidocaine for shock-refractory VF/pulseless VT.

Treatment recommendations

Amiodarone may be used for the treatment of shock-refractory or recurrent VF/pulseless VT in infants and children; if amiodarone is not available, lidocaine may be considered.

Knowledge gaps

Does the use of amiodarone compared with lidocaine improve outcome from shock-refractory or recurrent VF/pulseless VT in infants and children? Is lidocaine effective for the treatment of VF/pulseless VT in children?

AED use in infants[Peds-001A, Peds-001B]

Consensus on science

One LOE 4[300] and two LOE 5[288,301] studies showed that infants in cardiac arrest (in- and out-of-hospital) may have shockable rhythms. Evidence from three LOE 5[302–304] studies showed that many AED devices can safely and accurately distinguish between a shockable and nonshockable rhythm in infants and children.

The optimal energy dose for defibrillation in infants has not been established, but indirect data from five LOE 5 animal studies[287,294,305–307] showed that the young myocardium may be able to tolerate high-energy doses. In three LOE 5 animal studies a paediatric attenuator used with an adult-dose biphasic AED shock was as effective and less harmful than monophasic weight-based doses[290] or biphasic adult doses.[292,293]

Two LOE 4 case reports[308,309] described survival of infants with out-of-hospital cardiac arrest when AED use was coupled with bystander CPR and defibrillation using an AED. Two paediatric LOE 5 case reports[310,311] noted successful defibrillation with minimal myocardial damage and good neurological outcome using an AED with adult energy doses.

Treatment recommendations

For treatment of out-of-hospital VF/pulseless VT in infants, the recommended method of shock delivery by device is listed in order of preference below. If there is any delay in availability of the preferred device, the available device should be used. The AED algorithm should have demonstrated high specificity and sensitivity for detecting shockable rhythms in infants. The order of preference is as follows:

1. Manual defibrillator.
2. AED with dose attenuator.
3. AED without dose attenuator.

Knowledge gaps

Is there a lower limit of infant size or weight below which an AED should not be used?

Arrhythmia therapy[Peds-030]

The evidence on emergency treatment of arrhythmias was reviewed and the only change was the addition of procainamide as possible therapy for refractory supraventricular tachycardia (SVT).

Unstable VT

Consensus on science

There is insufficient evidence to support or refute the efficacy of electric therapy over drug therapy or the superiority of any drug for the emergency treatment of unstable VT in the paediatric age group. In two LOE 5 adult case series,[312,313] early electric cardioversion was effective for treatment of unstable VT.

In four small LOE 4 paediatric case series[298,299,314,315] amiodarone was effective in the management of VT. One prospective randomised multicentre safety and efficacy LOE 2 trial evaluating amiodarone for the treatment of paediatric tachyarrhythmias[316] found that 71% of children treated with amiodarone experienced cardiovascular side effects. Both efficacy and adverse events were dose-related.

Treatment recommendations

It is reasonable to use synchronised electric cardioversion as the preferred first therapy for paediatric VT with hypotension or evidence of poor perfusion. If drug therapy is used to treat unstable VT, amiodarone may be a reasonable choice, with careful haemodynamic monitoring performed during its slow delivery.

Knowledge gaps

What is the optimal dose of energy for synchronised cardioversion during treatment of unstable VT in paediatric patients?

Drugs for supraventricular tachycardia[Peds-031]

Consensus on science

Twenty-two LOE 4 studies in infants and children[317–338] demonstrated the effectiveness of adenosine for the treatment of haemodynamically stable or unstable SVT. One LOE 4 study[339] and four larger LOE 5 studies involving teenagers and adults[340–343] also demonstrated the efficacy of adenosine, although frequent but transient side effects were reported.

One LOE 2 study[344] showed highly successful (approximately 90%) treatment of SVT in infants and children using adenosine or

verapamil and superiority of these drugs to digitalis (61–71%). One LOE 5 randomised prospective adult study[345] and one LOE 5 meta-analysis, primarily involving adults but including some children,[346] demonstrated the effectiveness of verapamil and adenosine in treating SVT and highlighted the cost-effectiveness of verapamil over adenosine.

One LOE 4 randomised, prospective study[316] and 15 LOE 4 small case series and observational studies in infants and children[298,299,314,315,347–357] showed that amiodarone was effective in the treatment of supraventricular tachyarrhythmias. Generalization to paediatric SVT treatment with amiodarone may be limited, however, since most of these studies in children involved postoperative junctional tachycardia.

Rare but significant side effects have been reported in association with rapid administration of amiodarone. Bradycardia and hypotension were reported in one prospective LOE 4 study,[316] cardiovascular collapse was reported in two LOE 5 case reports,[358,359] and polymorphic VT was reported in one small LOE 4 case series.[360] Other LOE 5 case reports describe late side effects of pulmonary toxicity[359] and hypothyroidism.[362]

In one LOE 2 paediatric comparison control study[363] procainamide had a significantly higher success rate and an equal incidence of adverse effects when compared with amiodarone for treating refractory SVT. In five LOE 4 observational studies[364–368]and five LOE 5 case reports[369–373] procainamide effectively suppressed or slowed the rate in children with SVT. A wide variety of arrhythmias were studied, including ectopic atrial tachycardia, atrial flutter, and orthodromic reciprocating tachycardia.

In LOE 5 studies in children,[374] adults,[375–376] and animals,[377] hypotension from procainamide infusion resulted from vasodilation and not decreased myocardial contractility. Initial observational LOE 4 reports[378–380] and one LOE 4 case series[381] described successful treatment of paediatric SVT with verapamil. However, multiple small LOE 4 case series[344,382] and LOE 5 case reports[383,384] documented severe hypotension, bradycardia, and heart block causing haemodynamic collapse and death following IV administration of verapamil for SVT in infants. Two small LOE 4 paediatric case series[385,386] described esmolol and dexmedetomidine in the treatment of SVT.

Treatment recommendations

For infants and children with SVT with a palpable pulse, adenosine should be considered the preferred medication. Verapamil may be considered as alternative therapy in older children but should not be routinely used in infants. Procainamide or amiodarone given by a slow IV infusion with careful haemodynamic monitoring may be considered for refractory SVT.

Knowledge gaps

Does the use of alternate medications (e.g., esmolol, dexmedetomidine) in the treatment of SVT in infants and children improve outcome? What is the role of vagal maneuvers in the treatment of SVT?

Shock

The Task Force reviewed evidence related to several key questions about the management of shock in children. There is ongoing uncertainty about the indications for using colloid versus crystalloid in shock resuscitation. One large adult trial suggested that normal saline (isotonic crystalloid) is equivalent to albumin, although subgroup analysis suggested harm associated with the use of colloid in patients with traumatic brain injury. There were insufficient data to change the 2005 recommendations.

The optimal timing for intubation of children in shock remains unclear, although reports of children and adults with septic shock suggested potential beneficial effects of early intubation (before signs of respiratory failure develop) combined with a protocol-driven management approach. When children in septic shock were treated with a protocol that included therapy directed to normalizing central venous oxygen saturation, patient outcome appeared to improve.

Performing rapid sequence induction and tracheal intubation of a child with shock can cause acute cardiovascular collapse. Etomidate typically causes less haemodynamic compromise than other induction drugs and is therefore often used in this setting. However, data suggest that the use of this drug in children and adults with septic shock is associated with increased mortality that may be secondary to etomidate's inhibitory effects on corticosteroid synthesis. Administering stress-dose corticosteroids in septic shock remains controversial, with recent adult trials failing to show a beneficial effect.

Graded volume resuscitation for haemorrhagic shock[Peds-032]

Consensus on science

There are no paediatric studies of the timing or extent of volume resuscitation in haemorrhagic shock with hypotension. Nine LOE 5 adult[387–395] studies reported conflicting results with regard to the effect of timing and extent of volume resuscitation on outcome of haemorrhagic shock with hypotension.

Treatment recommendations

There is insufficient evidence as to the best timing or quantity for volume resuscitation in infants and children with haemorrhagic shock following trauma.

Knowledge gaps

What is the appropriate clinical indicator for volume resuscitation during treatment of haemorrhagic shock in infants and children?

Early ventilation in shock[Peds-038B]

Consensus on science

There are no studies investigating the role of intubation and assisted ventilation before the onset of respiratory failure in infants and children with shock. Two LOE 5 animal studies in septic shock[396,397] and one LOE 5 animal study in pericardial tamponade[398] showed improved haemodynamics and select organ perfusion with intubation before the onset of respiratory failure. One report of two adults (LOE 5[399]) described cardiac arrest following intubation of one adult with tamponade due to penetrating trauma and improvement in haemodynamics during spontaneous breathing in one mechanically ventilated adult patient with post-cardiac surgery tamponade.

One LOE 5 study of septic shock in adults[400] suggested a reduced mortality with early ventilation compared with historical controls who only received ventilation for respiratory failure. One LOE 5 study of animals in septic shock[401] showed that early assisted ventilation does not reduce oxygen extraction or prevent the development of lactic acidosis.

Treatment recommendations

There is insufficient evidence to support or refute the use of tracheal intubation of infants and children in shock before the onset of respiratory failure.

Knowledge gaps

Does the timing of respiratory support in infants and children with shock affect outcome?

Colloid versus crystalloid fluid administration[Peds-044A, Peds-044B]

Consensus on science

Evidence from three randomised blinded LOE 1 controlled trials in children with dengue shock syndrome[402–404] and one LOE 1 open randomised trial in children with septic shock[405] suggested no clinically important differences in survival from therapy with colloid versus therapy with isotonic crystalloid solutions for shock.

In one large LOE 5 randomised controlled trial of fluid therapy in adult ICU patients[406] and in six good-quality LOE 5 meta-analyses, predominantly of adults,[407–412] no mortality differences were noted when colloid was compared with hypertonic and isotonic crystalloid solutions, and no differences were noted between types of colloid solutions.

Three LOE 5 studies comparing the use of crystalloids and colloids for adults in shock suggested that crystalloid may have an associated survival benefit over colloid in subgroups of patients with shock, including general trauma,[409] traumatic brain injury,[413] and burns.[414] One randomised controlled LOE 5 study of children with severe malaria suggested better survival with colloid than with crystalloid infusion.[415]

Treatment recommendations

Isotonic crystalloids are recommended as the *initial* resuscitation fluid for infants and children with any type of shock. There is insufficient evidence to identify the superiority of any specific isotonic crystalloid over others.

Knowledge gaps

Does the use of any specific crystalloid solution (Ringer's lactate, normal saline, hypertonic saline) improve outcome for paediatric shock? Are there subgroups of children in shock whose outcome is improved with the use of colloid compared with crystalloid?

Vasoactive agents in distributive shock[Peds-045A, Peds-045B]

Consensus on science

One LOE 4 observational study[416] suggested that the course of paediatric septic shock physiology is dynamic and that serial assessments are required to titrate the type and dose of inotropes or vasopressor therapy to achieve optimal haemodynamic results. Evidence from four LOE 1 paediatric randomised controlled studies,[417–420] three LOE 5 adult randomised controlled studies,[421–423] and one LOE 5 adult systematic review[424] showed that no inotrope or vasopressor is superior in reducing mortality from paediatric or adult distributive shock.

Two LOE 1 paediatric randomised controlled studies[417,418] showed that children with "cold" (i.e., low cardiac index) septic shock improved haemodynamically with brief (4-h) administration of milrinone (bolus and infusion). One LOE 1 paediatric randomised controlled study[420] of vasodilatory shock compared the addition of vasopressin versus placebo to standard vasoactive agents and showed no change in duration of vasopressor infusion but observed a trend toward increased mortality.

Eleven small LOE 4 paediatric case series[425–435] showed improved haemodynamics but not survival when vasopressin or terlipressin was administered to children with refractory, vasodilatory, septic shock.

Treatment recommendations

There is insufficient evidence to recommend a specific inotrope or vasopressor to improve mortality in paediatric distributive shock. Selection of an inotrope or vasopressor to improve haemodynamics should be tailored to each patient's physiology and adjusted as clinical status changes.

Knowledge gaps

Does the use of any specific vasoactive agent improve outcome for infants and children with distributive shock?

Vasoactive agents in cardiogenic shock[Peds-046A]

Consensus on science

One LOE 4 paediatric case series[436] showed that critically ill children requiring inotropic support have wide variability in haemodynamic responses to different infusion rates of dobutamine. One LOE 2 blinded crossover study[437] found dopamine and dobutamine had equal haemodynamic effects in infants and children requiring post-cardiac surgical inotropic support but that dopamine at an infusion rate of $\geq 7\ \mu g\ kg^{-1}$ per minute increased pulmonary vascular resistance.

Six LOE 3 studies[438–443] showed that both dopamine and dobutamine infusions improve haemodynamics in children with cardiogenic shock.

Evidence from one LOE 1 paediatric placebo-controlled trial[444] showed that milrinone is effective in preventing low cardiac output syndrome in infants and children following biventricular cardiac repair. One LOE 4 study[445] showed that milrinone improved cardiac index in neonates with low cardiac output following cardiac surgery.

One small LOE 1 study[446] showed that children had better haemodynamic parameters and shorter ICU stays if they received milrinone compared with low-dose adrenaline plus nitroglycerin for inotropic support following repair of tetralogy of Fallot.

In two LOE 4 small case series,[447,448] when children with heart failure secondary to myocardial dysfunction were given levosimendan, they demonstrated improved ejection fraction, required a shorter duration of catecholamine infusions,[447] and showed a trend toward improved haemodynamics and reduced arterial lactate levels.[448]

In subgroup analysis from one LOE 5 randomised controlled trial in adults,[449] patients with cardiogenic shock treated with noradrenaline versus dopamine had an improved survival at 28 days. When all causes of shock were included, patients treated with noradrenaline also had fewer arrhythmias than those treated with dopamine (12% versus 24%).

Treatment recommendations

The catecholamine dose for inotropic support in cardiogenic shock must be individually titrated because there is a wide variability in clinical response. It is reasonable to use adrenaline, levosimendan, dopamine, or dobutamine for inotropic support in infants and children with cardiogenic shock. Milrinone may be beneficial for the prevention and treatment of low cardiac output following cardiac surgery.

There are insufficient data to support or refute the use of noradrenaline in paediatric cardiogenic shock.

Knowledge gaps

Does the use of any specific vasoactive agent improve outcome for infants and children with cardiogenic shock who have not undergone cardiac surgery?

Etomidate for intubation in hypotensive septic shock[Peds-047A, Peds-047B]

Consensus on science

One LOE 4 study of children with septic shock[450] showed that adrenal suppression occurred after the administration of a single dose of etomidate and persisted for at least 24 h. Evidence from two LOE 4[451,452] studies and one LOE 5[453] study showed that etomidate can be used to facilitate tracheal intubation in infants and children with minimal haemodynamic effect, but very few of these reports included patients with hypotensive septic shock. One LOE 4 study[450] suggested an association with mortality when etomidate is used to facilitate the intubation of children with septic shock.

One adult LOE 5 study[454] observed an increased mortality associated with the use of etomidate for intubation of patients in septic shock, even with steroid supplementation. Conversely, one underpowered adult LOE 5 study[455] in septic patients did not show an increase in mortality.

One multicentre adult LOE 5 comparative trial of etomidate versus ketamine for intubation[456] found no difference in organ failure over the first 72 h and no mortality difference, but this study included only a small number of patients with shock. Adrenal insufficiency was more common in etomidate-treated patients.

Treatment recommendations

Etomidate should not be routinely used when intubating an infant or child with septic shock. If etomidate is used in infants and children with septic shock, the increased risk of adrenal insufficiency should be recognised.

Knowledge gaps

If etomidate is used, does steroid administration improve outcome for infants and children with septic shock?

Corticosteroids in hypotensive shock[Peds-049A, Peds-049B]

Consensus on science

In six LOE 5 randomised controlled trials in adults with septic shock[454,457–461] the addition of low-dose hydrocortisone decreased the time to shock reversal. Three LOE 5 randomised controlled trials in adults with vasopressor-dependent septic shock[457,462,463] showed that survival was improved when low-dose hydrocortisone was administered, while one small adult LOE 5 randomised controlled trial[464] showed a trend toward increased survival.

One fair-quality, small LOE 1 study in children with septic shock[465] found that low-dose hydrocortisone administration resulted in no survival benefit. One fair-quality LOE 1 study of administration of low-dose hydrocortisone to children with septic shock[466] demonstrated earlier shock reversal. Data from one LOE 4 hospital discharge database[467] noted the association between the use of steroids in children with severe sepsis and decreased survival.

In one LOE 5 study in adults with septic shock[457] survival improved significantly with the use of low-dose hydrocortisone and fludrocortisone compared with placebo. Conversely four LOE 5 adult trials in septic shock[454,459–461] showed no survival benefit with low-dose corticosteroid therapy. In one large LOE 5 randomised controlled trial of adults in septic shock,[454] corticosteroid administration was associated with an increased risk of secondary infection.

Treatment recommendations

There is insufficient evidence to support or refute the routine use of stress-dose or low-dose hydrocortisone and/or other corticosteroids in infants and children with septic shock. Stress-dose corticosteroids may be considered in children with septic shock unresponsive to fluids and requiring vasoactive support.

Knowledge gaps

What is the appropriate "stress dose" of hydrocortisone for hypotensive septic shock? Should the dose of hydrocortisone be titrated to the degree of shock? Should an adrenocorticotrophin (ACTH) stimulation test be performed to determine if an infant or child in septic shock has adrenal insufficiency?

Diagnostic tests as guide to management of shock[Peds-050A, Peds-050B]

Consensus on science

In one LOE 1 randomised controlled trial in children with severe sepsis or fluid-refractory septic shock,[468] protocol-driven therapy that included targeting a superior vena caval oxygen saturation >70%, coupled with treating clinical signs of shock (prolonged capillary refill, reduced urine output, and reduced blood pressure), improved patient survival to hospital discharge in comparison to treatment guided by assessment of clinical signs alone.

Two LOE 5 studies of adults with septic shock, one a randomised controlled trial[469] and the other a cohort study,[470] documented improved survival to hospital discharge following implementation of protocol-driven early goal-directed therapy, including titration to a central venous oxygen saturation ($ScvO_2$) $\geq$70%. In one large multicentre LOE 5 adult study[471] evaluating the "Surviving Sepsis" bundle, early goal-directed therapy to achieve an $ScvO_2$ $\geq$70% was not associated with an improvement in survival, but venous oxygen saturations were measured in <25% of participants.

There are insufficient data on the utility of other diagnostic tests (e.g., pH, lactate) to help guide the management of infants and children with shock.

Treatment recommendations

A protocol-driven therapy, which includes titration to a superior vena caval oxygen saturation $\geq$70%, may be beneficial for infants and children (without cyanotic congenital heart disease) with fluid-refractory septic shock. No treatment recommendations can be made to target $ScvO_2$ saturation in the management of fluid-refractory septic shock in paediatric patients with cyanotic congenital heart disease or for other forms of paediatric shock.

Knowledge gaps

What is the optimal diagnostic test (i.e., lactate, $ScvO_2$) to guide management of paediatric shock? Does continuous versus intermittent $ScvO_2$ monitoring affect outcome?

Medications in cardiac arrest and bradycardia

The Task Force reviewed and updated evidence to support medications used during cardiac arrest and bradycardia, but no new recommendations were made. It was again emphasised that calcium and sodium bicarbonate should not be routinely used in paediatric cardiac arrest (i.e., should not be used without specific indications).

Calculating drug dose[Peds-017B]

Consensus on science

Eight LOE 5 studies[472–479] concluded that length-based methods are more accurate than age-based or observer (parent or provider) estimate-based methods in the prediction of body weight. Four LOE 5 studies[472,474,480,481] suggested that the addition of a category of body habitus to length may improve prediction of body weight.

Six LOE 5 studies[482–487] attempted to find a formula based on drug pharmacokinetics and physiology that would allow the calculation of a paediatric dose from the adult dose.

Treatment recommendations

In nonobese paediatric patients, initial resuscitation drug doses should be based on actual body weight (which closely approximates ideal body weight). If necessary, body weight can be estimated from body length.

In obese patients the initial doses of resuscitation drugs should be based on ideal body weight that can be estimated from length. Administration of drug doses based on actual body weight in obese patients may result in drug toxicity.

Subsequent doses of resuscitation drugs in both nonobese and obese patients should take into account observed clinical effects and toxicities. It is reasonable to titrate the dose to the desired therapeutic effect, but it should not exceed the adult dose.

Knowledge gaps

What is the most accurate method for calculating resuscitation drug doses for children? Does the accuracy of the estimated weight used to calculate drug dose affect patient outcome? Do specific resuscitation drugs require different adjustments for estimated weight, maturity and/or body composition?

Are formulas for scaling drug doses with formulas from adult doses superior to existing weight-based methods?

Adrenaline dose[Peds-018]

Consensus on science

No studies have compared adrenaline versus placebo administration for pulseless cardiac arrest in infants and children. One LOE 5 randomised controlled adult study[488] of standard drug therapy compared with no drug therapy during out-of-hospital cardiac arrest showed improved survival to hospital admission with any drug delivery but no difference in survival to hospital discharge.

Evidence from one LOE 1 prospective, randomised, controlled trial,[489] two LOE 2 prospective trials,[490,491] and two LOE 2 case series with concurrent controls[492,493] showed no increase in survival to hospital discharge or improved neurological outcome when adrenaline doses of >10 $\mu g\,kg^{-1}$ IV were used in out-of-hospital or in-hospital paediatric cardiac arrest. In one LOE 1 prospective trial[489] of paediatric in-hospital cardiac arrest comparing high-dose (100 $\mu g\,kg^{-1}$) with standard-dose adrenaline administered if cardiac arrest persisted after one standard dose of adrenaline, 24-h survival was reduced in the high-dose adrenaline group.

Evidence extrapolated from adult prehospital or in-hospital studies, including nine LOE 1 randomised trials,[494–502] three LOE 2 trials,[503–505] and three LOE 3 studies,[506–508] showed no improvement in survival to hospital discharge or neurological outcome when doses >1 mg of adrenaline were given.

Treatment recommendations

In infants and children with out-of-hospital or in-hospital cardiac arrest, the appropriate dose of IV adrenaline is 10 $\mu g\,kg^{-1}$ per dose (0.01 $mg\,kg^{-1}$) for the first and for subsequent doses. The maximum single dose is 1 mg.

Knowledge gaps

Does adrenaline administration improve outcome from cardiac arrest in infants and children? Are there specific patients or arrest types (e.g., prolonged arrest, asphyxial arrest, VF arrest) for which adrenaline is more effective?

Sodium bicarbonate during cardiac arrest[Peds-028]

Consensus on science

There are no randomised controlled studies in infants and children examining the use of sodium bicarbonate as part of the management of paediatric cardiac arrest. One LOE 2 multicentre retrospective in-hospital paediatric study[509] found that sodium bicarbonate administered during cardiac arrest was associated with decreased survival, even after controlling for age, gender, and first documented cardiac rhythm.

Two LOE 5 randomised controlled studies have examined the value of sodium bicarbonate in the management of arrest in other populations: one adult out-of-hospital cardiac arrest study[510] and one study in neonates with respiratory arrest in the delivery room.[511] Both failed to show an improvement in overall survival.

Treatment recommendations

Routine administration of sodium bicarbonate is not recommended in the management of paediatric cardiac arrest.

Knowledge gaps

Are there circumstances under which sodium bicarbonate administration improves outcome from paediatric cardiac arrest?

Vasopressin[Peds-020A, Peds-020B]

Consensus on science

In one paediatric LOE 3 study[512] vasopressin was associated with lower ROSC and a trend toward lower 24-h and discharge survival. In three paediatric LOE 4[513–515] and two adult LOE 5[516,517] case series/reports (nine patients) vasopressin[513] or its long-acting analogue, terlipressin,[514,515] administration was associated with ROSC in patients with refractory cardiac arrest (i.e., standard therapy failed).

Extrapolated evidence from six LOE 5 adult studies[518–523] and one LOE 1 adult meta-analysis[524] showed that vasopressin used either by itself or in combination with adrenaline during cardiac arrest does not improve ROSC, hospital discharge, or neurological outcome. Evidence from one LOE 5 animal study[525] of an infant asphyxial arrest model showed no difference in ROSC when terlipressin was administered alone or in combination with adrenaline as compared with adrenaline alone.

Treatment recommendations

There is insufficient evidence for or against the administration of vasopressin or its long-acting analogue, terlipressin, in paediatric cardiac arrest.

Knowledge gaps

Are there patient subgroups who might benefit from vasopressin (with or without other vasopressors) for paediatric cardiac arrest? Does the use of "early" versus "late" (i.e., rescue) vasopressin affect outcome in paediatric cardiac arrest? Is vasopressin effective when administered via a tracheal tube?

Calcium in cardiac arrest[Peds-021A, Peds-021B]

Consensus on science

Evidence from three LOE 2[509,526,527] studies in children and five LOE 5 adult studies[528–532] failed to document an improvement in survival to hospital admission, hospital discharge, or favorable neurological outcome when calcium was administered during cardiopulmonary arrest in the absence of documented hypocalcaemia, calcium channel blocker overdose, hypermagnesaemia, or hyperkalaemia. Four LOE 5 animal studies[533–536] showed no

improvement in ROSC when calcium, compared with adrenaline or placebo, was administered during cardiopulmonary arrest.

Two studies investigating calcium for in-hospital paediatric cardiac arrest suggested a potential for harm. One LOE 2 study examining data from the NRCPR[526] observed an adjusted odds ratio of survival to hospital discharge of 0.6 in children who received calcium, and one LOE 3 multicentre study[509] showed an odds ratio for increased hospital mortality of 2.24 associated with the use of calcium. One LOE 2 study of cardiac arrest in the PICU setting[527] suggested a potential for harm with the administration of calcium during cardiac arrest; the administration of one or more boluses was an independent predictor of hospital mortality.

Treatment recommendations

Routine use of calcium for infants and children with cardiopulmonary arrest is not recommended in the absence of hypocalcaemia, calcium channel blocker overdose, hypermagnesaemia, or hyperkalaemia.

Knowledge gaps

Are there indications for calcium administration that may be associated with improved outcome from paediatric cardiac arrest? Does the increased mortality risk associated with calcium administration reflect harm from calcium or does it simply identify patients who failed to respond to other ALS interventions and therefore were at a higher risk of death?

Atropine versus adrenaline for bradycardia[Peds-052A]

Consensus on science

Evidence from one LOE 3 study of in-hospital paediatric cardiac arrest[537] observed an improved odds of survival to discharge for those patients who received atropine based on multivariate analysis, whereas the use of adrenaline was associated with decreased odds of survival. Another large LOE 3 study[538] demonstrated no association between atropine administration and survival.

In one LOE 5 adult case series,[539] six of eight patients in cardiac arrest who did not respond to adrenaline did respond to atropine with a change to a perfusing rhythm; three survived to hospital discharge. An LOE 5 retrospective adult review[540] observed that a small number of asystolic patients who failed to respond to adrenaline did respond to atropine, but none survived to hospital discharge.

Four LOE 5 adult studies[541–544] showed a benefit of atropine in vagally mediated bradycardia. One small LOE 4 paediatric case series[545] showed that atropine is more effective than adrenaline in increasing heart rate and blood pressure in children with post-cardiac surgical hypotension and bradycardia (Bezold–Jarisch reflex mediated bradycardia).

Four LOE 5 adult[542,546–548] and four LOE 5 animal[549–552] studies showed no benefit from atropine used to treat bradycardia or cardiac arrest. One LOE 5 animal study[553] did show a benefit of atropine when used with adrenaline in cardiac arrest.

Treatment recommendations

Adrenaline may be used for infants and children with bradycardia and poor perfusion that is unresponsive to ventilation and oxygenation. It is reasonable to administer atropine for bradycardia caused by increased vagal tone or cholinergic drug toxicity. There is insufficient evidence to support or refute the routine use of atropine for paediatric cardiac arrest.

Knowledge gaps

What is the optimal dose of adrenaline for paediatric bradycardia? Is there a role for titrated doses? Does the use of adrenaline versus atropine improve outcome from paediatric bradycardia? Are there circumstances under which atropine administration improves outcome from paediatric cardiac arrest?

Extracorporeal cardiac life support[Peds-014, Peds-014B]

There is increasing evidence that extracorporeal cardiac life support (ECLS) can act as a bridge to maintain oxygenation and circulation in selected infants and children with cardiac arrest if they are transplant candidates or have a self-limited or treatable illness. When ECLS is initiated for the treatment of cardiac arrest, it is referred to as ECPR (extracorporeal CPR). ECPR can only be employed if the cardiac arrest occurs in a monitored environment with protocols and personnel for rapid initiation.

Consensus on science

One LOE 2[554] and 26 LOE 4 studies[555–580] reported favorable early outcome after ECPR in children with primary cardiac disease who were located in an ICU or other highly supervised environment using ECPR protocols at the time of the arrest.

One LOE 2[554] and two LOE 4[555,564] studies indicated poor outcome from ECPR in children with noncardiac diseases.

In one LOE 4 study[556] survival following ECPR in children was associated with shorter time interval between arrest and ECPR team activation and shorter CPR duration. Two LOE 4 studies[560,581] found insignificant improvements in outcome after ECPR in children following protocol changes leading to shorter durations of CPR. One LOE 2[554] and three LOE 4[555,559,565] studies found no relationship between CPR duration and outcome after ECPR in children.

Three small LOE 4 studies,[582–584] including a total of 21 children, showed favorable outcome with ECPR following out-of-hospital cardiac arrest associated with environmentally induced severe hypothermia (temperature $\leq$30 °C).

Treatment recommendations

ECPR may be beneficial for infants and children with cardiac arrest if they have heart disease amenable to recovery or transplantation and the arrest occurs in a highly supervised environment such as an ICU with existing clinical protocols and available expertise and equipment to rapidly initiate ECPR. There is insufficient evidence for any specific threshold for CPR duration beyond which survival with ECPR is unlikely. ECPR may be considered in cases of environmentally induced severe hypothermia (temperature $\leq$30 °C) for paediatric patients with out-of-hospital cardiac arrest if the appropriate expertise, equipment, and clinical protocols are in place.

Knowledge gaps

What are the long-term neurological outcomes of paediatric patients treated with ECPR? Is there an upper limit for the duration of standard CPR beyond which using ECPR will be of no benefit?

Post-resuscitation care

The Task Force reviewed evidence regarding hypothermia for paediatric patients who remain comatose following resuscitation from cardiac arrest. There is clear benefit for adult patients who remain comatose after VF arrest, but there is little evidence regarding effectiveness for infants (i.e., beyond the neonatal period) and young children who most commonly have asphyxial arrest.

Some patients with sudden death without an obvious cause have a genetic abnormality of myocardial ion channels (i.e., a channelopathy), which presumably leads to a fatal arrhythmia. Because this is an inherited abnormality, family members might be affected, but special tests are required for the detection of this inherited genetic defect.

Hypothermia[Peds-010A, Peds-010B]

Consensus on science

There are no randomised paediatric studies on induced therapeutic hypothermia following cardiac arrest.

Two prospective randomised LOE 5 studies of adults with VF arrest[585,586] and two prospective randomised LOE 5 studies of newborns with birth asphyxia[587,588] showed that therapeutic hypothermia (32–34 °C) up to 72 h after resuscitation has an acceptable safety profile and may be associated with better long-term neurological outcome.

One LOE 2 observational study[589] neither supports nor refutes the use of therapeutic hypothermia after resuscitation from paediatric cardiac arrest. However, patients in this study were not randomised, and the cooled patients were much sicker and younger than those not cooled.

Treatment recommendations

Therapeutic hypothermia (to 32–34 °C) may be beneficial for adolescents who remain comatose following resuscitation from sudden witnessed out-of-hospital VF cardiac arrest. Therapeutic hypothermia (to 32–34 °C) may be considered for infants and children who remain comatose following resuscitation from cardiac arrest.

Knowledge gaps

Does therapeutic hypothermia improve outcome following paediatric cardiac arrest? Is there a difference in effectiveness for VF arrest versus asphyxial arrest? What is the optimal protocol for cooling after paediatric cardiac arrest (timing, duration, goal temperature, rate of rewarming)?

Vasoactive drugs[Peds-024A, Peds-024B]

Consensus on science

There are no studies evaluating the role of vasoactive medications after ROSC in children. Evidence from two LOE 3 studies in children,[590,591] two LOE 5 studies in adults,[592,593] and two LOE 5 animal studies[594,595] documented that myocardial dysfunction and vascular instability are common following resuscitation from cardiac arrest.

Evidence from six LOE 5 animal studies[594–599] documented haemodynamic improvement when vasoactive medications (dobutamine, milrinone, levosimendan) were given in the post-cardiac arrest period. Evidence from one large LOE 5 paediatric[444] and four LOE 5 adult[600–603] studies of patients with low cardiac output or at risk for low cardiac output following cardiac surgery documented consistent improvement in haemodynamics when vasoactive medications were administered.

Treatment recommendations

It is reasonable to administer vasoactive medications to infants and children with documented or suspected cardiovascular dysfunction after cardiac arrest. These vasoactive medications should be selected and titrated to improve myocardial function and/or organ perfusion while trying to limit adverse effects.

Knowledge gaps

What is the optimal vasoactive drug regimen for postarrest myocardial dysfunction in infants and children?

Glucose[Peds-016]

Consensus on science

There is insufficient evidence to support or refute any specific glucose management strategy in infants and children following cardiac arrest. Although there is an association of hyperglycaemia and hypoglycaemia with poor outcome following ROSC after cardiac arrest, there are no studies that show causation and no studies that show that the treatment of either hyperglycaemia or hypoglycaemia following ROSC improves outcome.

Two studies of adult survivors of cardiac arrest, including one LOE 5 prospective observational study[604] and one LOE 5 randomised controlled trial comparing tight with moderate glucose control[605] observed no survival benefit with tight glucose control. Two studies of tight glucose control in adult surgical ICU patients, including one LOE 1 prospective randomised controlled trial[606] and one LOE 1 meta-analysis[607] observed reduced mortality with tight glucose control. Two LOE 5 meta-analyses comparing tight with moderate glucose control in adult ICU patients[608,609] and one LOE 5 randomised controlled trial comparing tight with moderate glucose control in adult medical ICU patients[610] observed no differences in survival. Three LOE 5 studies of tight glucose control in adult ICU patients, including one randomised controlled trial in cardiac surgical patients,[611] one multicentre randomised controlled trial in medical and surgical ICU patients,[612] and one cohort-controlled study of medical and surgical ICU patients[613] demonstrated increased mortality with tight glucose control.

One LOE 5 randomised controlled trial of critically ill children[614] observed an improvement in inflammatory biochemical markers and reduced ICU length of stay with tight glucose control. One study of tight glucose control of critically ill neonates[615] was terminated early for reasons of futility. Significant rates of hypoglycaemia are widely reported with the use of tight glucose control without explicit methodology or continuous glucose monitoring in critically ill neonates,[615] children,[614] and adults.[607,608,612]

Evidence from LOE 5 animal studies of neonatal cerebral ischaemia[616] and critically ill adults[617,618] suggest that hypoglycaemia combined with hypoxia and ischaemia is harmful and associated with higher mortality. Evidence from three LOE 5 animal studies[619–621] showed that prolonged hyperglycaemia after resuscitation is harmful to the brain. One LOE 5 animal study[622] showed that glucose infusion with associated hyperglycaemia after resuscitation worsened outcome, whereas another LOE 5 animal study[623] showed that moderate hyperglycaemia managed with insulin improved neurological outcome.

Treatment recommendations

It is appropriate to monitor blood glucose levels and avoid hypoglycaemia as well as sustained hyperglycaemia following cardiac arrest. There is insufficient evidence to recommend specific strategies to manage hyperglycaemia in infants and children with ROSC following cardiac arrest. If hyperglycaemia is treated following ROSC in children, blood glucose concentrations should be carefully monitored to reduce episodes of hypoglycaemia.

Knowledge gaps

Does the use of "tight" glucose control improve outcome following paediatric cardiac arrest?

Channelopathy[Peds-048A, Peds-048B]

Consensus on science

In four LOE 4 studies[624–627] 14–35% of young patients with sudden, unexpected death had no abnormalities found at autopsy.

In seven LOE 3 studies[628–634] mutations causing channelopathies occurred in 2–10% of infants with sudden infant death syndrome noted as the cause of death. In one LOE 3[635] and two LOE 4[636,637] studies 14–20% of young adults with sudden, unexpected death had no abnormalities on autopsy but had genetic mutations causing channelopathies. In four LOE 4 studies,[638–641] using clinical and laboratory (electrocardiographic, molecular-genetic screen-

ing) investigations, 22–53% of first- and second-degree relatives of patients with sudden, unexplained death had inherited, arrhythmogenic disease.

Treatment recommendations

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

Knowledge gaps

What is the population-based incidence of inherited arrhythmic deaths in patients with sudden, unexpected death and a negative autopsy? What are the most effective strategies (e.g., for emergency medicine physician, primary care provider, coroner, or others) to identify families at risk?

Special situations

New topics introduced in this document include resuscitation of infants and children with certain congenital cardiac abnormalities, namely single ventricle following stage I procedure and following the Fontan or bidirectional Glenn procedures (BDGs) as well as resuscitation of infants and children with cardiac arrest and pulmonary hypertension.

Life Support for trauma[Peds-041A, Peds-041B]

Consensus on science

Cardiac arrest due to major (blunt or penetrating) injury (out-of-hospital and in-hospital) in children has a very high mortality rate.[642,645] In one LOE 4[645] and one LOE 5[117] study there was no survival advantage to intubating child victims of traumatic cardiac arrest in the out-of-hospital setting. One LOE 2[646] and four LOE 4[647–650] studies suggested that there is minimal survival advantage associated with resuscitative thoracotomy with or without internal cardiac massage for blunt trauma-induced cardiac arrest in children. Two LOE 4 studies[648,649] suggested that survival in children with cardiac arrest from penetrating trauma is improved by thoracotomy if time from event to hospital is short and signs of life are restored in the field.

Treatment recommendations

There is insufficient evidence to make a recommendation for modification of standard resuscitation for infants and children suffering cardiac arrest due to major trauma, although consideration should be given to selectively performing a resuscitative thoracotomy in children with penetrating injuries who arrive at the hospital with a perfusing rhythm.

Knowledge gaps

What is the role of open-chest CPR for nontraumatic etiologies of paediatric cardiac arrest?

Single-ventricle post stage I repair[Peds-059]

Consensus on science

In one LOE 4 case series[651] cardiac arrest occurred frequently (in 20% of 112 patients) in infants following stage I repair for single-ventricle anatomy. Two LOE 5 case series of mechanically ventilated, chemically paralyzed patients with a single ventricle in the preoperative period[652,653] showed that excessive pulmonary blood flow may be attenuated in the short term by increasing the inspired fraction of CO_2 to achieve a $Paco_2$ of 50–60 mm Hg. In the same population, decreasing the fraction of inspired oxygen below 0.21 did not appear to improve systemic oxygen delivery. Three LOE 4 studies[654–656] showed that clinical identification of the prearrest state in patients with a single ventricle is difficult and may be aided by monitoring systemic oxygen extraction using superior vena caval oxygen saturation or near infrared spectroscopy of cerebral and splanchnic circulations.

One LOE 3 prospective, crossover design study[657] of infants following stage I repair showed that inspired carbon dioxide increased systemic oxygen delivery. Evidence from three LOE 4 studies of infants following stage I repair[658–660] showed that reducing systemic vascular resistance with agents such as phenoxybenzamine improved systemic oxygen delivery,[659] reduced the risk for cardiovascular collapse,[658] and improved survival.[660]

There is no evidence for or against any specific modification of standard resuscitation practice for cardiac arrest in infants with single-ventricle anatomy following stage I repair.

Five LOE 4 paediatric studies[555,558,578,661,662] showed that survival to hospital discharge for patients with single-ventricle anatomy following ECPR (see ECPR above) is comparable to that of other neonates undergoing cardiac surgery. In one LOE 4 study[578] survival following ECPR initiated as a consequence of systemic-to-pulmonary artery shunt block after stage I repair was consistently higher than for other etiologies of cardiac arrest.

Treatment recommendations

Standard resuscitation (prearrest and arrest) procedures should be followed for infants and children with single-ventricle anatomy following stage I repair. Neonates with a single ventricle before stage I repair who demonstrate shock caused by elevated pulmonary to systemic flow ratio (Qp-to-Qs ratio) might benefit from inducing mild hypercarbia ($Paco_2$ to 50–60 mm Hg); this can be achieved during mechanical ventilation by reducing minute ventilation, adding CO_2 to inspired air, or administering opioids with or without chemical paralysis.

Neonates in a prearrest state following stage I repair may benefit from α-adrenergic antagonists to treat or ameliorate excessive systemic vasoconstriction in order to improve systemic blood flow and oxygen delivery and reduce the likelihood of cardiac arrest. Assessment of systemic oxygen extraction by monitoring $SvcO_2$ or near infrared spectroscopy monitoring of cerebral and splanchnic circulation may help identify evolving haemodynamic changes in infants following stage I procedures; such haemodynamic changes may herald impending cardiac arrest.

Knowledge gaps

Is there benefit in using heparin or thrombolytics during cardiac arrest to open a potentially occluded systemic-to-pulmonary artery (PA) or right ventricle to pulmonary artery (RV-PA) shunt following stage I repair? What is the role of monitoring near infrared spectroscopy/$SvcO_2$ to guide resuscitation following stage I repair? Is there a potential benefit from the administration of milrinone during the prearrest state in infants with a single ventricle? Is it better to use a pure β-adrenergic agonist (isoprenaline) or an α- and β-agonist (adrenaline) to achieve ROSC after cardiac arrest fol-

lowing stage I repair? Does P_{ETCO_2} reflect pulmonary blood flow in single-ventricle physiology and can it be used to guide resuscitative procedures? Should the inspired oxygen concentration (100% versus room air) be different in infants with single-ventricle physiology during resuscitation from cardiac arrest? How does the Sano modification of Stage I repair (RV-PA conduit instead of a systemic-pulmonary artery shunt) affect response to therapies for cardiac arrest?

Single-ventricle post-Fontan and bidirectional Glenn procedures[Peds-055B]

Consensus on science

In one LOE 4 case series[663] ECLS was useful in resuscitating patients with Fontan circulation but was not successful in hemi-Fontan/BDG patients. One LOE 4 case report[664] described manual external abdominal compressions with closed chest cardiac compressions as an alternative for standard CPR following a modified Fontan procedure.

Evidence from four LOE 5 studies[665–668] of patients with BDG circulation who were not in cardiac arrest or shock supports increasing CO_2 tension and hypoventilation to improve cerebral, superior vena caval, and pulmonary blood flow in order to increase systemic oxygen delivery. In two LOE 5 studies[669,670] of patients with BDG circulation who were not in cardiac arrest or a prearrest state, excessive ventilation reduced cerebral oxygenation. In two LOE 5 studies[671,672] of patients following a Fontan procedure who were not in cardiac arrest or a prearrest state, negative-pressure ventilation improved stroke volume and cardiac output compared with intermittent positive-pressure ventilation.

One LOE 5 case series[673] of patients following a Fontan procedure who were not in cardiac arrest or a prearrest state showed that high-frequency jet ventilation improved pulmonary vascular resistance and cardiac index. However, another LOE 5 case series[674] found that high-frequency oscillation ventilation did not increase cardiac index or decrease pulmonary vascular resistance.

Changes in pulmonary blood flow typically reflect changes in cardiac output, but in infants and children with right-to-left shunts, an increase in right-to-left shunting that bypasses the lungs, as occurs in some infants and children with congenital heart disease or pulmonary hypertension, decreases the proportion of blood flowing through the pulmonary circulation, and as a result, the P_{ETCO_2} falls.[675] Conversely, increasing pulmonary blood flow, as happens following shunt insertion in infants with cyanotic heart disease, increases the P_{ETCO_2} and reduces the difference between the P_{aCO_2} and end-tidal CO_2.[84,85] Likewise, if there are intrapulmonary shunts that bypass the alveoli, there will be a greater difference between the P_{aCO_2} and P_{ETCO_2}.[83]

Treatment recommendations

In patients with Fontan or hemi-Fontan/BDG physiology who are in a prearrest state, hypercarbia achieved by hypoventilation may be beneficial to increase oxygenation and cardiac output, while negative-pressure ventilation, if available, may be beneficial by increasing cardiac output. During cardiopulmonary arrest it is reasonable to consider ECPR for patients with Fontan physiology. There is insufficient evidence to support or refute the use of ECPR in patients with hemi-Fontan/BDG physiology.

Knowledge gaps

What is the optimal method for cannulation for ECPR in patients with hemi-Fontan/BDG or Fontan physiology? What is the optimal CPR strategy (e.g., with or without manual external abdominal compression; with or without active chest decompression; with or without an impedance threshold device) for patients with hemi-Fontan/BDG or Fontan physiology? Is there an ideal compression–ventilation ratio during CPR for infants following hemi-Fontan/BDG or Fontan procedures? Are compression "boots" or a MAST (military antishock trousers) suit beneficial for patients in prearrest states or cardiac arrest following hemi-Fontan/BDG or Fontan procedures?

Pulmonary hypertension[Peds-056A]

Consensus on science

Two LOE 5 observational paediatric studies[676,677] showed that children with pulmonary hypertension are at increased risk for cardiac arrest. There are no studies that demonstrate the superiority of any specific therapy for resuscitation from cardiac arrest in infants and children with a pulmonary hypertensive crisis.

In one LOE 5 retrospective study in adults[678] standard CPR techniques were often unsuccessful in victims with pulmonary hypertension and cardiac arrest. Those who were successfully resuscitated had a reversible cause and received a bolus of IV iloprost or inhaled nitric oxide (NO) during the resuscitation.

One LOE 5 study of adults after cardiac transplant[679] and two LOE 5 studies in children with congenital heart disease[680,681] observed that inhaled NO and aerosolised prostacyclin or analogues appear to be equally effective in reducing pulmonary vascular resistance. In one LOE 5 study in children with pulmonary hypertension after cardiac surgery[682] inhaled NO and alkalosis appeared to be equally effective in reducing pulmonary vascular resistance. There is no evidence of benefit or harm of excessive ventilation for infants and children in cardiac arrest with pulmonary hypertension.

Four LOE 5 studies in pulmonary hypertensive adults and children with crises or cardiac arrest[683–686] showed that mechanical right ventricular support improved survival.

Treatment recommendations

Rescuers should provide conventional paediatric advanced life support, including oxygenation and ventilation for cardiac arrest associated with pulmonary hypertension. It may be beneficial to attempt to correct hypercarbia. If the administration of medications (IV or inhaled) to decrease pulmonary artery pressure has been interrupted, it may be advisable to reinstitute it.

Inhaled NO or aerosolised prostacyclin or analogue to reduce pulmonary vascular resistance should be considered. If unavailable, an IV bolus of prostacyclin may be considered.

Knowledge gaps

Is adrenaline harmful for resuscitation of paediatric patients with pulmonary hypertension who are in prearrest states or cardiac arrest? Is excessive ventilation of infants and children in prearrest states or cardiac arrest in the setting of pulmonary hypertension helpful or harmful? Does vasopressin improve outcome for cardiac arrest in the setting of pulmonary hypertensive crisis? Is a pure β-agonist, such as isoprenaline, effective or harmful during prearrest states or cardiac arrest associated with pulmonary hypertension? If used early in resuscitation, does the use of ECLS improve the outcome of the infant or child with pulmonary hypertension?

Prognosis and decision to terminate CPR[Peds-060]

Consensus on science

In one LOE 3[687] and one LOE 4[688] study, survival from in-hospital paediatric cardiac arrest in the 1980 s was approximately 9%. One LOE 1[538] and one LOE 3 paediatric study[689] showed that survival from in-hospital cardiac arrest in the early 2000 s was 16–18%. Three prognostic LOE 1 prospective observational paediatric stud-

ies from 2006[537,690,691] reported that survival from in-hospital cardiac arrest in 2006 was 26–27%.

One LOE 1 prospective study[300] showed that survival from all paediatric out-of-hospital cardiac arrest was 6% compared with 5% for adults. Survival in infants was 3%, and in children and adolescents survival was 9%. This study demonstrated that earlier poor survival rates were heavily influenced by poor infant survival (many of whom probably had sudden infant death syndrome and had probably been dead for some time).

Thirteen studies (LOE 1[300,301,537,538,690,692,693]; LOE 3[577,687,694]; LOE 4[688,695,696]) studies showed an association between several factors and survival from cardiac arrest. These factors include duration of CPR, number of doses of adrenaline, age, witnessed versus unwitnessed cardiac arrest, obesity,[697] and the first and subsequent cardiac rhythm. Thirteen studies (LOE 1[300,645]; LOE 2[698]; LOE 3[643,650,694,699–703]; LOE 4[704,705]) showed an association between mortality and causes of arrest such as submersion and trauma for out-of-hospital cardiac arrest. None of the associations reported in these studies allow prediction of outcome.

Treatment recommendations

There is insufficient evidence to allow a reliable prediction of success or failure to achieve ROSC or survival from cardiac arrest in infants and children.

Knowledge gaps

Are there reliable prognostic factors to guide decision making to terminate CPR in infants and children? Are there reliable clinical factors to predict neurological outcome following resuscitation from cardiac arrest in infants and children?

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Appendix A. Evidence-based worksheets for Part 10: paediatric basic and advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations

Task Force	WS ID	PICO title	Short title	Authors	URL
Peds	Peds-001A	In infants (<1 year, not including newly born) in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of AEDs (I) compared with standard management (which does not include use of AEDs) (C), improve outcomes (e.g., termination of rhythm, ROSC, survival) (O)?	AEDs in children less than 1 yr	Reylon Meeks	http://circ.ahajournals.org/site/C2010/Peds-001A.pdf
Peds	Peds-001B	In infants (<1 year, not including newly born) in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of AEDs (I) compared with standard management (which does not include use of AEDs) (C), improve outcomes (e.g., termination of rhythm, ROSC, survival) (O)?	AEDs in children less than 1 yr	Antonio Rodriguez-Nunez	http://circ.ahajournals.org/site/C2010/Peds-001B.pdf
Peds	Peds-002A	For infants and children in cardiac arrest, does the use of a pulse check (I) vs assessment for signs of life (C) improve the accuracy of diagnosis of paediatric CPA (O)?	Pulse check accuracy	Aaron Donoghue, James Tibballs	http://circ.ahajournals.org/site/C2010/Peds-002A.pdf
Peds	Peds-003	During cardiac arrest in infants or children (P), does the presence of family members during the resuscitation (I) compared to their absence (C) improve patient or family outcome measures (O)?	Family presence	Douglas Diekema	http://circ.ahajournals.org/site/C2010/Peds-003.pdf
Peds	Peds-004	In infants and children with respiratory failure who undergo tracheal intubation (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of devices (e.g., CO_2 detection device, CO_2 analyser or oesophageal detector device) (I) compared with usual management (C), improve the accuracy of diagnosis of airway placement (O)?	Verification of airway placement	Diana Fendya, Monica Kleinman	http://circ.ahajournals.org/site/C2010/Peds-004.pdf

Appendix A (*Continued*)

Task Force	WS ID	PICO title	Short title	Authors	URL
Peds	Peds-005A	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of end-tidal CO_2 (I), compared with clinical assessment (C), improve accuracy of diagnosis of a perfusing rhythm (O)?	End-tidal CO_2 to diagnose perfusing rhythm	Arno Zaritsky	http://circ.ahajournals.org/site/C2010/Peds-005A.pdf
Peds	Peds-005B	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of end-tidal CO_2 (I), compared with clinical assessment (C), improve accuracy of diagnosis of a perfusing rhythm (O)?	End-tidal CO_2 to diagnose perfusing rhythm	Anne-Marie Guerguerian	http://circ.ahajournals.org/site/C2010/Peds-005B.pdf
Peds	Peds-006B	In paediatric patients in clinical cardiac arrest (prehospital [OHCA] or in hospital [IHCA]) (P), does the use of a focused echocardiogram (I) compared with standard assessment, assist in the diagnosis of reversible causes of cardiac arrest?	Methods to diagnose perfusing rhythm	Christoph Eich, Faiqa Qureshi	http://circ.ahajournals.org/site/C2010/Peds-006B.pdf
Peds	Peds-007	In children requiring emergent intubation (prehospital, in-hospital) (P), does the use of cuffed ETTs (I) compared with uncuffed ETTs (C) improve therapeutic endpoints (e.g., oxygenation and ventilation) or reduce morbidity or risk of complications (e.g., need for tube change, airway injury, aspiration) (O)?	Cuffed vs uncuffed ETTs	Ashraf Coovadia	http://circ.ahajournals.org/site/C2010/Peds-007.pdf
Peds	Peds-008	In children requiring assisted ventilation (prehospital, in-hospital) (P), does the use of bag-valve-mask (I) compared with tracheal intubation (C) improve therapeutic endpoints (oxygenation and ventilation), reduce morbidity or risk of complications (e.g., aspiration), or improve survival (O)?	BVM vs intubation	Dominique Biarent	http://circ.ahajournals.org/site/C2010/Peds-008.pdf
Peds	Peds-009	In paediatric patients in cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of supraglottic airway devices (I) compared with bag-valve-mask alone (C), improve therapeutic endpoints (e.g., ventilation and oxygenation), improve quality of resuscitation (e.g., reduce hands-off time, allow for continuous compressions), reduce morbidity or risk of complications (e.g., aspiration) or improve survival (O)?	Supraglottic airway devices	Robert Bingham	http://circ.ahajournals.org/site/C2010/Peds-009.pdf
Peds	Peds-010A	For infants and children who have ROSC after cardiac arrest (P), does the use of induced hypothermia (I) compared with normothermia (C) improve outcome (survival to discharge, survival with good neurological outcome) (O)?	Induced hypothermia after ROSC	Robert Hickey	http://circ.ahajournals.org/site/C2010/Peds-010A.pdf
Peds	Peds-010B	For infants and children who have ROSC after cardiac arrest (P), does the use of induced hypothermia (I) compared with normothermia (C) improve outcome (survival to discharge, survival with good neurological outcome) (O)?	Induced hypothermia after ROSC	Jamie Hutchison	http://circ.ahajournals.org/site/C2010/Peds-010B.pdf
Peds	Peds-011B	In infants and children with cardiac arrest from a non-asphyxial or asphyxial cause (excluding newborns) (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of another specific C:V ratio by laypersons and HCPs (I) compared with standard care (15:2) (C), improve outcome (e.g., ROSC, survival) (O)?	Compression–ventilation ratio	Robert Bingham, Robert Hickey	http://circ.ahajournals.org/site/C2010/Peds-011B.pdf
Peds	Peds-012A	In infants and children (not including newborns) with cardiac arrest (out-of-hospital and in-hospital) (P), does the use of compression-only CPR (I) as opposed to standard CPR (ventilations and compressions) (C), improve outcome (O) (e.g., ROSC, survival)?	Compression-only CPR	Robert A. Berg, Dominique Biarent	http://circ.ahajournals.org/site/C2010/Peds-012A.pdf
Peds	Peds-013A	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) and a secure airway (P), does the use of a specific minute ventilation (combination of respiratory rate and tidal volume) depending on the aetiology of the arrest (I) as opposed to standard care (8–10 asynchronous breaths/min) (C), improve outcome (O) (e.g., ROSC, survival)?	Aetiology specific minute ventilation	Monica Kleinman	http://circ.ahajournals.org/site/C2010/Peds-013A.pdf

Appendix A (*Continued*)

Task Force	WS ID	PICO title	Short title	Authors	URL
Peds	Peds-013B	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) and a secure airway (P), does the use of a specific minute ventilation (combination of respiratory rate and tidal volume) depending on the aetiology of the arrest (I) as opposed to standard care (8–10 asynchronous breaths/min) (C), improve outcome (O) (e.g., ROSC, survival)?	Aetiology specific minute ventilation	Naoki Shimizu	http://circ.ahajournals.org/site/C2010/Peds-013B.pdf
Peds	Peds-014	In paediatric patients in cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P) does the use of rapid deployment ECMO or emergency cardiopulmonary bypass (I), compared with standard treatment (C), improve outcome (ROSC, survival to hospital discharge, survival with favorable neurological outcomes) (O)?"	ECMO	Marilyn Morris	http://circ.ahajournals.org/site/C2010/Peds-014.pdf
Peds	Peds-014B	In paediatric patients in cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P) does the use of rapid deployment ECMO or emergency cardiopulmonary bypass (I), compared with standard treatment (C), improve outcome (ROSC, survival to hospital discharge, survival with favorable neurological outcomes) (O)?	ECMO	Kate Brown	http://circ.ahajournals.org/site/C2010/Peds-014B.pdf
Peds	Peds-015	In paediatric patients in cardiac arrest, associated with or without asphyxia (prehospital [OHCA] or in-hospital [IHCA]) (P) does ventilation with a specific oxygen concentration (room air or a titrated concentration between 0.21 and 1.0) (I), compared with standard treatment (100% oxygen) (C), improve outcome (ROSC, survival to hospital discharge, survival with favorable neurological outcome) (O)?	Titrated oxygen vs 100% oxygen	Robert Hickey	http://circ.ahajournals.org/site/C2010/Peds-015.pdf
Peds	Peds-016	In infants and children with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of a specific strategy to manage blood glucose (e.g., target range) (I) as opposed to standard care (C), improve outcome (O) (e.g., survival)?	Glucose control following resuscitation	Duncan Macrae, Vijay Srinivasan	http://circ.ahajournals.org/site/C2010/Peds-016.pdf
Peds	Peds-017B	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of any specific alternative method for calculating drug dosages (I) compared with standard weight-based dosing (C), improve outcome (e.g., achieving expected drug effect, ROSC, survival, avoidance of toxicity) (O)?	Methods for calculating drug dosages	Ian Maconochie, Vijay Srinivasan	http://circ.ahajournals.org/site/C2010/Peds-017B.pdf
Peds	Peds-018	In adult and paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of any specific alternative dosing regimen for adrenaline (I) compared with standard recommendations (C), improve outcome (e.g., ROSC, survival to hospital discharge, survival with favorable neurological outcome) (O)?	Adrenaline dose	Amelia Reis	http://circ.ahajournals.org/site/C2010/Peds-018.pdf
Peds	Peds-019	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) due to VF/pulseless VT (P), does the use of amiodarone (I) compared with lidocaine (C), improve outcome (e.g., ROSC, survival to hospital discharge, survival with favorable neurological outcome) (O)?	Amiodarone vs lidocaine for VF/VT	Dianne Atkins	http://circ.ahajournals.org/site/C2010/Peds-019.pdf
Peds	Peds-020A	In adult and paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of vasopressin or vasopressin + adrenaline (I) compared with standard treatment recommendations (C), improve outcome (e.g., ROSC, survival to hospital discharge, or survival with favorable neurological outcome) (O)?	Vasopressin	Elise van der Jagt	http://circ.ahajournals.org/site/C2010/Peds-020A.pdf

Appendix A (*Continued*)

Task Force	WS ID	PICO title	Short title	Authors	URL
Peds	Peds-020B	In adult and paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of vasopressin or vasopressin + adrenaline (I) compared with standard treatment recommendations (C), improve outcome (e.g., ROSC, survival to hospital discharge, or survival with favorable neurological outcome) (O)?	Vasopressin	Dominique Biarent	http://circ.ahajournals.org/site/C2010/Peds-020B.pdf
Peds	Peds-021A	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of calcium (I) compared with no calcium (C), improve outcome (O) (e.g., ROSC, survival to hospital discharge, survival with favorable neurological outcome)?	Calcium	Allan de Caen	http://circ.ahajournals.org/site/C2010/Peds-021A.pdf
Peds	Peds-021B	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of calcium (I) compared with no calcium (C), improve outcome (O) (e.g., ROSC, survival to hospital discharge, survival with favorable neurological outcome)?	Calcium	Felipe Martinez, Sergio Pesutic, Sergio Rendich	http://circ.ahajournals.org/site/C2010/Peds-021B.pdf
Peds	Peds-022A	In paediatric patients with cardiac arrest due to primary or secondary VF or pulseless VT (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of more than one shock for the initial or subsequent defibrillation attempt(s) (I), compared with standard management (C), improve outcome (e.g., termination of rhythm, ROSC, survival to hospital discharge, survival with favorable neurological outcome) (O)?	Single or stacked shocks	Marc Berg	http://circ.ahajournals.org/site/C2010/Peds-022A.pdf
Peds	Peds-023A	In paediatric patients with cardiac arrest due to primary or secondary VF or pulseless VT (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of a specific energy dose or regimen of energy doses for the initial or subsequent defibrillation attempt(s) (I), compared with standard management (C), improve outcome (e.g., termination of rhythm, ROSC, survival to hospital discharge, survival with favorable neurological outcome) (O)?	Energy doses	Jonathan Egan	http://circ.ahajournals.org/site/C2010/Peds-023A.pdf
Peds	Peds-023B	In paediatric patients with cardiac arrest due to primary or secondary VF or pulseless VT (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of a specific energy dose or regimen of energy doses for the initial or subsequent defibrillation attempt(s) (I), compared with standard management (C), improve outcome (e.g., termination of rhythm, ROSC, survival to hospital discharge, survival with favorable neurological outcome) (O)?	Energy doses	Dianne Atkins	http://circ.ahajournals.org/site/C2010/Peds-023B.pdf
Peds	Peds-024A	In paediatric patients with ROSC after cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) who have signs of cardiovascular dysfunction (P), does the use of any specific cardioactive drugs (I) as opposed to standard care (or different cardioactive drugs) (C), improve physiological endpoints (oxygen delivery, haemodynamics) or patient outcome (e.g., survival to discharge or survival with favorable neurological outcome) (O)?	Cardioactive drugs post-resuscitation	Allan de Caen	http://circ.ahajournals.org/site/C2010/Peds-024A.pdf
Peds	Peds-024B	In paediatric patients with ROSC after cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) who have signs of cardiovascular dysfunction (P), does the use of any specific cardioactive drugs (I) as opposed to standard care (or different cardioactive drugs) (C), improve physiological endpoints (oxygen delivery, haemodynamics) or patient outcome (e.g., survival to discharge or survival with favorable neurological outcome) (O)?	Cardioactive drugs post-resuscitation	Mark Coulthard	http://circ.ahajournals.org/site/C2010/Peds-024B.pdf
Peds	Peds-025A	In paediatric patients with in-hospital cardiac or respiratory arrest (P), does use of EWSS/response teams/MET systems (I) compared with no such responses (C), improve outcome (e.g., reduce rate of cardiac and respiratory arrests and in-hospital mortality) (O)?	METs	Elise van der Jagt	http://circ.ahajournals.org/site/C2010/Peds-025A.pdf

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Task Force	WS ID	PICO title	Short title	Authors	URL
Peds	Peds-025B	In paediatric patients with in-hospital cardiac or respiratory arrest (P), does use of EWSS/response teams/MET systems (I) compared with no such responses (C), improve outcome (e.g., reduce rate of cardiac and respiratory arrests and in-hospital mortality) (O)?	METs	James Tibballs	http://circ.ahajournals.org/site/C2010/Peds-025B.pdf
Peds	Peds-026A	For intubated newborns within the first month of life (beyond the delivery room) who are receiving chest compressions (P), does the use of continuous chest compressions (without pause for ventilation) (I) vs chest compressions with interruptions for ventilation (C) improve outcome (time to sustained heart rate >100, survival to ICU admission, survival to discharge, survival with favorable neurological status) (O)?	Continuous chest compressions for intubated newborns outside of DR	Monica Kleinman	http://circ.ahajournals.org/site/C2010/Peds-026A.pdf
Peds	Peds-027A	For newborns within the first month of life (beyond the delivery room) who are not intubated and who are receiving CPR (P), does the use of a 3:1 compression to ventilation ratio (I), compared with a 15:2 compression to ventilation ratio (C) improve outcome (time to sustained heart rate >100, survival to ICU admission, survival to discharge, discharge with favorable neurological status) (O)?	3:1 vs 15:2 ratio for neonates outside of DR	Leon Chameides	http://circ.ahajournals.org/site/C2010/Peds-027A.pdf
Peds	Peds-028	In paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (including prolonged arrest states) (P), does the use of $NaHCO_3$ (I) compared with no $NaHCO_3$ (C), improve outcome (O) (e.g., ROSC, survival)?	Sodium bicarbonate	Stephen M. Schexnayder	http://circ.ahajournals.org/site/C2010/Peds-028.pdf
Peds	Peds-029	In infants and children in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific paddle/pad size/orientation and position (I) compared with standard resuscitation or other specific paddle/pad size/orientation and position) (C), improve outcomes (e.g., successful defibrillation, ROSC, survival) (O)?	Paddle size and placement for defibrillation	Dianne Atkins	http://circ.ahajournals.org/site/C2010/Peds-029.pdf
Peds	Peds-030	In infants and children with unstable ventricular tachycardia (prehospital and in-hospital) (P), does the use of any drug/combination of drugs/intervention (e.g., cardioversion) (I) compared with no drugs/intervention (C) improve outcome (e.g., termination of rhythm, survival) (O)?	Drugs for unstable tachycardia	Jeffrey Berman, Bradford Harris	http://circ.ahajournals.org/site/C2010/Peds-030.pdf
Peds	Peds-031	In infants and children with supraventricular tachycardia with a pulse (P), does the use of any drug or combination of drugs (I), compared with adenosine (C), result in improved outcomes (termination of rhythm, survival)?	Drugs for SVT	Ricardo Samson	http://circ.ahajournals.org/site/C2010/Peds-031.pdf
Peds	Peds-032	In infants and children with haemorrhagic shock following trauma (P), does the use of graded volume resuscitation (I) as opposed to standard care (C), improve outcome (haemodynamics, survival) (O)?	Graded volume resuscitation for traumatic shock	Jesus Lopez-Herce	http://circ.ahajournals.org/site/C2010/Peds-032.pdf
Peds	Peds-033	In paediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of one-hand chest compressions (I) compared with two-hand chest compressions (C) improve outcomes (e.g., ROSC, rescuer performance) (O)?	One-hand vs two-hand compressions	Sharon Kinney	http://circ.ahajournals.org/site/C2010/Peds-033.pdf
Peds	Peds-034	In infants with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of two-thumb chest compression without circumferential squeeze (I) compared to two-thumb chest compression with circumferential squeeze (C) improve outcome (e.g., ROSC, rescuer performance (O)?	Two thumb vs two finger	James Tibballs	http://circ.ahajournals.org/site/C2010/Peds-034.pdf
Peds	Peds-035	In infants and children with cardiac arrest (P), does establishing intraosseous access (I) compared to establishing conventional (non-intraosseous) venous access (C) improve patient outcome (e.g., ROSC, survival to hospital discharge (O)?	IO vs IV	Jonathan Duff	http://circ.ahajournals.org/site/C2010/Peds-035.pdf

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Task Force	WS ID	PICO title	Short title	Authors	URL
Peds	Peds-036	In infants and children with cardiac arrest (P), does the use of tracheal drug delivery (I) compared to intravenous drug delivery (C) worsen patient outcome (e.g., ROSC, survival to hospital discharge (O)?	ET vs IV drugs	Mioara D. Manole	http://circ.ahajournals.org/site/C2010/Peds-036.pdf
Peds	Peds-038B	In infants and children in shock, does early intubation and assisted ventilation compared to the use of these interventions only for associated respiratory failure lead to improved patient outcome (haemodynamics, survival?)	Intubation for shock (timing)	Amelia Reis	http://circ.ahajournals.org/site/C2010/Peds-038B.pdf
Peds	Peds-039A	In infants and children with respiratory failure who require emergent tracheal intubation (P), does the use of cricoid pressure or laryngeal manipulation (I), when compared with standard practice (C), improve or worsen outcome (e.g., success of intubation, aspiration risk, side effects) (O)?	Cricoid pressure and laryngeal manipulation	Lester Proctor	http://circ.ahajournals.org/site/C2010/Peds-039A.pdf
Peds	Peds-039B	In infants and children with respiratory failure who require emergent tracheal intubation (P), does the use of cricoid pressure or laryngeal manipulation (I), when compared with standard practice (C), improve or worsen outcome (e.g., success of intubation, aspiration risk, side effects) (O)?	Cricoid pressure and laryngeal manipulation	Ian Maconochie	http://circ.ahajournals.org/site/C2010/Peds-039B.pdf
Peds	Peds-040A	In infants and children in cardiac arrest (out-of-hospital and in-hospital) (P), does any specific compression depth (I) as opposed to standard care (i.e., depth specified in treatment algorithm) (C), improve outcome (O) (e.g., Blood pressure, ROSC, survival)? Note: BLS is doing their own worksheet.	Compression depth	Robert Michael Sutton	http://circ.ahajournals.org/site/C2010/Peds-040A.pdf
Peds	Peds-040B	In infants and children in cardiac arrest (out-of-hospital and in-hospital) (P), does any specific compression depth (I) as opposed to standard care (i.e., depth specified in treatment algorithm) (C), improve outcome (O) (e.g., Blood pressure, ROSC, survival)? Note: BLS is doing their own worksheet.	Compression depth	David Zideman	http://circ.ahajournals.org/site/C2010/Peds-040B.pdf
Peds	Peds-041A	In children and infants with cardiac arrest due to major (blunt or penetrating) injury (out-of-hospital and in-hospital) (P), does the use of any specific modifications to standard resuscitation (I) compared with standard resuscitation (C), improve outcome (O) (e.g., ROSC, survival)? e.g., open vs closed chest CPR, other examples	Traumatic arrest	Kennith Sartorelli	http://circ.ahajournals.org/site/C2010/Peds-041A.pdf
Peds	Peds-041B	In children and infants with cardiac arrest due to major (blunt or penetrating) injury (out-of-hospital and in-hospital) (P), does the use of any specific modifications to standard resuscitation (I) compared with standard resuscitation (C), improve outcome (O) (e.g., ROSC, survival)? e.g., open vs closed chest CPR, other examples	Traumatic arrest	Jesus Lopez-Herce	http://circ.ahajournals.org/site/C2010/Peds-041B.pdf
Peds	Peds-043A	In infants and children in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of self-adhesive defibrillation pads (I) compared with paddles (C), improve outcomes (e.g., successful defibrillation, ROSC, survival) (O)?	Hands-off defibrillation vs paddles	Mark Terry	http://circ.ahajournals.org/site/C2010/Peds-043A.pdf
Peds	Peds-043B	In infants and children in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of self-adhesive defibrillation pads (I) compared with paddles (C), improve outcomes (e.g., successful defibrillation, ROSC, survival) (O)?	Hands-off defibrillation vs paddles	Farhan Bhanji	http://circ.ahajournals.org/site/C2010/Peds-043B.pdf
Peds	Peds-044A	In infants and children with any type of shock (P), does the use of any specific resuscitation fluid or combination of fluids [eg: isotonic crystalloid, colloid, hypertonic saline, blood products] (I) when compared with standard care (C) improve patient outcome (haemodynamics, survival) (O)?	Resuscitation fluids	Sharon Mace	http://circ.ahajournals.org/site/C2010/Peds-044A.pdf

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Task Force	WS ID	PICO title	Short title	Authors	URL
Peds	Peds-044B	In infants and children with any type of shock (P), does the use of any specific resuscitation fluid or combination of fluids [eg: isotonic crystalloid, colloid, hypertonic saline, blood products] (I) when compared with standard care (C) improve patient outcome (haemodynamics, survival) (O)?	Resuscitation fluids	Richard Aickin	http://circ.ahajournals.org/site/C2010/Peds-044B.pdf
Peds	Peds-045A	In infants and children with distributive shock with and without myocardial dysfunction (P), does the use of any specific inotropic agent (I) when compared to standard care (C), improve patient outcome (haemodynamics, survival) (O)?	Distributive shock and inotropes	Ericka Fink, Alfredo Misraji	http://circ.ahajournals.org/site/C2010/Peds-045A.pdf
Peds	Peds-045B	In infants and children with distributive shock with and without myocardial dysfunction (P), does the use of any specific inotropic agent (I) when compared to standard care (C), improve patient outcome (haemodynamics, survival) (O)?	Distributive shock and inotropes	Loh Tsee Foong	http://circ.ahajournals.org/site/C2010/Peds-045B.pdf
Peds	Peds-046A	In infants and children with cardiogenic shock (P), does the use of any specific inotropic agent (I) when compared with standard care (C), improve patient outcome (haemodynamics, survival) (O)?	Cardiogenic shock and inotropes	Akira Nishisaki	http://circ.ahajournals.org/site/C2010/Peds-046A.pdf
Peds	Peds-047A	In infants and children with hypotensive septic shock (P), does the use of etomidate as an induction agent to facilitate intubation (I) compared with a standard technique without etomidate (C) improve patient outcome (haemodynamics, survival) (O)?	Etomidate and septic shock	Stephen M. Schexnayder	http://circ.ahajournals.org/site/C2010/Peds-047A.pdf
Peds	Peds-047B	In infants and children with hypotensive septic shock (P), does the use of etomidate as an induction agent to facilitate intubation (I) compared with a standard technique without etomidate (C) improve patient outcome (haemodynamics, survival) (O)?	Etomidate and septic shock	Jonathan Duff	http://circ.ahajournals.org/site/C2010/Peds-047B.pdf
Peds	Peds-048A	In infants and children who are undergoing resuscitation from cardiac arrest (P), does consideration of a channelopathy as the aetiology of the arrest (I), as compared with standard management (C), improve outcome (ROSC, survival to discharge, survival with favorable neurological outcome) (O)?	Channelopathies	Robert Hickey	http://circ.ahajournals.org/site/C2010/Peds-048A.pdf
Peds	Peds-048B	In infants and children who are undergoing resuscitation from cardiac arrest (P), does consideration of a channelopathy as the aetiology of the arrest (I), as compared with standard management (C), improve outcome (ROSC, survival to discharge, survival with favorable neurological outcome) (O)?	Channelopathies	William Scott	http://circ.ahajournals.org/site/C2010/Peds-048B.pdf
Peds	Peds-049A	In infants and children with hypotensive septic shock (P), does the use of corticosteroids in addition to standard care (I) when compare with standard care without the use of corticosteroids (C), improve patient outcome (e.g., Haemodynamics or survival) (O)?	Corticosteroids and septic shock	Arno Zaritsky	http://circ.ahajournals.org/site/C2010/Peds-049A.pdf
Peds	Peds-049B	In infants and children with hypotensive septic shock (P), does the use of corticosteroids in addition to standard care (I) when compare with standard care without the use of corticosteroids (C), improve patient outcome (e.g., Haemodynamics or survival) (O)?	Corticosteroids and septic shock	Mark Coulthard	http://circ.ahajournals.org/site/C2010/Peds-049B.pdf
Peds	Peds-050A	In infants and children with acute illness or injury (P), do specific diagnostic tests (laboratory data [mixed venous oxygen saturation, pH, lactate], (I) as opposed to clinical data (vital signs, capillary refill, mental status, end-organ function [urine output]) (C), increase the accuracy of diagnosis of shock (O)?	Diagnostic tests for shock	Alexis Topjian	http://circ.ahajournals.org/site/C2010/Peds-050A.pdf
Peds	Peds-050B	In infants and children with acute illness or injury (P), do specific diagnostic tests (laboratory data [mixed venous oxygen saturation, pH, lactate], (I) as opposed to clinical data (vital signs, capillary refill, mental status, end-organ function [urine output]) (C), increase the accuracy of diagnosis of shock (O)?	Diagnostic tests for shock	Sharon Kinney	http://circ.ahajournals.org/site/C2010/Peds-050B.pdf

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Task Force	WS ID	PICO title	Short title	Authors	URL
Peds	Peds-052A	In infants and children with cardiac arrest or symptomatic bradycardia that is unresponsive to oxygenation and/or ventilation (P), does the use of atropine (I), as compared with epinephrine or no atropine (C), improve patient outcome (return to age-appropriate heart rate, subsequent pulseless arrest, ROSC, survival) (O)?	Atropine vs adrenaline for bradycardia	Susan Fuchs, Sasa Kurosawa, Masahiko Nitta	http://circ.ahajournals.org/site/C2010/Peds-052A.pdf
Peds	Peds-055B	For infants and children with Fontan or hemi-Fontan circulation who require resuscitation from cardiac arrest or prearrest states (prehospital [OHCA] or in-hospital [IHCA]) (P), does any specific modification to standard practice (I) compared with standard resuscitation practice (C) improve outcome (e.g., ROSC, survival to discharge, survival with good neurological outcome(O)?	Resuscitation for hemi-Fontan/Fontan circulation	Desmond Bohn, Bradley Marino	http://circ.ahajournals.org/site/C2010/Peds-055B.pdf
Peds	Peds-056A	For infants and children in cardiac arrest with pulmonary hypertension (prehospital [OHCA] or in-hospital [IHCA]) (P), do any specific modifications to resuscitation techniques (I) compared with standard resuscitation techniques (C), improve outcome (ROSC, survival to discharge, favorable neurological survival) (O)?	Resuscitation of the patient with pulmonary hypertension	Ian Adatia, John Berger, David Wessel	http://circ.ahajournals.org/site/C2010/Peds-056A.pdf
Peds	Peds-057A	For infants and children who require tracheal intubation (prehospital or in hospital) (P) does the use of a specific formula to guide cuffed tracheal tube size (I), as opposed to the use of the existing formula of 3 + age/4 (C), achieve better outcomes (e.g., successful tube placement) (O)?	Formulae for cuffed ET tube size	Robert Bingham	http://circ.ahajournals.org/site/C2010/Peds-057A.pdf
Peds	Peds-057B	For infants and children who require tracheal intubation (prehospital or in hospital) (P) does the use of a specific formula to guide cuffed tracheal tube size (I), as opposed to the use of the existing formula of 3 + age/4 (C), achieve better outcomes (e.g., successful tube placement) (O)?	Formulas for predicting ET tube size	Eugene Freid	http://circ.ahajournals.org/site/C2010/Peds-057B.pdf
Peds	Peds-058B	In paediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of invasive monitoring (I) compared with clinical assessment (C), improve accuracy of diagnosis of a perfusing rhythm (O)?	Invasive monitoring for diagnosing perfusing rhythm	Antonio Rodriguez-Nunez	http://circ.ahajournals.org/site/C2010/Peds-058B.pdf
Peds	Peds-059	For infants and children with single ventricle, s/p stage I repair who require resuscitation from cardiac arrest or prearrest states (prehospital [OHCA] or in-hospital [IHCA]) (P), does any specific modification to standard practice (I) compared with standard resuscitation practice (C) improve outcome (e.g., ROSC, survival to discharge, survival with good neurological outcome)(O)?	Resuscitation of the patient with single ventricle	George Hoffman, Shane Tibby	http://circ.ahajournals.org/site/C2010/Peds-059.pdf
Peds	Peds-060	For paediatric patients (in any setting (P), is there a clinical decision rule (I) that enables reliable prediction of ROSC (or futile resuscitation efforts)? (PROGNOSIS)	Clinical decision rules to predict ROSC	Gabrielle Nuthall	http://circ.ahajournals.org/site/C2010/Peds-060.pdf

Appendix B.

CoSTR Part 10: writing group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Monica E. Kleinman	Children's Hospital Anesthesia Foundation: Non-profit organization—Senior Associate in Critical Care Medicine	None	None	None	None	None	None
Allan R. de Caen	Self-employed, Clinical Associate Professor Pediatric Critical Care Medicine, Stollery Children's Hospital/ University of Alberta	None	None	None	None	None	[a]Medical Expert for the Canadian Medical Protective Association
Dianne L. Atkins	University of Iowa: Medical School—Professor [b]Serving as a Worksheet editor for 2010 Guidelines Process. Compensation is paid partially to my institution (66%) and partially to me (34%). The salary from my institution is not altered by this	None	None	None	None	None	[a]Serving as a defense expert witness in a case of ventricular fibrillation in a 2 year old child. Compensation is paid directly to me
Marc D. Berg	University of Arizona—Assoc. Prof. Clinical Pediatrics; Attending Physician's Healthcare (UPH): Also serve on the UPH Board of Directors—Intensivist, Pediatric Critical Care Medicine	None	None	None	None	None	None
Robert A. Berg	U of Pennsylvania—Professor	None	None	None	None	None	None
Farhan Bhanji	Montreal Children's Hospital, McGill University—Assistant Professor of Pediatrics	None	None	None	None	None	None
Dominique Biarent	Hôpital Universitaire des Enfants reine Fabiola: PICU Director	None	None	None	None	None	[a]Medical expert consultant in one trial for an insurance co. (Fortis) Medical expert for legal proceedings [b]Grant of 25,000 euro from "Fondation Roi Baudoin" to the non profit organization "sauvez mon enfant" for a psychological research in the PICU (I am administrator of the non profit organization and promotor of research). The grant is not given to me but to the NPO [b]Grant of 67,500 euro from the Belgian "Loterie Nationale" to build a teaching lab to teach paediatric resuscitation to health care provider [b]Grant given to the non profit organization "sauvez mon enfant" for psychological research in the PICU (I am administrator of the non profit organization and promotor of the research) the grant is not given to me but to the NPO [b]Grant from Baxter Foundation to pay a psychologist in my PICU ($44.540). The grant is paid to the NPO
Robert Bingham	National Health Service of England—Consultant paediatric anaesthetist	None	None	None	None	None	[a]Occasional expert witness reports on pediatric resuscitation related topics

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Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Leon Chameides	Emeritus Director, Pediatric Cardiology, Connecticut Children's Medical Center; Clinical Professor, University of Connecticut	None	None	None	None	None	None
Ashraf Coovadia	Rahima Moosa Mother and Child Hospital: Paediatric Consultant (Attending) in Department of Paediatrics and Child Health—Adjunct Professor in Paediatrics	None	None	None	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing—Prof; AHA ECC Product Dev.—Senior Science Editor. I receive significant compensation from the AHA as consultant/SSE to provide protected time to serve as the co-editor of the 2010 ILCOR CoSTR and the 2010 AHA Guidelines for CPR and ECC	None	None	None	None	None	None
Robert W. Hickey	University of Pittsburgh—Associate Professor	[b]Salary support from NIH for examining cyclopentenone prostaglandin effects in ischaemic brain injury	None	None	None	None	[a]1–2×/year medical malpractice expert
Vinay M. Nadkarni	University of Pennsylvania School of Medicine, Children's Hospital of Philadelphia: Non-profit, Academic, University Hospital—Attending Physician, Anesthesia, Critical Care and Pediatrics	[b]NIH RO1: Therapeutic Hypothermia after Cardiac Arrest, Co-Investigator. [b]Center of Excellence Grant, Laerdal Foundation for Acute Care Medicine, PI: Understanding the mechanics and quality of CPR [a]NHTSA: Mechanics of Chest Compression in children, Co-Investigator	None	None	None	None	None
Amelia Reis	Hospital das Clinico Universidade de Sao Paulo, Pediatric Emergency Physician	None	None	None	None	None	None

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Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Antonio Rodriguez-Nunez	Hospital Clinico Universitario de Santiago de Compostela—Pediatric Emergency and Critical Care Division; University of Santiago de Compostela—Associate Professor of Pediatrics	None	None	None	None	None	None
James Tibballs	Royal Children's Hospital, Melbourne Healthcare, Physician ICU	None	None	None	None	None	None
Arno L. Zaritsky	Children's Hospital of The King's Daughters—Sr. VP for Clinical Services	None	None	None	None	[a]Data Safety Monitoring Board for NIH-sponsored trial of therapeutic hypothermia after pediatric cardiac arrest	None
David Zideman	Imperial College NHS Trust: United Kingdom Healthcare provider—Consultant Anaesthetist; London Organizing Committee of the Olympic Games—Lead Clinician for EMS	None	None	None	None	None	[a]Expert testimony on Cardiac Arrest under General Anaesthesis to Her Majesty's Coroner for Surrey (Expert witness fee for case review and court appearance) less than 1500 US dollars

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

CoSTR Part 10: worksheet collaborator disclosures

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership Interest	Consultant/ advisory board	Other
Ian Adatia	TBD	TBD	TBD	TBD	TBD	TBD	TBD
Richard Aickin	Auckland District Health Board Government Funded Healthcare Provider (primary care through to tertiary hospital services) for Auckland population and for national tertiary services. Director of Child Health	None	None	None	None	[a]New Zealand Health and Disability Commission: occasional expert reports provided with respect to alleged breaches of healthcare standards. 1-2 reports per year. Small personal payment received	[a]Expert Witness: Occasional expert testimony for Coroner's Court and criminal (Child protection) cases. Approx 1× year. No personal payment—small payment to Auckland District Health Board for my time.
John Berger	Children's National Medical Center Non-profit children's hospital Medical Director, Cardiac Intensive Care and Pulmonary Hypertension	[b]5 U 10 HD 049981—DL Wessel (PI) 12/1/09/–11/30/14 Sponsor: NIH/NICHD/NCMRR Pediatric Critical Care Research Network	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership Interest	Consultant/ advisory board	Other
		The major aims of the network are to reduce morbidity and mortality in pediatric critical illness and injury, and to provide a framework for the development of the scientific basis of pediatric critical care practice. I am responsible for conduct of network approved studies at CNMC. As a member of the network steering committee, I am responsible for contributing to design of studies, analysing results and disseminating research findings. Grant money comes to institution Role: Co-Investigator [a]Therapeutic Hypothermia after Pediatric Cardiac Arrest (THAPCA) Trials. PI:JT Berger 2009 Sponsor: of Michigan I am the site PI for the conduct of a randomized trial of therapeutic hypothermia in children who have had a cardiac arrest funded by NHLBI. Money comes to the institution Role: Consortium Site PI [a]Tracking Outcomes and Practice in Pediatric Pulmonary Hypertension. PI: JT Berger. 2008 Sponsor: Association in Pediatric Pulmonary Hypertension I am the site PI responsible for contributing subject data to a registry of pediatric pulmonary hypertension patients and their therapy Role: Site PI					
Jeffrey Berman	University of North Carolina:Faculty member UNC School of Medicine—Professor of Anesthesiology	None	None	None	None	None	None
Desmond Bohn	The Hospital for Sick Children, Toronto—Chief, Department of Critical Care Medicine	None	None	None	None	None	None
Kate Brown	Great Ormond Street Hospital for Children NHS Trust Hospital consultant in paediatric intensive care Consultant paediatric cardiac intensive care	None	None	None	None	None	None
Mark Coulthard	Queensland Health: State Health Employer Organisation—Paediatric Intensive Care Specialist	None	None	None	None	None	None
Douglas S. Diekema	Children's University Medical Group: Delivery of medical care in Children's Hospital of Seattle and the University of Washington—Professor of Pediatrics, Attending Physician, Emergency Department	None	None	None	None	None	None
Aaron Donoghue	University of Pennsylvania—Assistant Professor	None	None	None	None	None	None
Jonathan Duff	Alberta Health Services: Pediatric Intensivist	None	None	None	None	None	None
Jonathan Egan	The Children's hospital at Westmead, Sydney—Pediatric Intensivist	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership Interest	Consultant/ advisory board	Other
Christoph Eich	University Medical Centre of Göttingen, Germany: Attending Anesthesiologist, Intensivist and Emergency Physician	None	None	None	None	None	None
Diana G. Fendya	Children's National Medical Center, EMSC National Resource Center; Trauma/Acute Care Nursing Specialist	None	None	None	None	None	None
Ericka L. Fink	Children's Hospital of Pittsburgh of UPMC—Assistant Professor	[b]P.I., K23 from NINDS Duration of Hypothermia for Neuroprotection after Pediatric Cardiac Arrest Institution [b]P.I., Laerdal Foundation grant $21,365 Same topic Institution [a]Children's Hospital of Pittsburgh of UPMC Clinical and Translational Science Institute $6500 Same topic Institution	None	None	None	None	None
Loh Tsee Foong	TBD	TBD	TBD	TBD	TBD	TBD	TBD
Eugene B. Freid	Nemours Children's Clinics Health Care Organization Staff Anesthesiologist and Intensivist University of Florida Jacksonville Health Care Organization Pediatric Intensivist	None	None	[a]University of North Carolina—Speaker at Anesthesiology Refresher Course. 1000–1500/year honorarium sent to institution.	None	None	None
Susan Fuchs	Children's Memorial Hospital-Assoc Director, Div Pediatric Emergency Medicine	None	None	None	None	None	[a]Currently on the American Academy of Pediatrics Advanced Pediatric Life Support Steering Committee and Currently Co-chairperson of the AAP Pediatric Education for Prehospital Professional (PEPP) steering committee
Anne-Marie Guerguerian	The Hospital for Sick Children; Staff Physician	None	None	None	None	None	None
Bradford Harris	UNC at Chapel Hill; Assist Prof.	[b]5 P01 AT002620-02 (Peden) 09/30/04–06/30/09 5% NIH/NCCAM $1,660,813 Annual Direct Translational Research Center for CAM Therapy of Asthma The objective of this research is to identify antioxidant complementary and alternative medicine therapies for application in asthma [b]5 RO1 ES012706-02 (Peden) 09/01/04–07/31/09 5% NIH/NIEHS $209,314	None	[a]Assoc Clinical Research Professor on peds pharm	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership Interest	Consultant/ advisory board	Other
		[b]O3 and LPS-Induced Airway Inflammation in Humans in vivo The objective is to test three hypotheses to define the ways that O3 and LPS interact to exacerbate airway disease [b]5 R01 HL080337-02 (Peden) 05/06/05–04/30/09 5% NIH NHLBI/NIAID $350,000 Airway Biology of Acute Asthma: Translational Studies The major goal is to determine if asthma exacerbation and allergen challenge models allow for examination of innate/acquired immune interactions					
George Hoffman	Medical College of Wisconsin medical school Professor, Anesthesiology and Pediatrics	None	None	[a]Somanetics, Inc biomedical device manufac-turer 1653 East Maple Road Troy, MI 48083-4208 I have informally served in consul-tant/advisory capacity and have received honoraria for speaking	None	[a]Edwards Life Sciences, Inc biomedical device manufacturer One Edwards Way Irvine, CA 92614 I have served informally in con-sultant/advisory capacity	None
	[View] Children's Hospital of Wisconsin hospital Medical Director, Pediatric Anesthesiology					[a]Masimo, Inc biomedical device manufacturer 2852 Kelvin Ave. Irvine, CA, 92614 I have served informally in consul-tant/advisory capacity	
Jamie Hutchison	SickKIds Hospital Director Neurocritical Care	None	None	None	None	None	None
Sharon Kinney	University of Melbourne and Royal Children's Hospital Melbourne—Lecturer and MET Coordinator	None	None	None	None	None	None
Sasa Kurosawa	Shizuoka Children's Hospital Depertoment of Pediatric Emergency & General Pediatrics Doctor National Center for Child Health & Development Department of Health Policy, Research Institution researcher	None	None	None	None	None	None
Jesús López-Herce	Hospital General Universitario Gregorio Marañón—Pediatric Assistant	None	None	None	None	None	None
Sharon Mace	Clerveland Clinic—Physician employed fulltime by the hospital; Attending staff physician	None	None	[a]Baxter Healthcare Pharma-ceutical Speaker Bureau	None	[a]Baxter Healthcare Pharmaceutical Consultant, Advisory Board	None

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Ian Maconochie	Imperial Academic Health Sciences Centre, London:I run the pediatric emergency medicine department at St MAry's Hospital in Paddington, London—Lead Consultant in Pediatric Emergency medicine	None	[a]Postal for survey of UK departments about use of pain relief from Therakind, a company looking to obtain license for use of commonly used drugs from the medical regulatory authority in UK. Estimated payment was about 150 pounds sterling	None	None	[a]I am a deputy editor for The Emergency Medicine Journal, a commissioning editor for the Archives of Diseases of Childhood and sit on the editorial advisory panel for the British Medical Journal. I am editorial board member for Current Pediatric Reviews and Pediatric Emergency Medical Journal. The latter 2 I do not receive payment [a]I act as a consultant advisory to TSG associates in relation to major disaster management systems I have advised Therakind on the licensing of drugs in the pediatric population., ie approaching the medical regulatory authority to obtain a license on the use of a commonly used drug in the treatment of fitting children in UK	[a]I have acted as an expert witness in cases relating to the management of children who may have had non accidental injury
Duncan Macrae	The Royal Brompton and Harefield NHS Foundation Trust—Director of Children's Services	None	None	None	None	None	None
Mioara D. Manole	University of Pittsburgh: Pediatric Emergency Medicine Attending Physician; Assist Professor Pediatrics	[b]NIH K08HD58798-funds go to University [a]Children's Hosp of Pitts RAC grant-funds to University.	None	None	None	None	None
Bradley S. Marino	Cincinnati Children's Hospital Medical Center Associate Professor of Pediatrics	None	None	None	None	None	None
Felipe Martinez	Universidad de Valparaíso—Professor	None	None	None	None	None	None
Reylon Meeks	Blank Children's Hospital, Pleasant Hill Fire Dept., N Clinical Specialist	None	None	None	None	None	None
Alfredo Misraji	TBD	TBD	TBD	TBD	TBD	TBD	TBD
Marilyn Morris	Columbia University; Assistant Professor Pediatrics	None	None	None	None	None	[a]Expert witness $900 for 3 hour case for defense of child that received ECPR

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Akira Nishisaki	Children's Hosp of Philadelphia, non profit Tertiary Children's Hospital; Attending MD CCMedicine	None	None	None	None	None	None
Masahiko Nitta	Osaka Medical College—Assistant Professor	None	None	None	None	None	None
Gabrielle Nuthall	Auckland District Health Board: Pediatric Intensive Care Specialist	None	None	None	None	None	None
Sergio Pesutic Pérez	Centro de Formación en Apoyo Vital Director	None	None	None	None	None	None
Lester T. Proctor	University of Wisconsin-Madison College of Medicine and Public Health—Professor	None	None	None	None	None	None
Faiqa A. Qureshi	Children's Specialty Group—Physician	None	None	None	None	None	None
Sergio Rendich	Hospital Naval Almirante Nef—Pediatrician; Hospital Gustavo Fricke; Pediatrician—Intensive Care Unit; Universidad de Valparaíso Professor, Pediatrics Clínica Las Condes Critical Patient Unit Centro de Formación en Apoyo Vital; Instructor, NRP	None	None	None	None	None	None
Ricardo A. Samson	The University of Arizona: Faculty member within the Department of Pediatrics Chief of the Cardiology Section Provide clinical care, teaching and research related to the field of Pediatric Cardiology—Professor of Pediatrics	None	None	None	None	None	None
Kennith Sartorelli	University of Vermont Associate Professor of Surgery	None	None	None	None	None	None
Stephen M. Schexnayder	University of Arkansas for Medical Sciences—College of Medicine: Physician—Clinician Educator—Professor and Division Chief: AHA editor/writer	[a]Pharmacokinetics of pantoperazole in pediatrics patients (Pediatric Pharmacology Research Unit) Pharmacokinetics of esomeprazole in pediatric patients (Astra Zeneca)	None	[a]Contemporary Forums (Nursing conference) Pediatric Clinics of North America (guest editor)	None	None	[a]Expert witness in various medicolegal cases involving pediatric critical care and emergency medicine
William Scott	UT Southwestern Medical Center—Professor	None	None	None	None	None	None
Vijay Srinivasan	Children's Hospital of Philadelphia—Attending Physician, Pediatric Intensive Care Unit	[a]A Reproducible Method for Blood Glucose Control in Critically Ill Children (RC1 sub contract with Inter Mountain Medical Center, PI: Alan Morris), site PI: Vijay Srinivasan—submitted for NIH Challenge Grants	[a]PI: A Novel Application of Impedance Threshold Device technologies to optimize Fluid Removal during Hemodialysis in Children (unfunded research at CHOP—IRB Research Protocol No: 2007-12-5712)—have received impedance threshold devices for this study from Advanced Circulatory Systems, Inc, EdenPrairieMN	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership Interest	Consultant/ advisory board	Other
Robert M. Sutton	The Children's Hospital of Philadelphia Critical Care Attending	[a]Unrestricted research grant support from the Laerdal Foundation for Acute Care Medicine	None	None	None	None	None
Mark Terry	Johnson County Med-Act: County government ambulance service—Deputy Chief Operations	None	None	None	None	None	None
Shane Tibby	Guy's and St Thomas' NHS Foundation Trust, London National Health Service Hospital trust in United Kingdom Consultant in Pediatric Intensive Care	None	None	None	None	None	None
Alexis Topjian	The Children's Hospital of Philadelphia—attending physician	[a]Site PI for the Therapeutic hypothermia after cardiac arrest study. NIH funded study. Money goes to the institution	None	None	None	None	None
Elise W. van der Jagt	University of Rochester: Academic Institution including Medical School/Center—Professor of Pediatrics and Critical Care	[b]Project Title: Therapeutic Hypothermia After Pediatric Cardiac Arrest (THAPCA) Trials PI: Frank W. Moler, M.D. (University of Michigan) Proposed project period: 7/1/2009—6/30/2014 We are part of this multi-institutional grant but after the grant was funded, the initial institutions that would be involved were the higher volume/larger children's hospitals. At this time we are not receiving any funding from this grant.[a]PI andCo-Investigator/Site	None	None	None	None	None
David Wessel	Children's National Medical Center Senior Vice President	None	None	None	None	[b]IKARIA Holdings Inc. Pharmaceutical Consultant	None

This table represents the relationships of worksheet collaborators that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all worksheet collaborators are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.

[b] Significant.

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2. 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 6: pediatric basic and advanced life support. Circulation 2005;112. III-73–90.
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Part 11: Neonatal resuscitation
2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations☆

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Approximately 10% of newborns require some assistance to begin breathing at birth, and <1% require extensive resuscitation (LOE 4[1,2]). Although the vast majority of newborn infants do not require intervention to make the transition from intrauterine to extrauterine life, the large number of births worldwide means that many infants require some assistance to achieve cardiorespiratory stability. Newborn infants who are born at term and are breathing or crying and have good tone must be dried and kept warm. These actions can be provided with the baby lying on the mother's chest and should not require separation of mother and baby.

All others need to be assessed to determine their need for one or more of the following actions in sequence:

A. Initial steps in stabilization (dry and provide warmth, position, assess the airway, stimulate to breathe)
B. Ventilation
C. Chest compressions
D. Medications or volume expansion

Progression to the next step is initially based on simultaneous assessment of 2 vital characteristics: heart rate and respirations. Progression occurs only after successful completion of the preceding step. Approximately 30 s is allotted to complete each of the first two steps successfully, reevaluate, and decide whether to progress to the next (see Fig. 1).

Since publication of the *2005 International Consensus on CPR and ECC Science with Treatment Recommendations*,[3,4] several controversial neonatal resuscitation issues have been identified. The literature was researched and a consensus was reached on the assessment of oxygenation and role of supplementary oxygen, peripartum management of meconium, ventilation strategies, devices to confirm placement of an advanced airway (e.g., tracheal tube or laryngeal mask airway), medications, maintenance of body temperature, postresuscitation management, and considerations for withholding and discontinuing resuscitation. Educational techniques for teaching, assessing, and maintaining resuscitation knowledge and skills and issues regarding the personnel needed at cesarean sections were also debated. The following are the major new recommendations:

- Progression to the next step following the initial evaluation is now defined by the simultaneous assessment of 2 vital characteristics: heart rate and respirations. Oximetry should be used for evaluation of oxygenation because assessment of colour is unreliable.
- For babies born at term it is best to begin resuscitation with air rather than 100% oxygen.
- Administration of supplementary oxygen should be regulated by blending oxygen and air, and the concentration delivered should be guided by oximetry.
- The available evidence does not support or refute the routine tracheal suctioning of infants born through meconium-stained amniotic fluid, even when the newborn is depressed.
- The chest compression–ventilation ratio should remain at 3:1 for neonates unless the arrest is known to be of cardiac aetiology, in which case a higher ratio should be considered.
- Therapeutic hypothermia should be considered for infants born at term or near-term with evolving moderate to severe hypoxic–ischaemic encephalopathy, with protocol and follow-up coordinated through a regional perinatal system.
- It is appropriate to consider discontinuing resuscitation if there has been no detectable heart rate for 10 min. Many factors contribute to the decision to continue beyond 10 min.
- Cord clamping should be delayed for at least 1 min in babies who do not require resuscitation. Evidence is insufficient to recommend a time for clamping in those who require resuscitation.

☆ *Note from the writing group*: Throughout this article, the reader will notice combinations of superscripted letters and numbers (e.g., "Peripartum Suctioning[NRP-011A,NRP-012A]"). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.

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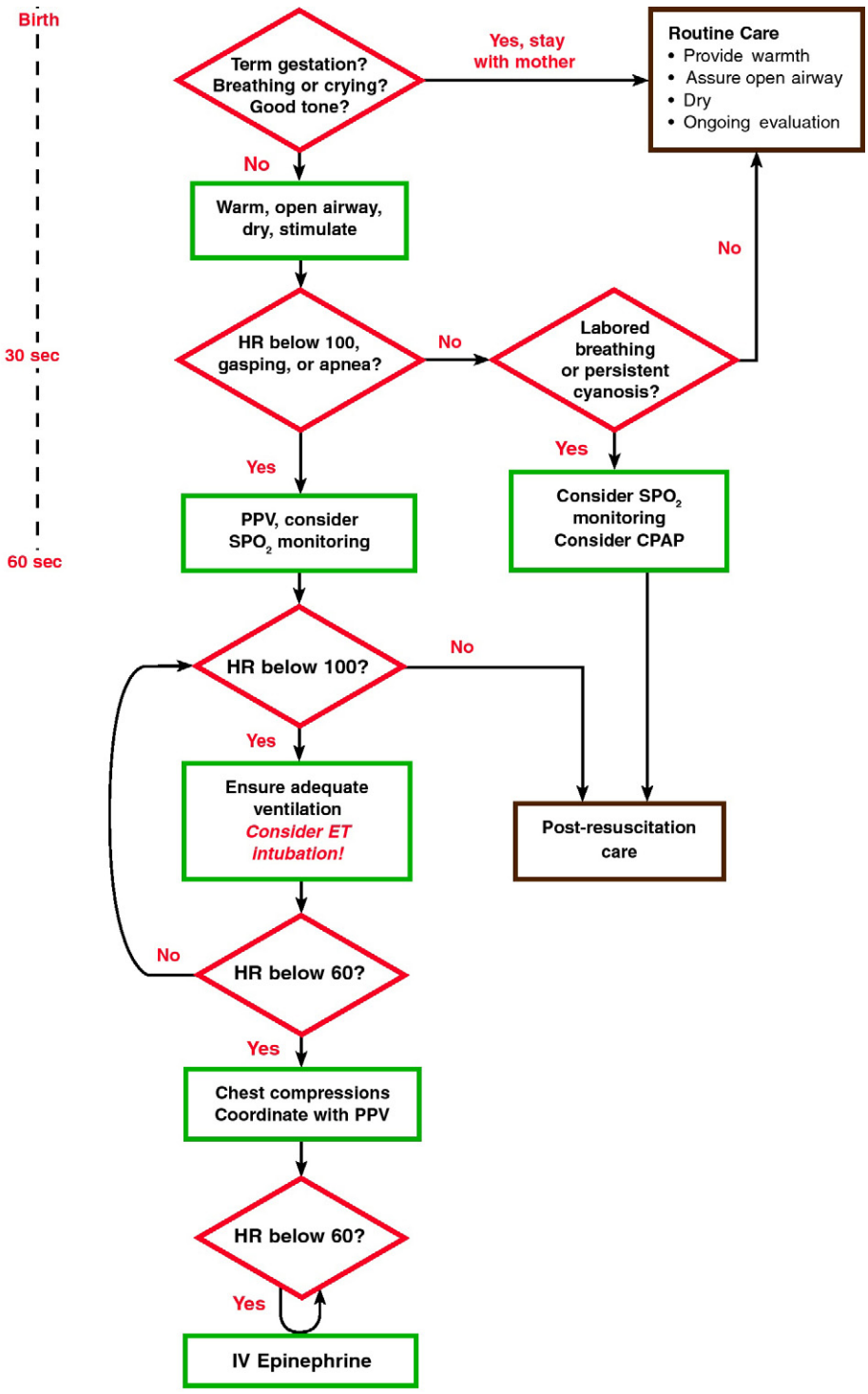

Fig. 1. Newborn Resuscitation Algorithm.

Initial assessment and intervention

Assessment of cardiorespiratory transition and need for resuscitation[NRP-001A,NRP-001B,NRP-014A,NRP-014B]

Consensus on science

A prompt increase in heart rate remains the most sensitive indicator of resuscitation efficacy (LOE 5[5]). Of the clinical assessments, auscultation of the heart is the most accurate, with palpation of the umbilical cord less so. However, both are relatively insensitive (LOE 2[6] and LOE 4[7]). Several studies have addressed the accuracy of pulse oximetry in measuring heart rate in the delivery room and have shown the feasibility of pulse oximetry during newborn resuscitation. However, none of these studies examined the impact of these measurements on resuscitation outcomes (LOE 4[7,8]). Pulse oximetry (Spo_2) and heart rate can be measured reliably after 90 s from birth with a pulse oximeter designed to reduce movement artefact and a neonatal probe (LOE 4[9,10]). Preductal values, obtained from the right wrist or hand, are higher than postductal values.[8,11] Applying the oximeter probe to the subject before connecting

it to the instrument will produce reliable results more quickly (LOE 4[10]).

There is clear evidence that an increase in oxygenation and improvement in colour may take many minutes to achieve, even in uncompromised babies. Furthermore, there is increasing evidence that exposure of the newly born to hyperoxia is detrimental to many organs at a cellular and functional level. For this reason colour has been removed as an indicator of oxygenation or resuscitation efficacy. The oximeter can be used to adjust the increase in oxygenation to that of the uncompromised baby born at term.

Treatment recommendations

Heart rate should remain the primary vital sign by which to judge the need for and efficacy of resuscitation. Auscultation of the precordium should remain the primary means of assessing heart rate. There is a high likelihood of underestimating heart rate with palpation of the umbilical pulse, but this is preferable to other palpation locations.

For babies who require ongoing resuscitation or respiratory support or both, the goal should be to use pulse oximetry. The sensor should be placed on the baby's right hand or wrist before connecting the probe to the instrument. Because of concerns about the ability to consistently obtain accurate measurements, pulse oximetry should be used in conjunction with and should not replace clinical assessment of heart rate during newborn resuscitation.

Use of supplementary oxygen[NRP-013A,NRP-013B,NRP-014A,NRP-014B]

Consensus on science

In term infants receiving resuscitation with intermittent positive-pressure ventilation, 100% oxygen conferred no advantage over air in the short term and resulted in increased time to first breath or cry or both (LOE 2[12,13]). Meta-analyses of these studies showed a decrease in mortality with the group for whom resuscitation was initiated with air.[14,15]

There is evidence in newborn animal models of asphyxia that exposure to high concentrations of oxygen at resuscitation does not confer any clinical advantage and is potentially harmful at the cellular level.[16,17] Two animal models of hypoxia–ischaemia and persistent bradycardia found that those resuscitated with room air rather than 100% oxygen developed untoward biochemical changes in the brain (LOE 5[18,19]).

In preterm infants at <32 weeks' gestation, if attempting to mimic the gradual rise in oxygen saturation of healthy term babies in the first 10 min after birth by titrating the concentration to the baby's saturation, initial use of air or 100% oxygen is more likely to result in hypoxaemia or hyperoxaemia, respectively, than initiation of resuscitation with 30% or 90% oxygen and titration to oxygen saturation (LOE 2[11,20]). There is insufficient evidence in babies born at 32–37 weeks' gestation to define the appropriate oxygen administration strategy.

Treatment recommendation

In term infants receiving resuscitation at birth with positive-pressure ventilation, it is best to begin with air rather than 100% oxygen. If despite effective ventilation there is no increase in heart rate or if oxygenation (guided by oximetry) remains unacceptable, use of a higher concentration of oxygen should be considered.

Because many preterm babies of <32 weeks' gestation will not reach target saturations in air, blended oxygen and air may be given judiciously and ideally guided by pulse oximetry. Both hyperoxaemia and hypoxaemia should be avoided. If a blend of oxygen and air is not available, resuscitation should be initiated with air.

Peripartum suctioning[NRP-011A,NRP-012A]

Peripartum suctioning was examined from 2 perspectives: (1) suctioning of the airway in depressed neonates born through clear amniotic fluid and (2) tracheal suctioning in depressed neonates born through meconium-stained amniotic fluid.

Suctioning of the upper airway

Consensus on science

There is no evidence to support or refute suctioning of the mouth and nose of depressed neonates at birth when the infant is born through clear amniotic fluid. In healthy neonates suctioning of the mouth and nose is associated with cardiorespiratory complications (LOE 1[21,22]). In infants who are intubated, sedated, or paralyzed following resuscitation, tracheal suctioning in the absence of secretions may result in a decrease in oxygenation, an increase in cerebral blood flow and intracranial pressure, and a decrease in compliance (LOE 5[23]).

Treatment recommendation

Routine intrapartum oropharyngeal and nasopharyngeal suctioning for infants born with clear or meconium-stained amniotic fluid is no longer recommended.

Tracheal suctioning

Consensus on science

Depressed infants born through meconium-stained amniotic fluid are at increased risk of developing meconium aspiration syndrome (LOE 4[24,25]). Although these infants are at increased risk of developing meconium aspiration syndrome, the use of tracheal suctioning has not been associated with a reduction in the incidence of meconium aspiration syndrome or mortality (LOE 4[26]; LOE 5[27]). No randomised controlled studies have compared intubation and tracheal suctioning and no tracheal suctioning in depressed infants.

Treatment recommendation

The available evidence does not support or refute the routine tracheal suctioning of depressed infants born through meconium-stained amniotic fluid.

Tracheal suctioning

Consensus on science

Depressed infants born through meconium-stained amniotic fluid are at increased risk of developing meconium aspiration syndrome (LOE 4[24,25]). Although these infants are at increased risk of developing meconium aspiration syndrome, the use of tracheal suctioning has not been associated with a reduction in the incidence of meconium aspiration syndrome or mortality (LOE 4[26]; LOE 5[27]). No randomised controlled studies have compared intubation and tracheal suctioning and no tracheal suctioning in depressed infants.

Treatment recommendation

The available evidence does not support or refute the routine tracheal suctioning of depressed infants born through meconium-stained amniotic fluid.

Ventilation strategies[NRP-028A,NRP-028B]

Ventilation strategies were examined from four perspectives: (1) characteristics of the initial assisted breaths and the role of positive end-expiratory pressure (PEEP), (2) continuous positive air pressure (CPAP) during or following resuscitation, (3) devices to assist ventilation, and (4) strategies when resources are limited.

Initial breaths

Consensus on science

Both longer and shorter inspiratory times are in clinical use for initial ventilation in term infants, but there are no randomised controlled trials comparing these 2 approaches. In a small case series in term infants, a prolonged initial inflation of five seconds produced a twofold increase in functional residual capacity compared with historic controls (LOE 4[28]). A single randomised controlled trial in preterm infants of a 10-s sustained inflation followed by nasal CPAP compared with bag-mask ventilation demonstrated decreased need for intubation in the first 72 h, shorter duration of ventilatory support, and reduced bronchopulmonary dysplasia (LOE 1[29]). Two other randomised controlled trials failed to show a benefit from delivery room application of a sustained initial inflation followed by nasal CPAP (LOE 1[30,31]). Multiple variables among the three randomised controlled trials, including mode of intervention (nasopharyngeal tube versus face mask, T-piece versus self-inflating bag), as well as the use of CPAP in the delivery room make it difficult to determine the effect of the initial sustained inflation on establishing a functional residual capacity in very preterm infants.

Pressure

There is no evidence to support the use of inflation pressures higher than those that are necessary to achieve improvement in heart rate or chest expansion. This can usually be achieved in term infants with an inflation pressure of 30 cm H_2O (LOE 4[28,32]) and in preterm infants with pressures of 20–25 cm H_2O (LOE 4[33]). Occasionally higher pressures are required (LOE 4[34]). In immature animals, ventilation at birth with high volumes associated with the generation of high peak inflation pressures for a few minutes causes lung injury, impaired gas exchange, and reduced lung compliance (LOE 5[35]).

Positive end-expiratory pressure

There is no evidence to support or refute the value of PEEP during resuscitation of term infants. In preterm infants one small study did not show a benefit from PEEP during initial stabilization in reducing the number of infants who required intubation in the delivery room (LOE 1[36]). In studies of intubated immature animals the use of PEEP during initial stabilization after birth improved functional residual capacity, oxygenation, and lung compliance and reduced lung injury (LOE 5[37,38]), but high levels of PEEP (8–12 cm H_2O) may reduce pulmonary blood flow and increase the risk of pneumothorax (LOE 5[39,40]).

Treatment recommendation

To establish initial lung inflation in apnoeic newborn infants, initiation of intermittent positive-pressure ventilation at birth can be accomplished with either shorter or longer inspiratory times. Initial peak inflating pressures necessary to achieve an increase in heart rate or movement of the chest are variable and unpredictable and should be individualised with each breath. If pressure is being monitored, an initial inflation pressure of 20 cm H_2O may be effective in preterm babies, but a pressure of 30–40 cm H_2O may be necessary in some term babies. If pressure is not being monitored, the minimal inflation required to achieve an increase in heart rate should be used. Providers should avoid creation of excessive chest wall movement during ventilation of preterm infants immediately after birth.

Although measured peak inflation pressure does not correlate well with volume delivered in the context of changing respiratory mechanics, monitoring of inflation pressure may help provide consistent inflations and avoid unnecessarily high pressures. If positive-pressure ventilation is required, an initial inflation pressure of 20–25 cm H_2O is adequate for most preterm infants. If prompt improvement in heart rate or chest movement is not obtained, then higher pressures to achieve effective ventilation may be needed. PEEP is likely to be beneficial during initial stabilization of apneic preterm infants who require positive-pressure ventilation and should be used if suitable equipment is available.

Continuous positive airway pressure[NRP-002A,NRP-002B]

Consensus on science

For spontaneously breathing preterm infants at ≥25 weeks' gestation who have signs of respiratory distress, there is no significant difference between starting CPAP or intubation and mechanical ventilation in the delivery room when considering death or oxygen requirement at 36 weeks postmenstrual age. In spontaneously breathing infants at 25–28 weeks' gestation, CPAP compared with intubation reduced the rates of mechanical ventilation from 100% to 46% and surfactant use from 77% to 38% (LOE 1[41]). In the same trial infants on CPAP had a significantly increased rate of pneumothorax (9% versus 3%) (LOE 1[41]). There is no evidence to support or refute the use of CPAP in the term baby.

For very preterm infants, a multifaceted intervention, including PEEP, giving a sustained inflation and starting CPAP in the delivery room reduces the need for intubation and rate of mechanical ventilation within 72 h and reduces incidence of bronchopulmonary dysplasia compared with positive-pressure ventilation with a self-inflating bag via a face mask (LOE 1[29]). When compared with historic controls, use of delivery room CPAP for very premature infants was associated with a decrease in the requirement for intubation, days on mechanical ventilation, and use of postnatal steroids (LOE 4[33]), although a small underpowered feasibility trial of delivery room CPAP/PEEP versus no CPAP/PEEP did not show a significant difference in immediate outcomes (LOE 1[36]).

Treatment recommendation

Spontaneously breathing preterm infants who have respiratory distress may be supported with CPAP or intubation and mechanical ventilation. The most appropriate choice may be guided by local expertise and preferences.

Assisted ventilation devices[NRP-015A,NRP-015B,NRP-015C,NRP-017A,NRP-017B]

Consensus on science

There are no clinical studies in newborns requiring positive pressure during resuscitation to support or refute the superiority

of the T-piece resuscitator over bag-mask ventilation in improving outcome. In mechanical models target inflation pressures are delivered more consistently when using T-piece resuscitators than with self-inflating bags or flow-inflating bags (LOE 5[42,43]). In mechanical models PEEP is maintained more consistently with T-piece resuscitators compared with self-inflating bags or flow-inflating bags (LOE 5[44]). In mechanical models the ability to deliver a sustained inflation is better with either a T-piece resuscitator or flow-inflating bag than with a self-inflating bag (LOE 5[42,45]).

Treatment recommendation

Ventilation of the newborn can be performed effectively with a flow-inflating bag, a self-inflating bag, or a pressure-limited T-piece resuscitator.

Laryngeal mask airway[NRP-017A,NRP-017B]

Consensus on science

In one randomised controlled trial (LOE 1[46]) providers had similar success providing effective ventilation with either the laryngeal mask airway or face mask among newborns in the delivery room. In one retrospective cohort study (LOE 2[47]) and three large case series (LOE 4[48]) effective ventilation was achieved quickly using a laryngeal mask airway in newborns weighing >2000 g or delivered at ≥34 weeks' gestation. In one randomised controlled trial (LOE 1[49]) and one retrospective cohort study (LOE 2[50]) providers had similar success providing effective ventilation using either the laryngeal mask airway or tracheal tube among newborns in the delivery room. Although a single cohort study (LOE 2[50]) suggests that newborns resuscitated with a laryngeal mask may require less respiratory support after initial resuscitation, this conclusion is subject to significant selection bias. In multiple small case reports effective ventilation was achieved with a laryngeal mask airway when both face mask ventilation and tracheal intubation were unsuccessful. There is limited evidence to evaluate the effectiveness of the laryngeal mask airway for newborns weighing <2000 g, delivered at <34 weeks' gestation, in the setting of meconium-stained amniotic fluid, during chest compressions, or for administration of emergency intratracheal medications.

Treatment recommendation

The laryngeal mask airway should be considered during resuscitation of the newborn if face mask ventilation is unsuccessful and tracheal intubation is unsuccessful or not feasible. The laryngeal mask airway may be considered as an alternative to a face mask for positive-pressure ventilation among newborns weighing >2000 g or delivered at ≥34 weeks' gestation. There is limited evidence, however, to evaluate its use for newborns weighing <2000 g or delivered at <34 weeks' gestation. The laryngeal mask airway may be considered as an alternative to tracheal intubation as a secondary airway for resuscitation among newborns weighing >2000 g or delivered at ≥34 weeks' gestation. The laryngeal mask airway has not been evaluated in the setting of meconium-stained amniotic fluid, during chest compressions, or for administration of emergency intratracheal medications.

Upper airway interface devices[NRP-003A,NRP-003B]

Consensus on science

Within classes of interfaces, reports conflict about the ability to maintain a seal with an anatomically shaped mask compared with a soft round mask (LOE 5[51,52]). Delivery of positive-pressure ventilation via nasal prongs has been shown to be superior to delivery via a triangular face mask for outcomes of chest compressions and intubation (LOE 2[53]). It is likely that differences in clinical outcomes that have been reported in several studies may be attributable to the targeted intervention (i.e., CPAP versus intermittent positive-pressure ventilation) rather than the interface. Nasal prongs may be a more effective device than face masks for providing respiratory support after birth (LOE 2[53]). There is insufficient evidence to support or refute the use of one type of mask over another for achieving clinical outcome, except that the Rendell-Baker style mask is suboptimal in achieving an adequate seal when used for newborns (LOE 5[54]).

Treatment recommendations

Nasal prongs are an alternative way of giving respiratory support. Whichever interface is used, providers should ensure that they are skilled in using the interface devices available at the institution. Different masks must be held in different ways to appropriately reduce leak.

Exhaled air ventilation[NRP-004A,NRP-004B]

Consensus on science

Mouth-to-mouth ventilation is less effective than a self-inflating bag or tube and mask in improving survival rates in newborns with birth asphyxia (LOE 3[55]). Use of mouth-to-mask ventilation at 30 insufflations per minute is as effective as self-inflating bag-mask ventilation in increasing heart rate in the first 5 min after birth (LOE 2[56]). Mask-to-tube ventilation may cause infection in newborn infants (LOE 5[57]). Two studies (LOE 5[58,59]) demonstrated that tube-to-mask ventilation can be easily taught and acceptable breaths delivered. However, tube-to-mask ventilation was more difficult to use (LOE 5[60]; LOE 3[55]).

Treatment recommendation

Bag-mask ventilation is preferable to mouth-to-mask ventilation or tube-to-mask ventilation during neonatal resuscitation, but one of the latter two should be used when bag-mask devices are not available. Precautions must be taken because mouth-to-mask and mouth tube-to-mask ventilation are less comfortable and more tiring than bag-mask ventilation for the newborn at birth and may be associated with increased risk of infection in the infant and healthcare provider.

Monitoring during and after intubation

Gas monitoring devices

Measurement of gas volume[NRP-005A,NRP-005B,NRP-005C]

Consensus of science

There are no studies that compare clinical outcomes in newborns after resuscitation with or without monitoring of gas volume. In preterm animal models the tidal volume used during initial ventilation after birth may alter subsequent lung function and induce inflammation, but other factors, including the use and level of PEEP, appear to interact with tidal volume in determining specific effects (LOE 5[61,62]). It is unclear whether the absolute tidal volumes used affected outcomes. Studies in manikins and animals (LOE 5[63,64]) suggest that providers cannot maintain constant pressures or assess delivered volume during manual ventilation. The position of the mask and degree of leak may be improved by the use of a volume monitor (LOE 5[65]).

Treatment recommendations

Ventilation during newborn resuscitation should aim to adequately inflate the lung while avoiding overinflation. There is

insufficient evidence to recommend routine use of tidal-volume monitoring in neonates receiving positive-pressure ventilation during resuscitation.

Use of exhaled CO_2 detectors to confirm tracheal tube placement[NRP-016A]

Consensus on science

Studies (LOE 2[66–68]) suggest that detection of exhaled CO_2 confirms tracheal intubation in neonates with cardiac output more rapidly and accurately than clinical assessment alone. False-negative readings have been reported during cardiac arrest (LOE 4[69]) despite models suggesting efficacy (LOE 5[70]). False-positive readings may occur with colorimetric devices contaminated with adrenaline (epinephrine), surfactant, and atropine (LOE 5[71]). Neonatal studies have excluded infants who need extensive resuscitation. There is no comparative information to recommend any one method for detection of exhaled CO_2 in the neonatal population.

Treatment recommendation

Detection of exhaled CO_2 in addition to clinical assessment is recommended as the most reliable method to confirm tracheal placement in neonates with spontaneous circulation.

Colorimetric CO_2 detection to assess ventilation in nonintubated patients[NRP-018A,NRP-018B,NRP-018C]

Consensus on science

The use of colorimetric exhaled CO_2 detectors during face mask ventilation of small numbers of preterm infants in the intensive care unit (LOE 4[72]) and the delivery room (LOE 4[73]) has been reported and may help identify airway obstruction. It is unclear whether the use of exhaled CO_2 detectors during bag-mask ventilation confers additional benefit over clinical assessment alone. No risks attributed to the use of exhaled CO_2 detectors have been identified. The use of exhaled CO_2 detectors with other interfaces (e.g., nasal airways, laryngeal masks) during positive-pressure ventilation in the delivery room has not been reported.

Treatment recommendation

There is insufficient evidence to recommend routine use of colorimetric exhaled CO_2 detectors during mask ventilation of newborns in the delivery room.

Circulatory support

Chest compressions[NRP-006A,NRP-006B,NRP-007A,NRP-007B]

Consensus on science

In animal studies of asphyxial models of cardiac arrest, piglets resuscitated with a combination of chest compressions and ventilations had better outcomes than those resuscitated with ventilations or compressions alone (LOE 5[74,75]). A further study in piglets suggested that sustained chest compressions had a deleterious effect on myocardial and cerebral perfusion, especially during prolonged resuscitation.[76]

A physiological mathematical modeling study suggested that using higher compression–ventilation ratios would result in underventilation of asphyxiated infants (LOE 5[77]). The model predicts that between three and five compressions to one ventilation should be most efficient for newborns.

Manikin studies confirm that the 3:1 compression–ventilation ratio provides more ventilations per minute when compared with higher ratios, but the resuscitation is perceived as being more physically taxing, especially when performed by a lone rescuer (LOE 5[78,79]). Adult manikin studies using two rescuers have shown that a 5:1 ratio provides better-quality chest compressions than a 15:2 ratio (LOE 5[80]) but can result in more missed ventilations per cycle (LOE 5[81]). A paediatric manikin study of mouth-to-mouth ventilation by a lone lay rescuer found equivalent minute ventilation for both the 15:2 and 5:1 ratios, but the 15:2 ratio produced more chest compressions per minute (LOE 5[82]). With two-rescuer CPR provided by nursing students, however, minute ventilation and compressions per minute were increased with the 5:1 ratio compared with the 10:2 and 15:2 ratios (LOE 5[83]). When the 15:2 ratio was compared with the 30:2 ratio in a one-rescuer model of medical personnel using adolescent, child, and infant manikins, more compression cycles could be achieved with the 30:2 ratio on all manikins with no apparent effect on quality of compressions (LOE 5[84]). Effect on ventilation, however, was not assessed. One study in children suggested that CPR with rescue breathing is preferable to CPR alone when the arrest is of noncardiac aetiology (LOE 5[85]). There are no data regarding the optimum compression–ventilation ratios in neonates or neonatal models of primary cardiac versus predominantly asphyxial arrest.

Evidence from randomised studies in swine models (LOE 5[86,87]), manikin studies (LOE 5[84,88]), small case series (LOE 4[89]), and cadavers (LOE 5[90]) support the current practice of favouring the two thumb-encircling hands technique of chest compressions when compared with the two-finger technique. The former method produces higher blood pressure, can sustain a consistent quality of compressions for a longer time, and is perceived as easier and less tiring for the provider. One manikin study involving a variety of medical or quasimedical personnel (LOE 5[91]) found no difference in a number of qualitative measures between the two techniques other than significantly fewer compressions were judged as too shallow with the two-thumb technique. One small case series in newborns found higher systolic blood pressure generated with the two-finger technique when compared with the two-thumb-encircling hands technique (LOE 4[92]). Both techniques, however, generated comparable and adequate diastolic pressures, a more important determinant of coronary perfusion. Compressions should be centred over the lower third of the sternum rather than the mid-sternum (LOE 5[93,94]). Chest compression depth should favour one third the external anterior–posterior diameter of the chest rather than deeper compressions (LOE 5[95]).

Treatment recommendation

There is no evidence from quality human, animal, manikin, or mathematical modelling studies to warrant a change from the current compression–ventilation ratio of 3:1. Strategies should be considered for optimising the quality of compressions and ventilations with as few interruptions as possible. Because ventilation is critical to reversal of newborn asphyxial arrest, any higher ratio that decreases minute ventilation should be introduced with caution. If the arrest is known to be of cardiac aetiology, a higher compression–ventilation ratio should be considered (e.g., 15:2).

Chest compressions in the newborn should be delivered by the two-thumb-encircling hands method as the preferred option. Compressions should be centred over the lower third of the sternum and should compress the chest one-third the anterior–posterior diameter. Any chest compressions should be performed in combination with adequate inflation breaths.

Medications and fluid administration

Adrenaline

Route and dose of adrenaline[NRP-008A,NRP-008B,NRP-009A,NRP-009B]

Consensus on science

Despite the widespread use of adrenaline during resuscitation, no controlled clinical trials have directly compared tracheal and intravenous administration of adrenaline among neonates with a heart rate of <60 beats per minute despite adequate ventilation and chest compressions. Limited evidence from neonatal case series or case reports (LOE 4[96,97]) indicates that adrenaline administered by the tracheal route using a wide range of doses (0.003 mg kg^{-1} to 0.25 mg kg^{-1}) may result in return of spontaneous circulation (ROSC) or an increase in heart rate when intravenous access is not available. These case series are limited by inconsistent standards for adrenaline administration and are subject to both selection and reporting bias.

Evidence from one case series using rigorously defined standards for adrenaline administration and outcomes reporting indicates that tracheal administration of adrenaline (0.01 mg/kg) is likely to be less effective than intravenous administration of the same dose (LOE 4[2]). This is consistent with evidence extrapolated from neonatal animal models indicating that higher doses (0.05–0.1 mg kg^{-1}) of tracheal adrenaline may be required to achieve increased blood adrenaline concentrations and a haemodynamic response equivalent to intravenous administration (LOE 5[98,99]). Evidence extrapolated from adult animal models indicates that blood concentrations of adrenaline are significantly lower following tracheal administration (LOE 5[100,101]), and tracheal doses ranging from 0.05 mg kg^{-1} to 0.1 mg kg^{-1} may be required to achieve ROSC (LOE 5[102]).

Although it has been widely assumed that adrenaline can be administered faster by the tracheal route than by the intravenous route, no clinical trials have evaluated this hypothesis. Two studies have reported cases of inappropriate early use of tracheal adrenaline before airway and breathing are established (LOE 4[96,97]). One case series describing in-hospital paediatric cardiac arrest suggested that survival was higher among infants who received their first dose of adrenaline by the tracheal route; however, the time required for first dose administration using the tracheal and intravenous routes was not provided (LOE 5[103]).

Despite the widespread use of adrenaline during resuscitation, no controlled clinical trials have evaluated the ideal dose of adrenaline among neonates with a heart rate of <60 beats per minute despite adequate ventilation and chest compressions. Evidence extrapolated from paediatric studies that included infants <1 year of age (LOE 5[104,105]) indicate no benefit from intravenous adrenaline doses ≥0.03 mg/kg. This is in contrast to a single paediatric case series using historic controls that indicated a marked improvement in ROSC using high-dose intravenous adrenaline (0.1 mg kg^{-1}) among children who had not responded to two doses of standard adrenaline (0.01 mg kg^{-1}) (LOE 5[106]). Further extrapolative evidence from a meta-analysis of five adult clinical trials indicates that high-dose intravenous adrenaline may increase ROSC but offers no benefit in survival to hospital discharge (LOE 5[107]). Evidence from a planned secondary analysis of a paediatric randomised controlled trial suggests an increased risk of mortality among children receiving high-dose intravenous adrenaline (0.1 mg kg^{-1}) (LOE 5[104]). Additional evidence from two paediatric animal studies (LOE 5[108,109]) indicates that intravenous adrenaline ≥0.1 mg kg^{-1} increased risk of post-resuscitation mortality and interfered with cerebral cortical blood flow and cardiac output. There are no published studies comparing standard- and high-dose tracheal adrenaline in the neonatal population with hypoxic–hypercarbic arrest, and the ideal dose for tracheal administration is unknown. Data from neonatal case series and animal models suggest that higher doses (0.05–0.1 mg kg^{-1}) of tracheal adrenaline may be required to achieve increased blood adrenaline concentrations and a haemodynamic response equivalent to intravenous administration (LOE 4[2,96]).

Treatment recommendation

If adequate ventilation and chest compressions have failed to increase the heart rate to >60 beats per minute, then it is reasonable to use adrenaline despite the lack of human neonatal data. If adrenaline is indicated, a dose of 0.01–0.03 mg kg^{-1} should be administered *intravenously* as soon as possible. If adequate ventilation and chest compressions have failed to increase the heart rate to >60 beats per minute and intravenous access is *not* available, then it is reasonable to administer tracheal adrenaline. If adrenaline is administered by the tracheal route, it is likely that a larger dose (0.05–0.1 mg kg^{-1}) will be required to achieve an effect similar to that of the 0.01 mg kg^{-1} intravenous dose. Higher intravenous doses cannot be recommended and may be harmful.

Volume expansion[NRP-029A,NRP-029B,NRP-029C]

Consensus on science

Multiple case series support the use of volume expansion in babies with a history of blood loss, including some who are unresponsive to chest compressions (LOE 4[110]). Many with pallor and tachycardia responded to volume expansion without having received chest compressions. In the absence of a history of blood loss there is limited evidence of benefit from administration of volume during resuscitation unresponsive to chest compressions/adrenaline (LOE 4[111]) and some suggestion of potential harm from animal studies (LOE 5[112,113]).

Treatment recommendation

Early volume replacement with crystalloid or red cells is indicated for babies with blood loss who are not responding to resuscitation. There is insufficient evidence to support the routine use of volume administration in the infant with no blood loss who is refractory to ventilation, chest compressions, and adrenaline. Because blood loss may be occult, a trial of volume administration may be considered in babies who do not respond to resuscitation.

Other drugs

Very rarely a narcotic antagonist (naloxone), sodium bicarbonate,[NRP-021A,NRP-021B] or vasopressors may be useful after resuscitation.

Naloxone[NRP-022A, NRP-022B]

Consensus on science

There are no data comparing naloxone with positive-pressure ventilation as the main intervention for opioid-exposed newborn infants who are apnoeic at birth. For newborns who are vigorous in the delivery room despite maternal use of opioids, naloxone subtly increases ventilation parameters (such as increased alveolar ventilation and improved CO_2 response curves) for a short time, but the clinical relevance of these observations is questionable (LOE 4[114]). Several other studies found no difference between vigorous treatment with naloxone and placebo or no drug treatment for newborns with outcomes of pH, Pco_2, Apgar scores, and neurological outcomes (LOE 5[115]). Studies examining naloxone have consistently demonstrated that it is frequently misused (LOE 4[116]).

Naloxone given to a baby born to an opioid-addicted mother has been associated with seizures (LOE 5[117]). There are concerns about short- and long-term safety of naloxone in neonates (LOE 5[118]). Naloxone is absorbed more effectively when given intravenously but has a shorter half-life compared with intramuscular administration.

Treatment recommendation

Naloxone is not recommended as part of the initial resuscitation for newborns with respiratory depression in the delivery room. For the clinical situation of a newborn with respiratory depression after maternal opiate exposure, the focus needs to remain on effective ventilation and airway support for the persistently apnoeic newborn.

Vascular access[NRP-020A]

Consensus on science

Multiple clinical series and case reports suggest that fluids and medications can be successfully delivered by the intraosseous route during resuscitation of neonates when equipment or personnel skilled in establishing venous access are not available or if other vascular access sites (especially intravenous) cannot be successfully established within several minutes (LOE 4[119,120]).

Treatment recommendation

Temporary intraosseous access to provide fluids and medications to resuscitate critically ill neonates may be indicated following unsuccessful attempts to establish intravenous vascular access or when caregivers are more skilled at securing intraosseous access.

Supportive therapy

Temperature control

Maintenance of body temperature[NRP-023A]

Consensus on science

A large body of evidence supports the wrapping of newborn infants of <28 weeks' gestation in polythene wraps or bags at birth without drying to reduce heat loss (LOE 1[121,122]). Some of these infants were hyperthermic on admission to the neonatal intensive care unit, but it is unclear whether this is because they were born hot or because they became overheated during stabilization and transfer. In the absence of polythene wrapping, use of exothermic mattresses maintained the temperature of newborn infants weighing <1500 g within the normal range (LOE 2[123]). A combination of exothermic mattresses and polythene wrapping during resuscitation is the most effective strategy to avoid hypothermia but may increase the risk of hyperthermia (LOE3[124]). Delivery room temperatures of at least 26 °C for newborns at <28 weeks' gestation in combination with polythene wraps or bags maintained temperatures most effectively (LOE 4[125]; LOE 3[126]).

Treatment recommendation

Newborn infants of <28 weeks' gestation should be completely covered in a polythene wrap or bag up to their necks without drying immediately after birth and then placed under a radiant heater and resuscitated or stabilised in a standard fashion. Infants should be kept wrapped until admission and temperature check. Hyperthermia should be avoided. Delivery room temperatures should be at least 26 °C for infants of <28 weeks' gestation.

Postresuscitation management

Temperature

Hyperthermia[NRP-031A,NRP-031B]

Consensus on science

Infants born to febrile mothers have been reported to have a higher incidence of perinatal respiratory depression, neonatal seizures, cerebral palsy, and increased risk of mortality (LOE 4[127,128]). There is no evidence to determine whether the fever or the cause of the fever increases the risk to the baby. In one study, neonatal fever at birth resolved spontaneously within 60 min (LOE 4[129]). Adult animal trials show decreased central nervous system injury with antipyretic therapy for hyperthermia (LOE 5[130]). In a randomised study high-dose corticosteroids lowered maternal temperature but were associated with an increased number of cases of asymptomatic bacteraemia in neonates (LOE 2[131]).

Treatment recommendation

There is insufficient evidence to support or refute the routine use of interventions to lower maternal fever to reduce neonatal morbidity and mortality. There should be an increased awareness that the presence of maternal hyperthermia may lead to a need for neonatal resuscitation. The goal is to achieve normothermia and avoid iatrogenic hyperthermia.

Therapeutic hypothermia[NRP-024A,NRP-024B]

Consensus on science

A large body of evidence from three large randomised studies (LOE 1[132-134]) and two small randomised trials (LOE 1[135,136]) demonstrated that induced hypothermia (33.5–34.5 °C) implemented within 6 h of birth in term infants at highest risk for brain injury (as defined by specific protocols) and with further treatment in neonatal intensive care units is associated with significantly fewer deaths and less neurodevelopmental disability at 18-month follow-up. The number needed to treat is nine.[137] Both cooling methods (systemic versus selective head cooling) were shown to be effective, but none of the studies compared them directly. The randomised trials produced remarkably consistent results despite using different methods of cooling.[138]

Treatment recommendations

Newly born infants born at or near-term with evolving moderate to severe hypoxic–ischaemic encephalopathy should be offered therapeutic hypothermia. Whole body cooling and selective head cooling are both appropriate strategies. Cooling should be initiated and conducted under clearly defined protocols with treatment in neonatal intensive care facilities and with the capability for multidisciplinary care. Treatment should be consistent with the protocols used in the randomised clinical trials (i.e., begin within 6 h of birth, continue for 72 h after birth, and rewarm over at least 4 h). Carefully monitor for known adverse effects of cooling, e.g., thrombocytopenia and hypotension. All treated infants should be followed up longitudinally.

General supportive care

Glucose[NRP-019A,NRP-019B]

Consensus on science

Newborns with lower blood glucose levels have a higher incidence of brain injury and adverse outcomes after a hypoxic–ischaemic insult, although no specific level associated with worse outcome has been identified (LOE 4[139];LOE 3[140]). Increased glucose levels after hypoxia–ischaemia do not appear to have adverse effects in studies of children (LOE 5[141]) or in animal studies (LOE 5[142]) and may be protective (LOE 5[143]). However, no randomised controlled trials have examined this question. Due to the paucity of data, no specific target glucose concentration range can be identified at present.

Treatment recommendation

Intravenous glucose infusion should be considered as soon as practical after resuscitation, with the goal of avoiding hypoglycaemia.

Timing of cord clamping[NRP-030A,NRP-030B,NRP-030C NRP-030D]

Consensus on science

For the uncomplicated birth at term there is evidence of a benefit to delaying cord clamping for a minimum time ranging from 1 min until the cord stops pulsating after delivery. Those with delayed clamping had improved iron status through early infancy but were more likely to receive phototherapy (LOE 1[144]). For an otherwise uncomplicated preterm birth, there is evidence of a benefit to delaying cord clamping for a minimum time ranging from 30 s to 3 min after delivery. Those who experienced delayed clamping in this group had higher blood pressures during stabilization and a lower incidence of intraventricular haemorrhage (LOE 1[145]) and received fewer blood transfusions[145] but were more likely to receive phototherapy (LOE 2[144]). There are limited data on the hazards or benefits of delayed cord clamping in the nonvigorous infant.[146,147]

Treatment recommendation

Delay in umbilical cord clamping for at least 1 min is recommended for newborn infants not requiring resuscitation. There is insufficient evidence to support or refute a recommendation to delay cord clamping in babies requiring resuscitation.

Withholding or discontinuing resuscitative efforts[NRP-025A,NRP-025B,NRP-025C,NRP-026A,NRP-026B,NRP-026C,RP-027A, NRP-027B]

Noninitiation of resuscitation

Consensus on science

For neonates at the margins of viability or those with conditions which predict a high risk of mortality or morbidity, attitudes and practice vary according to region and availability of resources (LOE 4[148]). Social science studies indicate that parents would like a larger role in the decisions to start resuscitation and continue life support of severely compromised newborns. Opinions among neonatal providers vary widely regarding the benefits and disadvantages of aggressive therapies in such newborns (LOE 4[149,150]). Some data are available to help identify conditions associated with high mortality and poor outcome (LOE 4[151,152]). Such conditions may include extreme prematurity and anomalies that predict extreme morbidity or early death. Treatment and outcome of infants at the margins of viability may be influenced by factors in addition to gestational age and birthweight.[153] Noninitiation of resuscitation and withdrawal of cardiorespiratory support are ethically equivalent.[154]

Treatment recommendation

When gestation, birth weight, or congenital anomalies are associated with almost certain early death and an unacceptably high morbidity is likely among the rare survivors, resuscitation is not indicated. In conditions associated with a high rate of survival and acceptable morbidity, resuscitation is nearly always indicated. In conditions associated with uncertain prognosis, when there is borderline survival and a relatively high rate of morbidity and when the burden to the child is high, the parents' views on resuscitation should be supported. There should be a consistent and coordinated approach from the obstetric and neonatal teams in applying these guidelines and in communicating with the parents in developing an agreed-upon management plan when possible. Once resuscitation is initiated it may be appropriate to subsequently decide to discontinue cardiorespiratory support and offer comfort care.

Discontinuation of resuscitation

Consensus on science

Available evidence, albeit from relatively small numbers of babies, suggests that babies born without a heart rate that has not returned by 10 min of age are likely to either die or have severe neurological disability (LOE 4[155,156]). It is not known whether there was significant selection bias in many of these studies, nor indeed that the babies included in these studies did receive "good-quality resuscitation." One study with a large contemporary cohort of infants (some randomised to postresuscitation hypothermia) indicates that in babies born without a detectable heart rate, the lack of ROSC after 10 min of age is associated with survival without severe neurological deficit in a small number of the survivors (LOE 4[157]). Data are not available regarding the number of infants who were deemed too sick for study entry or who died before enrollment. These factors may have resulted in a significant overestimation of the rate of intact survival among infants with an Apgar score of 0 at 10 min. In all reported series the cause of the asphyxia and the efficacy of the resuscitation process were not elucidated.

The evidence from seven LOE 5 studies[157,158] is insufficient to support or refute any recommendation regarding how much time should elapse with a heart rate of <60 but >0 beats per minute before discontinuing resuscitative efforts.

Treatment recommendation

In a newly born baby with no detectable heart rate which remains undetectable for 10 min, it is appropriate to then consider stopping resuscitation. The decision to continue resuscitation efforts when the infant has a heart rate of zero for longer than 10 min is often complex and may be influenced by issues such as the presumed aetiology of the arrest, gestation of the baby, potential reversibility of the situation, and the parents' previously expressed feelings about acceptable risk of morbidity.

The evidence of outcome when the heart rate is <60 beats per minute at birth and persists after 10 or 15 min of continuous and adequate resuscitative efforts at delivery is insufficient to guide decisions as to whether to withhold or to continue resuscitation.

Personnel needs at elective caesarean sections[NRP-010A,NRP-010B,NRP-010C]

Consensus on science

Retrospective studies show that delivery by Caesarean section at term under regional anaesthesia is associated with a small increase in risk of receiving bag-mask ventilation during neonatal resuscitation compared with unassisted vaginal birth. The number needed to treat equals 35 (LOE 4[159,160]). Five retrospective studies showed that delivery by Caesarean section at term under regional anaesthesia did not increase the risk of requirement for intubation during neonatal resuscitation compared with unassisted vaginal birth (LOE 4[161,162]). There is no evidence addressing this question in babies born at 34–36 weeks' gestation.

Treatment recommendations

When an infant without antenatally identified risk factors is delivered at term by Caesarean section under regional anaesthesia, a provider capable of performing bag-mask ventilation should be present at the delivery. It is not necessary for a provider skilled in neonatal intubation to be present at that delivery.

Educational techniques for teaching, assessing, and maintaining resuscitation knowledge and skills

Simulation[NRP-032A,NRP-032B,NRP-032C,EIT-019A,EIT-019B]

Consensus on science

There is a lack of uniformity in the definition of simulation as a learning methodology, determination of relevant outcomes, and use of appropriate measurement tools. Use of simulation as an adjunct to traditional education methodologies may enhance performance of healthcare professionals in actual clinical settings (LOE 1[163]; LOE 3[164]) and simulated resuscitations (LOE 1[165]; LOE 2[166]). Some studies did not show any difference in performance between standard training and simulation training in a clinical setting (LOE 1[167]) or using other means of evaluation (LOE 1[168]). No studies were found that revealed simulation-based training produced inferior results compared with traditional methodologies.

Treatment recommendations

Simulation should be used as a methodology in resuscitation education. The most effective interventions and evaluation methodologies remain to be defined.

Briefings and debriefings[NRP-033A,NRP-033B,EIT-001A,EIT-001B]

Consensus on science

Evidence from one prospective randomised controlled study (LOE 1[169]) and 17 other studies (LOE 3–4) of briefings and debriefings document improvement in the acquisition of content knowledge, technical skills, or behavioral skills required for effective and safe resuscitation. Only a single study (LOE 4[170]) revealed no effect of briefing/debriefing on performance, and no studies indicated that the use of briefings and debriefings had any negative effects.

Treatment recommendations

It is reasonable to recommend the use of briefings and debriefings during learning activities while caring for simulated patients and during clinical activities.

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Appendix A. Evidence-based worksheets for part 11: neonatal resuscitation: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations

Task force	WS ID	PICO title	Short title	Authors	URL
EIT	EIT-001A	For resuscitation teams (P), do briefings/debriefings (I), when compared to no briefings/debriefings (C), improve performance or outcomes (O)? (INTERVENTION)	Debriefing of CPR performance	Dana Edelson, Trevor Yuen	http://circ.ahajournals.org/site/C2010/EIT-001A.pdf
EIT	EIT-001B	For resuscitation teams (P), do briefings/debriefings (I), when compared to no briefings/debriefings (C), improve performance or outcomes (O)? (INTERVENTION)	Debriefing of CPR performance	Jasmeet Soar	http://circ.ahajournals.org/site/C2010/EIT-001B.pdf
EIT	EIT-019A	In participants undergoing BLS/ALS courses (P), does the inclusion of more realistic techniques (e.g., high fidelity manikins, in situ training) (I), as opposed to standard training (e.g., low fidelity, education centre) (C), improve outcomes (e.g., skills performance on manikins, skills performance in real arrests, willingness to perform, etc.) (O)?	High fidelity training	Jordan Duval-Arnould, Elizabeth Hunt	http://circ.ahajournals.org/site/C2010/EIT-019A.pdf
EIT	EIT-019B	In participants undergoing BLS/ALS courses (P), does the inclusion of more realistic techniques (e.g., high fidelity manikins, in situ training) (I), as opposed to standard training (e.g., low fidelity, education centre) (C), improve outcomes (e.g., skills performance on manikins, skills performance in real arrests, willingness to perform, etc.) (O)?	High fidelity training	Judith Finn	http://circ.ahajournals.org/site/C2010/EIT-019B.pdf
NRP	NRP-001A	For neonates requiring resuscitation (P), is any adjuct measure (e.g., CO_2 detection, pulse oximeter) as effective as the usual clinical findings (e.g., heart rate, chest movement) effective to improve outcome (O)?	Adjuncts: CO2 detection, pulse oximeter	John Kattwinkel	http://circ.ahajournals.org/site/C2010/NRP-001A.pdf
NRP	NRP-001B	For neonates requiring resuscitation (P), is any adjuct measure (e.g., CO_2 detection, pulse oximeter) as effective as the usual clinical findings (e.g., heart rate, chest movement) effective to improve outcome (O)?	Adjuncts: CO2 detection, pulse oximeter	Yacov Rabi	http://circ.ahajournals.org/site/C2010/NRP-001B.pdf
NRP	NRP-002A	In the neonates infant (preterm and term) receiving respiratory support (P), does the use of CPAP (I) versus no-CPAP or IPPV (C) improve outcome – specify (O)?	CPAP and IPPV	Colm O'Donnell	http://circ.ahajournals.org/site/C2010/NRP-002A.pdf
NRP	NRP-002B	In the neonates infant (preterm and term) receiving respiratory support (P), does the use of CPAP (I) versus no-CPAP or IPPV (C) improve outcome – specify (O)?	CPAP and IPPV	Douglas McMillan	http://circ.ahajournals.org/site/C2010/NRP-002B.pdf
NRP	NRP-003A	In neonatess receiving respiratory support (P) does the use of face mask interface (I) versus CPAP, NPCPAP, NC (C) (excluding intubation improve outcome) (O)?	Face mask interface versus CPAP etc	Colin Morley	http://circ.ahajournals.org/site/C2010/NRP-003A.pdf

Task force	WS ID	PICO title	Short title	Authors	URL
NRP	NRP-003B	In neonates receiving respiratory support (P) does the use of face mask interface (I) versus CPAP, NPCPAP, NC (C) (excluding intubation) improve outcome (O)?	Face mask interface versus CPAP etc	Yacov Rabi	http://circ.ahajournals.org/site/C2010/NRP-003B.pdf
NRP	NRP-004A	In neonates receiving resuscitation (P) does the use of mouth-to-mouth, mouth-to-mask, mouth tube to mask (I) as compared to a self-inflating bag (C) give equivalent outcomes (stable spontaneous breathing) (O), when devices for delivering PPV are not available?	Self-inflating bag versus mouth techniques	Nalini Singhal	http://circ.ahajournals.org/site/C2010/NRP-004A.pdf
NRP	NRP-004B	In neonates receiving resuscitation (P) does the use of mouth-to-mouth, mouth-to-mask, mouth tube to mask (I) as compared to a self-inflating bag (C) give equivalent outcomes (stable spontaneous breathing) (O), when devices for delivering PPV are not available?	Self-inflating bag versus mouth techniques	Maria Fernanda de Almeida	http://circ.ahajournals.org/site/C2010/NRP-004B.pdf
NRP	NRP-005A	In neonates receiving positive pressure ventilation (P) does the use of gas volume monitoring (I) versus clinical assessment with or without pressure monitoring (C) improve clinical outcome (O)?	Ventilation volume monitoring	Steven Ringer	http://circ.ahajournals.org/site/C2010/NRP-005A.pdf
NRP	NRP-005B	In neonates receiving positive pressure ventilation (P) does the use of gas volume monitoring (I) versus clinical assessment with or without pressure monitoring (C) improve clinical outcome (O)?	Ventilation volume monitoring	Khalid Aziz	http://circ.ahajournals.org/site/C2010/NRP-005B.pdf
NRP	NRP-005C	In neonates receiving positive pressure ventilation (P) does the use of gas volume monitoring (I) versus clinical assessment with or without pressure monitoring (C) improve clinical outcome (O)?	Ventilation volume monitoring	Jane McGowan	http://circ.ahajournals.org/site/C2010/NRP-005C.pdf
NRP	NRP-006A	In neonates receiving chest compressions (P) do other ratios (5:1, 15:2) (I) versus a 3:1 (C) improve outcomes (O)?	Compression ventilation ratio	Lindsay Mildenhall	http://circ.ahajournals.org/site/C2010/NRP-006A.pdf
NRP	NRP-006B	In neonates receiving chest compressions (P) do other ratios (5:1, 15:2) (I) versus a 3:1 (C) improve outcomes (O)?	Compression ventilation ratio	Myra Wyckoff	http://circ.ahajournals.org/site/C2010/NRP-006B.pdf
NRP	NRP-007A	In neonates (P) receiving chest compressions does the two thumb (I) versus two finger (C) method of administration improve outcome (O)?	Two thumb versus two finger	Lindsay Mildenhall	http://circ.ahajournals.org/site/C2010/NRP-007A.pdf
NRP	NRP-007B	In neonates (P) receiving chest compressions does the two thumb (I) versus two finger(C) method of administration improve outcome (O)?	Two thumb versus two finger	Myra Wyckoff	http://circ.ahajournals.org/site/C2010/NRP-007B.pdf
NRP	NRP-008A	Among neonates (<=28 days) with a HR <60 bpm despite adequate ventilation and chest compressions, does the IV route compared with the ET route of adrenaline administration: 1. increase heart rate >100 bpm faster, 2. increase ROSC, or 3. increase survival to discharge?	IV versus ET adrenaline	Jonathan Wyllie	http://circ.ahajournals.org/site/C2010/NRP-008A.pdf

Task force	WS ID	PICO title	Short title	Authors	URL
NRP	NRP-008B	Among neonates (<=28 days) with a HR <60 bpm despite adequate ventilation and chest compressions, does the IV route compared with the ET route of adrenaline administration: 1. increase heart rate >100 bpm faster, 2. increase ROSC, or 3. increase survival to discharge?	IV versus ET adrenaline	Gary Weiner	http://circ.ahajournals.org/site/C2010/NRP-008B.pdf
NRP	NRP-009A	Among neonates (<=28 days) with HR <60 bpm does HDE (IV >0.03 mg/kg or ET >0.1 mg/kg) compared with SDE: 1. increase HR >100 bpm faster, 2. increase ROSC, or 3. increase survival to discharge?	Adrenaline dose	Jonathan Wyllie	http://circ.ahajournals.org/site/C2010/NRP-009A.pdf
NRP	NRP-009B	Among neonates (<=28 days) with HR <60 bpm does HDE (IV >0.03 mg/kg or ET >0.1 mg/kg) compared with SDE: 1. increase HR >100 bpm faster, 2. increase ROSC, or 3. increase survival to discharge?	Adrenaline dose	Gary Weiner	http://circ.ahajournals.org/site/C2010/NRP-009B.pdf
NRP	NRP-010A	For infants delivered at >=34 weeks gestation (P), is delivery by elective c-section under regional anaesthesia (I) in comparison with unassisted vertex vaginal deliveries (C) associated with an increased risk of requirement for intubation during resuscitation (O)?	Prenatal prediction of respiratory compromise	Marilyn B. Escobedo	http://circ.ahajournals.org/site/C2010/NRP-010A.pdf
NRP	NRP-010B	For infants delivered at >=34 weeks gestation (P), is delivery by elective c-section under regional anaesthesia (I) in comparison with unassisted vertex vaginal deliveries (C) associated with an increased risk of requirement for intubation during resuscitation (O)?	Prenatal prediction of respiratory compromise	Ben Stenson	http://circ.ahajournals.org/site/C2010/NRP-010B.pdf
NRP	NRP-010C	For infants delivered at >=34 weeks gestation (P), is delivery by elective c-section under regional anaesthesia (I) in comparison with unassisted vertex vaginal deliveries (C) associated with an increased risk of requirement for intubation during resuscitation (O)?	Prenatal prediction of respiratory compromise	Dianne Atkins, Edgardo Szyld	http://circ.ahajournals.org/site/C2010/NRP-010C.pdf
NRP	NRP-011A	In depressed neonates with clear amniotic fluid (P) does suctioning of the mouth and nose (I) versus none (C) improve outcome (O)	Clear amniotic fluid	Sithembiso Velaphi, Dharmapuri Vidyasagar	http://circ.ahajournals.org/site/C2010/NRP-011A.pdf
NRP	NRP-012A	In depressed neonates born through meconium stained amniotic fluid (P), does tracheal suctioning (I) versus no suctioning (C) improve outcome (O)?	Stained amniotic fluid	Sithembiso Velaphi, Dharmapuri Vidyasagar	http://circ.ahajournals.org/site/C2010/NRP-012A.pdf
NRP	NRP-013A	When resuscitating or stabilizing newborns at birth (P), is there an oxygen administration strategy (I) that is superior to any other (C) in improving outcome (O)?	Oxygen administration	Jay Goldsmith	http://circ.ahajournals.org/site/C2010/NRP-013A.pdf

Task force	WS ID	PICO title	Short title	Authors	URL
NRP	NRP-013B	When resuscitating or stabilizing newborns at birth (P), is there an oxygen administration strategy (I) that is superior to any other (C) in improving outcome (O)?	Oxygen administration	Sam Richmond	http://circ.ahajournals.org/site/C2010/NRP-013B.pdf
NRP	NRP-014A	In neonates receiving resuscitation or stabilization (P), is the saturation demonstrated during normal birth (I) preferable to some other target (C), when considering outcome for premature and term neonates (O)?	Oxygen saturation target	John Kattwinkel	http://circ.ahajournals.org/site/C2010/NRP-014A.pdf
NRP	NRP-014B	In neonates receiving resuscitation or stabilization (P), is the saturation demonstrated during normal birth (I) preferable to some other target (C), when considering outcome for premature and term neonates (O)?	Oxygen saturation target	Colin Morley	http://circ.ahajournals.org/site/C2010/NRP-014B.pdf
NRP	NRP-015A	In neonates (P) receiving positive pressure during resuscitation, is positive pressure ventilation by T-piece resuscitator (I) superior to bag ventilation (C) for improving outcome – specify (O)?	T-piece resuscitator	David Boyle	http://circ.ahajournals.org/site/C2010/NRP-015A.pdf
NRP	NRP-015B	In neonates (P) receiving positive pressure during resuscitation, is positive pressure ventilation by T-piece resuscitator (I) superior to bag ventilation (C) for improving outcome – specify (O)?	T-piece resuscitator	Ben Stenson	http://circ.ahajournals.org/site/C2010/NRP-015B.pdf
NRP	NRP-015C	In neonates(P) receiving positive pressure during resuscitation, is positive pressure ventilation by T-piece resuscitator (I) superior to bag ventilation (C) for improving outcome – specify (O)?	T-piece resuscitator	David Field	http://circ.ahajournals.org/site/C2010/NRP-015C.pdf
NRP	NRP-016A	For neonates (P) following attempted tracheal intubation, is CO_2 detection (I) superior to clinical assessment (C) for confirming tracheal location (O)?	CO2 detection	Jonathan Wyllie	http://circ.ahajournals.org/site/C2010/NRP-016A.pdf
NRP	NRP-017A	For neonates requiring positive pressure ventilation (P), is LMA (I) an effective alternative to mask or tracheal ventilation (C) for improving outcome (O)? (achieving stable vital signs and reducing the need for subsequent tracheal intubation)?	LMA	Gary Weiner	http://circ.ahajournals.org/site/C2010/NRP-017A.pdf
NRP	NRP-017B	For neonates requiring positive pressure ventilation (P), is LMA (I) an effective alternative to mask or tracheal ventilation (C) for improving outcome (O)? (achieving stable vital signs and reducing the need for subsequent tracheal intubation)?	LMA	Enrique Udaeta	http://circ.ahajournals.org/site/C2010/NRP-017B.pdf

Task force	WS ID	PICO title	Short title	Authors	URL
NRP	NRP-018A	For non-intubated bradycardic neonates (P) requiring positive pressure ventilation, is the CO_2 monitoring device (I) more effective than chest rise, colour (C) for assessing adequate ventilation (O)?	Bradycardia and CO2 monitoring	Colm O'Donnell	http://circ.ahajournals.org/site/C2010/NRP-018A.pdf
NRP	NRP-018B	For non-intubated bradycardic neonates (P) requiring positive pressure ventilation, is the CO_2 monitoring device (I) more effective than chest rise, colour (C) for assessing adequate ventilation (O)?	Bradycardia and CO2 monitoring	Masanori Tamura	http://circ.ahajournals.org/site/C2010/NRP-018B.pdf
NRP	NRP-018C	For non-intubated bradycardic neonates (P) requiring positive pressure ventilation, is the CO_2 monitoring device (I) more effective than chest rise, colour (C) for assessing adequate ventilation (O)?	Bradycardia and CO_2 monitoring	Steven Ringer	http://circ.ahajournals.org/site/C2010/NRP-018C.pdf
NRP	NRP-019A	In neonates requiring resuscitation, (P) will the early use of supplemental glucose (I) during and/or following delivery room resuscitation, versus none (C) improve outcome (i.e., avoidance of hypoglycaemia, reduced long-term neurological morbidity) (O)?	Supplemental glucose	Jane McGowan	http://circ.ahajournals.org/site/C2010/NRP-019A.pdf
NRP	NRP-019B	In neonates requiring resuscitation, (P) will the early use of supplemental glucose (I) during and/or following delivery room resuscitation, versus none (C) improve outcome (i.e., avoidance of hypoglycaemia, reduced long-term neurological morbidity) (O)?	Supplemental glucose	Jeffrey Perlman	http://circ.ahajournals.org/site/C2010/NRP-019B.pdf
NRP	NRP-020A	In neonates requiring resuscitation, does the administration of emergency medications (P) by intra-osseous infusion (I) versus the intravenous route improve outcome (O)?	IO versus IV	William Engle	http://circ.ahajournals.org/site/C2010/NRP-020A.pdf
NRP	NRP-021A	In neonates requiring resuscitation and not responding to CPR (P), does the administration of sodium bicarbonate (I) versus no bicarbonate (C) improve outcome (O)?	Sodium bicarbonate	Jeffrey Perlman	http://circ.ahajournals.org/site/C2010/NRP-021A.pdf
NRP	NRP-021B	In neonates requiring resuscitation and not responding to CPR (P), does the administration of sodium bicarbonate (I) versus no bicarbonate (C) improve outcome (O)?	Sodium bicarbonate	Dianne Atkins, Sam Richmond	http://circ.ahajournals.org/site/C2010/NRP-021B.pdf
NRP	NRP-022A	In apneic neonates suspected of narcotic depression (P), does naloxone (I) when compared to effective ventilation without naloxone (C) improve outcome (O)?	Nalaxone	Ruth Guinsburg	http://circ.ahajournals.org/site/C2010/NRP-022A.pdf
NRP	NRP-022B	In apneic neonates suspected of narcotic depression (P), does naloxone (I) when compared to effective ventilation without naloxone (C) improve outcome (O)?	Nalaxone	Myra Wyckoff	http://circ.ahajournals.org/site/C2010/NRP-022B.pdf

Task force	WS ID	PICO title	Short title	Authors	URL
NRP	NRP-023A	In preterm neonates under radiant warmers (P), does increased room temperature, thermal mattress, or other intervention (I) as compared to plastic wraps alone (C) improve outcome (O)?	Warming adjuncts	Marilyn B. Escobedo, Michael Watkinson	http://circ.ahajournals.org/site/C2010/NRP-023A.pdf
NRP	NRP-024A	In term neonates at risk for hypoxic-ischaemic encephalopathy secondary to intra-partum hypoxia (P) does selective/whole body cooling (I) versus standard therapy (C), result in improved outcome (O)?	Hypothermia (induced)	Jeffrey Perlman	http://circ.ahajournals.org/site/C2010/NRP-024A.pdf
NRP	NRP-024B	In term neonates at risk for hypoxic-ischaemic encephalopathy secondary to intra-partum hypoxia (P) does selective/whole body cooling (I) versus standard therapy (C), result in improved outcome (O)?	Hypothermia (induced)	Peter Davis	http://circ.ahajournals.org/site/C2010/NRP-024B.pdf
NRP	NRP-025A	In term neonates without a detectable heart rate and no other signs of life (P) is 10 min (I) as opposed to 15 min or longer (C) of effective resuscitation a reliable measure of outcome (abnormal neurological examination and/or death) (O)?	Duration of CPR with asystole and outcome	Steve Byrne	http://circ.ahajournals.org/site/C2010/NRP-025A.pdf
NRP	NRP-025B	In term neonates without a detectable heart rate and no other signs of life (P) is 10 min (I) as opposed to 15 min or longer (C) of effective resuscitation a reliable measure of outcome (abnormal neurological examination and/or death) (O)?	Duration of CPR with asystole and outcome	Jay Goldsmith	http://circ.ahajournals.org/site/C2010/NRP-025B.pdf
NRP	NRP-025C	In term neonates without a detectable heart rate and no other signs of life (P) is 10 min (I) as opposed to 15 min or longer (C) of effective resuscitation a reliable measure of outcome (abnormal neurological examination and/or death) (O)?	Duration of CPR with asystole and outcome	Ruth Guinsburg	http://circ.ahajournals.org/site/C2010/NRP-025C.pdf
NRP	NRP-026A	In term neonates with a heart rate <60 and no other signs of life (P), is 10 min (I) as opposed to 15 min or longer (C) of effective resuscitation a reliable measure of outcome (abnormal neurological examination and/or death) (O)?	Duration of CPR with bradycardia and outcome	Steve Byrne	http://circ.ahajournals.org/site/C2010/NRP-026A.pdf
NRP	NRP-026B	In term neonates with a heart rate <60 and no other signs of life (P), is ten minutes (I) as opposed to 15 min or longer (C) of effective resuscitation a reliable measure of outcome (abnormal neurological examination and/or death) (O)?	Duration of CPR with bradycardia and outcome	Jay Goldsmith	http://circ.ahajournals.org/site/C2010/NRP-026B.pdf
NRP	NRP-026C	In term neonates with a heart rate <60 and no other signs of life (P), is ten minutes (I) as opposed to 15 min or longer (C) of effective resuscitation a reliable measure of outcome (abnormal neurological examination and/or death) (O)?	Duration of CPR with bradycardia and outcome	Ruth Guinsburg	http://circ.ahajournals.org/site/C2010/NRP-026C.pdf

Task force	WS ID	PICO title	Short title	Authors	URL
NRP	NRP-027A	In neonates at the limits of viability or anomalies associated with lethal outcomes (P) does the non initiation (I) versus initiation (C) of resuscitation result in an outcome that is ethically justified (O)	Futile resuscitation rules	Steve Byrne	http://circ.ahajournals.org/site/C2010/NRP-027A.pdf
NRP	NRP-027B	In neonates at the limits of viability or anomalies associated with lethal outcomes (P) does the non initiation (I) versus initiation (C) of resuscitation result in an outcome that is ethically justified (O)	Futile resuscitation rules	Jay Goldsmith	http://circ.ahajournals.org/site/C2010/NRP-027B.pdf
NRP	NRP-028A	In depressed neonates requiring positive pressure ventilation (P) does the administration of longer inspiratory times, higher inflation pressures, use of PEEP (I) as compared to standard management (C) improve outcome (O)?	Ventilation times and pressures	David Boyle	http://circ.ahajournals.org/site/C2010/NRP-028A.pdf
NRP	NRP-028B	In depressed neonates requiring positive pressure ventilation (P) does the administration of longer inspiratory times, higher inflation pressures, use of PEEP (I) as compared to standard management (C) improve outcome (O)?	Ventilation times and pressures	Ben Stenson	http://circ.ahajournals.org/site/C2010/NRP-028B.pdf
NRP	NRP-029A	In neonates requiring resuscitation and unresponsive to chest compressions/adrenaline (P) does the administration of volume (I) versus no volume (C) improve outcome (O)	Volume resuscitation with CPR	Susan Niermeyer	http://circ.ahajournals.org/site/C2010/NRP-029A.pdf
NRP	NRP-029B	In neonates requiring resuscitation and unresponsive to chest compressions/adrenaline (P) does the administration of volume (I) versus no volume (C) improve outcome (O)	Volume resuscitation with CPR	Douglas McMillan	http://circ.ahajournals.org/site/C2010/NRP-029B.pdf
NRP	NRP-029C	In neonates requiring resuscitation and unresponsive to chest compressions/adrenaline (P) does the administration of volume (I) versus no volume (C) improve outcome (O)	Volume resuscitation with CPR	Masanori Tamura	http://circ.ahajournals.org/site/C2010/NRP-029C.pdf
NRP	NRP-030A	In neonates (P), does delayed cord clamping cord or milking of the cord (I) versus standard management (C), improve outcome (O)	Umbilical cord clamping and milking	Susan Niermeyer	http://circ.ahajournals.org/site/C2010/NRP-030A.pdf
NRP	NRP-030B	In neonates (P), does delayed cord clamping cord or milking of the cord (I) versus standard management (C), improve outcome (O)	Umbilical cord clamping and milking	Dianne Atkins, Nalini Singhal	http://circ.ahajournals.org/site/C2010/NRP-030B.pdf
NRP	NRP-030C	In neonates (P), does delayed cord clamping cord or milking of the cord (I) versus standard management (C), improve outcome (O) (milking of the cord)	Umbilical cord clamping and milking	Gary Weiner	http://circ.ahajournals.org/site/C2010/NRP-030C.pdf
NRP	NRP-030D	In neonates (P), does delayed cord clamping cord or milking of the cord (I) versus standard management (C), improve outcome (O)?	Umbilical cord clamping and milking	Rintaro Mori	http://circ.ahajournals.org/site/C2010/NRP-030D.pdf

Task force	WS ID	PICO title	Short title	Authors	URL
NRP	NRP-031A	In neonates born to febrile mothers (P) does intervention to normalise temperature (I), compared to standard care (C) improve outcome (O)?	Maternal fever	Jeffrey Perlman	http://circ.ahajournals.org/site/C2010/NRP-031A.pdf
NRP	NRP-031B	In neonates born to febrile mothers (P) does intervention to normalise temperature (I), compared to standard care (C) improve outcome (O)	Maternal fever	Steven Ringer	http://circ.ahajournals.org/site/C2010/NRP-031B.pdf
NRP	NRP-032A	In participants undergoing resuscitation courses (P), does the inclusion of more realistic techniques (e.g., high fidelity manikins, in situ training) (I), as opposed to standard training (e.g., low fidelity, education centre) (C), improve outcomes (e.g., skills performance) (O)	Impact of realistic training on skills performance	Jane McGowan	http://circ.ahajournals.org/site/C2010/NRP-032A.pdf
NRP	NRP-032B	In participants undergoing resuscitation courses (P), does the inclusion of more realistic techniques (e.g., high fidelity manikins, in-situ training) (I), as opposed to standard training (e.g., low fidelity, education centre) (C), improve outcomes (e.g., skills performance) (O)	Impact of realistic training on skills performance	Louis Halamek	http://circ.ahajournals.org/site/C2010/NRP-032B.pdf
NRP	NRP-032C	In participants undergoing resuscitation courses (P), does the inclusion of more realistic techniques (e.g., high fidelity manikins, in situ training) (I), as opposed to standard training (e.g., low fidelity, education centre) (C), improve outcomes (e.g., skills performance) (O)	Impact of realistic training on skills performance	Khalid Aziz	http://circ.ahajournals.org/site/C2010/NRP-032C.pdf
NRP	NRP-033A	For hospital resuscitation teams (P), do team briefings/debriefings (I), when compared to no briefings/debriefings (C), improve team performance (O)? (INTERVENTION)	Impact of debreifing on team performance	Dianne Atkins, Nalini Singhal	http://circ.ahajournals.org/site/C2010/NRP-033A.pdf
NRP	NRP-033B	For hospital resuscitation teams (P), do team briefings/debriefings (I), when compared to no briefings/debriefings (C), improve team performance (O)? (INTERVENTION)	Impact of debreifing on team performance	Louis Halamek	http://circ.ahajournals.org/site/C2010/NRP-033B.pdf

Appendix B.

CoSTR Part 11: writing group disclosures

CoSTR Part 11: worksheet collaborator disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Jeffrey M. Perlman	Weill Cornell Medical College – Professor of Paediatrics	[a]NIH funding-Co-Investigator – Improving antimicrobial prescribing practices in the NICU	None	[b]University of Miami, and Cook County Chicago	None	None	None
Jonathan Wyllie	South Tees Foundation NHS Trust Health Service Provider NHS UK Consultant Neonatologist and Clinical Director of Neonatology	None	None	None	None	[b]Volunteer ICC Newborn Life Support ERC Volunteer author European Newborn Life Support Guidelines Volunteer author UK Newborn Resuscitation Guidelines Volunteer co-author Advanced Paediatric Life support Guidelines Volunteer member Advanced Life Support Group UK Volunteer acting chair Newborn Life Support Working Group for RC (UK) Volunteer British Association of Perinatal Medicine Neonatal Services and staffing working group	None
Dianne L. Atkins	University of Iowa; Prof. [a]I am a compensated worksheet editor for AHA 2010 Guidelines process. The compensation is divided: 2/3 to my institution and 1/3 directly to me. The amount paid to my institution does not alter my salary	None	None	None	None	None	None
Leon Chameides	Retired – Emeritus Director Paediatric Cardiology	None	None	None	None	None	None
Jay P. Goldsmith	Paediatrix Medical Group: single specialty multi-site group practice – Neonatologist	None	None	None	None	None	None
Ruth Guinsburg	Federal University of São Paulo – Full Professor of Paediatrics	None	None	None	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing – Professor; AHA ECC Product Development – Senior Science Editor–[a]the significant AHA compensation is designed to provide protected time for editing and writing responsibilities. I have a significant relationship with the AHA to support the mission of the AHA with the production of CoSTR and AHA Guidelines for CPR and ECC	None	None	None	None	None	None

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
John Kattwinkel	University of Virginia – Professor of Paediatrics	[b]American Academy of Paediatrics research grant to study resuscitators detection of compliance while administering positive pressure ventilation by resuscitation bag	None	None	None	None	[b]Expert Witness – Curley versus Gordon et al, Boston, MA
Colin Morley	Retired Professor of Neonatal Medicine	None	None	Honoraria [b]Japanese Neonatal Society [b]Neonatal Ventilation and Resuscitation-Zagreb, Croatia [b]UK Middles-borough Neonatal Symposium	None	[b]Drager Medical about equip design [b]Education video on neonatal CPAP	None
Sam Richmond	UK National Health Service – Consultant Neonatologist	None	None	None	None	None	None
Wendy M. Simon	American Academy of Paediatrics – Director of Life Support Programs	None	None	None	None	None	None
Nalini Singhal	University of Calgary – Professor	[b]AAP grant looking at effect of PEEP with and with oxygen on resuscitation Developing a International program for resuscitation, Helping Babies Breath	None	None	None	None	None
Edgardo Szyld	Fundasamin, Foundation for Womenĭs and Infant Health – Executive Director of a non profit institution (NGO)	None	None	None	None	None	None
Masanori Tamura	Saitama Medical Center, Saitama Medical University – Professor and Chairman, Department of Paediatrics	None	None	None	None	None	None

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/advisory board	Other
Sithembiso Velaphi	Univ of the Witwatersrand Univ-lecturer; Chris Hani Baragwanath hosp Govt hosp, principal specialist	None	None	None	None	None	None
Jonathan Wyllie	South Tees Foundation NHS Trust Health Service Provider NHS UK Consultant Neonatologist and Clinical Director of Neonatology	None	None	None	None	[b]Volunteer ICC Newborn Life Support ERC Volunteer author European Newborn Life Support Guidelines [b]Volunteer author UK Newborn Resuscitation Guidelines [b]Volunteer co-author Advanced Paediatric Life support Guidelines [b]Volunteer member Advanced Life Support Group UK [b]Volunteer acting chair Newborn Life Support Working Group for RC(UK) [b]Volunteer British Association of Perinatal Medicine Neonatal Services and staffing working group	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Significant.

[b] Modest.

Worksheet Collaborator Disclosures

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/ advisory board	Other
Khalid Aziz	University of Alberta – Associate Professor	None	None	None	None	None	None
David Boyle	Indiana University School of Medicine, Associate Professor of Paediatrics; University Paediatric Associates, Staff Neonatologist	None	None	None	None	None	None
Steven Byrne	South Tees Hospital Foundation NHS Trust: National Health Service Trust	None	None	None	None	None	None
Peter Davis	The Royal Women's Hospital, Melbourne, Australia – Staff Neonatologist	None	None	None	None	None	None
William Engle	Indiana University School of Medicine Professor of Paediatrics	None	None	None	None	None	None
Marilyn Escobedo	University of Oklahoma – Professor of Paediatrics	None	None	None	None	None	None
Maria Fernanda de Almeida	Federal University of São Paulo: Full time work (40h/week) at Neonatal Division – Department of Paediatrics – Assoc. Prof; Brazilian Paediatric Society: Voluntary work at Brazilian Neonatal Resuscitation Program – NRP Steering Committee – Co-chair	None	None	None	None	None	None
David Field	University of Leicester: Higher educational institution – UK Government funded – Professor of Neonatal Medicine	None	None	None	None	None	None
Judith Finn	University of Western Australia – Professor	[a]Multiple National Health and Medical Research Grants (NH&MRC), National Heart Foundation Australia and State Government grants of >$10,000 since 1999. No money came to me – all came to my University to employ research staff and meet research expenses. No grants were directly related to any topic on which I am undertaking a Worksheet and none involved the trialing of a commercial product	None	[b]Less than $1000 from the Japanese Resuscitation Council to speak at their JRC Conference in Osaka in 2009	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/ advisory board	Other
Louis P. Halamek	Stanford University – Associate Professor	[a]Laerdal Foundation I was the Principal Investigator on 3 year grant (2006–2009) funded by the Laerdal Foundation in the amount of $450,000 USD that was given to the simulation center I direct, the Center for Advanced Paediatric and Perinatal Education at Packard Children's Hospital at Stanford. This grant ended 2009-07-31. This grant was not a source of support for my salary	None	None	None	[b]I provide consultation services regarding simulation product design and function to Laerdal Medical, Inc., and Advanced Medical Simulation, Inc.	[b]I provide medicolegal consultation services to trial attorneys in the U.S., advising on questions relating to neonatal intensive care
Jane E. McGowan	St. Christopher's Paediatric Associates: Practice Group for Children's Hospital – part of Tenet Healthcare – Attending Neonatologist	None	None	[b]Received honorarium for giving at talk for the March of Dimes on "NRP and the Preterm Infant."	None	None	None
Douglas McMillan	Dalhousie University and Academic Paediatrics Incorporated: University and Department of Paediatrics Financial group – Professor and Head, Division of Neonatal Perinatal medicine	None	None	[a]Medical Legal consulting fees for different hospitals and the Canadian Medical Protective Association (occasionally for the plaintiff) – presently "remitted" to Academic Paediatrics Incorporated [b]Consulting fees for program reviews related to newborn care and associated education programs – presently "remitted" to Academic Paediatrics Incorp.	None	[b]Consulting fees for program reviews related to newborn care and associated education programs – presently "remitted" to Academic Paediatrics Incorporated	[b]Medical Legal consulting fees for different hospitals and the Canadian Medical Protective Association (occasionally for the plaintiff) – presently "remitted" to Academic Paediatrics Incorporated
Lindsay Mildenhall	Counties Manukau District Health Board Auckland New Zealand: Public Health Care Provider – Consultant Neonatologist	None	None	None	None	None	None
Rintaro Mori	Osaka Medical Center and Research Institute for Maternal and Child Health: a public children's hosp. run by a local government. – Division Director of Strategic Planning & Collaboration	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Susan Niermeyer	University of Colorado Denver School of Medicine: professor, clinical neonatologist – Professor of Paediatrics	[a]Editorship, Helping Babies Breathe, American Academy of Paediatrics 2008–2009 Salary support through contract with University of Colorado Denver School of Medicine	None	None	None	None	None
Colm O'Donnell	The National Maternity Hospital, Holles Street, Dublin 2, Ireland – Consultant Neonatologist; Our Lady's Children's Hospital, Crumlin, Dublin 12, Ireland – Consultant Neonatologist; University College Dublin, Ireland: University medical school – Clinical Lecturer	None	None	[b]I have received honoraria from Chiesi Pharma (makers of Curosurf) for speaking at 2 educational courses and 3 scientific meetings (i.e., on 5 occasions) in the last 2 years . The combined total of these honoraria is less than 1000 euros	None	None	None
Yacov Rabi	Alberta Health Services: Provide employment income for my role as a neonatologist – Physician; University of Calgary: Provides income for my role as an Assistant Professor of Medicine	None	[b]Supply of modified Neopuff circuits for a randomised control trial by Fisher Paykell.	None	None	None	None
Steven Ringer	Brigham and Women's Hospital: Non-profit Hospital–Chief, Newborn Medicine	None	None	[b]Vermont Oxford Neonatal Network Annual Meeting	None	[b]Alere Healthcare Advisory Board Consulting on Clinical Care guidelines. Nothing relevant to topics with which I am involved	[a]Expert Witness in medical legal proceedings (Malpractice cases). A number of different attorneys/insurance companies Money comes directly to me. Nothing relevant to questions under consideration
Jasmeet Soar	North Bristol NHS Trust: Government Hospital in UK – Consultant in Anaesthetics & Intensive Care Medicine	None	None	None	None	None	None
Ben Stenson	United Kingdom Public Health Service-Consultant Neonatologist	None	None	None	None	None	None
Enrique Udaeta	Medica Sur Lomas: Private Maternity Hospital – Director Department of Neonatology	None	None	None	None	None	None
Dharmapuri Vidyasagar	University of Illinois Professor emeritus Professor emeritus	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/ advisory board	Other
Michael Watkinson	NHS Heart of England Foundation Trust, Birmingham, UK This is an NHS hospital in England Consultant neonatologist	None	None	None	None	None	None
Gary M. Weiner	St. Joseph Mercy Hospital – Attending Neonatologist	None	[a]Received equipment on-loan (3 resuscitation mannequins, 2 sets of video recording equipment) from Laerdal Medical Corporation to be used to complete a research project evaluating educational methods for teaching neonatal resuscitation. The value of the on-loan equipment is approximately $35,000	None	None	None	None
Myra Wyckoff	UT Southwestern Medical Center at Dallas – Associate Professor of Paediatrics	[a]PI, American Academy of Paediatrics. Neonatal Resuscitation Program. The ergonomics of neonatal cardiac compressions. $71,030. January 2008–2009 The funding comes to the institution [b]Co-Investigator (Mentor), American Academy of Paediatrics Neonatal Resuscitation Program Young Investigator Grant. Effectiveness of Plastic Head Coverings for Hypothermia Prevention in Preterm Newborns. January 2009–January 2010, $10,000 The funding comes to the institution	None	[b]February 5, 2009 Paediatric Grand Rounds. University of Oklahoma Health Sciences Center. OKC, OK	None	None	None

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[a] Significant.

[b] Modest.

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Part 12: Education, implementation, and teams 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations☆

Jasmeet Soar (Co-chair)*,[1], Mary E. Mancini (Co-chair)[1], Farhan Bhanji, John E. Billi, Jennifer Dennett, Judith Finn, Matthew Huei-Ming Ma, Gavin D. Perkins, David L. Rodgers, Mary Fran Hazinski, Ian Jacobs, Peter T. Morley, on behalf of the Education, Implementation, and Teams Chapter Collaborators

Application of resuscitation science to improve patient care and outcomes requires effective strategies for education and implementation. Systematic reviews suggest that there are significant opportunities to improve education, enhance individual and team performance, and avoid delays in implementation of guidelines into practice. It is within this context that the International Liaison Consensus on Resuscitation (ILCOR) Education, Implementation, and Teams (EIT) Task Force was established and addressed 32 worksheet topics. Reviewers selected topics from the *2005 International Consensus on Cardiopulmonary Resuscitation* (*CPR*) and *Emergency Cardiovascular Care* (*ECC*) *Science With Treatment Recommendations*[1] and new topics identified by an expert group.

One challenge for the EIT Task Force was extrapolating outcomes from simulation studies to actual patient outcomes. During the evidence evaluation, if the PICO (*P*opulation, *I*ntervention, *C*omparator, *O*utcome) question outcomes were limited to training outcomes such as improved performance on a manikin or simulator, studies were classified to a level of evidence (LOE) according to study design (e.g., a randomised controlled trial [RCT] on a manikin would be LOE 1). Manikin or simulator studies were labeled as LOE 5 irrespective of the study design if the PICO question also included patient outcomes.

The following is a summary of key 2010 recommendations or changes related to EIT:

- Efforts to implement new resuscitation guidelines are likely to be more successful if a carefully planned, multifaceted implementation strategy is used. Education, while essential, is *only one* element of a comprehensive implementation strategy.
- All courses should be evaluated to ensure that they reliably achieve the program objectives. Training should aim to ensure that learners acquire and retain the skills and knowledge that will enable them to act correctly in actual cardiac arrests.
- Life support knowledge and skills, both basic and advanced, can deteriorate in as little as 3–6 months. Frequent assessments and, when needed, refresher training are recommended to maintain knowledge and skills.
- Short video/computer self-instruction courses with minimal or no instructor coaching, combined with hands-on practice (practice-while-you-watch), can be considered as an effective alternative to instructor-led basic life support (cardiopulmonary resuscitation [CPR] and automated external defibrillator [AED]) courses.
- Laypeople and healthcare providers (HCPs) should be trained to start CPR with chest compressions for adult victims of cardiac arrest. If they are trained to do so, they should perform ventilations. Performing chest compressions alone is reasonable for trained individuals if they are incapable of delivering airway and breathing maneuvers to cardiac arrest victims.
- AED use should not be restricted to trained personnel. Allowing use of AEDs by individuals without prior formal training can be beneficial and may be lifesaving. Since even brief training improves performance (e.g., speed of use, correct pad placement), it is recommended that training in the use of AEDs be provided.
- CPR prompt or feedback devices improve CPR skill acquisition and retention and may be considered during CPR training for laypeople and healthcare professionals. These devices may be considered for clinical use as part of an overall strategy to improve the quality of CPR.
- It is reasonable to wear personal protective equipment (PPE) (e.g., gloves) when performing CPR. CPR should not be delayed or withheld if PPE is not available unless there is a clear risk to the rescuer.
- Manual chest compressions should not continue during the delivery of a shock because safety has not been established.

☆ *Note from the writing group*: Throughout this article, the reader will notice combinations of superscripted letters and numbers (e.g., "Precourse Preparation[EIT-018A"]). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.

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Several important knowledge gaps were identified during the evidence review process:

- The optimal duration and type of initial training to acquire resuscitation knowledge and skills.
- The optimal frequency and type of refresher training required to maintain resuscitation knowledge and skills.
- The optimal use of assessment as a tool to promote the learning of resuscitation knowledge and skills.
- The impact of experience in actual resuscitation attempts on skill decay and the need for refresher training.
- The impact of specific training interventions on patient outcomes.
- A standardised nomenclature and definitions for different types of simulation training and terms such as 'high fidelity simulation,' 'feedback,' 'briefing' and 'debriefing.'
- The most effective and efficient methods of disseminating information about new resuscitation interventions or guidelines to reduce time to implementation.
- For cardiac resuscitation centres (facilities providing a comprehensive package of post resuscitation care), the optimal emergency medical services (EMS) system characteristics, safe patient transport interval (time taken to travel from scene to hospital), optimal mode of transport (e.g., ground ambulance, helicopter), and role of secondary transport (transfer from receiving hospital to a resuscitation centre).

The EIT Task Force organised its work into five major sections:

- Education—including who should be trained and how to prepare for training, the use of specific instructional strategies and techniques, retraining intervals, retention of knowledge and skills, and assessment methods.
- Risks and effects on the rescuer of CPR training and actual CPR performance.
- Rescuer willingness to respond.
- Implementation and teams—including a framework for implementation efforts as well as individual and team factors associated with success.
- Ethics and outcomes.

Education

Effective and efficient resuscitation education is one of the essential elements in the translation of guidelines into clinical practice. Educational interventions need to be population specific (e.g., lay rescuers, HCPs) and evaluated to ensure that they achieve the desired educational outcomes—not just at the end of the course but also during actual resuscitation events. Retention of knowledge and skills should be confirmed through assessment and not be assumed to persist for pre-established time intervals.

Populations

Who should be trained and how should they prepare for training?

Focused training[EIT-012A,EIT-012B]

For lay providers requiring basic life support training, does focusing training on high-risk populations, compared with no such targeting improve outcomes (e.g., bystander CPR, survival)?

Consensus on science

In three studies (LOE 1[2]; LOE 2[3,4]), people reported that they would be more willing to perform bystander CPR on family members than on nonrelatives.

One LOE 2 study[5] of people who called 911 found that unless family members had received CPR training, they were less likely to perform CPR than unrelated bystanders. Computer modeling (LOE 5)[6] suggested that very large numbers of older adults would need to be trained to achieve a sufficient increase in private residence bystander CPR rates to improve survival. Twelve studies (LOE 1[2,7–11]; LOE 2[3,12]; LOE 4[13,14]; LOE 5[15,16]) reported that training of patients and family members in CPR provided psychological benefit. Two LOE 1 studies[7,17] reported that negative psychological effects on patients can be avoided by providing social support.

Treatment recommendation

There is insufficient evidence to support or refute the use of training interventions that focus on high-risk populations. Training with social support reduces family member and patient anxiety, improves emotional adjustment, and increases feelings of empowerment.

Precourse preparation[EIT-018A]

For advanced life support providers undergoing advanced life support courses, does the inclusion of specific precourse preparation (e.g., e-learning and pretesting), as opposed to no such preparation, improve outcomes (e.g., same skill assessment but with less face-to-face [instructor] hands-on training)?

Consensus on science

Eight studies (LOE 1[18]; LOE 4[19]; LOE 5[20–25]) reported that a diverse range of precourse preparatory actions (e.g., computer-assisted learning, pretests, video-based learning, textbook reading) improved learner outcomes in advanced life support courses.

One large LOE 1 RCT[26] of use of a commercially available e-learning simulation program before attending an advanced life support course, compared with standard preparation with a course manual, did not improve either cognitive or psychomotor skill performance during cardiac arrest simulation testing.

Eighteen studies (LOE 2[27]; LOE 4[19,28]; LOE 5[20,25,29–41]) showed that alternative course delivery formats such as electronically delivered (CD or Internet-based) courses produced as good or better learner outcomes compared with traditional courses, and also reduced instructor-to-learner face-to-face time.

Treatment recommendation

Precourse preparation including, but not limited to, use of computer-assisted learning tutorials, written self-instruction materials, video-based learning, textbook reading, and pretests are recommended as part of advanced life support courses. Any method of precourse preparation that is aimed at improving knowledge and skills or reducing instructor-to-learner face-to-face time should be formally assessed to ensure equivalent or improved learning outcomes compared with standard instructor-led courses.

Instructional methods

There are multiple methods to deliver course content. This section examines specific instructional methods and strategies that may have an impact on course outcomes.

Alternative instructor methods[EIT-002A,EIT-002B]

For lay rescuers and HCPs, does the use of specific instructional methods (video/computer self-instruction), compared with traditional instructor-led courses, improve skill acquisition and retention?

Consensus on science

Twelve studies (LOE 1[42–47]; LOE 2 or 3[48–53]) demonstrated that basic life support skills can be acquired and retained at least as well and, in some cases, better using video-based self-instruction (practice-while-you-watch) compared with traditional instructor-led courses. Video-based self-instruction lasted from 8 to 34 min, whereas instructor-led courses were usually 4–6 h in duration. One LOE 1 study[54] demonstrated that prior viewing of a video on infant CPR before an instructor-led course improved skill acquisition.

When compared with traditional instructor-led CPR courses, various self-instructional and shortened programs have been demonstrated to be efficient (from the perspective of time) and effective (from the perspective of skill acquisition) in teaching CPR skills to various populations.

Treatment recommendation

Short video/computer self-instruction (with minimal or no instructor coaching) that includes synchronous hands-on practice (practice-while-you-watch) in basic life support can be considered as an effective alternative to instructor-led courses.

AED training interventions[EIT-013A,EIT-013B]

For basic life support providers (lay or HCP) requiring AED training, are there any specific training interventions, compared with traditional lecture/practice sessions, that increase outcomes (e.g., skill acquisition and retention, actual AED use)?

Consensus on science

One LOE 2 study[55] demonstrated that training delivered by laypeople is as effective as training by HCPs. One LOE 1 study[56] reported that instruction by nurses, as compared with physicians, resulted in better skill acquisition. Four studies (LOE 2[46,51,57]; LOE 4[58]) reported that the use of computer-based AED training improved skill acquisition and retention, particularly when combined with manikin practice. One LOE 1 study[47] supported the use of video-self instruction when compared with instructor-led training. Three LOE 1 studies[59–61] showed that the use of video self-instruction was less effective for some elements when compared with instructor-led training. One LOE 1 study[62] supported the use of a training poster and manikin for learning AED skills. Three studies (LOE 2[46,63]; LOE 4[64]) reported that laypeople and HCPs could use an AED without training. Three LOE 2 studies[65–67] reported that untrained individuals could deliver a shock with an AED. However, even minimal training (15-min lecture, 1-h lecture with manikin practice, or reading instructions) improved performance (e.g., time to shock delivery, correct pad placement, safety).

Treatment recommendation

AED use should not be restricted to trained personnel. Allowing use of AEDs by individuals without prior formal training can be beneficial and may be lifesaving. Since even brief training improves performance (e.g., speed of use, correct pad placement), it is recommended that training in the use of AEDs be provided. Laypeople can be used as AED instructors. Short video/computer self-instruction (with minimal or no instructor coaching) that includes synchronous hands-on practice in AED use (practice-while-you-watch) may be considered as an effective alternative to instructor-led AED courses.

Advanced life support leadership/team training[EIT-017A]

For advanced life support providers undergoing advanced life support courses, does the inclusion of specific leadership/team training, as opposed to no such specific training, improve outcomes (e.g., performance during cardiac arrest)?

Consensus on science

Four studies (LOE 1[68,69]; LOE 2[70,71]) of advanced life support in simulated in-hospital cardiac arrest and seven LOE 5 studies[72–78] of actual and simulated arrest demonstrated improved resuscitation team performance when specific team and/or leadership training was added to advanced life support courses.

Treatment recommendation

Specific teamwork training, including leadership skills, should be included in advanced life support courses.

Teaching chest compressions to achieve recoil[EIT-032]

Is there a method for teaching chest compressions, compared with current teaching, to achieve full chest recoil (complete release) after each compression?

Consensus on science

One LOE 5 clinical case series[79] documented a 46% incidence of incomplete chest recoil by professional rescuers using the 2005-recommended CPR technique. One LOE 4 study[80] electronically recorded chest recoil during in-hospital paediatric cardiac arrests, and found that leaning on the chest (>2.5 kg; an adult feedback threshold) occurred in 50% of chest compressions/decompressions using the recommended hand position, and that incomplete recoil was reduced with real-time automated feedback. Another LOE 4 in-hospital paediatric study[81] demonstrated a 23.4% incidence of incomplete recoil. One LOE 5 study[82] has shown that without specific training in complete chest recoil technique, 22% of trained rescuers leaned on the chest when performing CPR. Two LOE 5 studies[79,83] demonstrated that incomplete chest recoil was significantly reduced with three techniques (i.e., 'two-finger fulcrum,' 'five-finger fulcrum,' and 'hands-off') of lifting the heel of the hand slightly but completely off the chest during CPR in a manikin model. However, duty cycle and compression depth were reduced when professional and lay rescuers applied these techniques.

Treatment recommendation

There is insufficient evidence to recommend teaching any specific technique to optimise complete chest recoil during actual CPR.

Use of CPR prompt/feedback devices[EIT-005A,EIT-005B]

For lay rescuers and HCPs performing CPR, does the use of CPR prompt/feedback devices, compared with no device, improve acquisition, retention, and actual performance of CPR skills?

Consensus on science

Most devices considered in this review combine prompting (a signal to perform an action, e.g., metronome for compression rate) with feedback (after-event information about the effect of an action, e.g., visual display of compression depth). The effects have been considered together in this question and devices are referred to as prompt/feedback devices.

Seven LOE 5 manikin studies[84–90] demonstrated that CPR prompt/feedback devices either in addition to or in place of instructor-led training improved basic CPR skill acquisition (tested without use of the device). Another LOE 5 manikin study[85] showed that automated feedback might be less effective than instructor feedback for more complex skills (e.g., bag-mask ventilation).

Two LOE 5 manikin studies[84,87] showed improved skill retention when a CPR prompt/feedback device was used during initial training. An additional LOE 5 manikin study[89] showed that unsupervised refresher training with a CPR prompt/feedback device, compared with no refresher training, also improved skill retention. The LOE 5 follow-up arm of the manikin study of bag-mask

ventilation/CPR[85] continued to show poorer ventilation skills in the voice-activated manikin-feedback arm compared with the instructor-feedback arm.

Evidence from 21 manikin studies (LOE 5)[84,86,89–107] consistently demonstrated that CPR prompt/feedback devices used during CPR improved the quality of CPR performance on manikins. Three additional manikin studies (LOE 5) examined the utility of video/animations on mobile-phone devices: two studies showed improved checklist scores and quality of CPR[92,95] and faster initiation of CPR,[92] while the third study showed that participants using multimedia phone CPR instruction took longer to complete tasks than dispatcher-assisted CPR.[103] Two manikin studies (LOE 5)[108,109] that used two-way video communication to enable the dispatcher to review and comment on CPR in real time produced equivocal findings.

There are no studies demonstrating improved patient outcomes with CPR prompt/feedback devices. One study each in children (LOE 2)[110] and adults (LOE 2)[111] showed that metronomes improved chest compression rate and increased end-tidal carbon dioxide (thought to correlate with improved cardiac output and blood flow to the lungs). Five studies evaluating the introduction of CPR prompt/feedback devices in clinical practice (pre/post comparisons) found improved CPR performance (LOE 3)[80,112–115].

There may be some limitations to the use of CPR prompt/feedback devices. Two LOE 5 manikin studies[116,117] reported that chest-compression devices may overestimate compression depth if CPR is being performed on a compressible surface such as a mattress on a bed. One LOE 5 study[100] reported harm to a single participant when a hand got stuck in moving parts of the CPR feedback device. Another LOE 5 manikin study[118] demonstrated that additional mechanical work from the CPR provider was required to compress the spring in one of the pressure-sensing feedback devices. One case report (LOE 5)[119] documented soft tissue injury to a patient's chest when an accelerometer device was used for prolonged CPR.

Treatment recommendation

CPR prompt/feedback devices may be considered during CPR training for laypeople and HCPs. CPR prompt/feedback devices may be considered for clinical use as part of an overall strategy to improve the quality of CPR. Instructors and rescuers should be made aware that a compressible support surface (e.g., mattress) may cause a feedback device to overestimate depth of compression.

Training interventions[EIT-009A]

For adult and paediatric advanced life support providers, are there any specific training interventions (e.g., duration of session, interactive computer programs, e-learning, video self-instruction) compared with traditional lecture/practice sessions that increase outcomes (e.g., skill acquisition and retention)?

Consensus on science

There is limited evidence about interventions that enhance learning and retention from advanced life support courses. One LOE 3 study[120] suggested that the *2005 Guidelines* have helped to improve "no-flow" fraction (i.e., percent of total resuscitation time that compressions are not performed) but not other elements of quality of CPR performance. One LOE 1 study[121] demonstrated that clinical training before an advanced life support (ALS) course might improve long-term retention of ALS knowledge and skills. One LOE 5 advanced trauma life support (ATLS) study[122] suggested that postcourse experience might play a role in knowledge and skill retention. In one LOE 3 study[123] unscheduled mock-codes improved mock-code performance in hospital personnel. One LOE 2 study[124] found no difference in knowledge retention when live actors were used in ALS course training compared with manikins.

Treatment recommendation

There is insufficient evidence to recommend any specific training intervention, compared with traditional lecture/practice sessions, to improve learning, retention, and use of advanced life support skills.

Realistic training techniques[EIT-019A,EIT-019B]

For participants undergoing basic or advanced life support courses, does the inclusion of more realistic techniques (e.g., high-fidelity manikins, in situ training), as opposed to standard training (e.g., low-fidelity manikins, education centre), improve outcomes (e.g., skill performance on manikins, skill performance in an actual arrest, willingness to perform)?

Consensus on science

Studies report conflicting data on the effect of increasing realism (e.g., use of actual resuscitation settings, high-fidelity manikins) on learning, and few data on patient outcomes. Two studies (LOE 1[125]; LOE 2[126]) supported an improvement in performance of skills in actual arrest, but were underpowered to identify improved survival rate. One small LOE 1 study[127] showed no overall effect on performance, although the simulation-trained group demonstrated superior teamwork skills. Thirteen studies (LOE 1[125,128–132]; LOE 2[133–135]; LOE 3[136,137]; LOE 4[138,139]) reported an improvement in skills assessed using a manikin. Seven LOE 1 studies[140–146] reported no effect on skills assessed using a manikin. Eleven LOE 1 studies tested the effect of simulation fidelity on the participants' knowledge using multiple-choice questions; nine of these studies found no effect[124,127,128,130,140,141,143,144,147] and two of the 11 studies demonstrated an improvement in participant knowledge with the more realistic techniques.[148,149]

Two studies (LOE 3[136]; LOE 4[138]) that focused on resuscitation in trauma reported improved skill performance (on a manikin) with higher-fidelity simulation. One LOE 1 study[140] found no difference in skill performance or knowledge in advanced trauma life support (ATLS) with the use of high-fidelity simulation. One LOE 1 study[148] reported a significant increase in knowledge when using manikins or live patient models for trauma teaching compared with no manikins or no live models. In this study there was no difference in knowledge acquisition between using manikins or live patient models, although learners preferred using the manikins.

Four studies (LOE 1[128,140,141]; LOE 2[148]) reported that higher fidelity simulation was associated with improved learner satisfaction rate compared with a traditional curriculum. One LOE 1 study[144] questioned the cost-effectiveness of higher fidelity approaches compared with standard manikins.

Three studies (LOE 1[125]; LOE 2[134]; LOE 3[137]) reported that requiring learners to perform all of the steps of psychomotor skills in simulation as they would in an actual clinical situation could reveal inadequacies in training.

Treatment recommendation

There is insufficient evidence to support or refute the use of more realistic techniques (e.g., high-fidelity manikins, in situ training) to improve outcomes (e.g., skill performance on manikins, skill performance in actual arrest, willingness to perform) compared with standard training (e.g., low-fidelity manikins, education centre) in basic and advanced life support courses.

Course format and duration

Resuscitation training courses vary widely in their duration and how different elements of the curriculum are taught. This section examines the effect of course format and duration on learning outcomes.

Course duration[EIT-029A,EIT-029B]

For basic life support providers (lay or HCP), does a longer duration instructor-led course, compared with a shorter course, improve skill acquisition and retention?

Consensus on science

A single, randomised manikin LOE 1 study[150] demonstrated that a 7-h basic life support (with AED) instructor-led course resulted in better initial skill acquisition than a 4-h instructor-led course; and a 4-h instructor-led course resulted in better skill acquisition than a 2-h course. Retesting at 6 months after a 2-h course resulted in skill retention at 12 months that was equivalent to a 7-h course with no intermediate testing. This study[150] along with two LOE 2 manikin studies[151,152] demonstrated that for periods between 4 and 12 months, skill retention is higher for longer courses, but deterioration is at similar rates. The differences in learning outcomes for courses of different durations may not be significant, particularly if assessment and refresher training are undertaken.

Treatment recommendation

It is reasonable to consider shortening the duration of traditional instructor-led basic life support courses. Brief reassessment (e.g., at 6 months) should be considered to improve skills and retention. The optimal duration of an instructor-led basic life support course has not been determined. New course formats should be assessed to ensure that they achieve their objectives.

Nontraditional scheduling formats[EIT-020]

For participants undergoing advanced life support courses, does the use of nontraditional scheduling formats such as random scheduling (introducing station cases in a random manner) or modular courses, as opposed to traditional scheduling, improve outcomes (e.g., skills performance)?

Consensus on science

There are no published studies addressing the impact of different ALS course scheduling formats, compared with the traditional 2-day course format, that demonstrated improved learning outcomes (knowledge and skill acquisition and/or retention).

Treatment recommendation

There is insufficient evidence to support or refute the use of alternative advanced life support course scheduling formats compared with the traditional 2-day course format.

Retraining intervals

It is recognised that knowledge and skill retention declines within weeks after initial resuscitation training. Refresher training is invariably required to maintain knowledge and skills; however, the optimal frequency for refresher training is unclear. This section examines the evidence addressing the optimal frequency for refresher training to maintain adequate knowledge and skills.

Specific intervals for basic life support[EIT-010]

For basic life support providers (lay and HCP), are there any specific intervals for update/retraining, compared with standard practice (i.e., 12 or 24 monthly), that increase outcomes (e.g., skill acquisition and retention)?

Consensus on science

Six studies (LOE 1[44,87]; LOE 2[150]; LOE 4[47,153,154]) using different training approaches demonstrated that CPR skills (e.g., alerting EMS, chest compressions, ventilations) decay rapidly (within 3–6 months) after initial training. Two studies (LOE 1[155]; LOE 4[156]) reported skill decay within 7–12 months after initial training. Four studies (LOE 2[150]; LOE 4[157–159]) demonstrated that CPR performance was retained or improved with reevaluation, refresher, and/or retraining after as little as three months. Three LOE 2 studies[66,150,160] demonstrated that AED skills are retained longer than CPR skills. One LOE 2 study[160] reported higher levels of retention from a program that achieved initial training to a high (mastery) level. However, deterioration of CPR skills was still reported at three months.

Treatment recommendation

For basic life support providers (lay and HCP), skills assessment and, if required, a skills refresher should be undertaken more often than the current commonly recommended training interval of 12–24 months.

Specific intervals for advanced life support[EIT-011A,EIT-011B]

For adult and paediatric advanced life support providers, do any specific intervals for update/retraining, compared with standard practice (i.e., 12 or 24 months), increase outcomes (e.g., skill acquisition and retention)?

Consensus on science

One LOE 1 trial[161] and one LOE 3 study[162] suggested that refresher training may enhance resuscitation knowledge retention but did not maintain motor skills. Two RCTs (LOE 1)[163,164] showed no benefit of refresher training.

Nine studies (LOE 3[165]; LOE 4[153,166–172]) reported decreased resuscitation knowledge and/or skills performance when tested 3–6 months after initial training. Two LOE 4 studies[173,174] reported decreased performance when tested 7–12 months following training. One LOE 4 study[175] reported decay of practical skill performance when participants were tested 18 months after training.

Treatment recommendation

For advanced life support providers there should be more frequent assessment of skill performance and/or refresher training than is currently recommended in established advanced life support programs. There is insufficient evidence to recommend an optimal interval and form of assessment and/or refresher training.

Assessment

Written examination[EIT-004]

For students of adult and paediatric advanced level courses, does success in the written examination, compared with lack of success, predict success in completing the practical skills testing associated with the course or in cardiac arrest management performance in actual or simulated cardiac arrest events?

Consensus on science

Four observational studies (LOE P4)[176–179] did not support the ability of a written test to predict clinical skill performance in an advanced life support course. Twelve LOE P5 studies[180–191] supported using written tests as a predictor of nonresuscitation clinical skills, with variable levels of correlation ranging from 0.19 to 0.65. Three LOE P5 studies[192–194] were either neutral or did not support the ability of a written test to predict clinical skill performance.

Treatment recommendation

A written test in an advanced life support course should not be used as a substitute for demonstration of clinical skill performance.

Testing versus continuous assessment[EIT-021A]

For participants undergoing basic or advanced life support courses, does end-of-course testing, as opposed to continuous assessment and feedback, improve outcomes (e.g., improve learning/performance)?

Assessment versus no assessment[EIT-030A]

For lay and HCP, does the use of assessment, as opposed to no such assessment, improve CPR knowledge, skills, and learning/retention?

Consensus on science

No studies have compared outcomes of continuous versus end-of-course assessments for resuscitation training.

One LOE 1 manikin study[195] showed that including assessment during advanced life support training, compared with a control group without assessment, moderately improved performance at the 2-week postcourse scenario assessment. In another LOE 1 study[195] performance assessment after 6 months in the "testing" group compared with the control group failed to show a statistically significant improvement.

Treatment recommendation

Summative assessment at the end of advanced life support training should be considered as a strategy to improve learning outcomes. There is insufficient evidence to recommend an optimal method of assessment during life support training.

Education knowledge gaps

- Effect of targeting training to family and friends of those at "high risk" of cardiac arrest.
- Potential for tailoring preparation and training to individual learning styles.
- Optimal assessment tools and strategy to promote learning resuscitation skills.
- Optimal format and duration of self-instruction.
- Impact of resuscitation training on performance in actual cardiac arrest.
- Motivating bystanders to use AEDs.
- Optimal training (alternative, minimal, no training, standardised instructor-led training) for use of AEDs in actual events.
- Governmental, social, and political measures needed to improve public participation in life support programs.
- Optimal ways to teach and assess leadership and team skills.
- Specific techniques to optimise complete chest recoil during CPR without impacting depth, rate, and duty cycle of compression, including the use of prompt and feedback devices to acheive this.
- Optimal method for implementing feedback devices into practice.
- Specific advantages of prompt devices versus feedback devices and feedback timing (real time or immediately post-event).
- Optimal method for learning and retention of knowledge/skills.
- Standardization in simulation nomenclature and research methods.
- Influence of equipment or manikin fidelity, environmental fidelity, and psychological fidelity on learning outcomes.
- Optimal length of an instructor-led course.
- Comparison of different course formats (e.g., a 2-day course versus four half-day modules).
- Effect of ongoing clinical experience on retention of skills and need for assessment and/or refresher training.
- Optimal interval and form for assessment.
- Optimal format for refresher training when the need is identified.
- Effect of type of measurement/assessment.
- Effect of complexity on retention.
- Optimal intervals and strategies for refresher courses for various populations.
- Levels of knowledge/skill deterioration tolerable (clinically significant) before a refresher course is needed.
- Correlation between rescuer knowledge/skill competencies and patient survival.
- Modalities to increase knowledge/skill retention (clinical exposure, simulation, video learning).
- Economy and logistics of shorter intervals for update/retraining.
- Optimal form and timing of assessment to optimise learning, retention, and application of resuscitation skills.

Risks and effects on the rescuer of CPR training and actual CPR performance

The safety of rescuers is essential during training and actual CPR performance.

CPR and AED training and experience[EIT-014A,BLS-002A]

For providers (lay or HCP), does undertaking training/performing actual CPR or use of a defibrillator (manual or AED), compared with no such training/performance, increase harm to the rescuer?

Compression-only CPR[BLS-005A,BLS-005B]

For rescuers performing CPR on adults or children, does compression-only CPR, compared with traditional CPR, result in an increase in adverse outcomes (e.g., fatigue)?

Use of barrier devices[BLS-002A]

For rescuers performing CPR on adults or children (out-of-hospital and in-hospital), does the use of a barrier device, as opposed to no such use, improve outcomes (e.g., lower infection risk)?

Physical effects

Consensus on science

CPR is very rarely associated with adverse events to the rescuer during training or actual performance. An observational study (LOE 4)[196] reported one muscle strain during a large public access defibrillation trial.[197] One prospective observational study (LOE 4)[198] described five musculoskeletal injuries (four back-related) associated with performing chest compressions in 1265 medical emergency team (MET) call participants. Two retrospective

surveys of nurses and ambulance officers (LOE 4)[199,200] reported a high incidence of back symptoms attributed to performing CPR.

Three small simulation studies (LOE 4)[201–203] using a greater number of ventilations per minute than those provided with the currently recommended compression-ventilation ratio (30:2) described hyperventilation-related symptoms during rescue breathing. Five single or small case series (LOE 5)[204–208] described isolated adverse events from training or performing actual CPR (myocardial infarction, pneumothorax, chest pain, shortness of breath, nerve injury, allergy, vertigo). In one case report (LOE 5)[209] a rescuer suffered a puncture wound to her left hand from a victim's sternotomy wires when performing chest compressions.

One simulation study (LOE 5)[210] of six physicians (aged 25–40 years) and another study (LOE 5)[211] of 10 healthy medical students showed that performing chest compressions increased rescuer oxygen consumption. The authors considered that this increase in oxygen consumption was sufficient to cause myocardial ischaemia in individuals with coronary heart disease. A small randomised trial of cardiac rehabilitation patients (LOE 5)[9], however, reported no adverse physical events during CPR training.

Treatment recommendation

CPR training and actual performance is safe in most circumstances. Learners and rescuers should consider personal and environmental risks before starting CPR. Individuals undertaking CPR training should be advised of the nature and extent of the physical activity required during the training program. Learners who develop significant symptoms (e.g., chest pain, severe shortness of breath) during CPR should be advised to stop. Rescuers who develop significant symptoms during actual CPR should consider stopping CPR.

Rescuer fatigue

A single LOE 4 in-hospital patient study[212] of 3 min of continuous chest compressions with real-time feedback to the rescuer showed that the mean depth of compression deteriorated between 90 and 180 s, but compression rate was maintained. Three LOE 5 studies showed that some rescuers were unable to complete 5 min (laypeople),[213] 5–6 min (lay females),[214] or 18 min (HCPs)[215] of continuous chest compressions because of physical exhaustion. Two manikin studies (LOE 5)[215,216] demonstrated that performing chest compressions increases heart rate and oxygen consumption in HCPs. Two randomised manikin studies (LOE 5)[213,214] demonstrated that >5 to 10 min of continuous chest compressions by laypeople resulted in significantly less compression depth compared with standard 30:2 CPR, and no difference in compression rate. In one LOE 5 manikin study[217] experienced paramedics demonstrated no decline in chest compression quality below guideline recommendations during 10 min of BLS with any of three different compression–ventilation ratios (15:2, 30:2, and 50:2).

Four manikin studies (LOE 5)[218–221] showed a time-related deterioration in chest compression quality (mainly depth) during continuous compressions by HCPs. A single manikin study (LOE 5)[222] demonstrated that medical students performed better-quality chest compressions during the first 2 min of continuous chest compressions compared with 15:2 CPR, although there was deterioration in quality after 2 min. An LOE 5 manikin study[223] of HCPs showed that the number of effective compressions (depth >38 mm) was the same if the rescuer changed every minute or every 2 min during 8 min of continuous chest compressions. Fatigue was reported more frequently after a 2-min period of compressions.

Treatment recommendation

When performing chest compressions, if feasible, it is reasonable to consider changing rescuers after about 2 min to prevent rescuer fatigue (demonstrated by deterioration in chest compression quality—in particular, depth of compressions). The change of rescuers performing chest compressions should be done with minimum interruption to the compressions.

Risks during defibrillation attempts

Consensus on science

Harm to the rescuer or a bystander is extremely rare during defibrillation attempts. A large randomised trial of public access defibrillation (LOE 1)[197] and four prospective studies of first-responder AED use (LOE 4[224–226]; LOE 5[227]) demonstrated that AEDs can be used safely by laypeople and first responders. One LOE 4 manikin study[228] observed that laypeople using an AED touched the manikin during shock delivery in one third of defibrillation attempts.

An observational study (LOE 4)[229] of 43 patients undergoing cardioversion measured only a small current leakage through "mock rescuers" wearing polyethylene gloves and simulating chest compression during shock delivery. One LOE 5 systematic review[230] identified eight articles that reported a total of 29 adverse events associated with defibrillation. Only one case (LOE 5)[231] has been published since 1997. A 150-J biphasic shock was delivered during chest compressions. The rescuer doing chest compressions felt the electric discharge and did not suffer any harm. Seven cases were due to accidental or intentional defibrillator misuse (LOE 5)[232–236], one was due to device malfunction (LOE 5)[237], and four occurred during training/maintenance procedures (LOE 5)[237,238]. A case series (LOE 5)[237] identified 14 adverse events during actual resuscitation; all caused only minor harm.

The risks to individuals in contact with a patient during implanted cardioverter defibrillator (ICD) discharge are difficult to quantify. Four single case reports (LOE 5)[239–242] described shocks to the rescuer from discharging ICDs. ICD discharge was associated with a significant jolt to rescuers and in one case resulted in a peripheral nerve injury.[242]

Three animal studies suggested that the use of defibrillators in wet environments is safe (LOE 5)[243–245].

There are no reports of harm to rescuers from attempting defibrillation in wet environments.

Treatment recommendation

The risks associated with defibrillation are less than previously thought. There is insufficient evidence to recommend that continuing manual chest compressions during shock delivery for defibrillation is safe. It is reasonable for rescuers to wear gloves when performing CPR and attempting defibrillation (manual and/or AED) but resuscitation should not be delayed/withheld if gloves are not available.

There is insufficient evidence to make a recommendation about the safety of contacting a patient during ICD discharge. There is insufficient evidence to make a recommendation about the best method of avoiding shocks to the rescuer from an ICD discharge during CPR.

Although there are no reports of harm to rescuers, there is insufficient evidence to make a recommendation about the safety of defibrillation in wet environments.

Psychological effects

Consensus on science

One large prospective trial of PAD (LOE 4)[196] reported a few adverse psychological effects requiring intervention that were associated with CPR or AED use. One prospective analysis of stress reactions associated with a trial of public access defibrillation (LOE 4)[246] reported low levels of stress in those responding to an emergency in this setting. One prospective observational study of 1265 MET calls (LOE 4)[198] described "psychological injury" related to CPR performance in one rescuer. Two large retrospective questionnaire-based reports relating to performance of CPR by a bystander (LOE 4)[247,248] reported that nearly all respondents regarded their intervention as a positive experience. Two small retrospective studies of nurses involved in delivery of CPR (LOE 4[249]; LOE 5[250]) noted the stress involved and the importance of recognition and management of this stress.

Treatment recommendation

There are few reports of psychological harm to rescuers after involvement in a resuscitative attempt. There is insufficient evidence to support or refute any recommendation about minimizing the incidence of psychological harm to rescuers.

Disease transmission

Consensus on science

There are only a very few cases reported (LOE 5) where performing CPR has been implicated in disease transmission. Salmonella infantis,[251] panton-valentine leucocidin staphylococcus aureus,[252] severe acute respiratory syndrome (SARS),[253] meningococcal meningitis,[254] helicobacter pylori,[255] herpes simplex virus,[256,257] cutaneous tuberculosis,[258] stomatitis,[259] tracheitis,[260] shigella,[261] and streptococcus pyogenes[262] have been implicated. One report described herpes simplex virus infection as a result of training in CPR (LOE 5)[263]. One systematic review found that in the absence of high-risk activities, such as intravenous cannulation, there were no reports of transmission of hepatitis B, hepatitis C, human immunodeficiency virus (HIV), or cytomegalovirus during either training or actual CPR (LOE 5)[264].

Treatment recommendation

The risk of disease transmission during training and actual CPR performance is very low. Rescuers should take appropriate safety precautions, especially if a victim is known to have a serious infection (e.g., HIV, tuberculosis, hepatitis B virus, or SARS).

Barrier devices

Consensus on science

No human studies have addressed the safety, effectiveness, or feasibility of using barrier devices to prevent patient contact during rescue breathing. Nine clinical reports (LOE 5)[257,258,264–268] proposed or advocated the use of barrier devices to protect the rescuer from transmitted disease. Three LOE 5 studies[269–271] showed that barrier devices can decrease transmission of bacteria in controlled laboratory settings.

Treatment recommendation

The risk of disease transmission is very low and initiating rescue breathing without a barrier device is reasonable. If available, rescuers may consider using a barrier device. Safety precautions should be taken if the victim is known to have a serious infection (e.g., HIV, tuberculosis, hepatitis B virus, or SARS).

Knowledge gaps

- Actual incidence of disease transmission and other harm during CPR
- Safety of hands-on defibrillation
- Safest type of glove
- CPR in patients with ICDs
- Role of barrier devices

Rescuer willingness to respond

Increasing the willingness of individuals to respond to a cardiac arrest with early recognition, calling for help, and initiation of CPR is essential to improve survival rates.

Factors that increase outcomes[EIT-008A,EIT-008B]

Among bystanders (lay or HCP), are there any specific factors, compared with standard interventions, that increase outcomes (e.g., willingness to provide CPR or the actual performance of CPR [standard or chest compression only]) in adults or children with cardiac arrest (prehospital)?

Consensus on science

Sixteen LOE 4 studies[5,246,272–285] have suggested that many factors decrease the willingness of bystanders to start CPR, including bystander characteristics (panic, fear of disease or harming the victim or performing CPR incorrectly) and victim characteristics (stranger, being unkempt, evidence of drug use, blood, or vomit).

Two studies (LOE 1[131]; LOE 4[286]) have suggested that training rescuers to recognise gasping as a sign of cardiac arrest improves identification of cardiac arrest victims. Ten studies (LOE 2[10]; LOE 4[5,272,274,280–282,287–289]) showed increased bystander CPR rate in those trained in CPR, especially if training had occurred within five years. Three LOE 5 studies[272,275,290] showed that willingness to perform CPR was increased when emergency dispatchers provided telephone CPR instructions. Eight LOE 4 studies[273,277,280,284,285,287,291,292] provided evidence that potential rescuers would be more likely to start CPR if they had the option to use compression-only CPR.

Treatment recommendation

To increase willingness to perform CPR

- Laypeople should receive training in CPR. This training should include the recognition of gasping or abnormal breathing as a sign of cardiac arrest when other signs of life are absent.
- Laypeople should be trained to start resuscitation with chest compressions in adult and paediatric victims.
- If unwilling or unable to perform ventilations, rescuers should be instructed to continue compression-only CPR.
- EMS dispatchers should provide CPR instructions to callers who report cardiac arrest.
- When providing CPR instructions, EMS dispatchers should include recognition of gasping and abnormal breathing.

Knowledge gaps

- Optimal method for teaching recognition of cardiac arrest including gasping, agonal, and abnormal breathing.
- Optimal method for laypeople to recognise return of spontaneous circulation (ROSC).
- Optimal methods for mass education of laypeople.

Implementation and teams

The best scientific evidence for resuscitation will have little impact on patient outcomes if it is not effectively translated into clinical practice. Successful implementation is dependent on effective educational strategies to ensure that resuscitation providers have the necessary knowledge and skills in combination with the provision of necessary infrastructure and resources.[293] Education itself is only one strategy for implementing changes. This section addresses the need for a framework for successful implementation of guidelines, including broad implementation strategies that include educational activities.

Implementation strategies

Little is known about what strategies work best for implementing evidence-based guidelines in communities, institutions, or units. Implementation of the 2005 resuscitation guidelines in emergency medical services agencies was reported to take a mean of 416 ± 172 days in the Resuscitation Outcomes Consortium (ROC) sites[294] and 18 months in the Netherlands.[295] Identified barriers to rapid implementation included delays in getting staff trained, equipment delays, and organisational decision making.[294,295] This section provides insight into several elements that appear to facilitate successful implementation.

Implementation factors[EIT-022,EIT-022B]

In communities where processes/guidelines are being implemented, does the use of any specific factors, compared with no such use, improve outcomes (e.g., success of implementation)?

Consensus on science

Using the implementation of therapeutic hypothermia as an example, two LOE 3[296,297] and one LOE 5[298] single-institution interventional studies supported the use of a written protocol, pathway, or standard operating procedure as part of a comprehensive approach to implementing the therapeutic hypothermia guideline. One LOE 2 survey[299] and one LOE 3 single-institution intervention[300] also supported the use of written protocols, although Hay[300] only briefly described cointerventions used.

A wide spectrum of evidence supports the use of a comprehensive, multifaceted approach to guideline implementation, including identification and use of clinical champions, a consensus-building process, multidisciplinary involvement, written protocols, detailed process descriptions, practical logistic support, multimodality/multilevel education, and rapid cycle improvement (e.g., Plan, Do, Study, Act) to respond to problems as they arise. The evidence supporting this multifaceted approach includes one LOE 3 study,[296] one LOE 5 intervention description,[298] two LOE 5 theoretical reviews,[301,302] and four LOE 5 studies extrapolated from nonhypothermia nonarrest settings (2 RCTs,[303,304] one concurrent controlled trial,[305] and one retrospective controlled trial[306]).

Treatment recommendation

Institutions or communities planning to implement complex guidelines such as therapeutic hypothermia should consider using a comprehensive, multifaceted approach including clinical champions, a consensus-building process, multidisciplinary involvement, written protocols, detailed process description, practical logistic support, multimodality/multilevel education, and rapid cycle improvement methods.

Investigators studying implementation of guidelines should consider using a framework for implementing guidelines (e.g., Brach-AHRQ, 2008)[302] and report whether results were measured or estimated, and whether they were sustained.

Knowledge gaps

- Which specific factors (such as consensus-building, logistic support, rapid cycle improvement) are most critical for successful guidelines implementation?
- Differences between in-hospital and EMS implementations.
- Effectiveness of a multilevel approach (country, community, organisation, unit, individual).
- Importance of describing all cointerventions during implementation studies.
- Repeat surveys over time with same population to assess progress in implementation and to identify success factors and barriers.

Individual and team factors

Individual and team factors impact performance during resuscitative attempts. This section describes specific factors that have an impact on performance during simulated or actual cardiac arrest.

Prehospital physicians[ALS-SC-077]

In adult cardiac arrest (prehospital), does the performance of advanced life support procedures by experienced physicians, as opposed to standard care (without physicians), improve outcomes (e.g., ROSC, survival)?

Consensus on science

In adult cardiac arrest, physician presence during resuscitation, compared with paramedics alone, has been reported to increase compliance with guidelines (LOE 2[307]; LOE 4[308]) and physicians in some systems can perform advanced resuscitation procedures more successfully (LOE 2[307,309]; LOE 4[310–312]).

When compared within individual systems, four studies suggested improved survival to hospital discharge when physicians were part of the resuscitation team (LOE 2[313,314]; LOE 3[315,316]) and 10 studies suggested no difference in survival of the event (LOE 2)[307,313] or survival to hospital discharge (LOE 2)[307,315,317–323]. One study found lower survival of the event when physicians were part of the resuscitation team (LOE 2)[323].

Studies indirectly comparing resuscitation outcomes between physician-staffed and other systems are difficult to interpret because of the heterogeneity among systems, independent of physician-staffing (LOE 5)[324]. High survival rates after cardiac arrest have been reported from systems that employ experienced physicians as part of the EMS response (LOE 3[325,326]; LOE 4[310,312,327]) and these survival rates may be higher than in systems that rely on nonphysician providers (LOE 2[328]; LOE 3[325,326,329]). Other comparisons noted no difference in survival between systems using paramedics or physicians as part of the response (LOE 3)[330,331]. Well-organised nonphysician systems with highly trained

paramedics also reported high survival rates (LOE 5)[324]. There are no RCTs to address this question.

Treatment recommendation

There is insufficient evidence to make a recommendation for or against physician versus nonphysician providers of ALS during out-of-hospital CPR.

Knowledge gaps

More data are required to determine the training required to achieve best outcomes, the level of training and experience required to maintain competence in procedural skills, and the cost-effectiveness of physicians compared with nonphysicians.

Advanced life support checklists[EIT-031A,EIT-031B]

Does the use of a checklist during adult and paediatric advanced life support as opposed to no checklist, improve outcomes (e.g., compliance with guidelines, other outcomes)?

Consensus on science

Four LOE 5 randomised trials of cognitive aids/checklists for simulated basic life support,[92,95,332,333] three LOE 5 randomised trials of cognitive aids in simulated anaesthetic emergency or advanced resuscitation,[334–336] and one LOE 5 observational study[337] showed improvement in proxy outcomes (e.g., proper dosing of medications or performance of correct CPR procedures). One randomised[338] and one nonrandomised[339] trial (LOE 5) of cognitive aids showed improved recall of factual information important for effective advanced life support. Two LOE 4 surveys[340,341] on the use of checklists in actual resuscitations reported that physicians perceived cognitive aids to be useful. One LOE 5 retrospective analysis of actual anaesthesia emergency[342] suggested that a cognitive aid algorithm might be helpful in diagnosis and management. One LOE 5, three-armed study of simulated basic life support[333] demonstrated no difference in CPR performance between the short-checklist arm and the no-checklist arm, but a positive outcome in the long-checklist arm. One LOE 5 study of neonatal resuscitation[343] did not demonstrate any benefit from using a poster prompt.

Potential harm was found in one LOE 5 randomised trial of simulated basic life support[103] in which participants with a mobile-phone cognitive aid had >1-min delay in starting CPR. An LOE 5 simulated PALS study[344] showed potential harm because a significant portion of hand-held cognitive aid users applied the wrong algorithm. The outcome of using a cognitive aid such as a checklist may be specific to the aid or the situation.

Treatment recommendation

It is reasonable to use cognitive aids (e.g., checklists) during resuscitation, provided that they do not delay the start of resuscitative efforts. Aids should be validated using simulation or patient trials, both before and after implementation, to guide rapid cycle improvement.

Knowledge gaps

- The value of cognitive aids in simulated and actual resuscitation.
- Potential for unintended consequences associated with the use of a cognitive aid (especially delay to initiation of intervention or use of incorrect algorithm).
- Utility of specific cognitive aids with specific providers or in specific situations.
- Human factors issues in solo and team resuscitation.
- Optimal model for follow-up quality assurance (assessment of efficacy and rapid cycle improvement) after introduction of a cognitive aid.
- Transferability or generalizability of cognitive aids across settings.
- Can cognitive aids such as simple checklists be used without training?

Team briefings/debriefings[EIT-001A,EIT-001B]

For resuscitation teams, do briefings/debriefings, when compared to no briefings/debriefings, improve performance or outcomes?

HCP briefings/debriefings[NRP-033A,NRP-033B]

For HCPs, do briefings (before a learning or patient-care experience) and/or debriefings (after a learning or patient care experience), when compared to no briefings or debriefings, improve the acquisition of content knowledge, technical skills and behavioral skills required for effective and safe resuscitation?

Consensus on science

The terms 'briefing,' 'debriefing,' and 'feedback' are often used interchangeably in studies and have therefore been grouped as 'briefings/debriefings' in the Consensus on Science. Debriefings tend to occur after the event. Debriefing is an integral part of the actual training intervention in many studies. This makes it difficult to measure the effect of the debriefing.

Evidence from one LOE 1 prospective RCT[345] and 16 other studies (LOE 3–4)[71,73,93,125,126,132,346–355] documented improvement with briefings/debriefings in the acquisition of the content knowledge, technical skills, and/or behavioral skills required for effective and safe resuscitation. One LOE 4 study[356] revealed no effect of briefings/debriefings on performance. No studies indicated that the use of briefings/debriefings had any negative effect.

Treatment recommendation

It is reasonable to recommend the use of briefings and debriefings during both learning and actual clinical activities.

Knowledge gaps

- Relative benefits of team versus individual briefings/debriefings.
- Differential effectiveness of video, verbal, and other measures of feedback.
- Effects of briefings/debriefings on technical versus nontechnical skills.

System factors

This section describes broader resuscitation programs and implementation strategies that have an impact at a system level.

AED program factors[EIT-015]

In AED programs, what specific factors when included (e.g., linkage to 911 registries, location of program [including home]), compared with not included predict an effective outcome for the program?

Outcomes of AED programs[BLS-004B]

In adults and children with out-of-hospital cardiac arrest (including residential settings), does implementation of a public access AED program, as opposed to traditional EMS response, improve successful outcomes (e.g., ROSC)?

Consensus on science

One RCT (LOE 1)[197], four prospective controlled cohort studies (LOE 2)[357–360], one study using historical controls (LOE 3)[361], nine observational studies (LOE 4)[226,227,362–368], and one mathematical modeling study (LOE 5)[369] showed that AED programs are safe and feasible and significantly increase survival of out-of-hospital ventricular fibrillation (VF) cardiac arrest if the emergency response plan is effectively implemented and sustained.

For EMS programs, 10 studies (LOE 1[370]; LOE 2[358]; LOE 3[224,371,372]; LOE 4[373–377]) supported AED use; 11 studies (LOE 2[378,379]; LOE 3[380–383]; LOE 4[384–388]) were neutral, and two meta-analyses[359,389] suggested benefit.

For first-responder use, two studies (LOE 2[390]; LOE 3[391]) supported use of AEDs by fire or police first responders, but six studies (LOE 1[392]; LOE 2[393]; LOE 3[394–396]; LOE 4[397]) were neutral.

In public access trials, six studies (LOE 1[197]; LOE 2[357]; LOE 3[361,362]; LOE 4[365,367]) supported PAD. Two studies (LOE 3[398]; LOE 5[399]) were neutral. Five LOE 4 studies[226,363,364,400,401] demonstrated survival attributed to AED programs in casinos, airplanes, or airports. One LOE 4 study[402] was neutral.

For home AED deployment, three studies (LOE 1[197,403]; LOE 2[404]) showed that home AED programs are safe and feasible but were unlikely to result in a significant increase in survival of out-of-hospital VF cardiac arrest.

For on-site AEDs in public places, 11 studies (LOE 1[197]; LOE 2[357]; LOE 3[224,361,362]; LOE 4[226,363–366,405]) supported on-site AEDs. This approach demonstrates high survival at low deployment rates. Four studies (LOE 1[392]; LOE 2[406]; LOE 3[395,398]) did not demonstrate improvement in survival to discharge compared with EMS, despite better response time.

For mobile AEDs, three studies (LOE 2[357,358]; LOE 3[391]) reported that community first responders (CFRs) equipped with AEDs achieved improvement in survival when they arrived at the patient's side sooner than traditional EMS responders.

In one LOE 2 study[358] first responders were trained only in AED use; however, most survivors received CPR and AED, suggesting a role for CPR. There is no evidence to support a specific type of rescuer as better than another. One LOE 3 study[361] noted that even untrained bystanders achieved good results.

One LOE 3 study[398] reported that use of a restrictive dispatch protocol (unresponsive and not breathing) to summon first responders reduced the frequency of deployment, by reducing not only false alarms (false-positives) but also legitimate calls (true positives). In contrast, in one LOE 2 study[358] a less-restrictive dispatch protocol (unresponsive patient) yielded more false-positives as part of a wider involvement of first responders and increased survival. No difference in response interval appeared to be related to instrument of dispatch (telephone compared with pager).

Treatment recommendation

Implementation of AED programs in public settings should be based on the characteristics of published reports of successful programs in similar settings.

Home AED use for high-risk individuals who do not have an ICD is safe and feasible and may be considered on an individual basis, but has not been shown to change overall survival rates.

Because population-specific (e.g., rate of witnessed arrest) and program-specific (e.g., response time) characteristics affect survival, when implementing an AED program, community and program leaders should consider factors such as location, development of a team with responsibility for monitoring and maintaining the devices, training and retraining programs for those who are likely to use the AED, coordination with the local EMS agency, and identification of a group of paid or volunteer individuals who are committed to using the AED for victims of arrest.

Knowledge gaps

- Community or program characteristics of effective AED programs

Other specific worksheets that would be helpful are:

- Evaluating AED deployment strategies
- Should communities perform cardiac arrest surveillance to inform placement of public AEDs?

Recognition and prevention

Patients who have cardiac arrest often have unrecognised or untreated warning signs. This section describes strategies to predict, recognise, and prevent cardiorespiratory arrest, including the role of education.

Sudden death in apparently healthy children and young adults[EIT-007]

In apparently healthy children and young adults, does the presence of any warning signs available to the layperson or HCP (e.g., syncope, family history), as opposed to their absence, predict an increased risk of sudden death? (Exclude screening in athletes and patients with known ischaemic heart disease.)

Consensus on science

Specific symptoms in apparently healthy children and young adults. There are no studies specifically examining the nature of syncope in apparently healthy children and young adults and their risk of sudden cardiac death (SCD). In one LOE P3 study[481] a family history of syncope or SCD, palpitations as a symptom, supine syncope, and syncope associated with exercise and emotional stress were more common in patients with than without Long QT Syndrome (LQTS). Two LOE P5 studies in older adults[482,483] identified the absence of nausea and vomiting before syncope and electrocardiogram (ECG) abnormalities as independent predictors of arrhythmic syncope. Less than 5 s of warning signs before syncope and less than two syncope episodes were predictors of syncope due to ventricular tachycardia (VT) or atrioventricular (AV) block.

A postmortem case study (LOE 5)[484] highlighted that inexplicable drowning and drowning in a strong swimmer may be due to LQTS or *Catecholaminergic Polymorphic Ventricular Tachycardia* (CPVT). Two LOE P5 studies[485,486] identified an association between LQTS and presentation with seizure phenotype.

Screening for risk of SCD in apparently healthy young adults and children. Evidence from two large prospective screening trials (LOE P1)[487,488] failed to identify any symptoms alone as a predictor of SCD in apparently healthy children and young adults. There was strong evidence in one of these trials[487] for use of 12-lead ECG when screening for cardiac disease.

Prodromal symptoms in victims of sudden death and SCD. Eight LOE P5 studies[489–497] examined the prodromal symptoms in victims of sudden death and SCD. Many patients complained of cardiac symptoms including syncope/presyncope, chest pain, and palpitations before death.

Risk of SCD in patients with known cardiac disease. In patients with a known diagnosis of cardiac disease, 11 studies (LOE P4[498]; LOE P5[499–508]) showed that syncope (with or without prodrome—particularly recent or recurrent) was invariably identified as an independent risk factor for increased risk of death. Chest pain on exertion only, and palpitations associated with syncope only, were associated with hypertrophic cardiomyopathy, coronary abnormalities, Wolff-Parkinson-White, and arrhythmogenic right ventricular cardiomyopathy.

Screening of family members. Five LOE P4 studies[498,509–512] examining the systematic evaluation of family members of patients with cardiac diseases associated with SCD and victims of SCD demonstrated a high yield of families affected by syndromes associated with SCD.

Treatment recommendation

Children and young adults presenting with characteristic symptoms of arrhythmic syncope should have a specialist cardiology assessment, which should include an ECG and in most cases an echocardiogram and exercise test.

Characteristics of arrhythmic syncope include syncope in the supine position, occurring during or after exercise, with no or only brief prodromal symptoms, repetitive episodes, or in individuals with a family history of sudden death. In addition, nonpleuritic chest pain, palpitations associated with syncope, seizures (when resistant to treatment, occurring at night, or precipitated by exercise, syncope, or loud noise), and drowning in a competent swimmer should raise suspicion of increased risk. Systematic evaluation in a clinic specializing in the care of those at risk for SCD is recommended in family members of young victims of SCD or those with a known cardiac disorder resulting in an increased risk of SCD.

Knowledge gaps

- Efficacy, elements, and patient selection criteria for dedicated cardiac screening clinics for relatives of patients with inheritable cardiac disease or SCD victims.
- Outcomes in children and young people specifically investigated for cardiac symptoms potentially related to risk of SCD.
- Incidence of warning signs in those who have suffered sudden unexpected death in the young compared with those who died from other causes or a control population.
- Cardiac evaluation of children with seizure disorders without definite cerebral disease and recalcitrant to therapy.

Early recognition and response systems to prevent in-hospital cardiac arrests[EIT-024]

In adults admitted to hospital, does use of early warning systems/rapid response team (RRT) systems/MET systems, compared with no such responses, reduce cardiac and respiratory arrest?

Consensus on science

A single LOE 1 study involving 23 hospitals[513] did not show a reduction in cardiac arrest rate after introduction of an MET when analysed on an intention-to-treat basis. *Post hoc* analysis of that study[514] showed a significant inverse relationship between frequency of team activation and cardiac arrest and unexpected mortality rate. An LOE 2 multicentre study[515] did not show a reduction in cardiac arrest numbers after implementation of an MET. Seven additional LOE 3 studies[516–522] did not show a reduction in cardiac arrest rate associated with the introduction of an RRT/MET.

A meta-analysis[523] showed that RRT/MET systems were associated with a reduction in rate of cardiopulmonary arrest outside the ICU but not with a lower hospital mortality rate.

Seventeen LOE 3 single-centre studies[524–540] reported reduced numbers of cardiac arrests after the implementation of RRT/MET systems. None of these studies addressed the impact of confounding factors on study outcomes.

A single-centre LOE 3 study[541] was unable to demonstrate a reduction in cardiac arrest rates after the implementation of an early warning scoring system (EWSS). After implementing an EWSS, cardiac arrest rate increased among patients who had higher early warning scores, compared to similarly scored patients before the intervention.

Treatment recommendation

In adult patients admitted to hospital, there is insufficient evidence to support or refute the use of early warning/RRT/MET systems, compared with no such systems, to reduce cardiac and respiratory arrests and hospital mortality. However, it is reasonable for hospitals to provide a system of care that includes (a) staff education about the signs of patient deterioration; (b) appropriate and regular vital signs monitoring of patients; (c) clear guidance (e.g., via calling criteria or early warning scores) to assist staff in the early detection of patient deterioration; (d) a clear, uniform system of calling for assistance; and (e) a clinical response to calls for assistance.

There is insufficient evidence to identify the best methods for the delivery of these components and, based on current evidence, this should be based on local circumstances.

Prediction of cardiac arrest in adult patients in hospital[EIT-025]

In hospital inpatients (adult), does the presence of any specific factors, compared with no such factors, predict occurrence of cardiac arrest (or other outcome)?

Consensus on science

Outcome: cardiac arrest. One LOE P3 multicentre cross-sectional survey,[542] one LOE P2 multicentre matched case–control study using pooled outcomes (cardiac arrest, unplanned ICU admission, and death),[543] and two single-hospital retrospective case-control studies (LOE P3[544] and LOE P4[545]) supported the ability of alterations in physiological variables, singly or in combination, to predict the occurrence of cardiac arrest. Single variables included heart rate, respiratory rate, systolic blood pressure, and decrease in level of consciousness. Combined elements included variably pooled and scored data (Modified Early Warning Score [MEWS]) with different cut-off points (MET criteria and MEWS). Sensitivity ranged from 49% to 89% and specificity from 77% to 99%.

An LOE P3 multicentre prospective observational study[546] measured the incidence of cardiac arrest, unplanned ICU admissions, and deaths, with or without antecedents recorded on charts: 60% of primary events had antecedents, the most frequent being decreases in systolic blood pressure and Glasgow Coma Scale (GCS) score.

Opposing evidence from one LOE P2 multicentre matched case–control study[543] and one LOE P2 single-hospital retrospective case-control study[545] reported that single variables and cut-offs did not correlate with the occurrence of cardiac arrest. Data were insufficient to define which variables and cut-offs were the best predictors of the occurrence of cardiac arrest.

Outcome: unexpected ICU admission. One LOE P3 multicentre cross-sectional survey,[542] one LOE P2 multicentre matched case–control study using pooled outcomes (cardiac arrest,

unplanned ICU admission, and death),[543] one LOE P3 single-institution retrospective observational study,[547] and one LOE P2 single-centre prospective cohort study[548] suggested that for in-hospital patients, altered vital signs were associated with unplanned ICU admission. However, different criteria for ICU admission between studies make this a less useful end point.

Outcome: mortality (predicted on admission to hospital). Six studies (LOE P2[549]; LOE P3[550,551]; LOE P4[552–554]) supported the value of combinations of demographic, physiological, and/or laboratory variables recorded on admission in predicting death in specific patient populations.

Three studies (LOE P2[555] LOE P3[556] and LOE P4[557]) supported the value of combinations of demographic, physiological, and laboratory variables recorded on admission in predicting death in specific patient populations.

Eleven studies (LOE P1[558–563]; LOE P2[548,564,565] and LOE P3[566,567]) supported the value of different combinations of demographic, physiological, and/or laboratory value derangement recorded at admission to hospital in predicting death with a sensitivity and specificity in the range of 0.6–0.8, but the best combination of variables and cut-off levels is still to be identified.

Prediction during hospital stay on ordinary wards. Eleven studies (LOE P1 prospective multicentre observational[568]; LOE P1 prospective single-centre cohort[569,570]; LOE P3 multicentre cross-sectional survey[542,571]; LOE 2 multicentre matched case-control using pooled outcomes [cardiac arrest, unplanned ICU admission, and death][543]; LOE P2 single-centre prospective observational[572–574]; LOE P3 multicentre prospective in a selected population of patients with greater illness severity[575]; LOE P3 single-centre retrospective observational[576]) supported the ability of physiological derangements measured in adult ward patients to predict death. The more abnormalities, the higher the risk of death, with a positive predictive value ranging from 11% to 70%. The best combination of variables and cut-off points is still to be identified.

Best variables to predict outcome. One LOE P2 cohort study on existing datasets[577] and 3 LOE P1 single-centre prospective studies[561–563] evaluating different variables showed a marked variation in their sensitivity and positive predictive value. For aggregate-weighted scoring systems, inclusion of heart rate (HR), respiratory rate (RR), systolic blood pressure (SBP), AVPU (alert, vocalizing, pain, unresponsive), temperature, age, and oxygen saturation achieved the best predictive value (area under Receiver Operating Characteristic curve 0.782, 95% CI 0.767–0.797). For single-parameter track and trigger systems, cut-off points of HR <35 and >140 min^{-1}, RR <6 and >32 min^{-1}, and SBP <80 mm Hg achieved the best positive predictive value. The inclusion of age improved the predictive value of both aggregate and single-parameter scoring systems.

Treatment recommendation

Hospitals should use a system validated for their specific patient population to identify individuals at increased risk of serious clinical deterioration, cardiac arrest, or death, both on admission and during hospital stay.

Educational strategies to improve outcomes[EIT-026A]

For hospital staff, does the use of any specific educational strategies, compared with no such strategies, improve outcomes (e.g., early recognition and rescue of the deteriorating patient at risk of cardiac/respiratory arrest)?

For hospital staff, does the use of any specific educational strategies, compared with no such strategies, improve outcomes (e.g., early recognition and rescue of the deteriorating patient at risk of cardiac/respiratory arrest)?

Consensus on science

There are no RCTs addressing the impact of a specific educational intervention on improvement of outcomes such as the earlier recognition or rescue of the deteriorating patient at risk of cardiac/respiratory arrest.

One LOE 3 multicentre before-and-after study[578] found that the number of cardiac arrest calls decreased while prearrest calls increased after implementing a standardised educational program in two hospitals; the intervention was associated with a decrease in true arrests as well as an increase in initial survival after cardiac arrest and survival to discharge. A prospective LOE 3 single-centre trial[579] of a simulation-based educational program failed to yield such benefits.

Treatment recommendation

There is insufficient evidence to identify specific educational strategies that improve outcomes (e.g., early recognition and rescue of the deteriorating patient at risk of cardiac/respiratory arrest). Educational efforts have a positive impact on knowledge, skills, and attitudes/confidence, and increase the frequency of activation of a response, and should therefore be considered.

Knowledge gaps

- Optimal risk stratification on admission and during hospital stay for clinical deterioration or death.
- Methods to identify patients most likely to benefit from early treatment escalation.
- Importance of various components of the rapid response system—including education, monitoring, calling criteria, mechanism of calling, and response.
- Elements of required education—including calling criteria, clinical skills, and simulation training.
- Optimal frequency of vital signs monitoring to detect deterioration.
- Cost-benefits of physician-led versus nonphysicians teams.
- Cost-benefits of rapid response team versus patient team responses.
- Do RRT/MET systems (or their individual components) improve outcomes other than cardiac arrest (e.g., reduced hospital mortality, reduced length of stay)?
- Impact of other variables (e.g., time of day, monitoring status) on risk.

Ethics and outcomes

The decision to start, continue and terminate resuscitation efforts is based on the balance between the risks, benefits, and burdens these interventions place on patients, family members, and healthcare providers. There are circumstances where resuscitation is inappropriate and should not be provided. This includes when there is clear evidence that to start resuscitation would be futile or against the expressed wishes of the patient. Systems should be established to communicate these prospective decisions and simple algorithms should be developed to assist rescuers in limiting the burden of unnecessary, potentially painful treatments.

Decisions before cardiac arrest[EIT-016]

In adults and children with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]), does existence and use of advance directives (e.g., "living wills" and Do Not Attempt Resuscitation [DNAR]

orders), compared with no such directives, improve outcomes (e.g., appropriate resuscitative efforts)?

In adults and children with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]), does existence and use of advance directives (e.g., "living wills" and Do Not Attempt Resuscitation [DNAR] orders), compared with no such directives, improve outcomes (e.g., appropriate resuscitative efforts)?

Consensus on science

In adults with out-of-hospital cardiac arrest, five studies (LOE 4[580–582]; LOE 5[583,584]) supported the use of DNAR orders and Physician Orders for Life Sustaining Treatment (POLST) forms compared with no such directives to improve outcomes (e.g., appropriate resuscitative efforts). One LOE 4 study[585] supported the use of advance directives in the context of a communitywide approach. Three LOE 4 studies[586–588] were neutral. Four studies (LOE 2[589]; LOE 4[585,590,591]) supported the use of advance directives. Two studies (LOE 1[592]; LOE 2[593]) suggested that the presence of advance directives reduced resuscitation rates in patients.

In adult patients with cardiac arrest, 18 additional studies (LOE 1[594–597]; LOE 2[598–600]; LOE 4[601–606]; LOE 5[607–611]) did not support the use of advance directives (e.g., living wills), compared with no such directives, to improve outcome defined as resuscitative efforts based on patient preference. Evidence from one LOE 3 study[612] suggested that the presence of a DNAR order decreased CPR rates.

No study was found that specifically addressed these issues in children.

Treatment recommendation

Standardised orders for limitations on life-sustaining treatments (e.g., DNAR, POLST) should be considered to decrease the incidence of futile resuscitation attempts and to ensure that adult patient wishes are honored. These orders should be specific, detailed, transferable across healthcare settings, and easily understood. Processes, protocols, and systems should be developed that fit within local cultural norms and legal limitations to allow providers to honor patient wishes about resuscitation efforts.

Knowledge gaps

- Implementation of DNAR/POLST in patients who move among different healthcare settings (e.g., out-of-hospital and in-hospital).
- Relationship between DNAR/POLST decisions and patient preferences.
- Critical elements for prehospital DNAR.

Termination of resuscitation rules[EIT-003A]

For adult patients in any setting, is there a clinical decision rule that enables reliable prediction of ROSC (or futile resuscitation efforts)?

For adult patients in any setting, is there a clinical decision rule that enables reliable prediction of ROSC (or futile resuscitation efforts)?

Consensus on science

One high-quality LOE P1 prospective study in adults[613] demonstrated that the "basic life support termination of resuscitation rule" (no shockable rhythm, unwitnessed by EMS, and no ROSC) is predictive of death when applied by defibrillation-only emergency medical technicians (EMTs). The survival rate with the application of this rule is 0.5% (95% CI 0.2–0.9). Subsequent studies including two LOE P1 studies[614,615] showed external generalizability of this rule.

Additional adult studies (LOE P1[616]; LOE P2[617]; LOE P5[618]) showed associations with futility of certain variables such as no ROSC at scene, nonshockable rhythm, unwitnessed arrest, no bystander CPR, call response time, and patient demographics.

Two in-hospital studies (LOE P1[619]; LOE P2[620]) and one emergency department (ED) study (LOE P2)[621] showed that the reliability of termination of resuscitation rules is limited in these settings.

Treatment recommendation

Prospectively validated termination of resuscitation rules such as the "basic life support termination of resuscitation rule" are recommended to guide termination of prehospital CPR in adults.

Other rules for various provider levels, including in-hospital providers, may be helpful to reduce variability in decision-making; however, rules should be prospectively validated before implementation.

Knowledge gaps

- When to start CPR in neonatal, paediatric, and adult patients.
- When to stop CPR in paediatric and neonatal patients.
- Prospectively validated termination of resuscitation rule for advanced life support providers.

Quality of life

Part of the decision-making process in deciding for or against commencing resuscitation is the likelihood of success of the resuscitation attempt and the quality of life that can be expected following discharge from hospital.

Quality of life after resuscitation[EIT-006]

In cardiac arrest patients (in-hospital and out-of-hospital), does resuscitation produce a good quality of life for survivors after discharge from hospital?

In cardiac arrest patients (in-hospital and out-of-hospital), does resuscitation produce a good quality of life for survivors after discharge from hospital?

Consensus on science

Eight prospective cohort studies (LOE P1)[622–629], two 'follow-up of untreated control group in an RCT' studies (LOE P2)[630,631], eight retrospective cohort studies (LOE P3)[632–639], and 28 case series (LOE P4)[319,326,640–665] showed that quality of life is good in cardiac arrest survivors.

One prospective cohort study (LOE P1)[666], one 'follow-up of untreated control group in an RCT' study (LOE P2)[667], three retrospective cohort studies (LOE P3)[634,668,669], and 12 case series (LOE P4)[417,670–680] showed that cardiac arrest survivors experience problems in physical, cognitive, psychological, and social functioning that impact on quality of life to a varying degree.

Seven case series (LOE P4)[681–687] suggested that resuscitation led to high rate of cognitive impairment and poorer quality of life. Four of these seven studies evaluated populations in which cardiac arrest prognosis is considered poor: nursing home patients,[681] octogenarians,[686] out-of hospital paediatric cardiac arrests with on-going CPR on hospital arrival,[683] and patients who remain comatose after resuscitation from out-of-hospital cardiac arrest.[687]

Treatment recommendation

Resuscitation after cardiac arrest produces a good quality of life in most survivors. There is little evidence to suggest that resuscitation leads to a large pool of survivors with an unacceptable quality of life. Cardiac arrest survivors may experience problems including anxiety, depression, post-traumatic stress, and difficulties with cognitive function. Clinicians should be aware of these potential problems, screen for them, and if found, treat them. Interventional

resuscitation studies should be encouraged to include a follow-up evaluation (ideally at least 6 months post-event) that assesses general health-related quality of life with a validated instrument (e.g., Health Utility Index 3, EQ5D, SF36), affective disorder (anxiety and depression), post-traumatic stress disorder, and cognitive function.

Knowledge gaps

- The best approach for clinicians to use to measure quality of life for patients after resuscitation.
- Consensus on a recommended set of QoL dimensions and measures to facilitate comparison and integration of literature, and future research.
- Long-term QoL studies of resuscitated children.
- Impact on families of cardiac arrest survivors.

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Appendix A. Evidence-based worksheets for Part 12: education, implementation, and teams: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations

Task Force	WS ID	PICO title	Short title	Authors	URL
ALS	ALS-SC-077	In adult cardiac arrest (prehospital) (P), does the performance of ALS procedures by experienced physicians (I) as opposed to standard care (without physicians) (C), improve outcome (O) (e.g., ROSC, survival)?	ALS procedures	Michael Bernhard, Bernd W. Böttiger, Clifton Callaway, Joseph P. Ornato	http://circ.ahajournals.org/site/C2010/ALS-SC-077.pdf
BLS	BLS-002A	In rescuers (P), does performing CPR on adult and paediatric patients with cardiac arrest (out-of-hospital and in-hospital) (I) as opposed to not performing CPR (ventilations and compressions) (C), increase the likelihood of harm (O) (e.g., infection)?	Harm to rescuers from CPR	Sung Oh Hwang	http://circ.ahajournals.org/site/C2010/BLS-002A.pdf
BLS	BLS-004B	In adult and paediatric patients with out-of-hospital cardiac arrest (including residential settings) (P), does implementation of a public access AED program (I) as opposed to traditional EMS response (C), improve successful outcomes (O) (e.g., ROSC, survival)?	Public access AED programs	E. Brooke Lerner	http://circ.ahajournals.org/site/C2010/BLS-004B.pdf
BLS	BLS-005A	In rescuers performing CPR on adult or paediatric patients (P), does compression only CPR (I) when compared with traditional CPR (C) result in an increase in adverse outcomes (e.g., fatigue) (O)?	Rescuer fatigue in CC Only CPR	Michael Baubin, Anthony J. Handley	http://circ.ahajournals.org/site/C2010/BLS-005A.pdf
BLS	BLS-012A	In rescuers performing CPR on adult or paediatric patients (out-of-hospital and in-hospital) (P), does the use of barrier devices (I) as opposed to no such use (C), improve outcome (O) (e.g., lower infection risk)?	Barrier devices	E. Brooke Lerner	http://circ.ahajournals.org/site/C2010/BLS-012A.pdf
EIT	EIT-001A	For resuscitation teams (P), do briefings/debriefings (I), when compared to no briefings/debriefings (C), improve performance or outcomes (O)? (INTERVENTION)	Debriefing of CPR performance	Dana P. Edelson, Trevor Yuen	http://circ.ahajournals.org/site/C2010/EIT-001A.pdf

Task Force	WS ID	PICO title	Short title	Authors	URL
EIT	EIT-001B	For resuscitation teams (P), do briefings/debriefings (I), when compared to no briefings/debriefings (C), improve performance or outcomes (O)? (INTERVENTION)	Debriefing of CPR performance	Jasmeet Soar	http://circ.ahajournals.org/site/C2010/EIT-001B.pdf
EIT	EIT-002A	For LAY PROVIDERS and HCPs (P), does the use of specific instructional methods (video/computer self-instructions) (I), when compared with traditional instructor-led courses (C) improve skill acquisition and retention (O)? (INTERVENTION)	CPR instruction methods (self-instruction vs traditional)	Anthony J. Handley	http://circ.ahajournals.org/site/C2010/EIT-002A.pdf
EIT	EIT-002B	For LAY PROVIDERS and HCPs (P), does the use of specific instructional methods (video/computer self-instructions) (I), when compared with traditional instructor-led courses (C) improve skill acquisition and retention (O)? (INTERVENTION)	CPR instruction methods (self-instruction vs traditional)	Linda Denke, Mary Mancini	http://circ.ahajournals.org/site/C2010/EIT-002B.pdf
EIT	EIT-003A	For adult (in any setting (P), is there a clinical decision rule (I) that enables reliable prediction of ROSC (or futile resuscitation efforts)? (DIAGNOSIS)	Futile resuscitation rules	Jennifer Dennett	http://circ.ahajournals.org/site/C2010/EIT-003A.pdf
EIT	EIT-004	For students of advanced level resuscitation courses (such as ACLS and PALS) (P), does success in the written examination (I) when compared with lack of success (C), predict success in completing the practical skills testing associated with the course or in resuscitation management performance in actual or simulated resuscitation events (O)? (PROGNOSIS)	Written exam for advanced resuscitation courses	Farhan Bhanji, David L. Rodgers	http://circ.ahajournals.org/site/C2010/EIT-004.pdf
EIT	EIT-005A	In laypersons and HCPs performing CPR, does the use of CPR feedback devices when compared to no device improves CPR skill acquisition, retention, and real life performance? (INTERVENTION)	CPR feedback devices during training	Gavin D. Perkins, Joyce Yeung	http://circ.ahajournals.org/site/C2010/EIT-005A.pdf
EIT	EIT-005B	In laypersons and HCPs performing CPR, does the use of CPR feedback devices when compared to no device improves CPR skill acquisition, retention, and real life performance? (INTERVENTION)	CPR feedback devices during training	Reylon A. Meeks	http://circ.ahajournals.org/site/C2010/EIT-005B.pdf
EIT	EIT-006	In cardiac arrest patients (in-hospital and prehospital) [P] does resuscitation [I] produce a good Quality of Life (QoL) for survivors after discharge from the hospital. [O]? Prognosis	Quality of life after resuscitation	Stephen Brett, Vanessa Ellliott, David L. Rodgers	http://circ.ahajournals.org/site/C2010/EIT-006.pdf
EIT	EIT-007	In apparently healthy children and young adults (P), dose the presence of any warning signs available to the lay person or health care professional (e.g. syncope, family history) (I), as opposed to their absence (C), predict an increased risk of sudden death (O)? (Exclude screening in sportsmen and patients with known ischaemic heart disease)	Warning signs predict increased risk of sudden death	Rani Robson, Jonathan Skinner	http://circ.ahajournals.org/site/C2010/EIT-007.pdf

Task Force	WS ID	PICO title	Short title	Authors	URL
EIT	EIT-008A	In bystanders (lay or HCP) (P), are there any specific factors (I) compared with standard interventions (C) that increase outcomes (e.g., willingness to provide or the actual performance of CPR (standard or chest compression only) on adult or paediatric patients with cardiac arrest (prehospital [OHCA]) (O)?	Willingness to provide CPR	Judy Young	http://circ.ahajournals.org/site/C2010/EIT-008A.pdf
EIT	EIT-008B	In bystanders (lay or HCP) (P), are there any specific factors (I) compared with standard interventions (C) that increase outcomes (e.g., willingness to provide or the actual performance of CPR (standard or chest compression only) on adult or paediatric patients with cardiac arrest (prehospital [OHCA]) (O)?	Willingness to provide CPR	Tetsuo Hatanaka, Masami Ishikawa, Keiichi Tada	http://circ.ahajournals.org/site/C2010/EIT-008B.pdf
EIT	EIT-009A	In ALS/PALS providers (P), are there any specific training interventions (e.g., duration of session, interactive computer programmes/e-learning, video self-instruction) (I) compared with traditional lecture/practice sessions (C) that increase outcomes (e.g., skill aquisition and retention) (O)?	Comparison of training methods	Alessandro Barelli, Farhan Bhanji	http://circ.ahajournals.org/site/C2010/EIT-009A.pdf
EIT	EIT-010	In BLS providers (lay and HCP) (P), are any specific intervals for update/retraining (I) compared with standard practice (i.e., 12 or 24 monthly) (C) that increase outcomes (e.g., skill acquisition and retention) (O)?	Timing for BLS retraining	Maaret Castrén, Barbara Furry	http://circ.ahajournals.org/site/C2010/EIT-010.pdf
EIT	EIT-011A	In ALS and PALS providers (P), are any specific intervals for update/retraining (I) compared with standard practice (i.e., 12 or 24 monthly) (C) that increase outcomes (e.g., skill aquisition and retention) (O)?	Timing for advanced resuscitation retraining	Jane E. McGowan	http://circ.ahajournals.org/site/C2010/EIT-011A.pdf
EIT	EIT-011B	In ALS and PALS providers (P), are any specific intervals for update/retraining (I) compared with standard practice (i.e., 12 or 24 monthly) (C) that increase outcomes (e.g., skill aquisition and retention) (O)?	Timing for advanced resuscitation retraining	Matthew Huei-Ming Ma, Chih-Wei Yang, Zuisen Yen	http://circ.ahajournals.org/site/C2010/EIT-011B.pdf
EIT	EIT-012A	In lay providers requiring BLS training (P), does focusing training on high risk populations (I) compared with no such targeting (C) increase outcomes (e.g., bystander CPR, survival) (O)?	BLS training for high risk populations	Elaine Gilfoyle	http://circ.ahajournals.org/site/C2010/EIT-012A.pdf
EIT	EIT-012B	In lay providers requiring BLS training (P), does focusing training on high risk populations (I) compared with no such targeting (C) increase outcomes (e.g., bystander CPR, survival) (O)?	BLS training for high risk populations	Casandra L. Williams	http://circ.ahajournals.org/site/C2010/EIT-012B.pdf
EIT	EIT-013A	In BLS providers (lay or HCP) requiring AED training (P), are there any specific training interventions (I) compared with traditional lecture/practice sessions (C) that increase outcomes (e.g., skill aquisition and retention, actual AED use) (O)?	AED training methods	Deems Okamoto	http://circ.ahajournals.org/site/C2010/EIT-013A.pdf

Task Force	WS ID	PICO title	Short title	Authors	URL
EIT	EIT-013B	In BLS providers (lay or HCP) requiring AED training (P), are there any specific training interventions (I) compared with traditional lecture/practice sessions (C) that increase outcomes (e.g., skill aquisition and retention, actual AED use) (O)?	AED training methods	Gavin D. Perkins, Joyce Yeung	http://circ.ahajournals.org/site/C2010/EIT-013B.pdf
EIT	EIT-014A	In providers (lay or HCP)(P), does undertaking training/perform actual CPR or use of defibrillator (manual or AED) (I) compared with no such training/performance(C) increase harm (e.g., infection or other adverse events)(O)?—include electrical safety of defibrillation	CPR training and harm to rescuer	Franklin H.G. Bridgewater	http://circ.ahajournals.org/site/C2010/EIT-014A.pdf
EIT	EIT-015	In AED programs (P), does the inclusion of any specific factors (e.g., linkage to 911 registries, location of program [including home]) (I) compared with not including those factors (C) improve the outcome of the program (O)?	AED training content	David C. Parish, Andrea Scapigliati, Antoine Trammell	http://circ.ahajournals.org/site/C2010/EIT-015.pdf
EIT	EIT-016	In adult and paediatric patients with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does existence and use of advanced directives (e.g., "living wills" and "do not resuscitate" orders) (I) compared with no such directives (C), improve outcome (e.g., appropriate resuscitation efforts) (O)?	Advanced directives	Jennifer Dennett, Terri Schmidt	http://circ.ahajournals.org/site/C2010/EIT-016.pdf
EIT	EIT-017A	In ALS providers undergoing ALS courses (P), does the inclusion of specific leadership/team training (I), as opposed to no such specific training (C), improve outcomes (e.g., performance during cardiac arrests) (O)?	Team and leadership training	Robin P. Davies, Dana P. Edelson, Trevor Yuen	http://circ.ahajournals.org/site/C2010/EIT-017A.pdf
EIT	EIT-018A	In ALS providers undergoing ALS courses (P), does the inclusion of specific precourse preparation (e.g., e-learning and pre-testing) (I), as opposed to no such preparation (C), improve outcomes (e.g., same skill assessment, but with less face to face (instructor) hands on training) (O)?	Precourse preparation for advanced courses	Andrew Lockey, David L. Rodgers	http://circ.ahajournals.org/site/C2010/EIT-018A.pdf
EIT	EIT-019A	In participants undergoing BLS/ALS courses (P), does the inclusion of more realistic techniques (e.g., high fidelity manikins, in-situ training) (I), as opposed to standard training (e.g., low fidelity, education centre) (C), improve outcomes (e.g., skills performance on manikins, skills performance in real arrests, willingness to perform) (O)?	High fidelity training	Jordan Duval-Arnould, Elizabeth A. Hunt	http://circ.ahajournals.org/site/C2010/EIT-019A.pdf
EIT	EIT-019B	In participants undergoing BLS/ALS courses (P), does the inclusion of more realistic techniques (e.g., high fidelity manikins, in-situ training) (I), as opposed to standard training (e.g., low fidelity, education centre) (C), improve outcomes (e.g., skills performance on manikins, skills performance in real arrests, willingness to perform) (O)?	High fidelity training	Judith Finn	http://circ.ahajournals.org/site/C2010/EIT-019B.pdf

Task Force	WS ID	PICO title	Short title	Authors	URL
EIT	EIT-020	In participants undergoing ALS courses (P), does the use of random scheduling (introducing station cases in a random manner) (I), as opposed to block scheduling (grouping the agenda around specific station activities such as VF or bradycardias) (C), improve outcomes (e.g., skills performance) (O)? Other outcomes may need to be determined after review of the literature, include use of modular courses	ALS scenarios: random vs block	Ian Bullock, David L. Rodgers	http://circ.ahajournals.org/site/C2010/EIT-020.pdf
EIT	EIT-021A	In participants undergoing BLS/ALS courses (P), does end of course testing (I), as opposed to continuous assessment and feedback (C), improve outcomes (e.g., improve learning/performance) (O)?	End of course testing vs continuous feedback	Farhan Bhanji, Gavin D. Perkins	http://circ.ahajournals.org/site/C2010/EIT-021A.pdf
EIT	EIT-022	In communities where processes/guidelines are being implemented (P), does the use of any specific factors (I), compared with no such use (C), improve outcomes (e.g., success of implementation) (O)?	Implementation of community guidelines	John E. Billi, R. Van Harrison	http://circ.ahajournals.org/site/C2010/EIT-022.pdf
EIT	EIT-022B	In communities where processes/guidelines are being implemented (P), does the use of any specific factors (I), compared with no such use (C), improve outcomes (e.g., success of implementation) (O)?	Implementation of community guidelines	Patrick Chow-In Ko	http://circ.ahajournals.org/site/C2010/EIT-022B.pdf
EIT	EIT-023B	For resuscitation systems (prehospital and in-hospital) (P), does the use of a performance measurement systems (e.g., Utstein) improve and/or allow for comparison of system outcomes (patient level and system level variables) (O)?	Measuring performance of resuscitation systems	Judith Finn, Satoshi Takeda	http://circ.ahajournals.org/site/C2010/EIT-023B.pdf
EIT	EIT-024	In adult patients admitted to hospital (P), does use of EWSS/response teams/MET systems (I) compared with no such responses (C), improve outcome (e.g., reduce cardiac and respiratory arrests) (O)?	METs	Michael DeVita, Mary Beth Mancini, Nicola Poplett, Gary Smith, Jasmeet Soar	http://circ.ahajournals.org/site/C2010/EIT-024.pdf
EIT	EIT-025	In-hospital in-patients (adult) (P), does the presence of any specific factors (I) compared with no such factors (C), predict occurrence of cardiac arrest (or other outcome) (O)?	Predicting in-hospital cardiac arrest	Erga Cerchiari, Michael DeVita	http://circ.ahajournals.org/site/C2010/EIT-025.pdf
EIT	EIT-026A	In-hospital staff (P), does the use of any specific educational strategies (I) compare with no such strategies (C) improve outcomes (e.g., early recognition and rescue of the deteriorating patient (at risk of cardiac/respiratory arrest)) (O)?	Training strategies for hospital staff (to predict arrest?)	Geoffrey K. Lighthall, Anne Lippert	http://circ.ahajournals.org/site/C2010/EIT-026A.pdf
EIT	EIT-027	In adult and paediatric patients with out-of-hospital cardiac arrests (P), does transport to a specialist cardiac arrest centre (I) compared with no such directed transport (C), improve outcome (e.g., survival) (O)?	Cardiac arrest centres	Graham Nichol, Jasmeet Soar	http://circ.ahajournals.org/site/C2010/EIT-027.pdf
EIT	EIT-028A	What resuscitation training interventions are practical, feasible and effective in low income countries?	Resuscitation training in low income countries	Martin Botha	http://circ.ahajournals.org/site/C2010/EIT-028A.pdf
EIT	EIT-028B	What resuscitation training interventions are practical, feasible and effective in low income countries?	Resuscitation training in low income countries	Peter A. Meaney	http://circ.ahajournals.org/site/C2010/EIT-028B.pdf

Task Force	WS ID	PICO title	Short title	Authors	URL
EIT	EIT-029A	For BLS providers (lay or HCP) (P), does a longer-duration instructor-based course (I), compared with a shorter course (C), improve skill acquisition and retention (O)?	Duration of BLS courses	Anthony J. Handley	http://circ.ahajournals.org/site/C2010/EIT-029A.pdf
EIT	EIT-029B	For BLS providers (lay or HCP) (P), does a longer-duration instructor-based course (I), compared with a shorter course (C), improve skill acquisition and retention (O)?	Duration of BLS courses	Yasuhiro Kuroda	http://circ.ahajournals.org/site/C2010/EIT-029B.pdf
EIT	EIT-030A	For lay and HCP (P) does the use of assessment (I) as opposed to no such assessment (C) improve knowledge, skills and learning/retention(O)?	Impact of assessment on knowledge, skills and learning/retention	Farhan Bhanji, Gavin D. Perkins	http://circ.ahajournals.org/site/C2010/EIT-030A.pdf
EIT	EIT-031A	Does the use of a checklist during adult and paediatric advanced life support as opposed to no checklist improve outcomes (e.g., compliance with guidelines, other outcomes)?	Use of checklist during ACLS or PALS	Nicholas Brennan	http://circ.ahajournals.org/site/C2010/EIT-031A.pdf
EIT	EIT-031B	Does the use of a checklist during adult and paediatric advanced life support as opposed to no checklist improve outcomes (e.g., compliance with guidelines, other outcomes)?	Use of checklist during ACLS or PALS	Farhan Bhanji, Matthew Weiss	http://circ.ahajournals.org/site/C2010/EIT-031B.pdf
EIT	EIT-032	(P)In adult patients receiving chest compressions I—is there a method to teach chest compressions(C) compared with current teaching	Methods to teach chest compressions	Tom P. Aufderheide	http://circ.ahajournals.org/site/C2010/EIT-032.pdf
NRP	NRP-033A	For hospital resuscitation teams (P), do team briefings/debriefings (I), when compared to no briefings/debriefings (C), improve team performance (O)? (INTERVENTION)	Impact of debreifing on team performance	Dianne L. Atkins, Nalini Singhal	http://circ.ahajournals.org/site/C2010/NRP-033A.pdf
NRP	NRP-033B	For hospital resuscitation teams (P), do team briefings/debriefings (I), when compared to no briefings/debriefings (C), improve team performance (O)? (INTERVENTION)	Impact of debreifing on team performance	Louis P. Halamek	http://circ.ahajournals.org/site/C2010/NRP-033B.pdf

Appendix B.

CoSTR Part 12: writing group disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Ownership interest	Consultant/ advisory board	Other
Mary E. Mancini	University of Texas at Arlington—Professor	[a]I have received research in the last two years from AHA and Laerdal Medical to study effectiveness of BLS programs	None	[a]I received an honoraria from Datascope for speaking at national programs for ENA and AACN	[a]I have a small percentage (<5%) of a patent held by the University for a device that could potentially be used for CPR feedback. To date, it has not been developed	None	None
Jasmeet Soar	North Bristol NHS Trust: Government Hospital in UK—Consultant in Anaesthetics & Intensive Care Medicine	None	None	None	None	None	None

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Farhan Bhanji	McGill University—Assistant Professor of Pediatrics	None	None	None	None	None	None
John E. Billi	University of Michigan Medical School Associate Dean	None	None	None	None	None	None
Jennifer Dennett	Central Gippsland Health Service-Deputy Director of Nursing	None	None	None	None	None	None
Judith Finn	University of Western Australia—Professor	[b]Multiple National Health and Medical Research Grants (NH&MRC), National Heart Foundation Australia and State Government grants of >$10,000 since 1999. A—No money came to me—all came to my University to employ research staff and meet research expenses. No grants were directly related to any topic on which I am undertaking a Worksheet and none involved the trialing of a commercial product	None	[a]Less than $1000 from the Japanese Resuscitation Council to speak at their JRC Conference in Osaka in 2009	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing—Professor; AHA ECC Product Development—Senior Science Editor [a]I receive significant compensation from the AHA precisely to provide protected time so I can co-edit the 2010 CoSTR and the 2010 AHA Guidelines for CPR and ECC. Thus, although I have a significant relationship, I think the purpose of the relationship is to help ensure the quality, consistency and timeliness of the both documents because I can devote the time to follow-through. One of the CoSTR sections that I will be helping to write and that I will be editing is the Education, Implementation and Teams section	None	None	None	None	None	None
Ian Jacobs	University of Western Australia: Discipline of Emergency Medicine Teaching/Research academic—Professor; American Heart Association: Evaluation of evidence worksheets for C2010—Work Sheet Expert	[b]Chief investigator on numerous grants awarded by:	[b]Funds are received into the Discipline of Emergency Medicine —University of Western Australia from the Ambulance Service—Western Australia and Laerdal (Australia) to maintain the Cardiac Arrest Registry for Western Australia. Our role is to independently maintain, analyse and report outcomes of cardiac arrest in Western Australia. I oversee the operation of the registry and reporting of outcomes. These funds are not used in any way to provide any direct or indirect salary or other financial support	None	None	None	None

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
		(a) National Health and Medical Research Council (b) The Department of Health—Western Australia (c) The National Heart Foundation of Australia These funds are awarded to the University of Western Australia and none are used to provide any direct or indirect salary or other financial support					
Matthew Huei-Ming Ma	National Taiwan University Hospital: Patient care, education and research—Associate professor of emergency medicine	None	None	None	None	None	None
Peter T. Morley	Royal Melbourne Hosp; Univ of Melbourne; Dir of Medical Education; AHA EEE	None	None	None	None	None	None
Gavin D. Perkins	University of Warwick—Associate Professor Editor for Resuscitation Journal	[b]Department of Health National Institute of Health (NIH) Research—Research for patient benefit grant to investigate strategies to improve quality of CPR in clinical practice. Grant awarded to institution—I am the PI; [b]Resuscitation Council (UK)—Research Grant to support randomised controlled trial of e-learning versus standard advanced life support training. Grant awarded to my institution. I am the PI	None	None	None	None	None
David Rodgers	Healthcare Simulation Strategies—President and Owner	None	None	[a]Received $750 for presenting at Laerdal Medical Midwest Simulation User Network meeting in 2008	None	[b]Provide consultation services to Laerdal Medical in patient simulation education program development	[b]Spouse (Robin R. Roberts) is an employee of the American Heart Association

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

[a] Modest.
[b] Significant.

CoSTR Part 12: worksheet collaborator disclosures

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Dianne L. Atkins	Medical College of Wisconsin Associate Professor	None	[b]Title: Circulation Improving Resuscitation Care Trial Source: Zoll Medical Corporation Role: Consultant Principal Investigator: Lars Wik, M.D. Dates: 12/2006–8/2010 Total Funding to MCW: $345,000 (funding is received by my employer to support my time on this trial. My institution receives support for 20% of my time and the remaining funds are used for other members of our staff and supplies. My role is to advise them on human subject protection issues and to assist with data management and report generation for the trial)	None	[a]Stockholder in Medtronic, Pfizer, and General Electric	None	None
Tom P. Aufderheide	Medical College of Wisconsin: Medical School and University—Professor of Emergency Medicine	[b]Resuscitation Outcomes Consortium [b](NHLBI): money comes to my institution Immediate Trial[b] (NHLBI): money comes to my institution ResQTrial [b](NHLBI): money comes to my institution Neurological Emergency Treatment Trials [b](NETT) Network: money comes to my institution	[b]Automated external defibrillators donated to ROC by Zoll Medical for implementation of the PRIMED Trial: equipment given directly to Milwaukee County EMS for use in the trial	None	None	[a]Chairman of the Board for Citizen CPR Foundation and President: this is a volunteer position; I receive no money for this Secretary and Member of the Board for Take Heart America: I have received no money for this Consultant to Medtronic for an acute myocardial infarction study: money goes directly to my institution Consultant to [a]JoLife (consultancy terminated March, 2009): money goes directly to my institution [a]Member Basic Life Support Subcommitte for National American Heart Association: this is a volunteer position	None
Alessandro Barelli	Catholic University School of Medicine Rome Italy—MD	None	None	None	None	None	None
Michael Bernhard	Hospital of Fulda gAG, Germany medical doctor	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Martin Botha	Academy of Advanced Life Support: Education and training in emergency medicine and AHA courses—training manager	None	None	None	None	None	None
Bernd W. Bottiger	Uniklinik Köln MD, DEAA	None	None	None	None	None	None
Nicholas Brennan	Western Sussex Hospital NHS Trust NHS Hospital Consultant Paediatrician	None	None	None	None	None	None
Stephen Brett	Imperial College Healthcare NHS Trust—Consultant in Intensive care Medicine	None	None	None	None	None	None
Frank Bridgewater	The Queen Elizabeth Hospital, Woodville, South Australia: Tertiary Hospital—Visiting Surgeon	None	None	None	None	None	None
Ian Bullock	Royal College of Physicians, Chief Operating Officer	None	None	None	None	None	None
Clifton W. Callaway	[b]University of Pittsburgh: Associate Professor, UPMC; Evidence evaluation expert for AHA	[b]Grants to University of Pittsburgh: NHLBI—Resuscitation Outcomes Consortium HRSA—Development and Dissemination of Program Tools for Uncontrolled Donation After Cardiac Death (UDCD)	[b]Loan of an Arctic Sun cooling device (without disposables) to human physiology laboratory for experiments on hypothermia by Medivance, Inc.	None	[b]Co-inventor on patent about ventricular fibrillation waveform analysis, licensed by University of Pittsburgh to Medtronic ERS, Inc.	None	None
Maaret Castren	Karolinska Institutet, Stockholm, Sweden: University—Professor in Emergency Medicine; Södersjukhuset, Stockholm, Sweden: Hospital—Consultant	[a]Laerdal 2008 95 000 NOK Laerdal 2009 155 000 NOK	[a]Equipment for therapeutic hypothermia during resuscitation	None	None	None	None
Erga Cerchiari	Maggiore Hospital, Bologna, Italy—Director of Anaesthesia and Intensive Care	None	None	None	None	None	None
Robin Davies	Heart of England NHS Foundation Trust—Senior Resuscitation Officer; Resuscitation Council (UK): Charity—Lead Resuscitation Officer; University of Warwick Medical School—Associate Research Fellow	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Linda Denke	Collin College Education of senior nursing students Professor of Nursing	None	None	None	None	None	None
Michael DeVita	UPMC Health System healthcare organisation physician, associate medical director	None	None	None	None	None	None
Jordan Duval-Arnould	Johns Hopkins University—Senior Clinical Research Coordinator	None	None	None	None	None	None
Dana P. Edelson	University of Chicago—Assistant Professor	[b]Research Grants Pending NHLBI Career Development Award Strategies to Predict and Prevent In-Hospital Cardiac Arrest (IHCA) (1K23HL097157-01) To validate a clinical judgment based tool for predicting impending clinical deterioration of hospitalised floor patients and compare it to previously described physiology-based tools Role: PI (funds delivered to university) [b]2009—present Philips Healthcare Research Grant Advancements in Cardiopulmonary Resuscitation and Emergency Care during Haemodynamic Crisis To measure capnography and pulse pressure, using a novel plethysmographic sensor, in critically ill patients and correlate quality of CPR with these measures during CA Role: PI (funds delivered to university) [b]2008—present Philips Healthcare Research Grant Q-CPR Users and Development Research Alliance The purpose of this project is to establish a multi-centre registry of in-hospital resuscitation quality data and a network for clinical trials of resuscitation	[a]Philips Healthcare, Andover, MA	[a]Philips Healthcare, Andover, MA	None	[a]Triage Wireless, San Diego, CA	[a]Expert Witness—Hanna Campbell & Powell LLP, Akron, OH—Hankton v Beeson

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
		Role: Principal Investigator (funds delivered to university) [b]2008—present NIH Clinical Research Loan Repayment Granted two years of student loan; to evaluate the effects of integrated team debriefing using actual performance data to improve CPR quality and patient survival following IHCA Role: PI (funds delivered to loan servicing program) [b]2008–2009 NIH Agency for Healthcare Research and Quality Immersive Simulation Team Training—Impact on Rescue, Recovery and Safety Culture (5U18HS016664-02) The goal is to study the effects of simulation based training for Rapid Response Teams Role: Consultant (funds delivered to university)					
Vanessa Elliott	North West London NHS Trust—Medical Registrar	None	None	None	None	None	None
Barbara Furry	The Center of Excellence in Education—Director	None	None	None	None	None	None
Elaine Gilfoyle	Self-employed—clinician—Pediatric Intensivist	None	None	None	None	None	None
Anthony J. Handley	Self-employed—Consultant Physician	None	None	None	None	[a]Consultant Medical Adviser British Airways Consultant Medical Adviser Virgin Atlantic Airways Consultant Medical Advisor DC Leisure Management Ltd	[b]Variable income as Expert Witness. Direct payment. No single firm of lawyers—instructions as received
Van Harrison	University of Michigan Professor of Medical Education	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Tetsuo Hatanaka	Emergency Life-Saving Technique Academy—Professor	[b]Research grant for "Cardiovascular Disease H18-Heart-01: A Study on Automated External Defibrillator Program and System Development for Improved Survival from Emergency Cardiovascular Disease" from the Ministry of Health, Labor and Welfare, Japan. The grant money comes directly to me	None	[a]Several kinds of honoraria for scientific meetings	None	None	None
Elizabeth A. Hunt	Johns Hopkins University School Med. Pediatric intensivist, researcher & Dir of Johns Hopkins Med Simulation Center-Director, Assist. Prof.	[a]Co PI on AHA grant to study relationship between scripted debriefing & high fidelity simulation on learning during PALS course	None	None	None	None	None
Sung Oh Hwang	Yonsei University, Republic of Korea Professor	None	None	None	[b]Patent owner (Sung Oh Hwang)-US patent (US7214203),Cardiopulmonary resuscitation apparatus [b]Stock holder (Sung Oh Hwnag)-Humed Co, Republic of Korea	None	None
Masami Ishikawa	Kure Kyosai Hosp; MD	None	None	None	None	None	None
Patrick Chow-In Ko	National Taiwan University Hospital healthcare provider Attending Physician/Assistant Professor	None	None	None	None	None	None
Yasuhiro Kuroda	Kagawa University, Japan: Department of Emergency, Disaster, and Critical Care Medicine—Professor	None	None	None	None	None	None
E. Brooke Lerner	Medical College of Wisconsin—Associate Professor	[b] Grant funding All monies are given to my institution. I receive no funding directly 1. Injury Research Center, Medical College of Wisconsin 2009–2011 Development of a Study of EMS Findings Predictive of Ped. Trauma Center—understand the epidemiology of injured ped.pts transported by EMS. To develop a strong multi-center study proposal to determine which Field Triage Criteria are most predictive of trauma center need for ped. patients	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
		Role: Principal-Investigator 5% support 2.HRSA/MCHB Project #U03MC00007 2008-20011 EMS for Children Network Development Demonstration Project To form collaborative research partnerships among various EMS agencies and academic and hospital-based entities Role: Co-Investigator 10% support 3. CDC, Project #1R49CE001175-01 2008-2012 Injury Research Center at the Medical College of Wisconsin Grant addresses the burden of injury in the Great Lakes Region of the Midwest (Wisconsin, Minnesota, Illinois, Indiana, Michigan & Ohio). Role: Epidemiologist 5% support 4. CDC, Project #U17/CE001232-01 2007–2010 Identifying & Disseminating Best Practices by Collaboration of Public Health & the EMS-This cooperative agreement is to bring together national organisations to work on projects to gather &disseminate information on the response to terrorism related injuries. Main activities include developing a national guideline for mass casualty triage and identify communities that are models for integration of public health and emergency care.Role: Principal-Investigator 9% support 5. Zoll Medical Corporation, Project # N/A 2006-2010 Circulation Improving Resus. Care This project will determine if Auto-Pulse integrated CPR is superior or equivalent to manual CPR, in terms of the number of pts who survive to hospital discharge after OHCA.Role: Consultant 20% support					

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Geoffrey K. Lighthall	US Department of Veterans Affairs Physician	None	None	None	None	None	None
Anne Lippert	Danish Institute for Medical Simulation: Regional Institute for Medical education, development and research—Consultant	None	None	None	None	None	None
Andrew Lockey	Calderdale & Huddersfield NHS Foundation Trust—EM Consultant	None	None	None	None	None	None
Jane E. McGowan	SCPA/Tenet healthcare: Pediatric Practice Group—Attending Neonatologist	None	None	None	None	None	None
Peter A. Meaney	University of Pennsylvania, Children's Hospital of Philadelphia: Anaesthesia, Critical Care and Pediatrics—Assistant Professor	[a]Laerdal Foundation, Research grant "Development and Validation of a Quantitative Measurement Device to Assess Technical Basic Life Support Skills in Resource Limited Settings." No direct support to investigator [a]Operation Smile, Educational development grant, "Pilot Training Proposal: Acute Care Training for the Peri-Operative Pediatrician." No direct support to investigator	[a]Laerdal Corporation, Research equipment (study manikins) for "Development and Validation of a Quantitative Measurement Device to Assess Technical Basic Life Support Skills in Resource Limited Settings." No direct support to investigator	[a]Received $400 to give lecture on "Pediatric Rhythms and Outcomes for In-Hospital Cardiac Arrest" at San Antonio Trauma Symposium, August 2008	[a]I have multiple mutual funds as part of my retirement and college savings portfolios. Am unsure as to how much is allocated to any conflicted companies	None	None
Reylon A. Meeks	Blank Children's Hosp/Pleasant Hill FD; Clinical Nurse Specialist/Fire Chief	None	None	None	None	None	None
Graham Nichol	University of Washington State-Professor	[b]NHLBI, Bethesda, MD. Co-PI, Resuscitation Outcomes Consortium Data Coordinating Center [b]NHLBI, Bethesda, MD. PI, Randomised Trial of Haemofiltration After Resuscitation from Cardiac Arrest [b]NHLBI, Bethesda, MD. Co-I, Randomised Field Trial of Cold Saline IV After Resuscitation from Cardiac Arrest. [a]Asmund S Laerdal Foundation for Acute Medicine Stavanger, NO PI, Randomised Trial of CPR Training Aid in Community	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Deems Okamoto	Ballard Emergency Physicians—Independent contractor providing emergency medical evaluation and treatment as a physician	None	None	None	None	None	None
Joseph Ornato	Virginia Commonwealth University Health System academic health center Prof & Chmn, Emergency Medicine	[b]NIH Neurological Emergency Treatment Trials, Principal Investigator, VCU study site	None	[a]ZOLL Circulation (approximately 1 lecture a year at most for last 5 years)	None	[b]Significant Consultant Advisory Board: NIH sponsored Resuscitation Outcomes Consortium, Cardiac Co-Chair/Consultant [a]Advisory Board: ZOLL Circulation (unpaid, receive only travel reimbursement for approximately 1 advisory board meeting per year, usually on East Coast close to home)	[b]American Editor, Resuscitation
David Parish	Mercer University School of Medicine Education of medical students and residents, medical research, patient care and community service. Professor, Interim Chair, Internal Medicine	None	None	None	None	None	None
Nicola Polett	Portsmouth Hospitals NHS Trust Healthcare—NHS Resuscitation Manager	None	None	None	None	None	None
Rani Robson	North Bristol NHS Trust—Cardiology Registrar	None	None	None	None	None	None
Andrea Scapigliati	Catholic University School of Medicine, Rome, Italy—Assistant Professor	None	None	None	None	None	None
Terri Schmidt	Clackamas County Health Department—EMS Medical Director	None	None	None	None	None	[a]Iinvestigator in the Resuscitations Outcomes Consortium which is funded by the NIH and AHA among others. Funding is to the institution and I have no salary support on the grant

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
							[a]I am a member of the National POLST Taskforce and speak on the subject. I do not receive honoraria
Nalini Singhal	University of Calgary Professor	[a]AAP grant looking at effect of PEEP with and with oxygen on resuscitation Developing a International program for resuscitation, Helping Babies Breathe	None	None	None	None	None
Jonathan Skinner	Auckland District Health Board, New Zealand: National Health Service of New Zealand, Health Board—Specialist Pediatric Cardiologist	[b]"Cure Kids New Zealand" (Childrens Health Research New Zealand), provide an academic salary to me, via my employer Auckland District Health Board, comprising the equivalent of 20% of my standard (40 hour) working week to allow me to work in the clinical and research area of cardiac inherited diseases	None	None	None	None	None
Gary Smith	Portsmouth Hosp NHS Trust, NHS Hosp Consultant in Critical Care	None	None	[a]Wife is shareholder of Learning Clinics, my employer	None	[a]Research Advisor with employer that develops vital signs capture system	None
Keiichi Tada	Hiroshima City Hosp; MD	None	None	None	None	None	None
Satoshi Takeda	Jikei University School of Medicine Lecturer	None	None	None	None	None	[a]Expert Witness: Eisai Japan [a]Philips
Antoine Trammell	Emory University School of Medicine Attending physician, division of General Medicine Instructor of Medicine	None	None	None	None	None	None
Matthew Weiss	Montreal Children's Hospital through the McGill University Health Centre—Pediatric Intensive Care Fellow	None	None	None	None	None	None
Casie Williams	Alaska Native Medical Center: Not currently employed—Retired 9/30/2008—Nurse Educator	None	None	None	None	None	None
Chih-Wei Yang	Ochsner Health System—Director, Vascular Medicine	None	None	None	None	None	None
Zuisen Yen	TBD	TBD	TBD	TBD	TBD	TBD	TBD
Joyce Yeung	Heart of England NHs Foundation Trust, UK—Anaesthetic Specialist Registrar/Research Fellow	None	None	None	None	None	None

Worksheet collaborator	Employment	Research grant	Other research support	Speakers' bureau/ honoraria	Ownership interest	Consultant/ advisory board	Other
Judy Young	Anne Arundel Medical Center: Direct patient care nurse in the CCU (Critical Care Unit). Work part time for this facility—Staff Nurse, CCU; Brevard Community College—Adjunct Clinical Faculty, Department of Nursing.; Sebastian River Medical Center: Direct patient care to critical care patients in the ICU. Work part time for this facility—RN, ICU; Florida Legal Nurse Experts, LLC—Owner, Florida Legal Nurse Experts, LLC	None	None	None	None	None	None
Trevor Yuen	University of Chicago Med Center; Clinical Research Data Assistant	None	None	None	None	None	None

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[a] Modest.

[b] Significant.

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