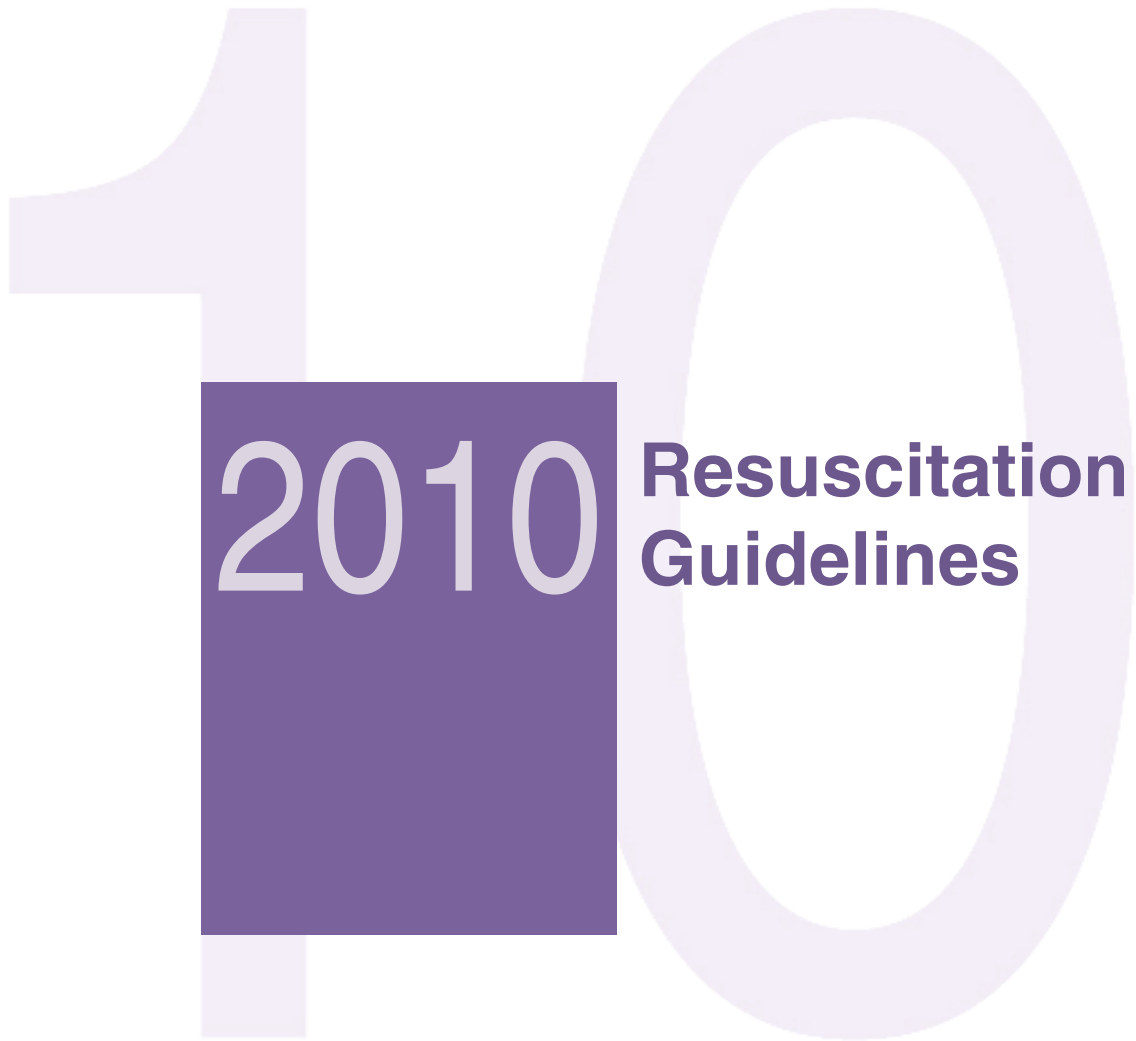


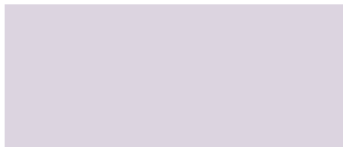


Resuscitation Council (UK)



2010

**Resuscitation
Guidelines**




October 2010

2010 Resuscitation Guidelines

October 2010

Edited by Jerry P. Nolan



Published by the Resuscitation Council (UK)
5th Floor, Tavistock House North
Tavistock Square
London WC1H 9HR



- Tel: 020 7388 4678
- Fax: 020 7383 0773
- E-mail: enquiries@resus.org.uk
- Website: www.resus.org.uk

Registered charity no. 286360

ISBN 978-1-903812-21-1

Copyright © Resuscitation Council (UK)

All rights reserved. No part of this publication may be reproduced or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise without the prior written permission of the Resuscitation Council (UK). Permission must also be obtained before any part of this publication is stored in any information storage or retrieval system of any nature.

Foreword

The Resuscitation Council (UK) is a registered charity formed in August 1981 by a group of medical practitioners from a variety of specialities who shared an interest in resuscitation. The objectives of the Council are:

- To produce guidelines for resuscitation procedures.
- To publish national standards for resuscitation and provide guidance on how they can be achieved.
- To promote the teaching of resuscitation in accordance with the established guidelines.
- To study and improve resuscitation teaching techniques.
- To encourage and fund research in resuscitation.
- To foster good working relations between all organisations involved in resuscitation.
- To design and publish training materials for high-quality resuscitation courses.
- To provide administrative support for resuscitation training courses.

Several members of the Executive Committee of the Resuscitation Council (UK) contribute to the activities of the European Resuscitation Council and the International Liaison Committee on Resuscitation; these bodies are responsible for training, research, and guideline setting worldwide.

Contents

Introduction	5
Jasmeet Soar, Jerry Nolan	
1. Summary of changes	8
Jerry Nolan, Jasmeet Soar	
2. Adult Basic Life Support	15
Anthony Handley, Michael Colquhoun	
3. The use of Automated External Defibrillators	29
Gavin Perkins, Michael Colquhoun	
4. Pre-hospital cardiac arrest	36
Fiona Jewkes, Jerry Nolan	
5. Prevention of in-hospital cardiac arrest and decisions about cardiopulmonary resuscitation	42
Jasmeet Soar, Gary Smith	
6. In-hospital resuscitation	48
Jasmeet Soar, Robin Davies	
7. Adult Advanced Life Support	58
Charles Deakin, Jerry Nolan, Gavin Perkins, Andrew Lockey	
8. Peri-arrest arrhythmias	81
David Pitcher, Gavin Perkins	

Contents (continued)

9. Paediatric Basic Life Support	91
Robert Bingham, Ian Maconochie, Sheila Simpson, Fiona Jewkes	
10. Paediatric Advanced Life Support	106
Robert Bingham, Ian Maconochie, Fiona Jewkes	
11. Newborn Life Support	118
Sam Richmond, Jonathan Wyllie	
12. References	128
Appendix: Conflict of interest declaration	153

Throughout this publication the masculine is used to denote the masculine or feminine. Text in blue indicates a web link, either internally to a page on the Resuscitation Council (UK) website, or externally to another site. Please refer to our website's [Terms of use](#) regarding external web links.

Introduction

Guidelines 2010 marks the 50th Anniversary of modern CPR.¹ These new guidelines are the culmination of many years of international collaboration to improve the practice and teaching of resuscitation medicine.² Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care provided the basis for the first international resuscitation guidelines.³ Representatives from the world's major resuscitation organisations reached this consensus only after exhaustive review of the published literature and extensive debate at consensus meetings. The review process was thorough and provided the best evidence-based approach to the resuscitation of patients of all ages. The guidelines that arose from this process were adopted internationally with only minor modifications required by local custom, practice, or availability of drugs.

This review process, led by the International Liaison Committee on Resuscitation (ILCOR), was repeated during 2004/5 and culminated in the 2005 International Consensus Conference on Emergency Cardiovascular Care (ECC) and Cardiopulmonary Resuscitation (CPR) Science with Treatment Recommendations (C2005). The summary science statements and treatment recommendations from this conference were published: *2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations (CoSTR)*.⁴ This document formed the scientific basis for the European Resuscitation Council (ERC) and Resuscitation Council (UK) (RC(UK)) Guidelines for Resuscitation 2005. Five years later, the whole review process has been repeated. The 2010 CoSTR⁵ is the product of the most recent Consensus on CPR Science Conference that took place in February 2010 and forms the basis of the 2010 ERC⁶ and RC(UK) Guidelines. Treatment recommendations derived directly from the ILCOR systematic reviews are formulated by consensus. The precise wording of the recommendations is agreed by an international task force of 8-12 individuals before being presented to an International Editorial Board of 25 individuals representing resuscitation organisations. Using a web-based system, writing group members have to vote for every treatment recommendation in their section of the consensus on CPR science document. This is consistent with nominal group consensus methods. Each section of the CoSTR manuscript is peer reviewed by four reviewers and individual responses are made to all comments from the reviewers; amendments are made to the final manuscript before publication in the journals *Circulation* and *Resuscitation*.

The ERC and RC(UK) derive their guidelines by taking ILCOR source statements and by applying nominal group methods, then translating them into treatment and practice recommendations that are relevant for European and UK populations respectively. The RC(UK) Guidelines are peer reviewed by the Executive Committee of the RC(UK),

which comprises 24 individuals and includes lay representation and representation of the key stakeholder groups.

These latest guidelines contain some treatment recommendations and changes in practice based on new scientific evidence that has accrued since 2005. Consistency in practice among countries provides the basis for the large trials necessary to establish best practice, and the further development of such international collaboration is encouraged. Similarly, consistent collection and reporting of audit data in registries that enable comparison between systems does much to improve practice and ensure that the victims of sudden cardiac arrest are given the best chance of successful resuscitation. These current guidelines reflect improvements in practice resulting from research and audit, encouraged by the co-operation that exists within the international resuscitation community.

The adult basic life support algorithm is unchanged from 2005. The adult advanced algorithms and paediatric resuscitation algorithms have been updated to reflect changes in the guidelines. The changes are relatively minor and every effort has been made to keep the algorithms simple, yet applicable to cardiac arrest victims in most circumstances.

Rescuers begin CPR if the victim is unconscious or unresponsive and not breathing normally (ignoring occasional gasps). A single compression-ventilation (CV) ratio of 30:2 is used by the single rescuer of an adult or child (excluding newborn) out of hospital, and for all adult CPR. This single ratio is designed to simplify teaching, promote skill retention, increase the number of compressions given, and decrease interruption to compressions. Once a defibrillator is attached, if a shockable rhythm is confirmed, a single shock is delivered. Irrespective of the resultant rhythm, chest compressions and ventilations (2 min with a CV ratio of 30:2) are resumed immediately after the shock to minimise the 'no-flow' time.

Several studies indicate that unnecessary interruptions to chest compressions occur frequently both in and out of hospital. Resuscitation instructors must emphasise the importance of minimising interruptions to chest compression.

Several of the treatment recommendations in these guidelines represent significant changes in the way resuscitation is delivered. It will take time for courses and training materials to be updated and for this change in practice to be disseminated to healthcare professionals and laypeople by resuscitation trainers. As this transition is made there will inevitably be some variation in practice between individuals and healthcare organisations. Healthcare organisations should implement those components of Guidelines 2010 relevant to them by the end of 2011.

Guidelines 2010 do not define the only way that resuscitation should be achieved; they merely represent a widely accepted view of how resuscitation can be undertaken both safely and effectively. The publication of new treatment recommendations does not imply that current clinical care is either unsafe or ineffective.

The process leading to the publication of the guidelines has entailed considerable work by many individuals over a protracted period. The RC(UK) would like to thank all the individuals and organisations that have contributed to the process and made this publication possible.

All those who undertook systematic reviews and participated in the 2010 Consensus on CPR Science Conference signed and adhered to a strict conflict of interest (COI) policy. The details of this ILCOR COI policy are published elsewhere.⁷ All the individuals contributing to the writing of these guidelines have signed and adhered to the [RC\(UK\) COI Policy](#). The COI declarations of all authors are listed in Appendix 1.

The RC(UK) Guidelines undergo a major revision every 5 years (synchronised with the International Consensus on Cardiopulmonary Resuscitation Science Conferences and new ERC Guidelines) with occasional interim amendments to reflect very important new science. These interim amendments are generally made only if delaying guideline changes until a major revision is thought to put patients at risk. The decision to publish interim 'advisory statements' is made by the ILCOR delegates. Thus, the next major review of these guidelines will be in 2015.

Abbreviations used

The following abbreviations have been used in these guidelines:

h	hour, hours
h ⁻¹	per hour
min	minute, minutes
min ⁻¹	per minute
s	second, seconds
s ⁻¹	per second.

Scope and purpose

The purpose of the Resuscitation Council (UK) guidance is to provide evidence-based interventions that are most likely to prevent cardiac arrest or increase the chances of the successful resuscitation (with full neurological recovery) of an adult, child or newborn baby in cardiac or respiratory arrest.

A cardiac arrest is the ultimate medical emergency – the correct treatment must be given immediately if the patient is to have any chance of surviving. The interventions that contribute to a successful outcome after a cardiac arrest can be conceptualised as a chain – the Chain of Survival.⁸



The key topics addressed by the guidance include:

- what a layperson should do if they witness a sudden collapse and a subsequently unresponsive victim;
- what healthcare professionals should do to prevent cardiac arrest;
- how healthcare professionals should treat cardiac arrest when it occurs;
- how healthcare professionals should provide life support until the patient's heart has been restarted;
- how healthcare professionals should treat the patient whose heart has been restarted (post resuscitation care);
- the process for facilitating decisions about resuscitation with patients, family members and/or carers.

Target audience

Basic life support (BLS) guidance is aimed at potentially all adults, especially those with a duty to respond to a medical emergency, and many children in the UK (i.e., anyone who might undertake training in, or have to perform actual cardiopulmonary resuscitation (CPR)). These people may be called upon at any time to attempt resuscitation on a relative, neighbour, colleague, or fellow traveller. Most of the remaining guidance relating to resuscitation is aimed at all healthcare professionals. The RC(UK) '[Standards for clinical practice and training](#)' document provides clear recommendations on the level of training required by individual groups of healthcare professionals.

Summary of main changes

Adult basic life support

The following changes in the basic life support (BLS) guidelines have been made to reflect the importance placed on chest compression, particularly good quality compressions, and to attempt to reduce the number and duration of pauses during chest compression:

- When obtaining help, ask for an automated external defibrillator (AED), if one is available.
- Compress the chest to a depth of 5-6 cm and at a rate of 100-120 min⁻¹.
- Do not stop to check the victim or discontinue CPR unless the victim starts to show signs of regaining consciousness, such as coughing, opening his eyes, speaking, or moving purposefully AND starts to breathe normally.
- Teach CPR to laypeople with an emphasis on chest compression, but include ventilation as the standard, particularly for those with a duty of care.

The use of Automated External Defibrillators

There are no major changes to the sequence of actions for AED users in Guidelines 2010. The following changes are aimed mainly at increasing the use of AEDs along with clarification on when to stop CPR:

- An AED can be used safely and effectively without previous training; its use should not be restricted to trained rescuers. Training should however be encouraged to help improve the time to shock delivery and correct pad placement
- 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 and AED courses. Such courses should be validated to ensure that they achieve equivalent outcomes to instructor-led courses
- When using an AED minimise interruptions in chest compression. Do not stop to check the victim or discontinue CPR unless the victim starts to show

signs of regaining consciousness, such as coughing, opening his eyes, speaking, or moving purposefully AND starts to breathe normally.

Pre-hospital cardiac arrest

A pre-hospital chapter has been included for the first time in the RC(UK) Guidelines. The chapter covers the following resuscitation topics of specific relevance to the pre-hospital emergency medical services (EMS):

- Telephone-advised cardiopulmonary resuscitation (CPR)
- CPR versus defibrillation first
- Pre-hospital airway management
- Rules for stopping resuscitation.

Prevention of cardiac arrest and decisions about cardiopulmonary resuscitation

Increasing importance is being given to prevention of cardiac arrest. Revisions to this chapter include:

- Recognition of those at risk of sudden cardiac death out of hospital.
- Much more detail on prevention strategies including the use of track-and-trigger systems.
- Emphasis on the use of structured communication e.g., Situation, Background, Assessment, Recommendation (SBAR).
- Reference to the current [Do-Not-Attempt-Resuscitation \(DNAR\) guidelines](#).

In-hospital resuscitation

Many of the changes to the 2010 guidelines on in-hospital resuscitation reflect changes that are described in more detail in the advanced life support (ALS) section. Highlights for in-hospital resuscitation include:

- Much greater emphasis on high-quality CPR with minimal interruptions in chest compressions.
- Defibrillation strategy is described in detail. The aim is to reduce the preshock pause to less than 5 s by planning ahead, compressing during charging, and using a very brief safety check.
- The importance of contributing data to the [National Cardiac Arrest Audit \(NCAA\)](#).

Adult advanced life support

There are several changes to the ALS guidelines and, for simplicity, these are grouped by topic.

Defibrillation

- There is increased emphasis on the importance of minimal interruption in high-quality chest compressions throughout any ALS intervention: chest compressions are paused briefly only to enable specific planned interventions.
- The recommendation for a specified period of CPR before out-of-hospital defibrillation following cardiac arrest unwitnessed by the EMS has been removed.
- Chest compressions are now continued while a defibrillator is charged – this will minimise the preshock pause.
- The role of the precordial thump is de-emphasised.
- There is inclusion of the use of up to three quick successive (stacked) shocks for ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) occurring in the cardiac catheterisation laboratory or in the immediate post-operative period following cardiac surgery.

Drugs

- Delivery of drugs via a tracheal tube is no longer recommended – if intravenous (IV) access cannot be achieved give drugs by the intraosseous (IO) route.
- When treating VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock and then every 3-5 min (during alternate cycles of CPR). Amiodarone 300 mg is also given after the third shock.
- Atropine is no longer recommended for routine use in asystole or pulseless electrical activity.

Airway

- There is reduced emphasis on early tracheal intubation unless achieved by highly skilled individuals with minimal interruption to chest compressions.
- There is increased emphasis on the use of capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of return of spontaneous circulation (ROSC).

Ultrasound

- The potential role of ultrasound imaging during ALS is recognised.

Post-resuscitation care

- There is recognition of the potential harm caused by hyperoxaemia after ROSC is achieved: once ROSC has been established and the oxygen saturation of arterial blood (SaO₂) can be monitored reliably (by pulse oximetry and/or arterial blood gas analysis), inspired oxygen is titrated to achieve a SaO₂ of 94 - 98%.
- There is much greater detail and emphasis on the treatment of the post-cardiac-arrest syndrome.
- There is recognition that implementation of a comprehensive, structured post-resuscitation treatment protocol may improve survival in cardiac arrest victims after ROSC.
- There is increased emphasis on the use of primary percutaneous coronary intervention in appropriate, but comatose, patients with sustained ROSC after cardiac arrest.
- There is revision of the recommendation for glucose control: in adults with sustained ROSC after cardiac arrest, blood glucose values >10 mmol l⁻¹ should be treated but hypoglycaemia must be avoided.
- Use of therapeutic hypothermia to include comatose survivors of cardiac arrest associated initially with non-shockable rhythms as well shockable rhythms. The lower level of evidence for use after cardiac arrest from non-shockable rhythms is acknowledged.
- There is 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.

Peri-arrest arrhythmias

There are relatively few changes from Guidelines 2005 in the treatment of peri-arrest arrhythmias:

- The assessment of patients in the context of peri-arrest arrhythmias uses the ABCDE approach.
- A single set of adverse features for tachy- and brady-arrhythmias has been introduced for consistency.

Paediatric basic life support

Changes in paediatric life support guidelines have been made partly in response to new scientific evidence, and partly to simplify them in order to assist teaching and retention. As in the past, there remains a paucity of good-quality evidence specifically on paediatric resuscitation, and some conclusions have had to be drawn from experimental work or extrapolated from adult data.

- Pulse palpation for 10 s is unreliable for determining the presence or absence of an effective circulation. This means that palpation of the pulse cannot be the sole determinant of the need for chest compressions. Healthcare providers therefore need to determine the presence or absence of 'signs of life', such as response to stimuli, normal breathing (rather than abnormal gasps) or spontaneous movement. They may also feel for a pulse but, if there are no other 'signs of life', they should only withhold CPR if they are certain there is a definite pulse. If the lay person considers that there are no 'signs of life', CPR should be started immediately.
- Although ventilation remains a very important component of CPR in asphyxial arrest, rescuers who are unable or unwilling to provide this should be encouraged to perform at least compression-only CPR. A child is far more likely to be harmed if bystanders do nothing at all.
- Chest compressions are frequently too shallow, so there has been a subtle, but important, change in the instruction on chest compressions from 'approximately one third' to 'at least one third' of the AP diameter of the chest. The mean one-third compression depths for infants and children are 4 and 5 cm respectively. In order to be consistent with the adult BLS guidelines the recommended compression rate is now 100 - 120 min⁻¹.

Paediatric advanced life support

There is concern that resuscitation from cardiac arrest is not performed as well as it might because the variations in guidelines for different age groups cause confusion to providers, and therefore poor performance. As in 2005, most of the changes in paediatric guidelines for 2010 have been made for simplification and to minimise differences between adult and paediatric guidance.

- Adrenaline is given after the third shock for shockable rhythms and then during every alternate cycle (i.e. every 3-5 min during CPR). In the non-shockable side of the algorithm adrenaline is still given initially as soon as vascular access is available.
- Amiodarone is given after the third shock for shockable rhythms. The dose is repeated after the fifth shock, if VF or pulseless VT is still present.
- Bag-mask ventilation remains the preferred method for achieving airway control and ventilation. If this fails, the laryngeal mask airway (or possibly other supraglottic airway device) is an acceptable alternative for suitably trained providers.
- Once spontaneous circulation has been restored, delivered oxygen should be titrated according to SaO₂ to limit the risk of hyperoxaemia.
- CO₂ detection (preferably with capnography) is encouraged even more strongly, not only to confirm placement of tracheal tubes but also to aid decision making during CPR and management of ventilation after ROSC.
- Recommendations for post-resuscitation care include consideration of induced hypothermia.

Newborn life support

The following are the main changes that have been made to the Newborn Life Support (NLS) guidelines in 2010:

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

Introduction

This chapter contains the guidelines for out-of-hospital, single rescuer, adult basic life support (BLS). Like the other guidelines in this publication, it is based on the document *2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations (CoSTR)*, which was published in October 2010. Basic life support refers to maintaining airway patency and supporting breathing and the circulation without the use of equipment other than a protective device.

It is important that those who may be present at the scene of a cardiac arrest, particularly lay bystanders, should have learnt the appropriate resuscitation skills and be able to put them into practice. Simplification of the BLS sequence continues to be a feature of these guidelines, but, in addition, there is now advice on who should be taught what skills, particularly chest-compression-only or chest compression and ventilation. Within this advice, allowance has been made for the rescuer who is unable or unwilling to perform rescue breathing, and for those who are untrained and receive telephone advice from the ambulance service.

Guidelines 2000 introduced the concept of checking for 'signs of a circulation'. This change was made because of the evidence that relying on a check of the carotid pulse to diagnose cardiac arrest is unreliable and time-consuming, mainly, but not exclusively, when attempted by non-healthcare professionals.⁹ Subsequent studies have shown that checking for breathing is also prone to error, particularly as agonal gasps are often misdiagnosed as normal breathing.¹⁰ In Guidelines 2010 the absence of normal breathing continues to be the main sign of cardiac arrest in a non-responsive victim. Once cardiopulmonary resuscitation (CPR) has started, it is now recommended that the rescuer should only stop CPR if the victim shows signs of regaining consciousness, such as coughing, opening his eyes, speaking, or moving purposefully, as well as breathing normally.

Guideline changes

It is well documented that interruptions in chest compression are common^{11, 12} and are associated with a reduced chance of survival.¹³ The 'perfect' solution is to deliver continuous compressions whilst giving ventilations independently. This is possible when the victim has an advanced airway in place, and is discussed in the adult advanced life support (ALS) chapter. Compression-only CPR is another way to increase the number of compressions given and will, by definition, eliminate pauses. It is effective for a

limited period only (probably less than 5 min)¹⁴ and is not recommended as the standard management of out-of-hospital cardiac arrest.

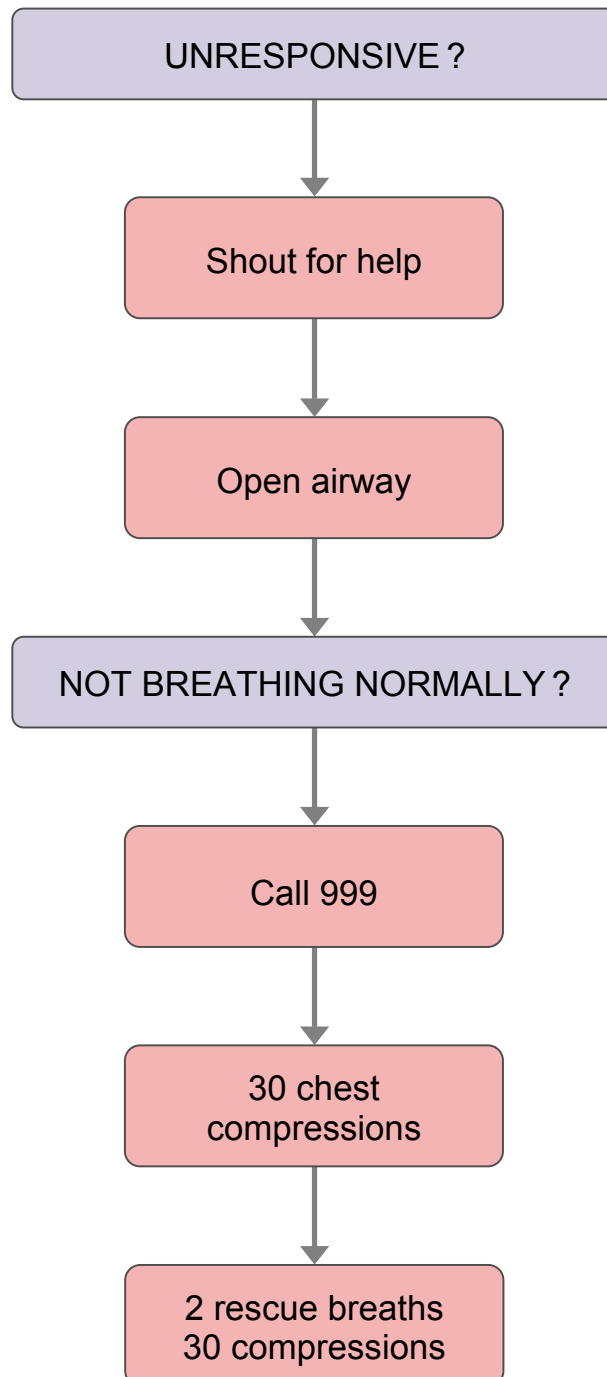
It is also known that chest compressions, both in hospital and outside, are often undertaken with insufficient depth and at the wrong rate.^{12, 15}

The following changes in the BLS guidelines have been made to reflect the importance placed on chest compression, particularly good quality compressions, and to attempt to reduce the number and duration of pauses in chest compression:

1. When obtaining help, ask for an automated external defibrillator (AED), if one is available.
2. Compress the chest to a depth of 5-6 cm and at a rate of 100-120 min⁻¹.
3. Give each rescue breath over 1 s rather than 2 s.
4. Do not stop to check the victim or discontinue CPR unless the victim starts to show signs of regaining consciousness, such as coughing, opening his eyes, speaking, or moving purposefully AND starts to breathe normally.
5. Teach CPR to laypeople with an emphasis on chest compression, but include ventilation as the standard, particularly for those with a duty of care.

In addition, advice has been added on the use of oxygen, and how to manage a victim who regurgitates stomach contents during resuscitation.

Adult basic life support algorithm



Adult basic life support sequence

Basic life support consists of the following sequence of actions:

1. **Make sure the victim, any bystanders, and you are safe.**
2. **Check the victim for a response.**
 - Gently shake his shoulders and ask loudly, 'Are you all right?'
- 3A. **If he responds:**
 - Leave him in the position in which you find him provided there is no further danger.
 - Try to find out what is wrong with him and get help if needed.
 - Reassess him regularly.
- 3B. **If he does not respond:**
 - Shout for help.
 - Turn the victim onto his back and then open the airway using head tilt and chin lift:
 - Place your hand on his forehead and gently tilt his head back.
 - With your fingertips under the point of the victim's chin, lift the chin to open the airway.
4. **Keeping the airway open, look, listen, and feel for normal breathing.**
 - Look for chest movement.
 - Listen at the victim's mouth for breath sounds.
 - Feel for air on your cheek.

In the first few minutes after cardiac arrest, a victim may be barely breathing, or taking infrequent, noisy, gasps. This is often termed agonal breathing and must not be confused with normal breathing.

Look, listen, and feel for **no more** than **10 s** to determine if the victim is breathing normally. If you have any doubt whether breathing is normal, act as if it is **not** normal.

- 5A. **If he is breathing normally:**
 - Turn him into the recovery position (**see below**).
 - Summon help from the ambulance service by mobile phone. If this is not possible, send a bystander. Leave the victim only if no other way of obtaining help is possible.
 - Continue to assess that breathing remains normal. If there is any doubt about the presence of normal breathing, start CPR (5B).

5B. If he is not breathing normally:

- Ask someone to call for an ambulance and bring an AED if available. If you are on your own, use your mobile phone to call for an ambulance. Leave the victim only when no other option exists for getting help.
- Start chest compression as follows:
 - Kneel by the side of the victim.
 - Place the heel of one hand in the centre of the victim's chest (which is the lower half of the victim's sternum (breastbone)).
 - Place the heel of your other hand on top of the first hand.
 - Interlock the fingers of your hands and ensure that pressure is not applied over the victim's ribs. Do not apply any pressure over the upper abdomen or the bottom end of the sternum.
 - Position yourself vertically above the victim's chest and, with your arms straight, press down on the sternum 5 - 6 cm.
 - After each compression, release all the pressure on the chest without losing contact between your hands and the sternum. Repeat at a rate of 100 - 120 min⁻¹.
 - Compression and release should take an equal amount of time.

6A. Combine chest compression with rescue breaths:

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

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

- Check the victim's mouth and remove any visible obstruction.
- Recheck that there is adequate head tilt and chin lift.
- Do not attempt more than two breaths each time before returning to chest compressions.

If there is more than one rescuer present, another should take over CPR about every 1-2 min to prevent fatigue. Ensure the minimum of delay during the changeover of rescuers, and do not interrupt chest compressions.

6B. Compression-only CPR

- If you are not trained to, or are unwilling to give rescue breaths, give chest compressions only.
- If chest compressions only are given, these should be continuous at a rate of 100 - 120 min⁻¹.
- Stop to recheck the victim only if he starts to show signs of regaining consciousness, such as coughing, opening his eyes, speaking, or moving purposefully AND starts to breathe normally; otherwise **do not interrupt resuscitation**.

7. Continue resuscitation until:

- qualified help arrives and takes over,
- the victim starts to show signs of regaining consciousness, such as coughing, opening his eyes, speaking, or moving purposefully AND starts to breathe normally, OR
- you become exhausted.

Further points related to basic life support

Risks to the rescuer and victim

The safety of both the rescuer and victim are paramount during a resuscitation attempt. There have been few incidents of rescuers suffering adverse effects from undertaking CPR, with only isolated reports of infections such as tuberculosis (TB) and severe acute respiratory distress syndrome (SARS). Transmission of HIV during CPR has never been reported.¹⁶

There have been no human studies to address the effectiveness of barrier devices during CPR; however, laboratory studies have shown that certain filters, or barrier devices with one-way valves, prevent transmission of oral bacteria from the victim to the rescuer during mouth-to-mouth ventilation. Rescuers should take appropriate safety precautions where feasible, especially if the victim is known to have a serious infection

such as TB or SARS. During an outbreak of a highly infectious condition (such as SARS), full protective precautions for the rescuer are essential.

Initial rescue breaths

During the first few minutes after non-asphyxial cardiac arrest the blood oxygen content remains high. Therefore, ventilation is less important than chest compression at this time.

It is well recognised that skill acquisition and retention are aided by simplification of the BLS sequence of actions. It is also recognised that rescuers are frequently unwilling to carry out mouth-to-mouth ventilation for a variety of reasons, including fear of infection and distaste for the procedure. For these reasons, and to emphasise the priority of chest compressions, it is recommended that, in adults, CPR should start with chest compressions rather than initial ventilations.

Jaw thrust

The jaw thrust technique is not recommended for lay rescuers because it is difficult to learn and perform. Therefore, the lay rescuer should open the airway using a head-tilt-chin-lift manoeuvre for both injured and non-injured victims.

Agonal gasps

Agonal gasps are present in up to 40% of cardiac arrest victims.¹⁰ Therefore laypeople should be taught to begin CPR if the victim is unconscious (unresponsive) and not breathing normally. It should be emphasised during training that agonal gasps occur commonly in the first few minutes after sudden cardiac arrest; they are an indication for starting CPR immediately and should not be confused with normal breathing.

Use of oxygen during basic life support

There is no evidence that oxygen administration is of benefit during basic life support in the majority of cases of cardiac arrest before healthcare professionals are available with equipment to secure the airway. Its use may lead to interruption in chest compressions, and is not recommended, except in cases of drowning (see below).

Mouth-to-nose ventilation

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

Mouth-to-tracheostomy ventilation

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

Bag-mask ventilation

Considerable practice and skill are required to use a bag and mask for ventilation. The lone rescuer has to be able to open the airway with a jaw thrust whilst simultaneously holding the mask to the victim's face. It is a technique that is appropriate only for lay rescuers who work in highly specialised areas, such as where there is a risk of cyanide poisoning or exposure to other toxic agents. There are other specific circumstances in which non-healthcare providers receive extended training in first aid, which could include training, and retraining, in the use of bag-mask ventilation. The same strict training that applies to healthcare professionals should be followed and the two-person technique is preferable.

Chest compression

In most circumstances it will be possible to identify the correct hand position for chest compression without removing the victim's clothes. If in any doubt, remove outer clothing.

Each time compressions are resumed on an adult, the rescuer should place his hands on the lower half of the sternum. It is recommended that this location be taught in a simple way, such as 'place the heel of your hand in the centre of the chest with the other hand on top.' This teaching should be accompanied by a demonstration of placing the hands on the lower half of the sternum.¹⁷ Use of the inter nipple line as a landmark for hand placement is not reliable.

Performing chest compression:

- a. Compress the chest at a rate of 100-120 min⁻¹.
- b. Each time compressions are resumed, place your hands without delay 'in the centre of the chest' (see above).
- c. Pay attention to achieving the full compression depth of 5-6 cm (for an adult).
- d. Allow the chest to recoil completely after each compression.
- e. Take approximately the same amount of time for compression and relaxation.
- f. Minimise interruptions in chest compression.
- g. Do not rely on a palpable carotid or femoral pulse as a gauge of effective arterial flow.
- h. 'Compression rate' refers to the speed at which compressions are given, not the total number delivered in each minute. The number delivered is

determined not only by the rate, but also by the number of interruptions to open the airway, deliver rescue breaths, and allow AED analysis.

Compression-only CPR

Studies have shown that compression-only CPR may be as effective as combined ventilation and compression in the first few minutes after non-asphyxial arrest. However, chest compression combined with rescue breaths is the method of choice for CPR by trained lay rescuers and professionals and should be the basis for lay-rescuer education. Lay rescuers who are unable or unwilling to provide rescue breaths, should be encouraged to give chest compressions alone. When advising untrained laypeople by telephone, ambulance dispatchers should give instruction on compression-only CPR.^{18, 19, 19a}

Regurgitation during CPR

Regurgitation of stomach contents is common during CPR, particularly in victims of drowning. If regurgitation occurs:

- Turn the victim away from you.
- Keep him on his side and prevent him from toppling on to his front.
- Ensure that his head is turned towards the floor and his mouth is open and at the lowest point, thus allowing vomit to drain away.
- Clear any residual debris from his mouth with your fingers; and immediately turn him on to his back, re-establish an airway, and continue rescue breathing and chest compressions at the recommended rate.

Teaching CPR

Compression-only CPR has potential advantages over chest compression and ventilation, particularly when the rescuer is an untrained or partially-trained layperson. However, there are situations where combining chest compressions with ventilation is better, for example in children, asphyxial arrests, and prolonged arrests.^{20, 21} Therefore, CPR should remain standard care for healthcare professionals and the preferred target for laypeople, the emphasis always being on minimal interruption in compressions.

A simple, education-based approach is recommended:

- Ideally, full CPR skills should be taught to all citizens.
- Initial or limited-time training should always include chest compression.
- Subsequent training (which may follow immediately or at a later date) should include ventilation as well as chest compression.

CPR training for citizens should be promoted, but untrained lay people should be encouraged to give chest compressions only, when possible and appropriate with telephone advice from an ambulance dispatcher.

Those laypeople with a duty of care, such as first aid workers, lifeguards, and child minders, should be taught chest compression and ventilation.

Over-the-head CPR

Over-the-head CPR for a single rescuer and straddle CPR for two rescuers may be considered for resuscitation in confined spaces.

Recovery position

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

The RC(UK) recommends the following sequence of actions to place a victim in the recovery position:

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

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

Choking

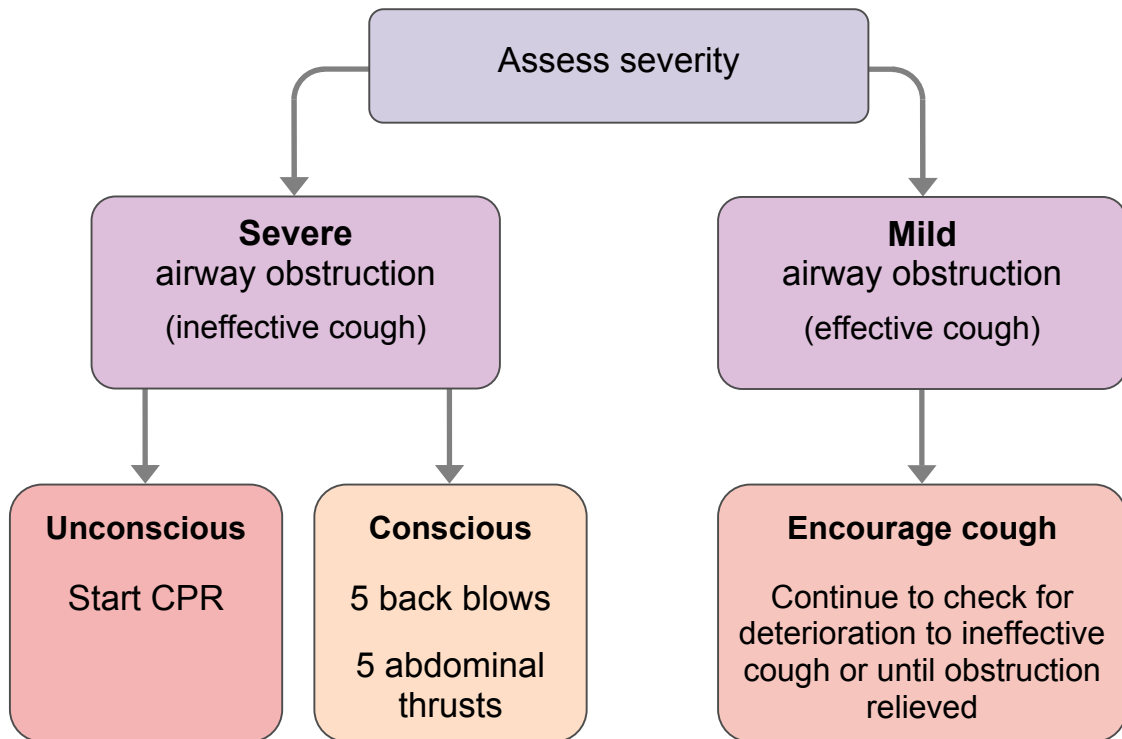
Recognition

Because recognition of choking (airway obstruction by a foreign body) is the key to successful outcome, it is important not to confuse this emergency with fainting, heart attack, seizure, or other conditions that may cause sudden respiratory distress, cyanosis, or loss of consciousness.

Foreign bodies may cause either mild or severe airway obstruction. The signs and symptoms enabling differentiation between mild and severe airway obstruction are summarised in the table below. It is important to ask the conscious victim 'Are you choking?'

General signs of choking	
<ul style="list-style-type: none"> • Attack occurs while eating • Victim may clutch his neck 	
Signs of severe airway obstruction	Signs of mild airway obstruction
<p><i>Response to question 'Are you choking?'</i></p> <ul style="list-style-type: none"> • Victim unable to speak • Victim may respond by nodding <p><i>Other signs</i></p> <ul style="list-style-type: none"> • Victim unable to breathe • Breathing sounds wheezy • Attempts at coughing are silent • Victim may be unconscious 	<p><i>Response to question 'Are you choking?'</i></p> <ul style="list-style-type: none"> • Victim speaks and answers yes <p><i>Other signs</i></p> <ul style="list-style-type: none"> • Victim is able to speak, cough, and breathe

Adult choking treatment algorithm



Sequence for the treatment of adult choking

(This sequence is also suitable for use in children over the age of 1 year)

- 1. If the victim shows signs of mild airway obstruction:**
 - Encourage him to continue coughing, but do nothing else.
- 2. If the victim shows signs of severe airway obstruction and is conscious:**
 - Give up to five back blows.
 - Stand to the side and slightly behind the victim.
 - Support the chest with one hand and lean the victim well forwards so that when the obstructing object is dislodged it comes out of the mouth rather than goes further down the airway.
 - Give **up to** five sharp blows between the shoulder blades with the heel of your other hand.

- Check to see if each back blow has relieved the airway obstruction. The aim is to relieve the obstruction with each blow rather than necessarily to give all five.
- If five back blows fail to relieve the airway obstruction give up to five abdominal thrusts.
 - Stand behind the victim and put both arms round the upper part of his abdomen.
 - Lean the victim forwards.
 - Clench your fist and place it between the umbilicus (navel) and the bottom end of the sternum (breastbone).
 - Grasp this hand with your other hand and pull sharply inwards and upwards.
 - Repeat up to five times.
- If the obstruction is still not relieved, continue alternating five back blows with five abdominal thrusts.

3. If the victim becomes unconscious:

- Support the victim carefully to the ground.
- Call an ambulance immediately.
- Begin CPR (from 5B of the adult BLS sequence). Healthcare providers, trained and experienced in feeling for a carotid pulse, should initiate chest compressions even if a pulse is present in the unconscious choking victim.

Following successful treatment for choking, foreign material may nevertheless remain in the upper or lower respiratory tract and cause complications later. Victims with a persistent cough, difficulty swallowing, or with the sensation of an object being still stuck in the throat should therefore be referred for an immediate medical opinion.

Resuscitation of children and victims of drowning

Both ventilation and compression are important for victims of cardiac arrest when the oxygen stores become depleted: about 2 - 4 min after collapse from ventricular fibrillation (VF), and immediately after collapse for victims of asphyxial arrest. Previous guidelines tried to take into account the difference in causation, and recommended that victims of identifiable asphyxia (drowning; trauma; intoxication) and children should receive 1 min of CPR before the lone rescuer left the victim to get help. But most cases of sudden cardiac arrest out of hospital occur in adults and are of cardiac origin due to VF (even though many of these will have changed to a non-shockable rhythm by the time of the first rhythm analysis). These additional recommendations, therefore, added to the complexity of the guidelines whilst applying to only a minority of victims.

Many children do not receive resuscitation because potential rescuers fear causing harm. This fear is unfounded; it is far better to use the adult BLS sequence for resuscitation of a child than to do nothing. For ease of teaching and retention, laypeople

should be taught to use the adult sequence for children who are not responsive and not breathing normally, with the single modification that the chest should be compressed by one third of its depth. However, the following minor modifications to the adult sequence will make it even more suitable for use in children:

- Give 5 initial rescue breaths before starting chest compressions (adult BLS sequence of actions 5B).
- If you are on your own, perform CPR for 1 min before going for help.
- Compress the chest by one third of its depth. Use two fingers for an infant under 1 year; use one or two hands for a child over 1 year as needed to achieve an adequate depth of compression.

The same modifications of five initial breaths, and 1 min of CPR by the lone rescuer before getting help, may improve outcome for victims of drowning. This modification should be taught only to those who have a specific duty of care to potential drowning victims (e.g. lifeguards). If supplemental oxygen is available, and can be brought to the victim and used without interruption in CPR (e.g., by attaching to a resuscitation face mask), it may be of benefit.

Drowning is easily identified. It can be difficult, on the other hand, for a layperson to recognise when trauma or intoxication has caused cardiorespiratory arrest. If either cause is suspected the victim should be managed according to the standard BLS protocol.

Introduction

This chapter contains guidelines for the use of automated external defibrillators (AEDs) by laypeople, first responders and healthcare professionals responding with an AED outside hospital. These guidelines are appropriate for all types of AED, including those that are fully automatic. Guidelines for in-hospital use of AEDs are provided in the electrical therapies section of the advanced life support guidelines.

In the UK approximately 30,000 people sustain cardiac arrest outside hospital and are treated by emergency medical services (EMS) each year.²² Electrical defibrillation is well established as the only effective therapy for cardiac arrest caused by ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT). The scientific evidence to support early defibrillation is overwhelming; the delay from collapse to delivery of the first shock is the single most important determinant of survival. If defibrillation is delivered promptly, survival rates as high as 75% have been reported.^{23, 24} The chances of successful defibrillation decline at a rate of about 10% with each minute of delay;²⁵ basic life support will help to maintain a shockable rhythm but is not a definitive treatment.

The Resuscitation Council (UK) recommends strongly a policy of attempting defibrillation with the minimum of delay in victims of VF/VT cardiac arrest.

Guideline changes

There are no major changes to the sequence of actions for AED users in Guidelines 2010. The ILCOR Consensus on Science and Treatment Recommendations²⁶ makes the following recommendations which are relevant to the RC(UK) AED guidelines:

1. An AED can be used safely and effectively without previous training. Therefore, the use of an AED should not be restricted to trained rescuers. However, training should be encouraged to help improve the time to shock delivery and correct pad placement.
2. 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 and AED courses. Such courses should be validated to ensure that they achieve equivalent outcomes to instructor led courses.²⁶

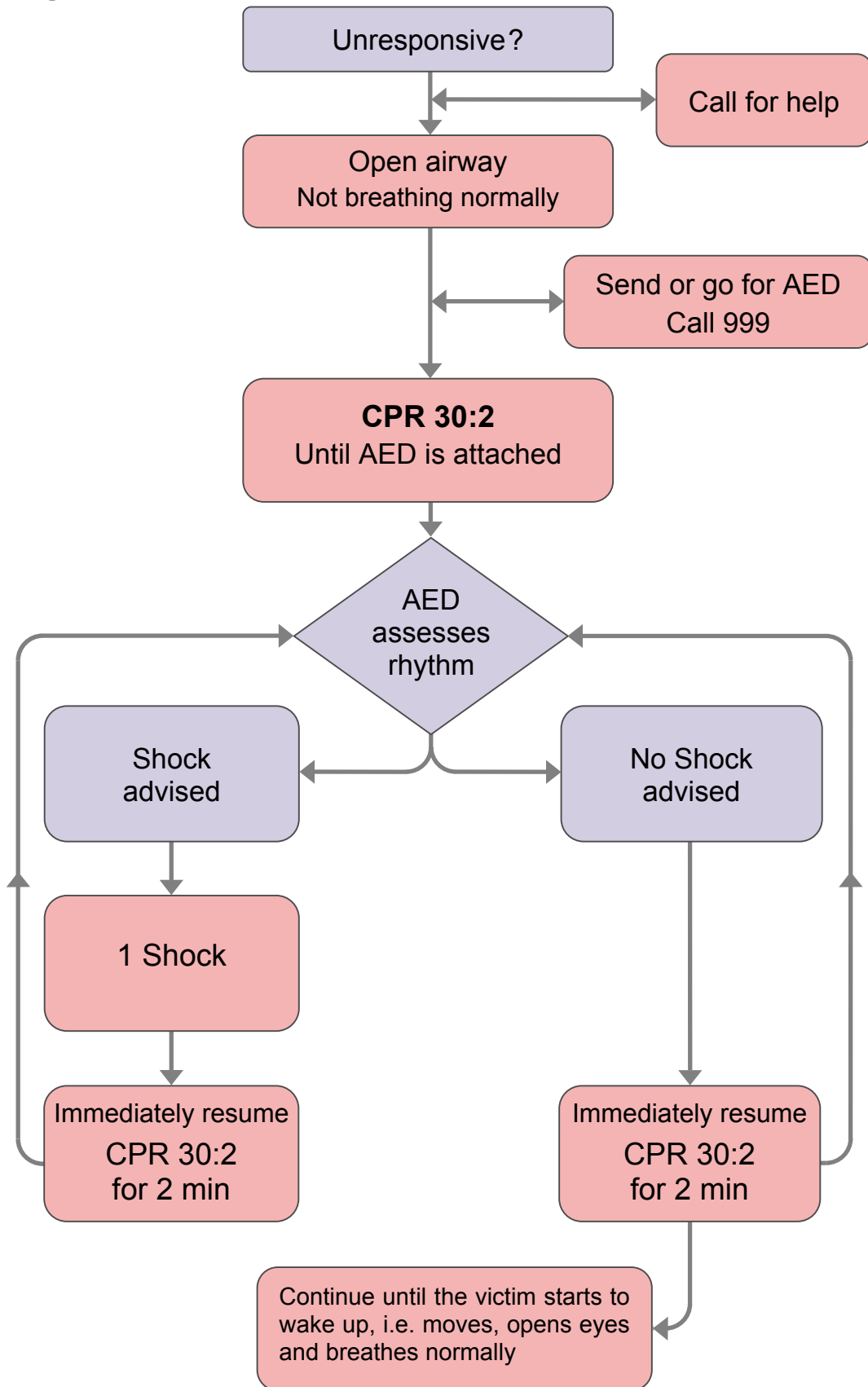
3. When using an AED minimise interruptions in chest compression. Do not stop to check the victim or discontinue cardiopulmonary resuscitation (CPR) unless the victim starts to show signs of regaining consciousness, such as coughing, opening his eyes, speaking, or moving purposefully AND starts to breathe normally.

Types of automated external defibrillator

AEDs are sophisticated, reliable, safe, computerised devices that deliver electric shocks to victims of cardiac arrest when the ECG rhythm is one that is likely to respond to a shock. Simplicity of operation is a key feature: controls are kept to a minimum, voice and visual prompts guide rescuers. Modern AEDs are suitable for use by both lay rescuers and healthcare professionals.

All AEDs analyse the victim's ECG rhythm and determine the need for a shock. The semi-automatic AED indicates the need for a shock, which is delivered by the operator, while the fully automatic AED administers the shock without the need for intervention by the operator. Some semi-automatic AEDs have the facility to enable the operator (normally a healthcare professional) to override the device and deliver a shock manually, independently of prompts.

AED algorithm



Sequence of actions when using an automated external defibrillator

The following sequence applies to the use of both semi-automatic and automatic AEDs in a victim who is found to be unconscious and not breathing normally.

1. **Follow the adult BLS sequence as described in the basic life support chapter. Do not delay starting CPR unless the AED is available immediately.**
2. **As soon as the AED arrives:**
 - If more than one rescuer is present, continue CPR while the AED is switched on. If you are alone, stop CPR and switch on the AED.
 - Follow the voice / visual prompts.
 - Attach the electrode pads to the patient's bare chest.
 - Ensure that nobody touches the victim while the AED is analysing the rhythm.
- 3A. **If a shock is indicated:**
 - Ensure that nobody touches the victim.
 - Push the shock button as directed (fully-automatic AEDs will deliver the shock automatically).
 - Continue as directed by the voice / visual prompts.
 - Minimise, as far as possible, interruptions in chest compression.
- 3B. **If no shock is indicated:**
 - Resume CPR immediately using a ratio of 30 compressions to 2 rescue breaths.
 - Continue as directed by the voice / visual prompts.
4. **Continue to follow the AED prompts until:**
 - qualified help arrives and takes over OR
 - the victim starts to show signs of regaining consciousness, such as coughing, opening his eyes, speaking, or moving purposefully AND starts to breathe normally OR
 - you become exhausted.

Placement of AED pads

Place one AED pad to the right of the sternum (breast bone), below the clavicle (collar bone). Place the other pad in the left mid-axillary line, approximately over the position of the V6 ECG electrode. It is important that this pad is placed sufficiently laterally and that it is clear of any breast tissue.

Although most AED pads are labelled left and right, or carry a picture of their correct placement, it does not matter if their positions are reversed. It is important to teach that if this happens 'in error', the pads should not be removed and replaced because this wastes time and they may not adhere adequately when re-attached.

The victim's chest must be sufficiently exposed to enable correct pad placement. Chest hair will prevent the pads adhering to the skin and will interfere with electrical contact. Shave the chest only if the hair is excessive, and even then spend as little time as possible on this. Do not delay defibrillation if a razor is not immediately available.

Defibrillation if the victim is wet

As long as there is no direct contact between the user and the victim when the shock is delivered, there is no direct pathway that the electricity can take that would cause the user to experience a shock. Dry the victim's chest so that the adhesive AED pads will stick and take particular care to ensure that no one is touching the victim when a shock is delivered.

Defibrillation in the presence of supplemental oxygen

There are no reports of fires caused by sparking where defibrillation was delivered using adhesive pads. If supplemental oxygen is being delivered by a face mask, remove the face mask and place it at least one metre away before delivering a shock. Do not allow this to delay shock delivery.

Minimise interruptions in CPR

The importance of early, uninterrupted chest compressions is emphasised throughout these guidelines. Interrupt CPR only when it is necessary to analyse the rhythm and deliver a shock. When two rescuers are present, the rescuer operating the AED applies the electrodes while the other continues CPR. The AED operator delivers a shock as soon as the shock is advised, ensuring that no one is in contact with the victim.

CPR before defibrillation

Provide good quality CPR while the AED is brought to the scene. Continue CPR whilst the AED is turned on, then follow the voice and visual prompts. Giving a specified period of CPR, as a routine before rhythm analysis and shock delivery, is not recommended.

Voice prompts

The sequence of actions and voice prompts provided by an AED are usually programmable and it is recommended that they be set as follows:

- deliver a single shock when a suitable rhythm is detected;
- no rhythm analysis immediately after the shock;
- a voice prompt for resumption of CPR immediately after the shock;
- a period of 2 min of CPR before further rhythm analysis.

AED use by healthcare professionals

All healthcare professionals should consider the use of an AED to be an integral component of BLS. Early defibrillation should be available throughout all hospitals, outpatient medical facilities and clinics. Sufficient staff should be trained to enable a first shock to be provided within 3 min of collapse anywhere in the hospital. Hospitals should monitor collapse-to-first-shock intervals and monitor resuscitation outcomes.

The RC(UK) advises that untrained employees working in healthcare establishments not be prevented from using an AED if they are confronted with a patient in cardiac arrest. The administration of a defibrillatory shock should not be delayed while waiting for more highly trained personnel to arrive. The same principle should apply to individuals whose certified period of qualification has expired.

Further information on AED use by healthcare professionals is provided in the in-hospital cardiac arrest chapter of these guidelines.

Storage and use of AEDs

AEDs should be stored in locations that are immediately accessible to rescuers; they should not be stored in locked cabinets as this may delay deployment. Use of the [UK standardised AED sign](#) is encouraged, to highlight the location of an AED. People with no previous training have used AEDs safely and effectively. While it is highly desirable that those who may be called upon to use an AED should be trained in their use, and keep their skills up to date, circumstances can dictate that no trained operator (or a trained operator whose certificate of training has expired) is present at the site of an emergency. Under these circumstances no inhibitions should be placed on any person willing to use an AED.

Children

Standard AED pads are suitable for use in children older than 8 years. Special paediatric pads, that attenuate the current delivered during defibrillation, should be used in children aged between 1 and 8 years if they are available; if not, standard adult-sized pads should be used. The use of an AED is not recommended in children aged less than 1 year. However, if an AED is the only defibrillator available its use should be considered (preferably with the paediatric pads described above).

Public access defibrillation (PAD)

Public access defibrillation is the term used to describe the use of AEDs by laypeople.²⁷ Two basic strategies are used. In the first, AEDs are installed in public places and used by people working nearby. Impressive results have been reported with survival rates as high as 74% with fast response times often possible when an AED is nearby.²³

In a complementary strategy, first responders are dispatched by an ambulance control centre when they might reach a patient more quickly than a conventional ambulance. The greater delay in defibrillation resulting from the need for such responders to travel to a patient has been associated with more modest success rates. However, this strategy does enable treatment of people who arrest at home, the commonest place for cardiac arrest to occur.

Further information may be found on the [RC\(UK\) web site](#).

Introduction

A pre-hospital chapter has been included for the first time in the Resuscitation Council (UK) Guidelines. The aim is to bring together those resuscitation topics of specific relevance to the pre-hospital emergency medical services. These topics comprise:

- telephone-advised cardiopulmonary resuscitation (CPR);
- CPR versus defibrillation first;
- pre-hospital airway management;
- rules for stopping resuscitation.

Telephone-advised CPR

Telephone-advised CPR has been included in the 2010 RC(UK) Guidelines because:

- there is widespread use of telephone triage systems that include advice for a rescuer attending a cardiac arrest victim;
- the wide availability of mobile phones makes it likely that there will be a phone available at the site where the victim has collapsed;
- there is robust research examining best practice of both the diagnosis of cardiac arrest by telephone and also the content and delivery of subsequent instructions provided to rescuers;^{18, 19, 28}
- in adults, telephone-advised compression-only CPR produces better survival rates than telephone-advised conventional CPR (chest compressions and mouth-to-mouth ventilation);^{19a}
- the time to first chest compression can be reduced significantly if a lay bystander can deliver compressions while waiting for professionals to arrive.

Telephone triage guidelines

Telephone triage is used throughout the UK to grade the urgency of emergency calls to the emergency medical services (EMS) and is becoming an integral part of the chain of survival for victims of cardiac arrest. As part of the call, if appropriate, call handlers will offer BLS instructions to the caller.²⁹⁻³¹ After out-of-hospital cardiac arrest, help from the EMS will be accessed by telephone and because of the widespread use of mobile phones it is common for a phone to be available at the point where resuscitation is

taking place. The opportunity to provide instructions by phone on how to give BLS enables the time to the first cardiac compression to be reduced dramatically, compared with waiting for the arrival of the EMS. The shorter the time until cardiac compressions are commenced, the higher the survival rate. However, significant delays in giving advice over the phone and/or poor quality CPR will limit the benefits.³¹

Standardised advice to bystanders should increase the chance that the cardiac arrest will be diagnosed and treated correctly. Further research on this topic will help to improve outcome.

Telephone triage systems

In the UK, call handlers who answer 999 calls generally have no background medical training apart from that provided when they were trained to use the system. They read the triage questions from a screen and the deviation allowed from the precise wording in either the question or the advice supplied, varies from supplier to supplier.

Wording

The wording of both the questions asked and the instructions offered must be understood not only by the caller, but also by the call handler who is relaying the questions and advice from a screen. Instructions must be brief, clear and memorable. Medical jargon must be avoided. The call handler is permitted some flexibility in wording to clarify the meaning to the caller but too much flexibility may give rise to ambiguity and lengthen the call.

Diagnosis

The diagnosis of cardiac arrest may be difficult over the telephone. Palpation of the carotid pulse by laypeople is unreliable for the diagnosis of cardiac arrest.³² Absence of breathing can be a better indicator of cardiorespiratory arrest, but many cardiac arrest victims gasp initially (agonal breathing) and this is often misinterpreted by the lay rescuer as breathing.¹⁰ Consequently, the call handler should ask if the victim is “breathing normally” instead of simply “breathing”. A few cardiac arrest victims will display some seizure activity. Seizure activity as a feature of cardiac arrest can cause confusion and delay the correct diagnosis. Asking whether the patient is a known epileptic may help to reduce the risk of patients with epilepsy receiving bystander CPR inadvertently.³³

These challenges may be compounded by a caller (rescuer) who is very distressed, may not have any support, and who may fear providing medical intervention for reasons unknown to the dispatcher.

Education of call handlers

Call handlers must be trained in the management of cardiac arrest and choking in all age groups, so that they can appreciate the instructions that they are giving. Giving the call handlers background information about the decay in survival by delaying BLS will make them aware of the urgency of the instructions. They also need to be trained to handle very distressed callers.

Telephone-advised compression-only CPR

When EMS response times are short (less than 5 min), there is some evidence that compression-only CPR produces at least equivalent outcomes to conventional CPR (chest compressions and mouth-to-mouth ventilation).¹⁴ In adults, telephone-advised compression-only cardiopulmonary resuscitation (CPR) produces better survival rates than conventional CPR.^{19, 19a, 28, 30} Rescuers may be more willing to give resuscitation if they do not have to provide ventilation.³⁴ In children 70% of out-of-hospital cardiac arrests are asphyxial in origin and survival rates are better if they are provided with both chest compressions and ventilations.²¹ However, after cardiac arrest from a primary cardiac cause, even in children there is no difference in survival after compression-only or conventional CPR – either technique produces better survival rates than no CPR.²¹ Telephone-advised CPR guidelines should provide instruction in compression-only CPR for both adults and children because it is quicker and easier to describe.³⁵ A rescuer receiving telephone advice is unlikely to provide effective ventilation and this would simply delay effective chest compressions.

CPR versus defibrillation first

Several studies have examined whether a period of CPR before defibrillation is beneficial, particularly in patients with an unwitnessed arrest or prolonged collapse without resuscitation. A review of evidence for the 2005 guidelines resulted in the recommendation that it was reasonable for EMS personnel to give a period of about 2 min of CPR (i.e. about five cycles at 30:2) before defibrillation in patients with prolonged collapse (> 5 min).³⁶ This recommendation was based on clinical studies in which response times exceeded 4-5 min and in which a period of 1.5 to 3 min of CPR by paramedics or EMS physicians before shock delivery improved return of spontaneous circulation (ROSC), survival to hospital discharge^{37, 38} and one-year survival³⁸ for adults with out-of-hospital VF/VT compared with immediate defibrillation.

In contrast, in two randomised controlled trials, a period of 1.5 to 3 min of CPR by EMS personnel before defibrillation did not improve ROSC or survival to hospital discharge in patients with out-of-hospital VF/VT, regardless of EMS response interval.^{39, 40} Four other studies have also failed to demonstrate significant improvements in overall ROSC or survival to hospital discharge with an initial period of CPR,^{37, 38, 41, 42} although one did show a higher rate of favourable neurological outcome.⁴¹

The duration of collapse is frequently difficult to estimate accurately and there is evidence that performing chest compressions while fetching and charging a defibrillator improves the probability of survival.⁴³ For these reasons, in any cardiac arrest that they have not witnessed, EMS personnel should provide good-quality CPR while a defibrillator is fetched, applied and charged, but routine delivery of a specified period of CPR (e.g. 2 or 3 min) before rhythm analysis and shock delivery is no longer recommended.

Pre-hospital airway management

There is insufficient evidence to support or refute the use of any specific technique to maintain an airway and provide ventilation in adults with pre-hospital or in-hospital cardiac arrest. Tracheal intubation has been perceived as the optimal method of providing and maintaining a clear and secure airway during cardiac arrest but data are accumulating on the problems associated with pre-hospital intubation. It is now strongly recommended that tracheal intubation should be used only when trained personnel are available to carry out the procedure with a high level of skill and confidence. In the absence of experienced personnel the use of supraglottic airway devices (SADs) during CPR is probably more rational. However, there are only poor-quality data on the pre-hospital use of these devices during cardiac arrest. The use of SADs is discussed in more detail in the advanced life support (ALS) chapter.

Tracheal intubation

The perceived advantages of tracheal intubation over bag-mask ventilation include: enabling ventilation without interrupting chest compressions,⁴⁴ enabling effective ventilation (particularly when lung and/or chest compliance is poor), minimising gastric inflation and therefore the risk of regurgitation, protection against pulmonary aspiration of gastric contents, and the potential to free a rescuer's hands for other tasks.

Use of the bag-mask is more likely to cause gastric distension, which, theoretically, is more likely to cause regurgitation and aspiration. However, there are no reliable data to indicate that the incidence of aspiration is any higher in cardiac arrest patients ventilated using a bag-mask compared with those ventilated via a tracheal tube.

The disadvantages of tracheal intubation over bag-valve-mask ventilation include:

- The risk of an unrecognised misplaced tracheal tube – in patients with out-of-hospital cardiac arrest, the documented incidence ranges from 0.5% to 17% (emergency physicians 0.5%;⁴⁵ paramedics 2.4%,⁴⁶ 6%,^{47, 48} 9%,⁴⁹ 17%,⁵⁰).
- A prolonged period without chest compressions while intubation is attempted: in a study of pre-hospital intubation by paramedics during 100 cardiac arrests, the total duration of the interruptions in CPR associated with tracheal intubation attempts was 110 s and in 25% the interruptions were for more than 3 min.⁵¹

- A comparatively high failure rate: intubation success rates correlate with the experience of the intubator.⁵²

Healthcare personnel who undertake pre-hospital intubation should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills. Rescuers must weigh the risks and benefits of intubation against the need to provide effective chest compressions. The intubation attempt may require some interruption of chest compressions but, once an advanced airway is in place, ventilation will not require interruption of chest compressions. Personnel skilled in advanced airway management should be able to undertake laryngoscopy without stopping chest compressions; a brief pause in chest compressions will be required only as the tube is passed through the vocal cords. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until return of spontaneous circulation.^{53, 54} No intubation attempt should interrupt chest compressions for more than 10 s; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After intubation, confirm correct tube placement and secure the tube adequately.

Confirmation of the correct placement of the tracheal tube

Waveform capnography is the most sensitive and specific way to confirm and monitor continuously the position of a tracheal tube in victims of cardiac arrest and should supplement clinical assessment (auscultation and visualisation of the tube passing between the vocal cords).⁵⁵ Waveform capnography will not discriminate between tracheal and bronchial placement of the tube – careful auscultation is essential. Existing portable monitors make capnographic initial confirmation and continuous monitoring of tracheal tube position feasible in almost all settings where intubation is performed, including out of hospital.

Rules for stopping resuscitation

Following out-of-hospital cardiac arrest, failure of ALS-trained EMS personnel to achieve ROSC at the scene is associated with an extremely low probability of survival. The rare exception, where the transfer to hospital of a patient with ongoing CPR results in long-term good quality survival, is usually associated with special circumstances, such as pre-existing hypothermia or drug overdose. For this reason, attempts have been made to formulate and validate rules for stopping resuscitation that allow EMS personnel to stop the resuscitation attempt and pronounce life extinct without transporting the victim to hospital. One such rule recommends stopping CPR when there is no return of spontaneous circulation, no shocks are administered, and the arrest is not witnessed by EMS personnel.⁵⁶ However, this rule was validated with defibrillation-only emergency medical technicians in Canada and may not apply to an EMS system staffed by paramedics. In the UK, [guidelines from the Joint Royal Colleges Ambulance Service Liaison Committee](#) (2006), advise that ambulance clinicians may stop resuscitation if **all** of the following criteria are met:

- 15 min or more has passed since the onset of collapse.
- No bystander CPR was given before arrival of the ambulance.
- There is no suspicion of drowning, hypothermia, poisoning/overdose, or pregnancy.
- Asystole is present for more than 30 s on the ECG monitor screen.

Pre-hospital resuscitation attempts are also generally discontinued if the rhythm remains asystole despite 20 min of advanced life support (ALS) except in cases of drowning and hypothermia.

Introduction

Prevention of cardiac arrest is the first link in the chain of survival.⁸ This chapter of the Resuscitation Council (UK) guidelines stresses the importance of preventing cardiac arrest in all age groups, and the decision-making process when cardiopulmonary resuscitation (CPR) is inappropriate. This update is based on the European Resuscitation Council Guidelines 2010,⁵⁵ and includes updates based on NICE Clinical Guideline 50,⁵⁷ and the Joint Statement from the British Medical Association (BMA), RC(UK), and the Royal College of Nursing (RCN) on [decisions relating to CPR](#). The recent General Medical Council publication, '[Treatment and care towards the end of life: good practice in decision making](#)', also includes advice on decisions relating to CPR.

Prevention of out-of-hospital cardiac arrest

Recognising cardiac chest pain

Most sudden cardiac death (SCD) victims have a history of cardiac disease and warning symptoms, most commonly chest pain, in the hour before cardiac arrest.⁵⁸ Early recognition of cardiac chest pain and rapid activation of the EMS is vitally important.^{58, 59} When a call to the EMS is made before a victim collapses, arrival of the ambulance is significantly sooner after collapse, and the chance of survival is higher.

Prevention of sudden cardiac death out of hospital

Coronary artery disease is the commonest cause of SCD. Non-ischaemic cardiomyopathy and valvular disease account for some other SCD events. A small percentage of SCDs are caused by inherited abnormalities (e.g. long and short QT syndromes, Brugada syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy) and by congenital heart disease.

In patients with a known diagnosis of cardiac disease, syncope (with or without prodrome – particularly recent or recurrent) is an independent risk factor for increased risk of death.⁵⁵ Apparently healthy children and young adults who suffer SCD may also have symptoms and signs (e.g. syncope/pre-syncope, chest pain, palpitation, heart murmur) that should alert healthcare professionals to seek expert help to prevent cardiac arrest. Features that indicate a high probability of arrhythmic syncope include:

- syncope in the supine position;

- syncope occurring during or after exercise (although syncope after exercise is often vasovagal);
- syncope with no or only brief prodromal symptoms;
- repeated episodes of unexplained syncope;
- syncope in individuals with a family history of sudden death or inherited cardiac condition.

Assessment in a clinic specialising in the care of those at risk for SCD is recommended in family members of young victims of SCD or those with a known cardiac disorder resulting in an increased risk of SCD. Specific and detailed [guidance for the care of individuals with transient loss of consciousness](#) is available from the National Institute for Health and Clinical Excellence (NICE).

Prevention of in-hospital arrest

Rates of survival and complete physiological recovery following in-hospital cardiac arrest are poor in all age groups. For example, fewer than 20% of adult patients having an in-hospital cardiac arrest will survive to go home.⁶⁰ Cardiac arrest is rare in both pregnant women and children, but outcomes after in-hospital arrest are also poor. Prevention of in-hospital cardiac arrest requires staff education, monitoring of patients, recognition of patient deterioration, a system to call for help and an effective response.⁶¹

Adults

Most adult survivors of in-hospital cardiac arrest have a witnessed and monitored ventricular fibrillation (VF) arrest and are defibrillated immediately.⁶⁰ The underlying cause of arrest in this group is usually primary myocardial ischaemia. In comparison, cardiac arrest in patients in unmonitored ward areas is usually a predictable event not caused by primary cardiac disease.⁶² In this group, cardiac arrest often follows a period of slow and progressive physiological deterioration involving unrecognised or inadequately treated hypoxaemia and hypotension.⁶³ The underlying cardiac arrest rhythm is usually asystole or PEA, and the chance of survival to hospital discharge is extremely poor.

Regular monitoring and early, effective treatment of seriously ill patients appear to improve clinical outcomes and prevent some cardiac arrests. Closer attention to patients who sustain a 'false' cardiac arrest (a cardiac arrest call to a patient who does not require basic or advanced life support) may also improve outcome, as up to one third of these patients die during their hospital stay.⁶⁴

Deficiencies in acute care

Analysis of the critical events preceding many adult cardiac arrests demonstrates many significant antecedents, usually related to abnormalities of the airway, breathing, and circulation.⁶³ Additional factors include a failure to use a systematic approach to the

assessment of critically ill patients, poor communication, lack of teamwork, and insufficient use of treatment limitation plans.

Hospital processes may also have significant effects on patient outcome. For example, patients who are discharged from intensive care units (ICU) to general wards at night have an increased risk of in-hospital death compared with those discharged during the day and those discharged to high-dependency units.⁶⁵ Higher nurse-patient staffing ratios are also associated with reduction in cardiac arrest rates, as well as rates of pneumonia, shock, and death. These suggest that adequate patient monitoring and assessment are crucial to preventing adverse outcomes.

Recognition of ‘at-risk’, or critically ill, adult patients

When patients deteriorate, they display common signs that represent failing respiratory, cardiovascular, and nervous systems. This is the basis for monitoring patients’ vital signs. Abnormal physiology is common on general wards,⁶⁶ yet the important physiological observations of sick patients are measured and recorded less frequently than is desirable.⁶² To assist in the early detection of critical illness, every patient should have a documented plan for vital signs monitoring that identifies which variables need to be measured and the frequency of measurement.⁶⁷

In recent years, early warning scores (EWS), or ‘calling-criteria’ have been adopted by many hospitals to assist in the early detection of critical illness.⁶⁸ EWS systems allocate points to routine vital sign measurements on the basis of their deviation from an arbitrarily agreed ‘normal’ range. The weighted score of one or more vital sign observations, or more often the total EWS, is used to alert ward staff or critical care outreach teams to the deteriorating condition of the patient. Systems that incorporate ‘calling-criteria’ activate a response when one or more routinely measured physiological variables reach an extremely abnormal value.

The sensitivity, specificity, and accuracy of EWS or calling-criteria systems to identify sick patients have been validated for death but not for other outcomes such as hospital length of stay, cardiac arrest, or need for higher care.^{69, 70} Several studies have identified abnormalities of heart rate, blood pressure, respiratory rate, and conscious level as possible markers of impending critical events. However, as not all important vital signs are, or can be, recorded continuously in general ward areas, the ability of these systems to predict cardiac arrest remains unconfirmed. Gaps in vital sign data recording are common; the use of EWS, calling-criteria and rapid response systems can increase the completeness of vital sign monitoring.

The clinical response

The medical and nursing response to a patient’s abnormal physiology needs to be both appropriate and speedy, yet this is not always the case.⁷¹ Traditionally, the response to cardiac arrest has been reactive, with a cardiac arrest team attending the patient after the cardiac arrest. The use of such teams appears to improve survival in circumstances where no coordinated cardiac arrest response previously existed. However, their impact

in other settings is questionable. For example, in one study only patients who had return of spontaneous circulation before the cardiac arrest team arrived were alive at hospital discharge.⁷² In some hospitals the role of the cardiac arrest team has been changed to include that of a medical emergency team (MET). This team responds not only to cardiac arrests, but also to patients with acute physiological deterioration. The MET usually comprises medical and nursing staff from intensive care and general medicine and responds to specific calling criteria. MET interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids.

The results of research into the benefits of introducing a MET are variable.⁷³ Studies with historical control groups show a reduction in cardiac arrests, deaths and unanticipated intensive care unit admissions, improved detection of medical errors, treatment-limitation decisions, and reduced postoperative ward deaths. A cluster-randomised controlled trial of the MET system demonstrated that the introduction of a MET increased the calling incidence for the team, but did not reduce the incidence of cardiac arrest, unexpected death, or unplanned ICU admission.⁷⁴

In the UK, a system of pre-emptive ward care, based predominantly on individual or teams of nurses known as critical care outreach, has developed.⁷⁵ Although the data on the effects of outreach care are also inconclusive, it has been suggested that outreach teams may reduce ward deaths, postoperative adverse events, ICU admissions and readmissions, and increase survival.^{76, 77}

The role of education in cardiac arrest prevention

The recognition that many cardiac arrests may be preventable has led to the development of postgraduate courses specifically designed to prevent physiological deterioration, critical illness, and cardiac arrest (e.g. Acute Life Threatening Events – Recognition and Treatment: ALERT).⁷⁸ Early evidence suggests that they can improve knowledge and change attitudes about acute care. The Immediate Life Support and Advanced Life Support Courses also include sections related to this important topic and appear to be effective at reducing arrests.⁷⁹ Other courses focus on managing sick patients in the first 24 hours of critical illness when more direct critical care expertise is not available immediately. It is recognised that training in acute and critical care should commence early, and many countries have established curricula for inclusion in undergraduate medical education programmes. Simulation is also being used increasingly to train staff in the prevention of patient deterioration.

Pregnant patients

The latest report of the triennial Confidential Enquiry into Maternal and Child Health (CEMACH) makes several recommendations to prevent deaths associated with pregnancy, including the need for hospitals to implement, audit, and regularly update multidisciplinary guidelines for the management of women at risk of, or who develop, complications in pregnancy.⁸⁰ It also recommends that clinical protocols and local referral pathways, including patient transfer, should be developed for pregnant women with pre-existing medical conditions, a history of psychiatric illness, and serious

complications of pregnancy (sepsis, pre-eclampsia and eclampsia, obstetric haemorrhage). Maternity teams should be trained to recognise and manage medical emergencies, and to demonstrate their competency in scenario-based training using simulation. CEMACH has recommended the use of obstetric early warning scoring systems for all obstetric admissions in all clinical settings. Outreach services for maternity have also been described elsewhere.⁸¹

Children

In children, cardiopulmonary arrest is more often caused by profound hypoxaemia and hypotension than primary cardiac disease. Ventricular fibrillation is less common than asystole or pulseless electrical activity. As with adults, there may be opportunities to introduce strategies that will prevent arrest.

There is already evidence of marked, often untreated, abnormalities of common vital signs in the 24 hours prior to the admission of children to an ICU, similar to those reported in adults.⁸² Recognition of the seriously ill child relies on determination of the normal and abnormal age-related values for vital signs, and reassessing them in the context of the progression of the child's condition. As in adults, serial measurement of heart rate, respiratory rate, temperature, blood pressure, and conscious level, particularly following any clinical intervention, must be performed and acted upon. Intervention at an early stage in an unwell child reduces significantly the risk of developing irreversible shock. Systemic blood pressure decreases at a late stage in shock in the child compared with the adult, and should not be used as the sole determinant of whether or not treatment is required.

Paediatric emergency teams, responding to early warning scores, have been established in some hospitals and appear to reduce the incidence of cardiac arrest.^{83, 84}

Resuscitation decisions

Cardiopulmonary resuscitation was originally developed to save the lives of people dying unexpectedly – 'hearts too young to die'. In-hospital death now often involves attempted CPR, even when the underlying condition and general health of the patient makes success unlikely. However, even when there is clear evidence that cardiac arrest or death are likely, ward staff rarely make decisions about the patient's resuscitation status. Improved knowledge, training, and do-not-attempt-resuscitation (DNAR) decision-making should improve patient care and prevent futile CPR attempts. Patients for whom CPR will not prolong life, but may merely prolong the dying process, should be identified early. Medical emergency teams may have an important role in improving end-of-life and DNAR decision-making.⁸⁵⁻⁸⁷

A DNAR decision should be considered when the patient:

- does not wish to have CPR, or
- will not survive cardiac arrest even if CPR is attempted.

The decision-making process should be based on current guidance from the BMA, RC(UK) and RCN. [A standardised form](#) should be used to record and communicate DNAR decisions. A DNAR decision (also referred to as DNACPR) refers specifically to CPR and not to other treatment.

Recommended strategies for the prevention of avoidable in-hospital cardiac arrests

1. Place critically ill patients, or those at risk of clinical deterioration, in areas where the level of care is matched to the level of patient sickness.
2. Monitor regularly such patients using simple vital sign observations (e.g. pulse, blood pressure, respiratory rate, conscious level, temperature and SpO₂). Match the frequency and type of observations to the severity of illness of the patient.
3. Use an EWS system or 'calling criteria' to identify patients who are critically ill, at risk of clinical deterioration or cardiopulmonary arrest, or both.
4. Use a patient vital signs chart that encourages and permits the regular measurement and recording of vital signs and, where used, early warning scores.
5. Ensure that the hospital has a clear policy that requires a timely, appropriate, clinical response to deterioration in the patient's clinical condition.
6. Introduce into each hospital a clearly identified response to critical illness. This will vary between sites, but may include an outreach service or resuscitation team (e.g., MET) capable of responding to acute clinical crises. This team should be alerted, using an early warning system, and the service must be available 24 hours a day.
7. Ensure that all clinical staff are trained in the recognition, monitoring, and management of the critically ill patient, and that they know their role in the rapid response system.
8. Empower staff to call for help when they identify a patient at risk of deterioration or cardiac arrest. Use a structured communication tool to ensure effective handover of information between staff (e.g. SBAR - Situation-Background-Assessment-Recommendation).
9. Agree a hospital DNAR policy, based on current national guidance, and ensure that all clinical staff understand it. Identify patients who do not wish to receive CPR and those for whom cardiopulmonary arrest is an anticipated terminal event for whom CPR would be inappropriate.
10. Audit all cardiac arrests, 'false arrests', unexpected deaths, and unanticipated intensive care unit admissions, using a common dataset. Audit the antecedents and clinical responses to these events. All hospitals should consider joining the [National Cardiac Arrest Audit](#).

6

In-hospital resuscitation

Introduction

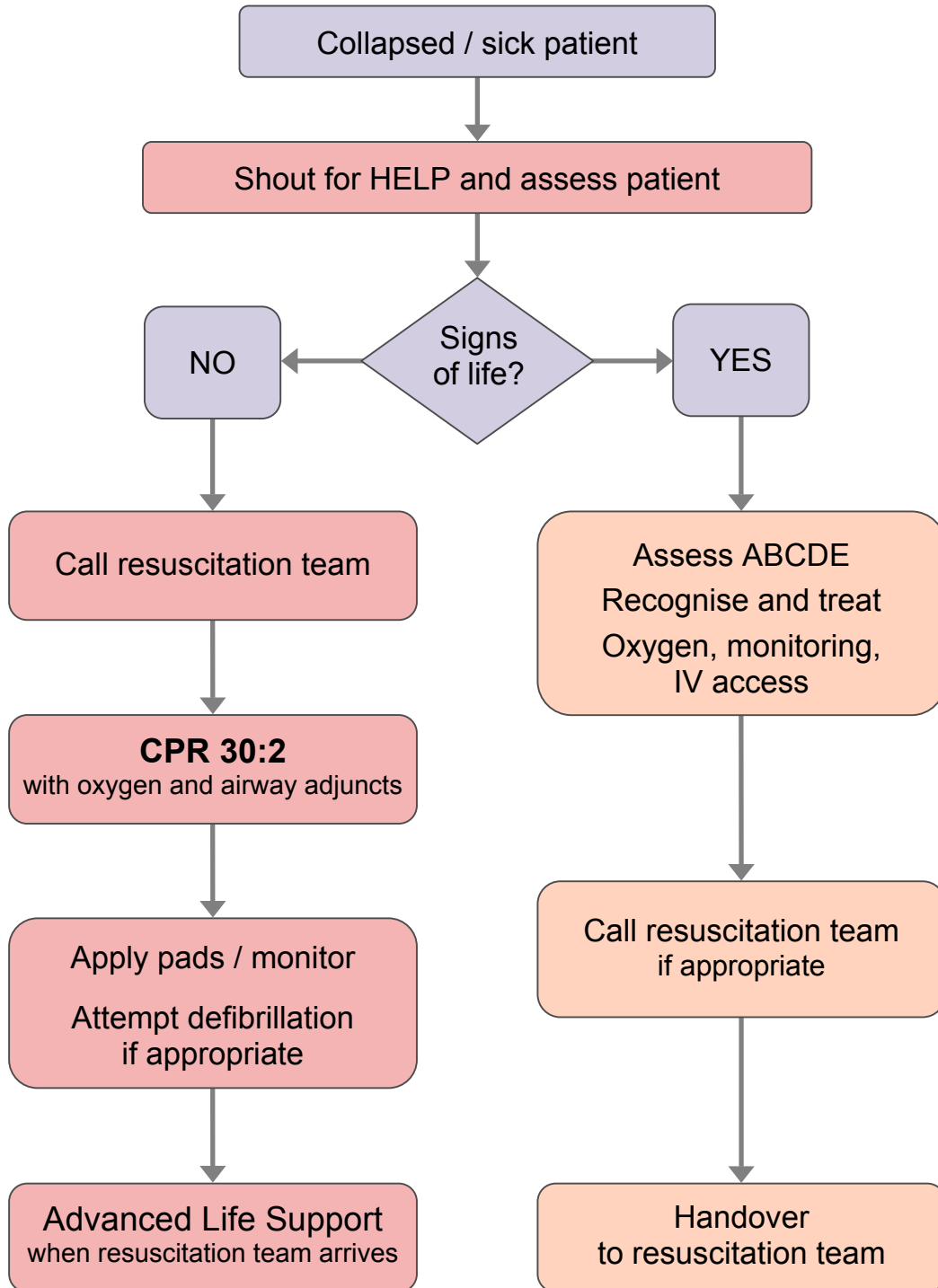
These guidelines are aimed primarily at healthcare professionals who are first to respond to an in-hospital cardiac arrest and may also be applicable to healthcare professionals in other clinical settings.

After in-hospital cardiac arrest the division between basic life support (BLS) and advanced life support (ALS) is arbitrary; in practice, the resuscitation process is a continuum. For all in-hospital cardiac arrests, ensure that:

- cardiorespiratory arrest is recognised immediately;
- help is summoned using a standard telephone number (e.g., 2222),⁸⁸
- cardiopulmonary resuscitation (CPR) is started immediately using adjuncts, for example a pocket mask, and, if indicated, defibrillation attempted as rapidly as possible and certainly within 3 min.

All in-hospital cardiac arrests should be reviewed as part of an audit and quality improvement process. Details should be recorded after each event. The [National Cardiac Arrest Audit](#) enables hospitals to collect standardised data, and monitor changes in cardiac arrest activity.

In-hospital resuscitation algorithm



Sequence for 'collapsed' patient in-hospital

1, Ensure personal safety

2. Check the patient for a response

- When a healthcare professional sees a patient collapse, or finds a patient apparently unconscious in a clinical area, he should first shout for help, then assess if the patient is responsive by gently shaking his shoulders and asking loudly, 'Are you all right?'
- It will be possible to undertake several actions simultaneously if other members of staff are nearby.

3A. If the patient responds:

- Urgent medical assessment is required. Call for help according to local protocols. This may be a resuscitation team (e.g. medical emergency team (MET)).
- While waiting for the team, assess the patient using the ABCDE (Airway Breathing Circulation Disability Exposure) approach.
- Give the patient oxygen – use pulse oximetry to guide oxygen therapy.⁸⁹
- Attach monitoring (minimum of: pulse oximetry, ECG and blood pressure) and record vital signs.⁶⁷
- Obtain venous access.
- Prepare for handover to team using SBAR (Situation, Background, Assessment, Recommendation)⁹⁰ or RSVP (Reason, Story, Vital signs, Plan)⁹¹ communication framework.

3B. If the patient does not respond:

- Shout for help (if this has not already been done).
- Turn the patient onto his back.
- Open the airway using head tilt and chin lift.
- If you suspect that there is a cervical spine injury, try to open the airway using a jaw thrust. Maintaining an airway and adequate ventilation is the overriding priority in managing a patient with a suspected spinal injury. If this is unsuccessful, use just enough head tilt to clear the airway. Use manual in-line stabilisation to minimise head movement if sufficient rescuers are available. Efforts to protect the cervical spine must not jeopardise oxygenation and ventilation.
- Keeping the airway open, look, listen, and feel to determine if the victim is breathing normally. This should be a rapid check and should take **less than 10 s**:
 - Listen at the victim's mouth for breath sounds.
 - Look for chest movement.
 - Feel for air on your cheek.

- Agonal breathing (occasional gasps, slow, laboured, or noisy breathing) is common immediately after cardiac arrest and is not normal breathing – it is a sign of cardiac arrest and should not be mistaken for a sign of life.
- Those experienced in clinical assessment may wish to assess the carotid pulse for less than 10 s. This may be performed simultaneously with checking for breathing or after the breathing check.
- The exact sequence will depend on the training of staff and their experience in assessment of breathing and circulation.

4A. If the patient has a pulse or other signs of life:

- Urgent medical assessment is required. Depending on the local protocols this may take the form of a resuscitation team.
- While awaiting this team, assess the patient using the ABCDE approach.
- Follow the steps in 3A above whilst waiting for the team.
- The patient is at high risk of further deterioration and cardiac arrest and needs continued observation until the team arrives.

4B. If there is no pulse or other sign of life:

- One person starts CPR as others call the resuscitation team and collect the resuscitation equipment and a defibrillator. If only one member of staff is present, this will mean leaving the patient.
- Give 30 chest compressions followed by 2 ventilations.
- Minimise interruptions and ensure high-quality compressions.
- The correct hand position for chest compression is the middle of the lower half of the sternum.
- The recommended depth of compression is at least 5 cm (not more than 6 cm) and the rate is at least 100 compressions min^{-1} (not more than 120 min^{-1}). Allow the chest to completely recoil in between each compression.
- If available, use a prompt and/or feedback device to help ensure high quality chest compressions.
- The person providing chest compressions should change about every 2 min, or earlier if unable to continue high quality chest compressions. This change should be done with minimal interruption to compressions.
- Maintain the airway and ventilate the lungs with the most appropriate equipment immediately at hand. A pocket mask, which may be supplemented with an oral airway, is usually readily available. Alternatively, use a supraglottic airway device (e.g. laryngeal mask airway (LMA)) and self-inflating bag, or bag-mask, according to local policy.
- Tracheal intubation should be attempted only by those who are trained, competent and experienced in this skill. Waveform capnography should be available routinely for confirming tracheal tube placement (in the presence of a cardiac output) and subsequent monitoring of an intubated patient.

Waveform capnography can also be used to monitor the quality of CPR (see ALS guidelines).

- Use an inspiratory time of 1 s and give enough volume to produce a normal chest rise. Add supplemental oxygen as soon as possible.
- Once the patient's trachea has been intubated or a supraglottic airway device has been inserted, continue chest compressions uninterrupted (except for defibrillation or pulse checks when indicated), at a rate of at least 100 min⁻¹, and ventilate the lungs at approximately 10 breaths min⁻¹. Avoid hyperventilation (both excessive rate and tidal volume), which may worsen outcome.
- If there is no airway and ventilation equipment available, consider giving mouth-to-mouth ventilation. If there are clinical reasons to avoid mouth-to-mouth contact, or you are unwilling or unable to do this, do chest compressions until help or airway equipment arrives. A pocket mask or bag mask device should be available rapidly in all clinical areas.
- When the defibrillator arrives, apply self-adhesive defibrillation pads to the patient and analyse the rhythm. These should be applied whilst chest compressions are ongoing. The use of adhesive pads will enable more rapid assessment of heart rhythm than attaching ECG electrodes.⁹²
- If using an automated external defibrillator (AED) switch on the machine and follow the AED's audio-visual prompts.
- For manual defibrillation, minimise the interruption to CPR to deliver a shock. Using a manual defibrillator it is possible to reduce the pause between stopping and restarting of chest compressions to less than 5 s.
- Plan what to do if the rhythm is shockable before CPR is stopped. Safety issues should also be addressed and planned for while chest compressions are ongoing.
- Pause briefly to assess the heart rhythm. With a manual defibrillator, if the rhythm is ventricular fibrillation/pulseless ventricular tachycardia (VF/VT), charge the defibrillator and restart chest compressions. Once the defibrillator is charged and everyone apart from the person doing compressions is clear, pause the chest compressions, rapidly ensure that all rescuers are clear of the patient and then deliver the shock. Restart chest compressions immediately after shock delivery. This sequence should be planned before stopping compressions.
- Continue resuscitation until the resuscitation team arrives or the patient shows signs of life. Follow the universal algorithm for ALS (see ALS guidelines).
- Once resuscitation is underway, and if there are sufficient staff present, prepare intravenous cannulae and drugs likely to be used by the resuscitation team (e.g., adrenaline).

- Identify one person to be responsible for handover to the resuscitation team leader. Use a structured communication tool for handover (e.g., SBAR, RSVP).^{90, 91} Locate the patient's records and ensure that they are available immediately the resuscitation team arrives.

4C. If the patient is not breathing but has a pulse (respiratory arrest):

- Ventilate the patient's lungs (as described above) and check for a pulse every 10 breaths (about every minute).

Only those competent in assessing breathing and a pulse will be able to make the diagnosis of respiratory arrest. If there are any doubts about the presence of a pulse, start chest compression and continue until more experienced help arrives.

5. If the patient has a monitored and witnessed cardiac arrest:

If a patient has a monitored and witnessed cardiac arrest in the cardiac catheter laboratory or early after cardiac surgery:

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/VT, give up to three quick successive (stacked) shocks if necessary. Start chest compressions immediately after the third shock and continue CPR for 2 min.
- This three-shock strategy may also be considered when a conscious patient has a witnessed VF/VT cardiac arrest and is already monitored using adhesive defibrillator electrodes with a manual defibrillator.
- A precordial thump in these settings works rarely⁹³⁻⁹⁵ and may succeed only if given within seconds of the onset of a shockable rhythm.⁹⁶ Delivery of a precordial thump must not delay calling for help or accessing a defibrillator. It is therefore appropriate therapy only when several clinicians are present at a witnessed, monitored arrest, and when a defibrillator is not immediately to hand. In practice, this is only likely to be in a critical care environment such as the emergency department or ICU, or in the cardiac catheter laboratory or pacing room.

Background notes

Hospital and staff factors

The exact sequence of actions after in-hospital cardiac arrest depends on several factors including:

- location (clinical or non-clinical area; monitored or unmonitored patients);
- skills of staff who respond;
- number of responders;
- equipment available;
- hospital system for response to cardiac arrest and medical emergencies (e.g. MET, cardiac arrest team).

Location

Monitored arrests are usually diagnosed rapidly. Ward patients may have had a period of deterioration and an unwitnessed arrest.^{62, 63} Ideally, all patients who are at high risk of cardiac arrest should be cared for in a monitored area where facilities for immediate resuscitation are available. Patients, visitors, or staff may also have a cardiac arrest in non-clinical areas (e.g. car parks, corridors). The Resuscitation Council (UK) has published [guidance for safer handling during resuscitation in healthcare settings](#).

Delay in attempting defibrillation may occur when patients sustain cardiac arrest in unmonitored hospital beds and in outpatient departments.⁹⁷ In these areas several minutes may elapse before resuscitation teams arrive with a defibrillator and deliver shocks. Despite limited evidence, AEDs should be considered for the hospital setting as a way to facilitate early defibrillation (a goal of less than 3 min from collapse), especially in areas where healthcare providers have no rhythm recognition skills or where they use defibrillators infrequently.

Skills of staff who respond

All healthcare professionals should be able to recognise cardiac arrest, call for help, and start resuscitation. Staff should do what they have been trained to do. For example, staff in critical care and emergency medicine may have more advanced resuscitation skills than staff who are not involved regularly in resuscitation in their normal clinical role. Hospital staff who attend a cardiac arrest may have different competencies in managing the airway, breathing, and circulation. Rescuers should use those resuscitation skills they have been trained to do.

The RC(UK) Immediate Life Support (ILS) course is aimed at the majority of healthcare professionals who attend cardiac arrests rarely but have the potential to be first responders or resuscitation team members.⁹⁸ A recent study found that the number of cardiac arrest calls decreased while pre-arrest calls increased after implementing a programme that included ILS teaching in two hospitals. This was associated with an increase in initial survival after cardiac arrest and survival to discharge.⁷⁹ The course teaches healthcare professionals the skills that, if used whilst awaiting the arrival of the resuscitation team, are most likely to result in successful resuscitation.

The RC(UK) Advanced Life Support (ALS) course is aimed at doctors and senior nurses working in acute areas of the hospital and those who may be resuscitation team leaders and members.^{99, 100} The course is also suitable for senior paramedics and some hospital technicians.

During training and clinical practice there should be a greater emphasis on non-technical skills (NTS).¹⁰¹ These consist of situational awareness, decision making, team working, including team leadership and task management. Tools such as SBAR or RSVP should be used to ensure rapid effective communication and handovers.

Number of responders

The single responder must ensure that help is on its way. If other staff are nearby, several actions can be undertaken simultaneously. Hospital staffing tends to be at its lowest during the night and at weekends. This may influence patient monitoring,

treatment and outcomes. Data from the US National Registry of CPR Investigators shows that survival rates from in-hospital cardiac arrest are lower during nights and weekends.¹⁰² Several studies show that higher nurse staffing is associated with lower rates of failure-to-rescue, and reductions in incidence of cardiac arrest, pneumonia, shock and death.¹⁰³⁻¹⁰⁵

Equipment available

Ideally, the equipment used for CPR (including defibrillators) and the layout of equipment and drugs should be standardised throughout the hospital.^{106, 107} A review by the RC(UK) of serious patient safety incidents associated with CPR and patient deterioration reported to the National Patient Safety Agency showed that equipment problems are a common contributing cause. All resuscitation equipment must be checked on a regular basis to ensure it is ready for use. AEDs should be considered for clinical and non-clinical areas where staff do not have rhythm recognition skills or rarely need to use a defibrillator.

Hospitals and teams that regularly treat cardiac arrests should have monitoring and equipment for transferring patients after they have been resuscitated. This includes portable monitors with a minimum of pulse oximetry, ECG, non-invasive blood pressure and waveform capnography for ventilated patients. For further information, refer to the Intensive Care Society's [Guidelines for the Transport of the Critically ill Adult](#).

Resuscitation team

The resuscitation team may take the form of a traditional cardiac arrest team, which is called only when cardiac arrest is recognised. Alternatively, hospitals may have strategies to recognise patients at risk of cardiac arrest and summon a team (e.g., MET) before cardiac arrest occurs. The term 'resuscitation team' reflects the range of response teams. In-hospital cardiac arrests are rarely sudden or unexpected. A strategy of recognising patients at risk of cardiac arrest may enable some of these arrests to be prevented, or may prevent futile resuscitation attempts in those patients who are unlikely to benefit from CPR (See prevention of in-hospital cardiac arrest and decisions about CPR chapter).

Surveys show that resuscitation teams rarely have formal pre- and post-event briefings (briefings and debriefings).¹⁰⁸⁻¹¹⁰ Resuscitation team members should meet for introductions and plan before they attend actual events. Team members should also debrief after each event based on what they actually did during the resuscitation. Ideally this should be based on data collected during the event.¹¹¹

National Cardiac Arrest Audit

All in-hospital cardiac arrests should be reviewed and audited. The National Cardiac Arrest Audit (NCAA) is a UK-wide database of in-hospital cardiac arrests and is supported by the RC(UK) and the Intensive Care National Audit & Research Centre (ICNARC). NCAA monitors and reports on the incidence of and outcome from, in-hospital cardiac arrests in order to inform practice and policy. It aims to identify and foster improvements in the prevention, care delivery and outcomes from cardiac arrest.

Participating in NCAA means that your hospital is collecting and contributing to national, standardised data on cardiac arrest, enabling improvements in patient care.^{57, 112, 113}

Diagnosis of cardiac arrest

Trained healthcare staff cannot assess the breathing and pulse sufficiently reliably to confirm cardiac arrest.¹¹⁴⁻¹²³ Agonal breathing (occasional gasps, slow, laboured or noisy breathing) is common in the early stages of cardiac arrest and is a sign of cardiac arrest and should not be confused as a sign of life/circulation.^{10, 124-126} Agonal breathing can also occur during chest compressions as cerebral perfusion improves, but is not indicative of a return of spontaneous circulation (ROSC). Delivering chest compressions to a patient with a beating heart is unlikely to cause harm.¹²⁷ However, delays in diagnosis of cardiac arrest and starting CPR will adversely effect survival and must be avoided.

High-quality CPR

The quality of chest compressions during in-hospital CPR is frequently sub-optimal.^{12, 15} The importance of uninterrupted chest compressions cannot be over-emphasised. Even short interruptions to chest compressions are disastrous for outcome and every effort must be made to ensure that continuous, effective chest compression is maintained throughout the resuscitation attempt. The person providing chest compressions should be changed every 2 min, but without causing long pauses in chest compressions.

Defibrillation strategy

The length of the pre-shock pause (the interval between stopping chest compressions and delivering a shock) is inversely related to the chance of successful defibrillation. Every 5-second increase in the duration of the pre-shock pause almost halves the chance of successful defibrillation, therefore it is critical to minimise the pause.¹³ The lengthy 'top-to-toe' safety check (e.g., "head, middle, bottom, self, oxygen away") performed after the defibrillator has charged and before shock delivery, taught and used in clinical practice commonly, will therefore significantly diminish the chances of successful defibrillation. Previous RC(UK) guidance and teaching materials state that the pre-shock pause should be less than 10 s; we believe that it is possible to reduce this to less than 5 s without endangering rescuers.

Rescuers should not compromise on safety. Actions should be planned before stopping chest compressions. If there are delays caused by difficulties in rhythm analysis or if individuals are still in contact with the patient, chest compressions should be restarted whilst plans are made to decide what to do when compressions are next stopped. Rescuers should wear gloves during CPR attempts. If they are not immediately available this should not delay starting CPR. Wearing gloves may decrease the risk of accidental shocks to rescuers although this requires further study.¹²⁸

Although there are no data supporting a three-shock strategy, it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs early in the electrical phase, immediately after onset of VF/VT. In circumstances where rapid early defibrillation is feasible (cardiac catheter laboratory, in monitored cardiac surgery patients, patients who have a witnessed and monitored VF/VT and are already connected to a defibrillator) three rapid defibrillation attempts may achieve ROSC without the need for chest compressions.

Introduction

This chapter on adult advanced life support (ALS) adheres to the same general principles as Guidelines 2005, but incorporates some important changes. The guidelines in this chapter apply to healthcare professionals trained in ALS techniques. Laypeople, first responders, and automated external defibrillator (AED) users are referred to the basic life support (BLS) and AED chapters.

Guideline changes

Defibrillation

- There is increased emphasis on the importance of minimally-interrupted high-quality chest compressions throughout any ALS intervention: chest compressions are paused briefly only to allow specific interventions.
- The recommendation for a specified period of cardiopulmonary resuscitation (CPR) before out-of-hospital defibrillation, following cardiac arrest unwitnessed by the emergency medical services (EMS), has been removed.
- Chest compressions are now continued while a defibrillator is charged – this will minimise the pre-shock pause.
- The role of the precordial thump is de-emphasised.
- The use of up to three quick successive (stacked) shocks is now recommended for ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) occurring in the cardiac catheterisation laboratory or in the immediate post-operative period following cardiac surgery.

Drugs

- Delivery of drugs via a tracheal tube is no longer recommended – if intravenous (IV) access cannot be achieved give drugs by the intraosseous (IO) route.
- When treating VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock and then every 3-5 min (during alternate cycles of CPR). In the 2005 Guidelines, adrenaline was given just before the third shock. This subtle change in the timing of adrenaline administration is to separate the timing of drug delivery from

attempted defibrillation. It is hoped that this will result in more efficient shock delivery and less interruption in chest compressions. Amiodarone 300 mg is also given after the third shock.

- Atropine is no longer recommended for routine use in asystole or pulseless electrical activity (PEA).

Airway

- There is reduced emphasis on early tracheal intubation unless achieved by highly skilled individuals with minimal interruption to chest compressions.
- There is increased emphasis on the use of capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of return of spontaneous circulation (ROSC).

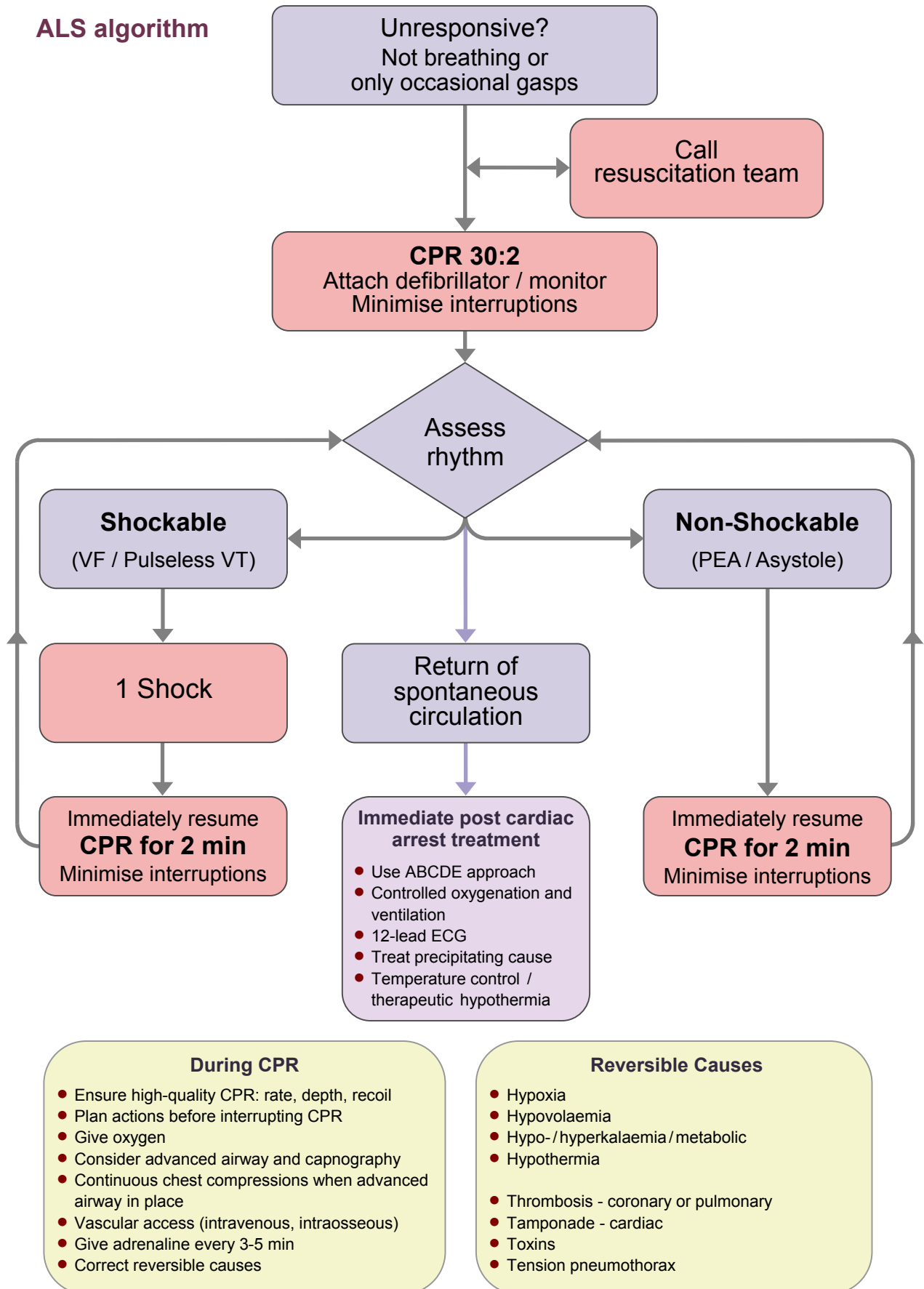
Ultrasound

- The potential role of ultrasound imaging during ALS is recognised.

Post-resuscitation care

- The potential harm caused by hyperoxaemia after ROSC is achieved is now recognised: once ROSC has been established and the oxygen saturation of arterial blood (SaO₂) can be monitored reliably (by pulse oximetry and/or arterial blood gas analysis), inspired oxygen is titrated to achieve a SaO₂ of 94 - 98%.
- There is much greater detail and emphasis on the treatment of the post-cardiac-arrest syndrome.
- There is recognition that implementation of a comprehensive, structured post-resuscitation treatment protocol may improve survival in cardiac arrest victims after ROSC.
- There is increased emphasis on the use of primary percutaneous coronary intervention in appropriate, but comatose, patients with sustained ROSC after cardiac arrest.
- The recommendation for glucose control has been revised: in adults with sustained ROSC after cardiac arrest, blood glucose values >10 mmol l⁻¹ should be treated but hypoglycaemia must be avoided.
- Use of therapeutic hypothermia now includes comatose survivors of cardiac arrest associated initially with non-shockable rhythms as well as shockable rhythms. The lower level of evidence for use after cardiac arrest from non-shockable rhythms is acknowledged.
- It is recognised 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.

ALS algorithm



Arrhythmias associated with cardiac arrest are divided into two groups: shockable rhythms (VF/VT) and non-shockable rhythms (asystole and PEA). The principle difference in management is the need for attempted defibrillation in patients with VF/VT. Subsequent actions, including chest compression, airway management and ventilation, vascular access, administration of adrenaline, and the identification and correction of reversible factors, are common to both groups. The ALS algorithm provides a standardised approach to the management of adult patients in cardiac arrest.

Shockable rhythms (VF/VT)

The first monitored rhythm is VF/VT in approximately 25% of cardiac arrests, both in⁶⁰ or out of hospital.^{42, 129, 130} VF/VT will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA.⁶⁰

Treatment of shockable rhythms (VF/VT)

1. Confirm cardiac arrest – check for signs of life or if trained to do so, breathing and pulse simultaneously.
2. Call resuscitation team.
3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads – one below the right clavicle and the other in the V6 position in the midaxillary line.
4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.
5. Stop chest compressions; confirm VF from the ECG.
6. Resume chest compressions immediately; simultaneously, the designated person selects the appropriate energy on the defibrillator (150-200 J biphasic for the first shock and 150-360 J biphasic for subsequent shocks) and presses the charge button.
7. While the defibrillator is charging, warn all rescuers other than the individual performing the chest compressions to “stand clear” and remove any oxygen delivery device as appropriate. Ensure that the rescuer giving the compressions is the only person touching the patient.
8. Once the defibrillator is charged, tell the rescuer doing the chest compressions to “stand clear”; when clear, give the shock.
9. Without reassessing the rhythm or feeling for a pulse, restart CPR using a ratio of 30:2, starting with chest compressions.
10. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.
11. Pause briefly to check the monitor.
12. If VF/VT, repeat steps 6 - 11 above and deliver a second shock.
13. If VF/VT persists repeat steps 6 - 8 above and deliver a third shock. Resume chest compressions immediately and then give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR.

14. Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/VT persists.
15. Give further adrenaline 1 mg IV after alternate shocks (i.e., approximately every 3-5 min).

If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of return of spontaneous circulation (ROSC):

- Check a central pulse and end-tidal CO₂ trace if available
- If there is evidence of ROSC, start post-resuscitation care.
- If no signs of ROSC, continue CPR and switch to the non-shockable algorithm.

If asystole is seen, continue CPR and switch to the non-shockable algorithm.

The interval between stopping compressions and delivering a shock must be minimised and certainly should not exceed a few seconds (ideally less than 5 s). Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation.

If an organised rhythm is seen during a 2-minute period of CPR, do not interrupt chest compressions to palpate a pulse unless the patient shows signs of life (this may include a sudden increase in end-tidal carbon dioxide [ETCO₂] if this is being monitored) suggesting ROSC. If there is any doubt about the existence of a pulse in the presence of an organised rhythm, resume CPR. If the patient has ROSC, begin post-resuscitation care.

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm⁹³⁻⁹⁵ and is only likely to succeed if given within the first few seconds of the onset of a shockable rhythm.⁹⁶ There is more success with pulseless VT than with VF. Delivery of a precordial thump must not delay calling for help or accessing a defibrillator. It is therefore appropriate therapy only when several clinicians are present at a witnessed, monitored arrest, and when a defibrillator is not immediately to hand. In practice, this is likely to be in a monitored environment such as the emergency department resuscitation room, ICU, CCU, cardiac catheter laboratory or pacemaker room.

A precordial thump should be undertaken immediately after confirmation of cardiac arrest and only by healthcare professionals trained in the technique. Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, then retract the fist immediately to create an impulse-like stimulus. There are a very few reports of a precordial thump converting a perfusing rhythm to a non-perfusing rhythm.¹³¹

Explanation for the changes in the treatment of VF/VT

Defibrillation strategy

► Single versus three-shock strategy

Experimental studies show that relatively short interruptions in chest compression to deliver rescue breaths or perform rhythm analysis are associated with reduced survival.¹³² Interruptions in chest compression also reduce the chances of converting VF to another rhythm.¹³³ The interruptions to CPR associated with a three-shock protocol, combined with the improved first shock efficacy (for termination of VF/VT) of biphasic defibrillators, prompted the recommendation of a single-shock strategy in the Guidelines 2005. Subsequent studies have shown a significantly lower hands-off-ratio with the one-shock protocol¹³⁴ and some,^{53, 135, 136} but not all,^{134, 137} have suggested a significant survival benefit from this single-shock strategy. However, all studies except one¹³⁷ were before-after studies and all introduced multiple changes in the protocol, making it difficult to attribute a possible survival benefit to one of the changes.

If VF/VT occurs during cardiac catheterisation or in the early post-operative period following cardiac surgery (when chest compressions could disrupt vascular sutures), consider delivering up to three-stacked shocks before starting chest compressions.¹³⁸ This three-shock strategy may also be considered for an initial, witnessed VF/VT cardiac arrest if the patient is already connected to a manual defibrillator – these circumstances are rare. Although there are no data supporting a three-shock strategy in any of these circumstances, it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs immediately after onset of VF.

► Defibrillation energy

The initial shock from a biphasic defibrillator should be no lower than 120 J for rectilinear biphasic waveforms, and 150 J for biphasic truncated exponential waveforms. For uniformity, it is recommended that the initial biphasic shock should be at least 150 J. Because of the lower efficacy of monophasic defibrillators for terminating VF/VT, and the use of a single-shock strategy, the recommended initial energy level for the first shock using a monophasic defibrillator remains at 360 J.

For second and subsequent shocks, the 2005 guidelines did not distinguish between a fixed and an escalating energy protocol. Since then, several studies have demonstrated that although an escalating strategy reduces the number of shocks required to restore an organised rhythm compared with fixed-dose biphasic defibrillation, rates of ROSC or survival to hospital discharge are not increased.¹³⁹⁻¹⁴² Conversely, a fixed-dose biphasic protocol has demonstrated high success rates with a three-shock fixed-dose protocol.¹⁴³ If an initial shock has been unsuccessful it may be worth attempting the second and subsequent shocks with a higher energy level if the defibrillator is capable of delivering a higher energy, but both fixed and escalating strategies are acceptable, based on current evidence.

Manufacturers should display the effective waveform energy range on the face of the biphasic device. If you are unaware of the effective energy range of the device, use the highest available energy for the first and subsequent shocks.

Fine VF

Fine VF that is difficult to distinguish from asystole is very unlikely to be shocked successfully into a perfusing rhythm. Continuing good quality CPR may improve the amplitude and frequency of the VF and improve the chance of successful defibrillation to a perfusing rhythm. Delivering repeated shocks in an attempt to defibrillate what is thought to be fine VF will increase myocardial injury, both directly from the electric current and indirectly from the interruptions in coronary blood flow.

Adrenaline

Despite the widespread use of adrenaline during resuscitation, and several studies involving vasopressin, there is no placebo-controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases neurologically-intact survival to hospital discharge. Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs. Despite the lack of human data, the use of adrenaline is still recommended, based largely on animal data and increased short-term survival in humans.^{144, 145} The alpha-adrenergic actions of adrenaline cause vasoconstriction, which increases myocardial and cerebral perfusion pressure. The higher coronary blood flow increases the frequency and amplitude of the VF waveform and should improve the chance of restoring a circulation when defibrillation is attempted.¹⁴⁶⁻¹⁴⁸ Although adrenaline improves short-term survival, animal data indicate that it impairs the microcirculation^{149, 150} and can cause post-cardiac-arrest myocardial dysfunction,^{151, 152} both of which might impact on long-term outcome. The optimal dose of adrenaline is not known, and there are no data supporting the use of repeated doses. There are few data on the pharmacokinetics of adrenaline during CPR. The optimal duration of CPR and number of shocks that should be given before giving drugs is unknown. On the basis of expert consensus, for VF/VT give adrenaline after the third shock once chest compressions have resumed, and then repeat every 3-5 min during cardiac arrest (alternate cycles). Do not interrupt CPR to give drugs.

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as the absence of any palpable pulse in the presence of cardiac electrical activity that would be expected to produce a cardiac output. These patients often have some mechanical myocardial contractions but they are too weak to produce a detectable pulse or blood pressure – this is sometimes described as ‘pseudo-PEA’. PEA may be caused by reversible conditions that can be treated if they are identified and corrected. Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively.

Sequence of actions for PEA

- Start CPR 30:2.
- Give adrenaline 1 mg as soon as intravascular access is achieved.
- Continue CPR 30:2 until the airway is secured, then continue chest compressions without pausing during ventilation.
- Consider possible reversible causes of PEA and correct any that are identified.
- Recheck the patient after 2 min:
 - If there is still **no** pulse and no change in the ECG appearance:
 - Continue CPR.
 - Recheck the patient after 2 min and proceed accordingly.
 - Give further adrenaline 1 mg every 3-5 min (alternate loops).
 - If VF/VT, change to the shockable rhythm algorithm.
 - If a pulse is present, start post-resuscitation care.

Sequence of actions for asystole

- Start CPR 30:2.
- Without stopping CPR, check that the leads are attached correctly.
- Give adrenaline 1 mg as soon as intravascular access is achieved.
- Continue CPR 30:2 until the airway is secured, then continue chest compression without pausing during ventilation.
- Consider possible reversible causes of PEA and correct any that are identified.
- Recheck the rhythm after 2 min and proceed accordingly.
- If VF/VT, change to the shockable rhythm algorithm.
- Give adrenaline 1 mg IV every 3-5 min (alternate loops).

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because the patient may respond to cardiac pacing when there is ventricular standstill with continuing P waves. There is no value in attempting to pace true asystole.

Atropine

Atropine antagonises the action of the parasympathetic neurotransmitter acetylcholine at muscarinic receptors. Therefore, it blocks the effect of the vagus nerve on both the sinoatrial (SA) node and the atrioventricular (AV) node, increasing the sinus rate and facilitating AV node conduction.

The 2005 guidelines recommended the administration of a single 3 mg dose of atropine for asystole and slow PEA ($< 60 \text{ min}^{-1}$); however, during cardiac arrest asystole is usually caused by primary myocardial pathology rather than excessive vagal tone and there is no evidence that routine use of atropine is beneficial in the treatment of asystole or PEA. Several recent studies have failed to demonstrate any benefit from atropine in

out-of-hospital or in-hospital cardiac arrests¹⁵³⁻¹⁵⁹ and its routine use for asystole or PEA is no longer recommended.

During CPR

During the treatment of persistent VF/VT or PEA/asystole, there should be an emphasis on giving good quality chest compression between defibrillation attempts, whilst recognising and treating reversible causes (4 Hs and 4 Ts), and whilst obtaining a secure airway and intravascular access. Healthcare providers must practise efficient coordination between CPR and shock delivery. The shorter the interval between stopping chest compressions and shock delivery, the more likely it is that the shock will be successful. Reduction in the interval from compression to shock delivery by even a few seconds can increase the probability of shock success. Providing CPR with a CV ratio of 30:2 is tiring; change the individual undertaking compressions every 2 min.

Potentially reversible causes

Potential causes or aggravating factors for which specific treatment exists must be sought during any cardiac arrest.¹³⁸ For ease of memory, these are divided into two groups of four, based upon their initial letter, either H or T:

- Hypoxia
- Hypovolaemia
- Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia, and other metabolic disorders
- Hypothermia

- Tension pneumothorax
- Tamponade
- Toxic substances
- Thromboembolism (pulmonary embolus/coronary thrombosis)

The four 'Hs'

Minimise the risk of **hypoxia** by ensuring that the patient's lungs are ventilated adequately with 100% oxygen. Make sure that there is adequate chest rise and that there are bilateral breath sounds. Using the techniques described below, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by **hypovolaemia** is usually due to severe haemorrhage; this may be precipitated by trauma, gastrointestinal bleeding, or rupture of an aortic aneurysm. Restore intravascular volume rapidly with fluid, coupled with urgent surgery to stop the haemorrhage.

Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia, and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history, e.g. renal failure. A 12-lead ECG may be diagnostic. Intravenous calcium

chloride is indicated in the presence of hyperkalaemia, hypocalcaemia, and calcium-channel-blocking drug overdose.

Suspect **hypothermia** in any drowning incident; use a low-reading thermometer.

The four 'Ts'

A **tension pneumothorax** may be the primary cause of PEA and may follow attempts to insert a central venous catheter. The diagnosis is made clinically and/or by use of ultrasound. Decompress rapidly by needle thoracocentesis or urgent thoracostomy, and then insert a chest drain.

Cardiac **tamponade** is difficult to diagnose because the typical signs of distended neck veins and hypotension are obscured by the arrest itself. Rapid transthoracic echocardiography with minimal interruption to chest compression can be used to identify a pericardial effusion. Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for resuscitative thoracotomy.

In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or **toxic** substances may be revealed only by laboratory investigations. Where available, the appropriate antidotes should be used, but most often treatment is supportive.

The commonest cause of **thromboembolic** or mechanical circulatory obstruction is massive pulmonary embolus. If cardiac arrest is likely to be caused by pulmonary embolism, consider giving a thrombolytic drug immediately. Thrombolysis may be considered in adult cardiac arrest, on a case-by-case basis, following initial failure of standard resuscitation in patients in whom an acute thrombotic aetiology for the arrest is suspected. Ongoing CPR is not a contraindication to thrombolysis. Thrombolytic drugs may take up to 90 min to be effective; only administer a thrombolytic drug if it is appropriate to continue CPR for this duration.

Use of ultrasound imaging during advanced life support

Several studies have examined the use of ultrasound during cardiac arrest to detect potentially reversible causes. Although no studies have shown that use of this imaging modality improves outcome, there is no doubt that ultrasound imaging provides information that may help to identify reversible causes of cardiac arrest (e.g. cardiac tamponade, pulmonary embolism, ischaemia (regional wall motion abnormality), aortic dissection, hypovolaemia, pneumothorax).¹⁶⁰ When ultrasound imaging and appropriately trained clinicians are available use them to assist with assessment and treatment of potentially reversible causes of cardiac arrest. The integration of ultrasound into advanced life support requires considerable training to ensure that interruptions to chest compressions are minimised. A sub-xiphoid probe position has been recommended.¹⁶⁰⁻¹⁶² Placement of the probe just before chest compressions are paused for a planned rhythm assessment enables a well-trained operator to obtain views within 10 s.

Intravenous fluids

Hypovolaemia is a potentially reversible cause of cardiac arrest: infuse fluids rapidly if hypovolaemia is suspected. In the initial stages of resuscitation there are no clear advantages to using colloid: use 0.9% sodium chloride or Hartmann's solution. Avoid dextrose; this is redistributed rapidly away from the intravascular space and causes hyperglycaemia, which may worsen neurological outcome after cardiac arrest. Try to ensure normovolaemia, but in the absence of hypovolaemia, infusion of an excessive volume of fluid is likely to be harmful during CPR.¹⁶³ Use intravenous fluid to flush peripherally injected drugs into the central circulation.

Open-chest cardiac compression

Open-chest cardiac compression may be indicated for patients with cardiac arrest caused by trauma, in the early postoperative phase after cardiothoracic surgery, or when the chest or abdomen is already open, for example during surgery following trauma.¹³⁸

Signs of life

If signs of life (such as regular respiratory effort, coughing, purposeful movement or eye opening) reappear during CPR, or readings from the patient's monitors (e.g. sudden increase in ET_{CO}₂ or blood pressure monitored from an arterial cannula) are compatible with ROSC, stop CPR and check the monitors briefly. Do not confuse agonal respiration (gaspings), which is common in the immediate few seconds following a cardiac arrest or during good quality CPR, for signs of life. If an organised cardiac rhythm is present, check for a pulse. If a pulse is palpable, continue post-resuscitation care, treatment of peri-arrest arrhythmias, or both. If no pulse is present, continue CPR.

Defibrillation

Strategies before defibrillation

Pads versus paddles

Self-adhesive defibrillation pads have practical benefits over hand-held paddles for routine monitoring and defibrillation.¹⁶⁴ They are safe and effective and, given the change in defibrillation strategy with the 2010 guidelines, are much preferred to standard defibrillation paddles.¹⁶⁵ Use self-adhesive pads in peri-arrest situations and in clinical situations where patient access is difficult. They have a similar transthoracic impedance¹⁶⁶ (and therefore efficacy) to manual paddles and enable the operator to defibrillate the patient from a safe distance rather than leaning over the patient as occurs with paddles. Pads enable a shock to be delivered more rapidly than with paddles.⁹²

Safe use of oxygen

In an oxygen-enriched atmosphere, sparks from defibrillator paddles applied poorly can cause a fire. Taking the following precautions can minimise this risk:

- Remove any oxygen mask or nasal cannulae and place them at least 1 m away from the patient's chest during defibrillation.

- Leave the ventilation bag connected to the tracheal tube or other airway adjunct. Alternatively, disconnect the ventilation bag from the tracheal tube and move it at least 1 m from the patient's chest during defibrillation.
- The use of self-adhesive defibrillation pads, rather than manual paddles, may minimise the risk of sparks occurring.

Chest hair

It may be necessary to shave the area intended for electrode placement, but do this rapidly and do not delay defibrillation if a razor is not available immediately.

Electrode position

Place the right (sternal) electrode to the right of the sternum, below the clavicle. Place the apical paddle in the mid-axillary line, approximately over the V6 ECG electrode position. This electrode should be clear of any breast tissue. It is important that this electrode is placed sufficiently laterally.

Antero-posterior electrode placement is an acceptable alternative to the traditional right pectoral-apical position for defibrillation and is the preferred choice for cardioversion of atrial fibrillation.

An implanted medical device (e.g. permanent pacemaker or implantable cardioverter defibrillator (ICD)) may be damaged during defibrillation if current is discharged through electrodes placed directly over the device. If possible, place the electrode away from the device if necessary by using an alternative electrode position.

Airway management and ventilation

Most of the principles of airway and ventilation management remain unchanged from Guidelines 2005. There is reduced emphasis on early tracheal intubation unless achieved by highly skilled individuals with minimal interruption to chest compressions. There is increased emphasis on the use of capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of ROSC.

Patients requiring resuscitation often have an obstructed airway. In these cases, prompt assessment, with control of the airway and ventilation of the lungs, is essential. Without adequate oxygenation it may be impossible to restore a spontaneous cardiac output. In a witnessed cardiac arrest in the vicinity of a defibrillator, attempted defibrillation takes precedence over opening of the airway.

Give high-flow oxygen until ROSC is achieved and reliable monitoring of the oxygen saturation of arterial blood enables the inspired oxygen to be adjusted.

Basic airway manoeuvres and airway adjuncts

Assess the airway. Use head tilt and chin lift, or jaw thrust to open the airway. Simple airway adjuncts (oropharyngeal or nasopharyngeal airways) are often helpful, and sometimes essential, to maintain an open airway.

Ventilation

Provide artificial ventilation as soon as possible in any patient in whom spontaneous ventilation is inadequate or absent. Expired air ventilation (rescue breathing) is effective but the rescuer's expired oxygen concentration is only 16-17%, so it must be replaced as soon as possible by ventilation with oxygen-enriched air. A pocket resuscitation mask enables mouth-to-mask ventilation and some enable supplemental oxygen to be given. Use a two-hand technique to maximise the seal with the patient's face. A self-inflating bag can be connected to a face mask, tracheal tube, or supraglottic airway device (SAD). The two-person technique for bag-mask ventilation is preferable. Deliver each breath over approximately 1 s and give a volume that corresponds to normal chest movement; this represents a compromise between giving an adequate volume, minimising the risk of gastric inflation, and allowing adequate time for chest compression. During CPR with an unprotected airway, give two ventilations after each sequence of 30 chest compressions. Once a tracheal tube or SAD has been inserted, ventilate the lungs at a rate of about 10 breaths min^{-1} and continue chest compression without pausing during ventilation.

Alternative airway devices

The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest. But there is evidence that, without adequate training and experience, the incidence of complications, such as unrecognised oesophageal intubation (6 - 17% in several studies involving paramedics) is unacceptably high.¹⁶⁷ Prolonged attempts at tracheal intubation are harmful; the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Several alternative airway devices have been considered for airway management during CPR. There are published studies on the use during CPR of the Combitube, the classic laryngeal mask airway (cLMA), the Laryngeal Tube (LT) and the i-gel, but none of these studies have been powered adequately to enable survival to be studied as a primary endpoint. Instead, most researchers have studied insertion and ventilation success rates. The SADs are easier to insert than a tracheal tube and, unlike tracheal intubation, can generally be inserted without interrupting chest compressions.¹⁶⁸ There are no data supporting the routine use of any specific approach to airway management during cardiac arrest. The best technique is dependent on the precise circumstances of the cardiac arrest and the competence of the rescuer. The Combitube is rarely, if ever, used in the UK and is no longer included in these guidelines.

Laryngeal mask airway (LMA)

A laryngeal mask airway is relatively easy to insert, and ventilation using an LMA is more efficient and easier than with a bag-mask. If gas leakage is excessive, chest

compression will have to be interrupted to enable ventilation. Although an LMA does not protect the airway as reliably as a tracheal tube, pulmonary aspiration is uncommon when using an LMA during cardiac arrest.

i-gel

The cuff of the i-gel is made of thermoplastic elastomer gel and does not require inflation; the stem of the i-gel incorporates a bite block and a narrow oesophageal drain tube. It is used commonly for maintenance of the airway during anaesthesia. The ease of insertion of the i-gel and its favourable leak pressure make it theoretically very attractive as a resuscitation airway device for those inexperienced in tracheal intubation. Use of the i-gel during cardiac arrest has been reported but more data on its use in this setting are awaited.

Laryngeal Tube

The laryngeal tube (LT) was introduced in 2001. A disposable version of the laryngeal tube (LT-D) is available and has been used during resuscitation following pre-hospital cardiac arrest.¹⁶⁹ The LT is not in common use in the UK.

Tracheal intubation

The pros and cons of tracheal intubation have been discussed in the pre-hospital chapter. As with pre-hospital tracheal intubation, intubation in hospital should be attempted only by trained personnel able to carry out the procedure with a high level of skill and confidence. No intubation attempt should interrupt chest compressions for more than 10 s; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After intubation, confirm tube placement and secure the tube adequately.

Confirmation of correct placement of the tracheal tube

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine use of primary and secondary techniques to confirm correct placement of the tracheal tube should reduce this risk.

Primary assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and heard clearly) and over the epigastrium (breath sounds should not be heard). Clinical signs of correct tube placement (condensation in the tube, chest rise, breath sounds on auscultation of lungs, and inability to hear gas entering the stomach) are not completely reliable. Secondary confirmation of tracheal tube placement by an exhaled carbon dioxide (CO₂) or oesophageal detection device should reduce the risk of unrecognised oesophageal intubation but the performance of the available devices varies considerably. Furthermore, none of the secondary confirmation techniques will differentiate between a tube placed in a main bronchus and one placed correctly in the trachea, so careful primary assessment to ensure equal expansion of both lungs and equally clear breath sounds over each remains important.

There are inadequate data to identify the optimal method of confirming tube placement during cardiac arrest, and all devices should be considered as adjuncts to other confirmatory techniques.¹⁷⁰ There are no data quantifying their ability to monitor tube position after initial placement.

Carbon dioxide detector devices measure the concentration of exhaled carbon dioxide from the lungs. The persistence of exhaled CO₂ after six ventilations indicates placement of the tracheal tube in the trachea or a main bronchus.⁴⁵ During cardiac arrest pulmonary blood flow may be so low that there is insufficient exhaled CO₂, so the CO₂ detector does not identify a correctly placed tracheal tube. When exhaled CO₂ is detected during cardiac arrest it indicates reliably that the tube is in the trachea or main bronchus. A variety of electronic as well as simple, inexpensive, colorimetric CO₂ detectors are available for both in-hospital and out-of-hospital use. End-tidal CO₂ detectors that include a waveform graphical display (capnographs) are the most reliable for verification of tracheal tube position during cardiac arrest.

Based on the available data, the accuracy of colorimetric CO₂ detectors, oesophageal detector devices and non-waveform capnometers does not exceed the accuracy of auscultation and direct visualisation for confirming the tracheal position of a tube in victims of cardiac arrest. Waveform capnography is the most sensitive and specific way to confirm and continuously monitor the position of a tracheal tube in victims of cardiac arrest and should supplement clinical assessment (auscultation and visualisation of the tracheal tube passing between the vocal cords). Waveform capnography will not discriminate between tracheal and bronchial placement of the tube – careful auscultation is essential. Existing portable monitors make capnographic initial confirmation and continuous monitoring of tracheal tube position feasible in almost all settings where intubation is performed, including out of hospital, emergency departments, and in-hospital locations. In the absence of a waveform capnograph it may be preferable to use a supraglottic airway device when advanced airway management is indicated.

Cricothyroidotomy

If it is impossible to ventilate an apnoeic patient with a bag-mask, or to pass a tracheal tube or alternative airway device, delivery of oxygen through a cannula or surgical cricothyroidotomy may be life-saving. Surgical cricothyroidotomy provides a definitive airway that can be used to ventilate the patient's lungs until semi-elective intubation or tracheostomy is performed. Needle cricothyroidotomy is a much more temporary procedure providing only short-term oxygenation.

Assisting the circulation

Intravascular access

Peripheral versus central venous drug delivery

Peripheral venous cannulation is quicker, easier to perform, and safer. Drugs injected peripherally must be followed by a flush of at least 20 ml of fluid. Only those who are skilled and competent in the technique should attempt central venous line insertion and this must be achieved with minimal interruption to chest compressions.

Intraosseous route

If intravenous access cannot be established within the first 2 min of resuscitation, consider gaining IO access. Intraosseous access has traditionally been used for children because of the difficulties in gaining intravenous access, but this route has now become established as a safe and effective route for drug and fluid delivery in adults too.¹⁷¹⁻¹⁷³ Tibial and humeral sites are readily accessible and provide equal flow rates for fluids.¹⁷² Intraosseous delivery of resuscitation drugs will achieve adequate plasma concentrations.

Tracheal route

Resuscitation drugs can also be given via a tracheal tube, but the plasma concentrations achieved using this route are very variable and generally considerably lower than those achieved by the IV or IO routes, particularly with adrenaline. Large volumes of intratracheal fluid impair gas exchange. With the ease of gaining IO access and the lack of efficacy of tracheal drug administration, tracheal administration of drugs is no longer recommended.

Drugs

The use of adrenaline has been discussed above.

Anti-arrhythmic drugs

As with vasopressors, the evidence that anti-arrhythmic drugs are of benefit in cardiac arrest is limited. No anti-arrhythmic drug given during human cardiac arrest has been shown to increase survival to hospital discharge, although amiodarone has been shown to increase survival to hospital admission after shock-refractory VF/VT.^{174, 175} There are no data on the use of amiodarone for shock-refractory VF/VT when single shocks are used. Despite the lack of human long-term outcome data, the balance of evidence is in favour of the use of some anti-arrhythmic drugs for the management of arrhythmias in cardiac arrest.

Amiodarone

Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Also atrioventricular conduction is slowed, and a similar effect is seen in accessory pathways. The hypotension that occurs with intravenous amiodarone is related to the rate of delivery and is due more to the solvent (Polysorbate 80 and benzyl alcohol), which causes histamine release, rather than the drug itself.¹⁷⁶ An aqueous amiodarone

preparation that is free from these side effects has recently been approved for use in the United States.

On the basis of expert consensus, if VF/VT persists, give amiodarone 300 mg by bolus injection (flushed with 20 ml of 0.9% sodium chloride or 5% dextrose)¹⁷⁷ after the third shock. A further dose of 150 mg may be given for recurrent or refractory VF/VT, followed by an infusion of 900 mg over 24 h. Lidocaine 1 mg kg⁻¹ may be used as an alternative if amiodarone is not available, but do not give lidocaine if amiodarone has been given already.

Magnesium

Although the benefits of giving magnesium in known hypomagnesaemic states are recognised, the benefit of giving magnesium routinely during cardiac arrest is unproven. Studies in adults in and out of hospital¹⁷⁸⁻¹⁸³ have failed to demonstrate any increase in the rate of ROSC when magnesium is given routinely during CPR. Give an initial intravenous dose of 2 g (= 8 mmol, 4 ml of 50% magnesium sulphate) for refractory VF if there is any suspicion of hypomagnesaemia (e.g. patients on potassium-losing diuretics); it may be repeated after 10-15 min. Other indications are:

- ventricular tachyarrhythmias in the presence of possible hypomagnesaemia;
- torsade de pointes VT;
- digoxin toxicity.

Bicarbonate

Cardiac arrest results in combined respiratory and metabolic acidosis because pulmonary gas exchange ceases and cellular metabolism becomes anaerobic. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state;¹⁸⁴ analysis of central venous blood may provide a better estimation of tissue pH. Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. It has the following effects:

- it exacerbates intracellular acidosis;
- it produces a negative inotropic effect on ischaemic myocardium;
- it presents a large, osmotically active, sodium load to an already compromised circulation and brain;
- it produces a shift to the left in the oxygen dissociation curve, further inhibiting release of oxygen to the tissues.

Giving sodium bicarbonate routinely during cardiac arrest and CPR (especially in out-of-hospital cardiac arrest), or after ROSC, is not recommended. Give sodium bicarbonate (50 mmol) if cardiac arrest is associated with hyperkalaemia or tricyclic antidepressant overdose. Repeat the dose according to the clinical condition of the patient and the results of repeated blood gas analysis.

Calcium

Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. There are no data supporting any beneficial action for calcium after most cases of cardiac arrest. High plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may impair cerebral recovery. Give calcium during resuscitation only when indicated specifically, i.e., in cardiac arrest caused by hyperkalaemia, hypocalcaemia, or overdose of calcium channel-blocking drugs.

The initial dose of 10 ml 10% calcium chloride (6.8 mmol Ca²⁺) may be repeated if necessary. Calcium can slow the heart rate and precipitate arrhythmias. In cardiac arrest, calcium may be given by rapid intravenous injection. In the presence of a spontaneous circulation give it slowly. Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.

Mechanical CPR

At best, standard manual CPR produces coronary and cerebral perfusion that is just 30% of normal.¹⁸⁵ Several CPR techniques and devices may improve haemodynamics or short-term survival when used by well-trained providers in selected cases. However, the success of any technique or device depends on the education and training of the rescuers and on resources (including personnel). Although manual chest compressions are often performed very poorly,¹⁸⁶⁻¹⁸⁸ no adjunct has consistently been shown to be superior to conventional manual CPR.

Impedance threshold device (ITD)

The impedance threshold device (ITD) is a valve that limits air entry into the lungs during chest recoil between chest compressions; this decreases intrathoracic pressure and increases venous return to the heart. A recent meta-analysis demonstrated improved ROSC and short-term survival but no significant improvement in either survival to discharge or neurologically intact survival to discharge associated with the use of an ITD in the management of adult out-of-hospital cardiac arrest patients.¹⁸⁹ In the absence of data showing that the ITD increases survival to hospital discharge, its routine use in cardiac arrest is not recommended.

Lund University cardiac arrest system (LUCAS) CPR

The Lund University cardiac arrest system (LUCAS) is a gas-driven sternal compression device that incorporates a suction cup for active decompression. Although animal studies showed that LUCAS-CPR improves haemodynamic and short-term survival compared with standard CPR,^{190, 191} there are no published randomised human studies comparing LUCAS-CPR with standard CPR.

Load-distributing band CPR (AutoPulse)

The load-distributing band (LDB) is a circumferential chest compression device comprising a pneumatically actuated constricting band and backboard. Although the use of LDB CPR improves haemodynamics,¹⁹²⁻¹⁹⁴ results of clinical trials have been conflicting.^{195, 196}

The current status of LUCAS and AutoPulse

Two large prospective randomised multicentre studies are currently underway to evaluate AutoPulse and LUCAS. In hospital, mechanical devices have been used to support patients undergoing primary coronary intervention (PCI)^{197, 198} and CT scans¹⁹⁹ and also for prolonged resuscitation attempts (e.g., hypothermia,^{200, 201} poisoning, fibrinolytic therapy for pulmonary embolism, prolonged transport etc) where rescuer fatigue may impair the effectiveness of manual chest compression. The role of mechanical devices in all situations requires further evaluation before firm recommendations on their use can be made.

Post-resuscitation care

The post-cardiac-arrest syndrome

Successful ROSC is the just the first step toward the goal of complete recovery from cardiac arrest. The post-cardiac-arrest syndrome, which comprises post-cardiac-arrest brain injury, post-cardiac-arrest myocardial dysfunction, the systemic ischaemia / reperfusion response, and persistence of the precipitating pathology, often complicates the post-resuscitation phase.²⁰² The severity of this syndrome will vary with the duration and cause of cardiac arrest. It may not occur at all if the cardiac arrest is brief. Post-cardiac-arrest brain injury manifests as coma, seizures, myoclonus, varying degrees of neurocognitive dysfunction and brain death. Post-cardiac-arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypercarbia, hyperoxia, pyrexia, hyperglycaemia and seizures. Significant myocardial dysfunction is common after cardiac arrest but typically recovers by 2-3 days.^{203, 204} The whole body ischaemia/reperfusion that occurs with resuscitation from cardiac arrest activates immunological and coagulation pathways contributing to multiple organ failure and increasing the risk of infection.^{205, 206} Thus, the post-cardiac-arrest syndrome has many features in common with sepsis, including intravascular volume depletion and vasodilation.^{207, 208}

Airway and breathing

Hypoxaemia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Several animal studies indicate that hyperoxaemia causes oxidative stress and harms post-ischaemic neurones.²⁰⁹ A clinical registry study documented that post-resuscitation hyperoxaemia was associated with worse outcome, compared with both normoxaemia and hypoxaemia.²¹⁰ As soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94 -98%. Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. There are no data to support the targeting of a specific arterial PCO₂ after resuscitation from cardiac arrest, but it is reasonable to adjust ventilation to achieve normocarbia and to monitor this using the end-tidal PCO₂ and arterial blood gas values.

Circulation

It is well recognised that post-cardiac-arrest patients with ST elevation myocardial infarction (STEMI) should undergo early coronary angiography and percutaneous coronary intervention (PCI) but, because chest pain and/or ST elevation are relatively poor predictors of acute coronary occlusion in these patients,²¹¹ this intervention should be considered in all post-cardiac-arrest patients who are suspected of having coronary artery disease as the cause of their arrest.^{55, 211, 212} Several studies indicate that the combination of therapeutic hypothermia and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction.²¹²⁻²¹⁶

Post-cardiac arrest myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias.²⁰³ If treatment with appropriate fluids and vasoactive drugs is insufficient to support the circulation, consider insertion of an intra-aortic balloon pump.^{212, 213} In the absence of definitive data, target the mean arterial blood pressure to achieve an adequate urine output ($1 \text{ ml kg}^{-1} \text{ h}^{-1}$) and normal or decreasing plasma lactate values, taking into consideration the patient's usual blood pressure (if known), the cause of the arrest and the severity of any myocardial dysfunction.²⁰²

Disability (optimising neurological recovery)

Control of seizures

Seizures or myoclonus or both occur in 5% to 15% of adult patients who achieve ROSC and 10% to 40% of those who remain comatose.²¹⁷⁻²²⁰ Seizures increase cerebral metabolism by up to 3-fold and may cause cerebral injury: treat promptly and effectively with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate. No studies address directly the use of prophylactic anticonvulsant drugs after cardiac arrest in adults.

Glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome.^{221, 222} A large randomised trial of intensive glucose control ($4.5 - 6.0 \text{ mmol l}^{-1}$) versus conventional glucose control (10 mmol l^{-1} or less) in general ICU patients reported increased 90-day mortality in patients treated with intensive glucose control.²²³ Another recent study and two meta-analyses of studies of tight glucose control versus conventional glucose control in critically ill patients showed no significant difference in mortality but found tight glucose control was associated with a significantly increased risk of hypoglycaemia.²²⁴⁻²²⁶ Severe hypoglycaemia is associated with increased mortality in critically ill patients,²²⁷ and comatose patients are at particular risk from unrecognised hypoglycaemia. Based on the available data, following ROSC blood glucose should be maintained at $\leq 10 \text{ mmol l}^{-1}$.²²⁸ Hypoglycaemia should be avoided. Strict glucose control should not be implemented in adult patients with ROSC after cardiac arrest because of the increased risk of hypoglycaemia.

Temperature control

Treatment of hyperpyrexia

A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest.²²⁹ Several studies document an association between post-cardiac-arrest pyrexia and poor outcomes.²³⁰⁻²³² Although the effect of elevated temperature on outcome is not proved, it seems prudent to treat any hyperthermia occurring after cardiac arrest with antipyretics or active cooling.

Therapeutic hypothermia

Animal and human data indicate that mild hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia.^{233, 234} Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen by about 6% for each 1°C reduction in temperature²³⁵ and this may reduce the release of excitatory amino acids and free radicals.²³³ Hypothermia blocks the intracellular consequences of excitotoxin exposure (high calcium and glutamate concentrations) and reduces the inflammatory response associated with the post-cardiac-arrest syndrome.

All studies of post-cardiac-arrest therapeutic hypothermia have included only patients in coma. There is good evidence supporting the use of induced hypothermia in comatose survivors of out-of-hospital cardiac arrest caused by VF. One randomised trial²³⁶ and a pseudo-randomised trial²³⁷ demonstrated improved neurological outcome at hospital discharge or at 6 months in comatose patients after out-of-hospital VF cardiac arrest. Cooling was initiated within minutes to hours after ROSC and a temperature range of 32-34°C was maintained for 12-24 h. Extrapolation of these data to other cardiac arrests (e.g., other initial rhythms, in-hospital arrests, paediatric patients) seems reasonable but is supported only by data derived from non-randomised trials.^{212, 238-241}

The practical application of therapeutic hypothermia is divided into three phases: induction, maintenance, and rewarming.²⁴² Animal data indicate that earlier cooling after ROSC produces better outcomes.²⁴³ External and/or internal cooling techniques can be used to initiate cooling. An infusion of 30 ml kg⁻¹ of 4°C 0.9% sodium chloride or Hartmann's solution decreases core temperature by approximately 1.5°C. Other methods of inducing and/or maintaining hypothermia include: simple ice packs and/or wet towels; cooling blankets or pads; water or air circulating blankets; water circulating gel-coated pads; intravascular heat exchanger; and cardiopulmonary bypass.

In the maintenance phase, a cooling method with effective temperature monitoring that avoids temperature fluctuations is preferred. This is best achieved with external or internal cooling devices that include continuous temperature feedback to achieve a set target temperature. Plasma electrolyte concentrations, effective intravascular volume and metabolic rate can change rapidly during rewarming, as they do during cooling. Thus, rewarming must be achieved slowly: the optimal rate is not known, but the consensus is currently about 0.25-0.5 °C increase in body temperature per hour.²⁴⁰ The well-recognised physiological effects of hypothermia need to be managed carefully.²⁴²

Prognostication

Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury.²⁴⁴ A quarter of those dying after admission to ICU following in-hospital cardiac arrest die from neurological injury. A means of predicting neurological outcome that can be applied to individual patients immediately after ROSC is required. Many studies have focused on prediction of poor long term outcome (vegetative state or death), based on clinical or test findings that indicate irreversible brain injury, to enable clinicians to limit care or withdraw organ support. The implications of these prognostic tests are such that they should have 100% specificity or zero false positive rate, i.e., no individuals eventually have a 'good' long-term outcome despite the prediction of a poor outcome.

Clinical examination

There are no clinical neurological signs that predict poor outcome (Cerebral Performance Category [CPC] 3 or 4, or death) reliably less than 24 h after cardiac arrest. In adult patients who are comatose after cardiac arrest, and who have not been treated with hypothermia and who do not have confounding factors (such as hypotension, sedatives or muscle relaxants), the absence of both pupillary light and corneal reflex at ≥ 72 h predicts poor outcome reliably.²²⁰ Absence of vestibulo-ocular reflexes at ≥ 24 h^{245, 246} and a GCS motor score of 2 or less at ≥ 72 h²²⁰ are less reliable. Other clinical signs, including myoclonus, are not recommended for predicting poor outcome. The presence of myoclonic status in adults is strongly associated with poor outcome,^{219, 220, 247-249} but rare cases of good neurological recovery from this situation have been described and accurate diagnosis is problematic.

Biochemical markers

Serum (e.g. neuronal specific enolase, S100 protein) or cerebrospinal fluid (CSF) biomarkers alone are insufficient as predictors of poor outcomes in comatose patients after cardiac arrest with or without treatment with therapeutic hypothermia.

Neurophysiological studies

No neurophysiological study predicts outcome for a comatose patient reliably within the first 24 h after cardiac arrest. If somatosensory evoked potentials (SSEP) are measured after 24 h in comatose cardiac arrest survivors not treated with therapeutic hypothermia, bilateral absence of the N20 cortical response to median nerve stimulation predicts poor outcome (death or CPC 3 or 4).²⁵⁰ Very few hospitals in the UK have the resources to enable SSEPs to be measured.

Imaging studies

Many imaging modalities (magnetic resonance imaging [MRI], computed tomography [CT], single photon emission computed tomography [SPECT], cerebral angiography, transcranial Doppler, nuclear medicine, near infra-red spectroscopy [NIRS]) have been studied to determine their utility for prediction of outcome in adult survivors of cardiac arrest.²⁵¹ There are no high-level studies that support the use of any imaging modality to predict outcome of comatose cardiac arrest survivors.

Impact of therapeutic hypothermia on prognostication

There is inadequate evidence to recommend a specific approach to prognosticating poor outcome in post-cardiac-arrest patients treated with therapeutic hypothermia. There are no clinical neurological signs, neurophysiological studies, biomarkers, or imaging modalities that can predict neurological outcome reliably in the first 24 h after cardiac arrest. Potentially reliable prognosticators of poor outcome in patients treated with therapeutic hypothermia after cardiac arrest include bilateral absence of N20 peak on SSEP \geq 24 h after cardiac arrest and the absence of both corneal and pupillary reflexes 3 or more days after cardiac arrest.^{247, 252} Given the limited available evidence, decisions to limit care should not be made based on the results of a single prognostication tool.

Organ donation

Post-cardiac-arrest patients who do not survive represent an opportunity to increase the organ donor pool, either after brain death²⁵³ or as non-heart-beating donors.²⁵⁴

Cardiac arrest centres

There is wide variation in patient survival rates among hospitals caring for patients after resuscitation from cardiac arrest.²⁵⁵⁻²⁵⁸ There is indirect evidence that regional cardiac systems of care improve outcome after STEMI.²⁵¹ The implication from all these data is that specialist cardiac arrest centres and systems of care may be effective but direct evidence is awaited.²⁵⁹⁻²⁶¹

Introduction

Cardiac arrhythmias are relatively common in the peri-arrest period. They are common in the setting of acute myocardial infarction and may precipitate ventricular fibrillation (VF) or follow successful defibrillation. The treatment algorithms described in this chapter have been designed to enable the non-specialist advanced life support (ALS) provider to treat the patient effectively and safely in an emergency; for this reason they have been kept as simple as possible. If patients are not acutely ill there may be several other treatment options, including the use of drugs (oral or parenteral) that will be less familiar to the non-expert. In this situation there will be time to seek advice from cardiologists or other senior doctors with the appropriate expertise.

Guideline changes

There are relatively few changes from Guidelines 2005. Initial assessment of patients with suspected peri-arrest arrhythmias now uses the ABCDE approach (see the preventing cardiac arrest chapter). A single set of adverse features for tachy- and brady-arrhythmias has been introduced for consistency.

Sequence of actions

Assess the patient using the ABCDE approach. In all cases, give oxygen and insert an intravenous cannula and assess the patient for adverse features. Whenever possible, record a 12-lead ECG; this will help determine the precise rhythm, either before treatment or retrospectively, if necessary with the help of an expert. Correct any electrolyte abnormalities (e.g. K^+ , Mg^{++} , Ca^{++}).

When you assess and treat any arrhythmia address two factors: the condition of the patient (stable versus unstable – determined by the absence or presence respectively of adverse features) and the nature of the arrhythmia.

Adverse features

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

- Shock – hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness.
- Syncope – transient loss of consciousness due to global reduction in blood flow to the brain.
- Myocardial ischaemia – typical ischaemic chest pain and/or evidence of myocardial ischaemia on 12-lead ECG.
- Heart failure – pulmonary oedema and/or raised jugular venous pressure (with or without peripheral oedema and liver enlargement).

Treatment options

Depending on the nature of the underlying arrhythmia and clinical status of the patient (in particular the presence or absence of adverse features) immediate treatments can be categorised under four headings:

1. Electrical (cardioversion for tachyarrhythmia or pacing for bradyarrhythmia)
2. Simple clinical intervention (e.g., vagal manoeuvres, fist pacing)
3. Pharmacological (drug treatment)
4. No treatment needed

Most drugs act more slowly and less reliably than electrical treatments, so electrical treatment is usually the preferred treatment for an unstable patient with adverse features.

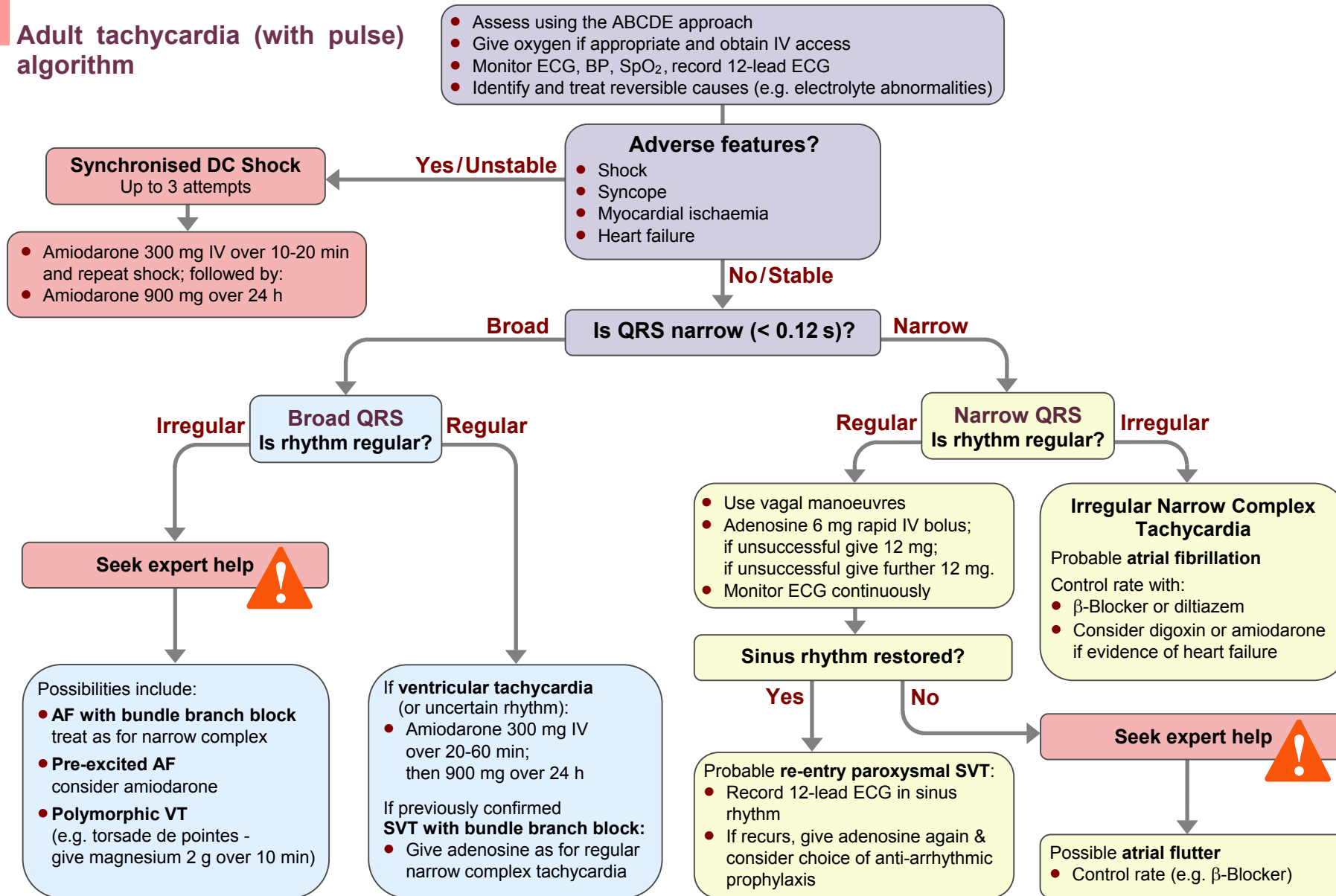
If a patient develops an arrhythmia during, or as a complication of some other condition (e.g. infection, acute myocardial infarction, heart failure), make sure that the underlying condition is assessed and treated appropriately, involving relevant experts if necessary. Once an arrhythmia has been treated successfully, repeat a 12-lead ECG to detect any abnormalities that may require treatment in the longer term.

Tachycardias

If the patient is unstable

If the patient is unstable and deteriorating (i.e., has adverse features caused by the tachycardia) synchronised cardioversion is the treatment of choice. In patients with otherwise normal hearts, serious signs and symptoms are uncommon if the ventricular rate is < 150 min⁻¹. Patients with impaired cardiac function, structural heart disease or other serious medical conditions (e.g. severe lung disease) may be symptomatic and unstable during arrhythmias with heart rates between 100 and 150 min⁻¹. If cardioversion fails to restore sinus rhythm, and the patient remains unstable, give amiodarone 300 mg IV over 10 - 20 min and re-attempt electrical cardioversion. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 h.

Adult tachycardia (with pulse) algorithm



Synchronised cardioversion

Carry out cardioversion under general anaesthesia or conscious sedation, administered by a healthcare professional competent in the technique being used. Ensure that the defibrillator is set to synchronised mode. For a broad-complex tachycardia or atrial fibrillation, start with 120-150 J biphasic shock (200 J monophasic) and increase in increments if this fails. Atrial flutter and regular narrow-complex tachycardia will often be terminated by lower energies: start with 70-120 J biphasic (100 J monophasic).

If the patient is stable

If there are no adverse features consider using drug treatment in the first instance (if any treatment is required). Assess the ECG and determine the QRS duration. If the QRS duration is greater than 0.12 s (3 small squares on standard ECG paper speed of 25 mm s⁻¹), this is a broad-complex tachycardia. If the QRS duration is less than 0.12 s, it is a narrow-complex tachycardia.

Broad-complex tachycardia

Broad-complex tachycardias (QRS \geq 0.12 s) are usually ventricular in origin. Broad-complex tachycardias may be also caused by supraventricular rhythms with aberrant conduction (bundle branch block). In the unstable, peri-arrest patient assume that the rhythm is ventricular in origin and attempt synchronised cardioversion as described above. Conversely, if a patient with broad-complex tachycardia is stable, the next step is to determine if the rhythm is regular or irregular.

Regular broad-complex tachycardia

A regular broad-complex tachycardia is likely to be VT or a supraventricular rhythm with bundle branch block. In a stable patient, if there is uncertainty about the source of the arrhythmia, give adenosine using the strategy described below.

If the broad complex tachycardia is thought to be VT, treat with amiodarone 300 mg intravenously over 20-60 min, followed by an infusion of 900 mg over 24 h. If a regular broad-complex tachycardia is known to be a supraventricular arrhythmia with bundle branch block, and the patient is stable, use the strategy indicated for narrow-complex tachycardia (below).

Irregular broad-complex tachycardia

This is most likely to be atrial fibrillation (AF) with bundle branch block, but careful examination of a 12-lead ECG (if necessary by an expert) may enable confident identification of the rhythm. Other possible causes are AF with ventricular pre-excitation (in patients with Wolff-Parkinson-White (WPW) syndrome), or polymorphic VT (e.g. torsade de pointes), but polymorphic VT is unlikely to be present without adverse features. Seek expert help with the assessment and treatment of irregular broad-complex tachyarrhythmia.

Treat torsade de pointes VT immediately by stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium

sulphate 2 g IV over 10 min (= 8 mmol, 4 ml of 50% magnesium sulphate). Obtain expert help, as other treatment (e.g. overdrive pacing) may be indicated to prevent relapse once the arrhythmia has been corrected. If adverse features develop, which is common, arrange immediate synchronised cardioversion. If the patient becomes pulseless, attempt defibrillation immediately (ALS algorithm).

Narrow-complex tachycardia

Examine the ECG to determine if the rhythm is regular or irregular.

Regular narrow-complex tachycardias include:²⁶²

- sinus tachycardia;
- AV nodal re-entry tachycardia (AVNRT) – the commonest type of regular narrow-complex tachyarrhythmia;
- AV re-entry tachycardia (AVRT) – due to WPW syndrome;
- atrial flutter with regular AV conduction (usually 2:1).

An irregular narrow-complex tachycardia is most likely to be AF or sometimes atrial flutter with variable AV conduction ('variable block').

Regular narrow-complex tachycardia

Sinus tachycardia

Sinus tachycardia is not an arrhythmia. This is a common physiological response to stimuli such as exercise or anxiety. In a sick patient it may occur in response to many conditions including pain, infection, anaemia, blood loss, and heart failure. Treatment is directed at the underlying cause; trying to slow sinus tachycardia that has occurred in response to most of these conditions will make the situation worse. Do not attempt to treat sinus tachycardia with cardioversion or anti-arrhythmic drugs.

AVNRT and AVRT (paroxysmal supraventricular tachycardia)

AV nodal re-entry tachycardia is the commonest type of paroxysmal supraventricular tachycardia (SVT), often seen in people without any other form of heart disease. It is relatively uncommon in the peri-arrest setting. It causes a regular, narrow-complex tachycardia, often with no clearly visible atrial activity on the ECG. The heart rate is commonly well above the typical range of sinus rhythm at rest (60-100 min⁻¹). It is usually benign, unless there is additional, co-incidental, structural heart disease or coronary disease, but it may cause symptoms that the patient finds frightening.

AV re-entry tachycardia occurs in patients with the WPW syndrome, and is also usually benign, unless there is additional structural heart disease. The common type of AVRT is a regular narrow-complex tachycardia, usually having no visible atrial activity on the ECG.

Atrial flutter with regular AV conduction (often 2:1 block)

This produces a regular narrow-complex tachycardia. It may be difficult to see atrial activity and identify flutter waves in the ECG with confidence, so the rhythm may be indistinguishable, at least initially, from AVNRT or AVRT.

Typical atrial flutter has an atrial rate of about 300 min^{-1} , so atrial flutter with 2:1 conduction produces a tachycardia of about 150 min^{-1} . Much faster rates (160 min^{-1} or more) are unlikely to be caused by atrial flutter with 2:1 conduction. Regular tachycardia with slower rates (e.g. 125-150) may be due to atrial flutter with 2:1 conduction, usually when the rate of the atrial flutter has been slowed by drug therapy.

Treatment of regular narrow-complex tachycardia

If the patient is unstable, with adverse features caused by the arrhythmia, attempt synchronised electrical cardioversion. It is reasonable to give adenosine to an unstable patient with a regular narrow-complex tachycardia while preparations are being made for synchronised cardioversion. However, do not delay electrical cardioversion if adenosine fails to restore sinus rhythm.

In the absence of adverse features:

- Start with vagal manoeuvres. Carotid sinus massage or the Valsalva manoeuvre will terminate up to a quarter of episodes of paroxysmal SVT. Record an ECG (preferably multi-lead) during each manoeuvre. If the rhythm is atrial flutter, slowing of the ventricular response will often occur and reveal flutter waves.
- If the arrhythmia persists and is not atrial flutter, give adenosine 6 mg as a rapid intravenous bolus. Use a relatively large cannula and large (e.g., antecubital) vein. Warn the patient that they will feel unwell and probably experience chest discomfort for a few seconds after the injection. Record an ECG (preferably multi-lead) during the injection. If the ventricular rate slows transiently, but then speeds up again, look for atrial activity, such as atrial flutter or other atrial tachycardia, and treat accordingly. If there is no response to adenosine 6 mg, give a 12 mg bolus. If there is no response give one further 12 mg bolus. Apparent lack of response to adenosine will occur if the bolus is given too slowly or into a peripheral vein.
- Vagal manoeuvres or adenosine will terminate almost all AVNRT or AVRT within seconds. Failure to terminate a regular narrow-complex tachycardia with adenosine suggests an atrial tachycardia such as atrial flutter (unless the adenosine has been injected too slowly or into a small peripheral vein).
- If adenosine is contra-indicated, or fails to terminate a regular narrow complex tachycardia without demonstrating that it is atrial flutter, consider giving a calcium-channel blocker, for example verapamil 2.5 - 5 mg intravenously over 2 min.

Irregular narrow-complex tachycardia

An irregular narrow-complex tachycardia is most likely to be AF with an uncontrolled ventricular response or, less commonly, atrial flutter with variable AV block. Record a 12-lead ECG to identify the rhythm. If the patient is unstable, with adverse features caused by the arrhythmia, attempt synchronised cardioversion.²⁶³

If there are no adverse features, treatment options include:

- rate control by drug therapy;
- rhythm control using drugs to encourage chemical cardioversion;
- rhythm control by electrical cardioversion;
- treatment to prevent complications (e.g. anticoagulation).

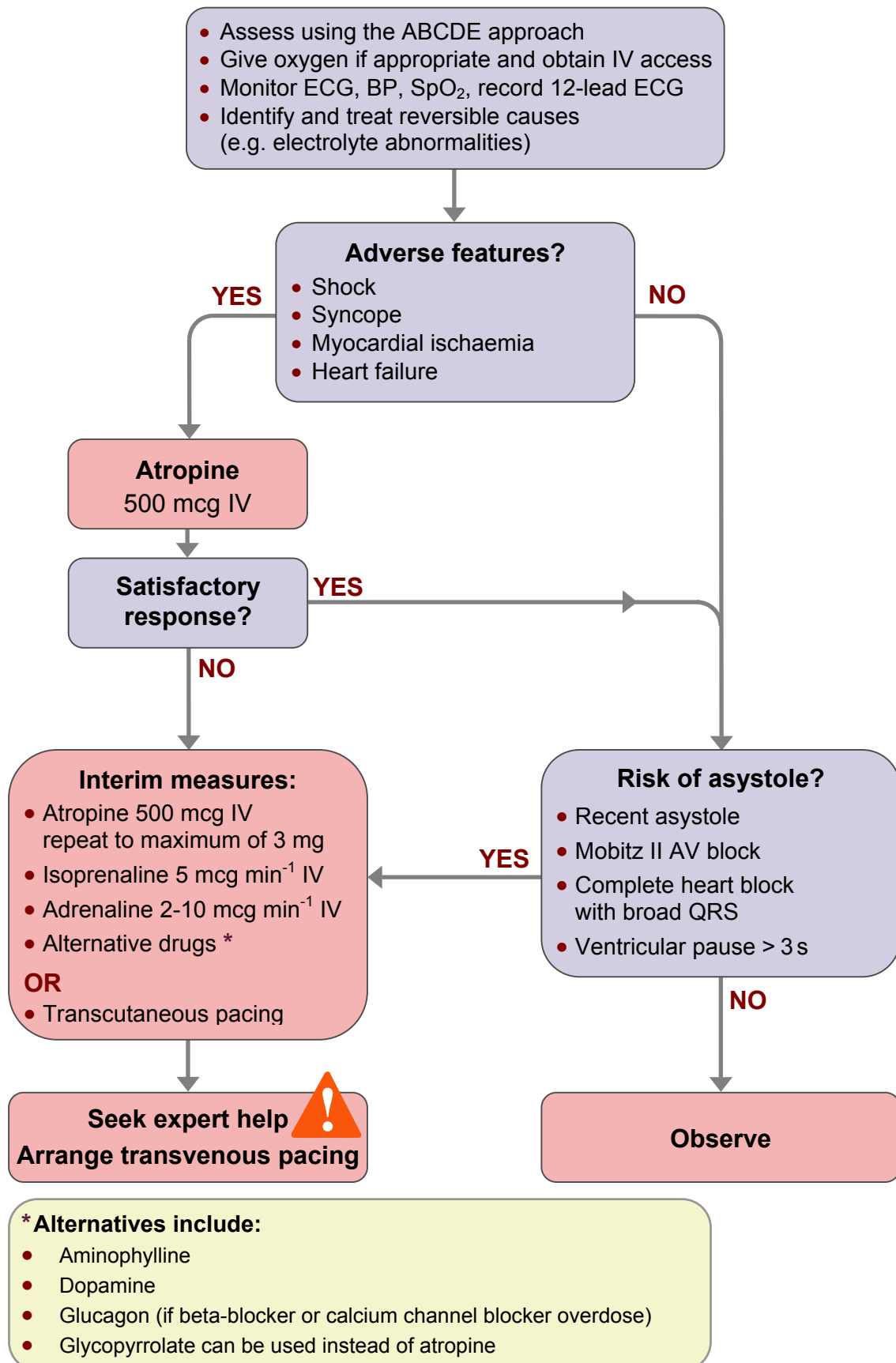
Obtain expert help to determine the most appropriate treatment for the individual patient. The longer a patient remains in AF the greater is the likelihood of atrial thrombus developing. In general, patients who have been in AF for more than 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least three weeks, or unless trans-oesophageal echocardiography has shown the absence of atrial thrombus. If the clinical situation dictates that cardioversion is needed more urgently, give either regular low-molecular-weight heparin in therapeutic dose or an intravenous bolus injection of unfractionated heparin followed by a continuous infusion to maintain the activated partial thromboplastin time (APTT) at 1.5 to 2 times the reference control value. Continue heparin therapy and commence oral anticoagulation after successful cardioversion. Seek expert advice on the duration of anticoagulation, which should be a minimum of 4 weeks, often substantially longer.

If the aim is to control heart rate, the usual drug of choice is a beta-blocker. Diltiazem or verapamil may be used in patients in whom beta blockade is contraindicated or not tolerated. An intravenous preparation of diltiazem is available in some countries but not in the UK. Digoxin may be used in patients with heart failure. Amiodarone may be used to assist with rate control but is most useful in maintaining rhythm control. Magnesium is also used but the data supporting this are limited. When possible seek expert help in selecting the best choice of treatment for rate control in each individual patient.

If the duration of AF is less than 48 h and rhythm control is considered appropriate, chemical cardioversion may be attempted. Seek expert help with the use of drugs such as flecainide. Do not use flecainide in the presence of heart failure, known left ventricular impairment or ischaemic heart disease, or a prolonged QT interval. Amiodarone (300 mg intravenously over 20-60 min followed by 900 mg over 24 h) may also be used but is less likely to achieve prompt cardioversion. Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

Seek expert help if any patient with AF is known or found to have ventricular pre-excitation (WPW syndrome). Avoid using adenosine, diltiazem, verapamil, or digoxin in patients with pre-excited AF or atrial flutter as these drugs block the AV node and cause a relative increase in pre-excitation.

Adult bradycardia algorithm



Bradycardia

Bradycardia is defined as a heart rate of $< 60 \text{ min}^{-1}$. It may be:

- physiological (e.g., in athletes);
- cardiac in origin (e.g., atrioventricular block or sinus node disease);
- non-cardiac in origin (e.g., vasovagal, hypothermia, hypothyroidism, hyperkalaemia);
- drug-induced (e.g., beta blockade, diltiazem, digoxin, amiodarone).

Assess the patient with bradycardia using the ABCDE approach. Consider the potential cause of the bradycardia and look for adverse features. Treat any reversible causes of bradycardia identified in the initial assessment. If adverse signs are present start to treat the bradycardia. Initial treatment is pharmacological, with pacing being reserved for patients unresponsive to pharmacological treatment or with risks factors for asystole.

Pharmacological treatment

If adverse signs are present, give atropine, 500 mcg, intravenously and, if necessary, repeat every 3-5 min to a total of 3 mg. Doses of atropine of less than 500 mcg have been reported to cause paradoxical slowing of the heart rate.²⁶⁴ In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart rate.²⁶⁵ Use atropine cautiously in the presence of acute coronary ischaemia or myocardial infarction; increased heart rate may worsen ischaemia or increase the zone of infarction. Do not give atropine to patients with cardiac transplants. Their hearts are denervated and will not respond to vagal blockade by atropine, which may cause paradoxical sinus arrest or high-grade AV block in these patients.²⁶⁶

If bradycardia with adverse signs persist despite atropine, consider cardiac pacing. If pacing cannot be achieved promptly consider the use of second-line drugs. Seek expert help to select the most appropriate choice. In some clinical settings second-line drugs may be appropriate before the use of cardiac pacing. For example consider giving intravenous glucagon if a beta-blocker or calcium channel blocker is a likely cause of the bradycardia. Consider using digoxin-specific antibody fragments for bradycardia due to digoxin toxicity. Consider using theophylline (100-200 mg by slow intravenous injection) for bradycardia complicating acute inferior wall myocardial infarction, spinal cord injury or cardiac transplantation.

Pacing

Transcutaneous pacing

Initiate transcutaneous pacing immediately if there is no response to atropine, or if atropine is unlikely to be effective. Transcutaneous pacing can be painful and may fail to achieve effective electrical capture (i.e. a QRS complex after the pacing stimulus) or fail to achieve a mechanical response (i.e. palpable pulse). Verify electrical capture on the monitor or ECG and check that it is producing a pulse. Reassess the patient's condition (ABCDE). Use analgesia and sedation as necessary to control pain; sedation may compromise respiratory effort so continue to reassess the patient at frequent intervals.

Fist pacing

If atropine is ineffective and transcutaneous pacing is not immediately available, fist pacing can be attempted while waiting for pacing equipment.²⁶⁷⁻²⁶⁹ Give serial rhythmic blows with the closed fist over the left lower edge of the sternum to stimulate the heart at a rate of 50-70 min⁻¹.

Transvenous pacing

Seek expert help to assess the need for temporary transvenous pacing and to initiate this when appropriate. Temporary transvenous pacing should be considered if there is documented recent asystole (ventricular standstill of more than 3 s), Mobitz type II AV block; complete (third-degree) AV block (especially with broad QRS or initial heart rate <40 beats min⁻¹).

Introduction

Changes in paediatric life support guidelines have been partly in response to new scientific evidence, and partly to simplify them in order to assist teaching and retention. As in the past, there remains a paucity of good quality evidence specifically on paediatric resuscitation, and some conclusions have had to be drawn from experimental work or extrapolated from adult data. Cardiorespiratory arrest is much less common in children than in adults and providers who are not specialists in paediatric practice will manage most cases initially. These considerations have emphasised the importance of providing simple, practical guidance, as well as rigorous assessment and incorporation of the best available scientific data.

There remains a strong focus on simplification where possible, based on the knowledge that many children receive no resuscitation at all because rescuers fear doing harm as they have not been taught paediatric resuscitation. Bystander resuscitation significantly improves outcome in children²¹ and there is evidence from experimental models that doing either chest compression or expired air ventilation alone may result in a better outcome than doing nothing. It follows that outcomes could be improved if bystanders who would otherwise do nothing, were encouraged to begin resuscitation, even if they do not follow an algorithm targeted specifically at children. However, there are distinct differences between the arrest of cardiac origin, seen predominately in adults, and the asphyxial arrest, which occurs commonly in children. Therefore, a separate paediatric algorithm is justified for healthcare professionals who have a duty to respond to paediatric emergencies and who are in a position to receive enhanced training.

Guideline changes

Recognition of cardiorespiratory arrest – healthcare provider and lay person

Pulse palpation for 10 s cannot give a reliable measurement of the presence or absence of an effective circulation. This means that palpation of the pulse cannot be the sole determinant of the need for chest compressions. Healthcare providers therefore need to determine the presence or absence of 'signs of life', such as response to stimuli, normal breathing (rather than abnormal gasps) or spontaneous movement. They may also perform pulse palpation but, if there are no other 'signs of life', they should only withhold CPR if they are certain that there is a definite pulse. The decision to start CPR should

take less than 10 s from the time of beginning the initial assessment of the child's circulatory status and if there is still doubt after that time, CPR should be initiated.

If the layperson considers that there are no 'signs of life', CPR should be started immediately.

Compression:Ventilation ratios – healthcare provider and lay person

Although ventilation remains a very important component of CPR in asphyxial arrest, rescuers who are unable or unwilling to provide this should be encouraged to perform at least compression-only CPR. A child is far more likely to be harmed if bystanders do nothing at all.

The CV ratio for resuscitation at birth remains 3:1 but there is uncertainty about the best ratio for use in neonates outside of the delivery room. The 2010 International Consensus on CPR Science²⁷⁰ suggests that the ratio should depend on aetiology but a literal application of this would be complex and confusing to apply in practice. The best approach is for individual units to decide on the CPR teaching that they provide for dealing with neonates based on the likely pathology that they encounter and the type of resuscitation that they perform most commonly.

Chest compression quality

Anthropomorphic and radiographic measurements in children have demonstrated that compression of the chest by one-third the AP diameter is feasible and safe. Data in adults and older children suggest that chest compressions are frequently too shallow, so there has been a subtle, but important, change in the instruction on chest compressions from "approximately one-third" to "at least one-third" of the AP diameter of the chest. A post-mortem review found that physical damage following CPR in children was very rare. It is reasonable to advise, "don't be afraid to push too hard".

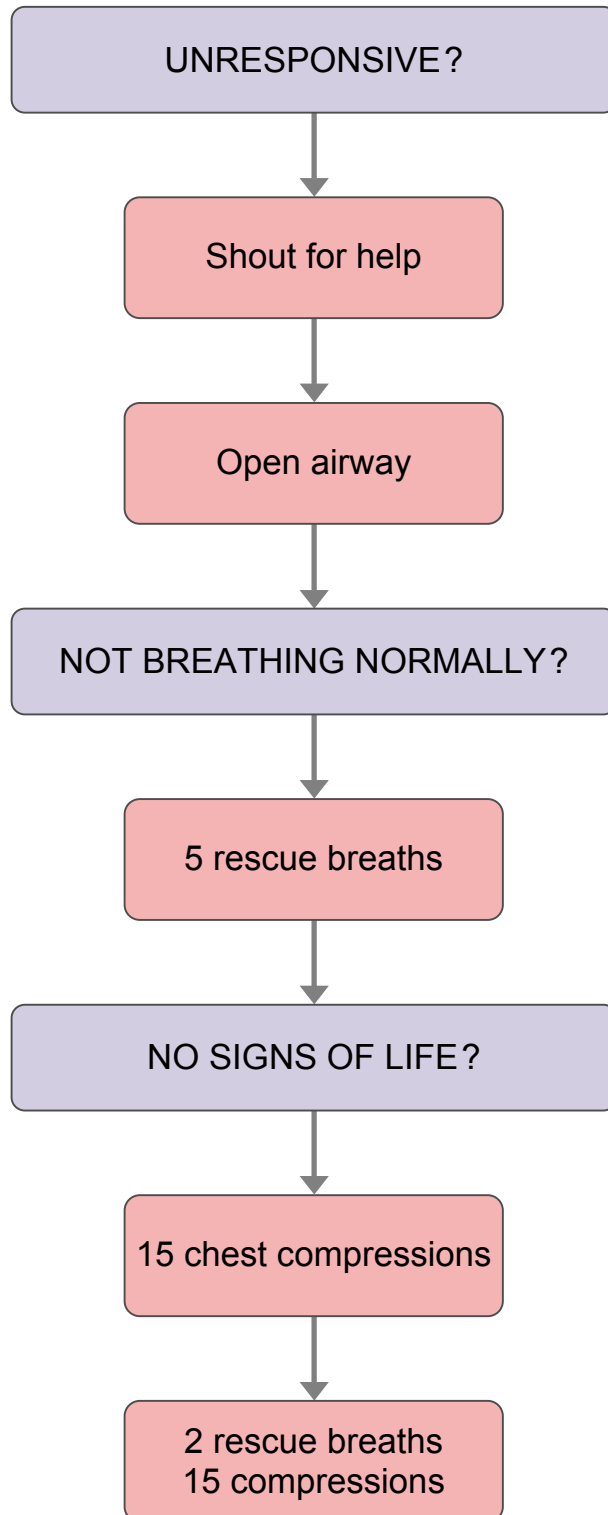
Training and feedback devices are being developed for adults but require absolute, rather than relative, dimensions. In order to facilitate the same for children, the measurement data above indicate that the mean dimensions for one-third compression depths for infants and children are 4 and 5 cm respectively.

In order to be consistent with the adult BLS guidelines the advice on compression rate has been amended to 'at least 100 but not greater than 120 min⁻¹'.

Automated external defibrillators (AEDs) in infants

Although there are only few data, evidence from case reports now favours the use of an AED (preferably with an attenuator) in infants with shockable rhythms, if no manually adjustable machine is available.^{271, 272} Shockable rhythms are unusual in children under 1 year of age and the main focus of resuscitation should be on good-quality CPR.

Paediatric Basic Life Support
(Healthcare professionals with a duty to respond)



Call resuscitation team

Infant and child BLS sequence

Rescuers who have been taught adult BLS, and have no specific knowledge of paediatric resuscitation, should use the adult sequence. The following modifications to the adult sequence will, however, make it more suitable for use in children:

- Give 5 initial rescue breaths before starting chest compression (adult sequence step 5B).
- If you are on your own, perform CPR for 1 min before going for help.
- Compress the chest by at least one-third of its depth. Use two fingers for an infant under 1 year; use one or two hands for a child over 1 year as needed to achieve an adequate depth of compression.

(See adult BLS chapter)

The following is the sequence that should be followed by those with a duty to respond to paediatric emergencies (usually healthcare professional teams):

- 1. Ensure the safety of rescuer and child.**
- 2. Check the child's responsiveness:**
 - Gently stimulate the child and ask loudly, 'Are you all right?'
 - Do not shake infants, or children with suspected cervical spine injuries.
- 3A. If the child responds by answering or moving:**
 - Leave the child in the position in which you find him (provided he is not in further danger).
 - Check his condition and get help if needed.
 - Reassess him regularly.
- 3B. If the child does not respond:**
 - Shout for help.
 - Turn the child onto his back and open the airway using head tilt and chin lift:
 - Place your hand on his forehead and gently tilt his head back.
 - With your fingertip(s) under the point of the child's chin, lift the chin. Do not push on the soft tissues under the chin as this may block the airway.
 - If you still have difficulty in opening the airway, try the jaw thrust method: place the first two fingers of each hand behind each side of the child's mandible (jaw bone) and push the jaw forward.

Have a low threshold for suspecting injury to the neck. If you suspect this, try to open the airway using chin lift or jaw thrust alone. If this is unsuccessful, add head tilt a small

amount at a time until the airway is open. Establishing an open airway takes priority over concerns about the cervical spine.

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

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

5A. If the child is breathing normally:

- Turn the child onto his side into the recovery position (see below).
- Send or go for help – call the relevant emergency number. Only leave the child if no other way of obtaining help is possible.
- Check for continued normal breathing.

5B. If the breathing is not normal or absent:

- Carefully remove any obvious airway obstruction.
- Give 5 initial rescue breaths.
- While performing the rescue breaths note any gag or cough response to your action. These responses, or their absence, will form part of your assessment of 'signs of life', described below.

Rescue breaths for a child over 1 year:

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

Rescue breaths for an infant:

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

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

- Open the child's mouth and remove any visible obstruction. Do not perform a blind finger sweep.
- Ensure that there is adequate head tilt and chin lift but also that the neck is not over extended.
- If head tilt and chin lift has not opened the airway, try the jaw thrust method.
- Make up to 5 attempts to achieve effective breaths. If still unsuccessful, move on to chest compression.

6. Assess the child's circulation (signs of life):

Take no more than 10 s to:

- Look for signs of life. These include any movement, coughing, or normal breathing (not abnormal gasps or infrequent, irregular breaths).
- If you check the pulse **take no more than 10 s**:
 - In a **child aged over 1 year** – feel for the carotid pulse in the neck.
 - In **an infant** – feel for the brachial pulse on the inner aspect of the upper arm.
 - For both infants and children the femoral pulse in the groin (mid way between the anterior superior iliac spine and the symphysis pubis) can also be used.

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

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

- Re-assess the child frequently.

7B. If there are no signs of life, unless you are CERTAIN that you can feel a definite pulse of greater than 60 min⁻¹ within 10 s

- Start chest compression.
- Combine rescue breathing and chest compression.

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

- To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Compress the sternum one finger's breadth above this.
- Compression should be sufficient to depress the sternum by at least one-third of the depth of the chest.
- Don't be afraid to push too hard. Push "hard and fast".
- Release the pressure completely, then repeat at a rate of 100 - 120 min⁻¹
- After 15 compressions, tilt the head, lift the chin, and give two effective breaths.
- Continue compressions and breaths in a ratio of 15:2.

The best method for compression varies slightly between infants and children.

Chest compression in infants:

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

Chest compression in children aged over 1 year:

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

8. Continue resuscitation until:

- The child shows signs of life (normal breathing, cough, movement or definite pulse of greater than 60 min⁻¹).
- Further qualified help arrives.
- You become exhausted.

When to call for assistance

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

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

Recovery position

An unconscious child whose airway is clear and who is breathing normally should be turned onto his side into the recovery position. There are several recovery positions; each has its advocates. The important principles to be followed are:

- The child should be placed in as near a true lateral position as possible with his mouth dependant to enable free drainage of fluid.
- The position should be stable. In an infant, this may require the support of a small pillow or a rolled-up blanket placed behind his back to maintain the position.
- There should be no pressure on the chest that impairs breathing.
- It should be possible to turn the child onto his side and to return him back easily and safely, taking into consideration the possibility of cervical spine injury.
- The airway should be accessible and easily observed.
- The adult recovery position is suitable for use in children.

Explanatory notes

Definitions

- A **newborn** is a child just after birth.
- A **neonate** is a child in the first 28 days of life.
- An **infant** is a child under 1 year.
- A **child** is between 1 year and puberty.

The differences between adult and paediatric resuscitation are largely based on differing aetiology, with primary cardiac arrest being more common in adults whereas children usually suffer from secondary cardiac arrest. The onset of puberty, which is the physiological end of childhood, is the most logical landmark for the upper age limit for use of paediatric guidelines. This has the advantage of being simple to determine in contrast to an age limit, as age may be unknown at the start of resuscitation. Clearly, it is inappropriate and unnecessary to establish the onset of puberty formally; if the rescuer believes the victim to be a child then he should use the paediatric guidelines. If a misjudgment is made, and the victim turns out to be a young adult, little harm will accrue as studies of aetiology have shown that the paediatric pattern of arrest continues into early adulthood.

It is necessary to differentiate between infants and older children, as there are some important differences between these two groups.

Compression:ventilation ratios

The 2010 International Liaison Committee on Resuscitation (ILCOR) Consensus on Science with Treatment Recommendations (CoSTR), continues with the 2005 recommendation that the CV ratio should be based on whether one or more rescuers are present.²⁷¹ The 2010 CoSTR recommends that lay rescuers, who usually learn only single-rescuer techniques, should be taught to use a ratio of 30 compressions to 2 ventilations. This is the same ratio as recommended for adults and enables anyone trained in BLS techniques to resuscitate children with minimal additional information. Two or more rescuers with a duty to respond should learn a ratio with more rescue breaths (15:2), as this has been validated by experimental and mathematical studies.²⁷³²⁷⁴ This latter group, who would normally be healthcare professionals, should receive enhanced training targeted specifically at the resuscitation of children.

Although the 2010 CoSTR recommendation is based on the number of rescuers present, it would certainly negate the main benefit of simplicity if lay rescuers were taught a different ratio for use if there were two of them. Similarly, those with a duty to respond, who would normally be taught to use a ratio of 15:2, do not have to use the 30:2 ratio if they are alone, unless they are not achieving an adequate number of compressions because of difficulty in the transition between ventilation and compression.

Chest compression technique

The 2010 guidelines prioritise chest compressions. Subtle changes in wording (*at least* one-third depth, *at least* 100 min⁻¹) encourage harder and faster compressions. The evidence suggests that rescuers are too gentle and slow and that harm is unlikely, either through the use of excessive force or through performing chest compressions in a victim who has a spontaneous circulation. Interruptions are minimised by not stopping compressions during defibrillator charging, immediate resumption after shock delivery and continuing without a pause for breaths, once the trachea is intubated.

Automated external defibrillators

Since the publication of Guidelines 2005 there have been continuing reports of safe and successful use of AEDs in children less than 8 years and further studies demonstrating that AEDs are capable of identifying arrhythmias accurately in children and are extremely unlikely to advise a shock inappropriately. **Nevertheless, if there is any possibility that an AED may need to be used in children, the purchaser should check that the performance of the particular model has been tested in paediatric arrhythmias.**

Many manufacturers now supply purpose-made paediatric pads or programmes, which typically attenuate the output of the machine to 50-75 J.²⁷⁵ These devices are recommended for children between 1 and 8 years. If no such system or manually adjustable machine is available, an unmodified adult AED may be used.

Although shockable rhythms are extremely unusual in infants, there are rare case reports of the successful use of AEDs in this age group. For an infant in a shockable rhythm, the risk:benefit ratio favours the use of an AED (ideally with an attenuator) if a manually adjustable model is not available.

Choking

Recognition of choking

No new evidence on this subject was presented during the 2010 Consensus Conference. Back blows, chest thrusts and abdominal thrusts all increase intra-thoracic pressure and can expel foreign bodies from the airway. In half of the episodes more than one technique is needed to relieve the obstruction. There are no data to indicate which measure should be used first or in which order they should be applied. If one is unsuccessful, try the others in rotation until the object is cleared.

When a foreign body enters the airway the child reacts immediately by coughing in an attempt to expel it. A spontaneous cough is likely to be more effective and safer than any manoeuvre a rescuer might perform. However, if coughing is absent or ineffective, and the object completely obstructs the airway, the child will become asphyxiated rapidly. Active interventions to relieve choking are therefore required only when

coughing becomes ineffective, but they then must be commenced rapidly and confidently.

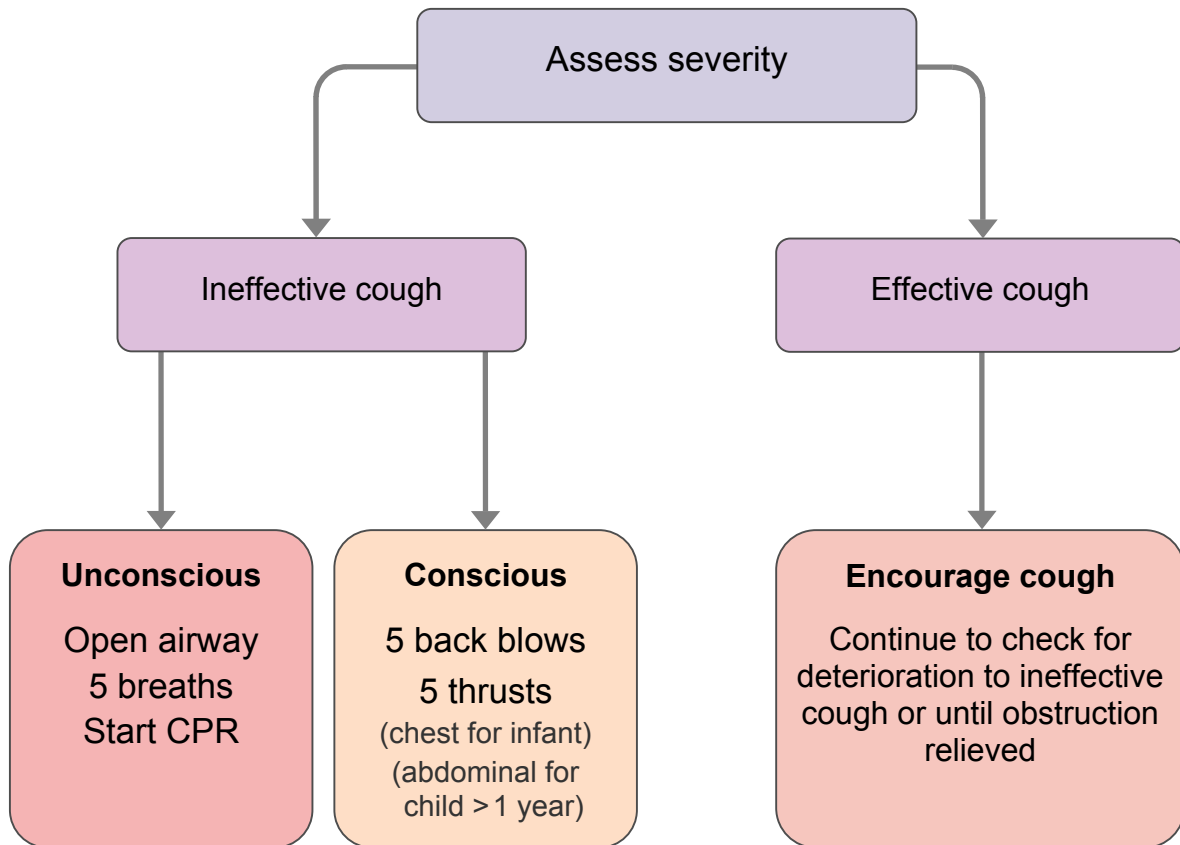
The majority of choking events in children occur during play or whilst eating, when a carer is usually present. Events are therefore frequently witnessed, and interventions are usually initiated when the child is conscious.

Choking is characterised by the sudden onset of respiratory distress associated with coughing, gagging, or stridor. Similar signs and symptoms may also be associated with other causes of airway obstruction, such as laryngitis or epiglottitis, which require different management. Suspect choking caused by a foreign body if:

- the onset was very sudden;
- there are no other signs of illness;
- there are clues to alert the rescuer, for example a history of eating or playing with small items immediately prior to the onset of symptoms.

General signs of choking	
<ul style="list-style-type: none"> • Witnessed episode • Coughing or choking • Sudden onset • Recent history of playing with or eating small objects 	
Ineffective coughing	Effective cough
<ul style="list-style-type: none"> • Unable to vocalise • Quiet or silent cough • Unable to breathe • Cyanosis • Decreasing level of consciousness 	<ul style="list-style-type: none"> • Crying or verbal response to questions • Loud cough • Able to take a breath before coughing • Fully responsive

Paediatric Choking Treatment Algorithm



Relief of choking

Safety and summoning assistance

Safety is paramount. Rescuers should avoid placing themselves in danger and consider the safest action to manage the choking child:

- If the child is coughing effectively, then no external manoeuvre is necessary. Encourage the child to cough, and monitor continuously.
- If the child's coughing is, or is becoming, ineffective, **shout for help** immediately and determine the child's conscious level.

Conscious child with choking

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

Back blows

► In an infant:

- Support the infant in a head-downwards, prone position, to enable gravity to assist removal of the foreign body.
- A seated or kneeling rescuer should be able to support the infant safely across his lap.
- Support the infant's head by placing the thumb of one hand at the angle of the lower jaw, and one or two fingers from the same hand at the same point on the other side of the jaw.
- Do not compress the soft tissues under the infant's jaw, as this will exacerbate the airway obstruction.
- Deliver up to 5 sharp back blows with the heel of one hand in the middle of the back between the shoulder blades.
- The aim is to relieve the obstruction with each blow rather than to give all 5.

► In a child over 1 year:

- Back blows are more effective if the child is positioned head down.
- A small child may be placed across the rescuer's lap as with an infant.
- If this is not possible, support the child in a forward-leaning position and deliver the back blows from behind.

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

Chest thrusts for infants:

- Turn the infant into a head-downwards supine position. This is achieved safely by placing your free arm along the infant's back and encircling the occiput with your hand.
- Support the infant down your arm, which is placed down (or across) your thigh.
- Identify the landmark for chest compression (lower sternum approximately a finger's breadth above the xiphisternum).
- Deliver up to 5 chest thrusts. These are similar to chest compressions, but sharper in nature and delivered at a slower rate.
- The aim is to relieve the obstruction with each thrust rather than to give all 5.

Abdominal thrusts for children over 1 year:

- Stand or kneel behind the child. Place your arms under the child's arms and encircle his torso.
- Clench your fist and place it between the umbilicus and xiphisternum.
- Grasp this hand with your other hand and pull sharply inwards and upwards.
- Repeat up to 4 more times.
- Ensure that pressure is not applied to the xiphoid process or the lower rib cage as this may cause abdominal trauma.
- The aim is to relieve the obstruction with each thrust rather than to give all 5.

Following chest or abdominal thrusts, reassess the child:

- If the object has not been expelled and the victim is still conscious, continue the sequence of back blows and chest (for infant) or abdominal (for children) thrusts.
- Call out, or send, for help if it is still not available.
- Do not leave the child at this stage.

If the object is expelled successfully, assess the child's clinical condition. It is possible that part of the object may remain in the respiratory tract and cause complications. If there is any doubt, seek medical assistance.

Unconscious child with choking

- If the choking child is, or becomes, unconscious place him on a firm, flat surface.
- Call out, or send, for help if it is still not available.
- Do not leave the child at this stage.

Airway opening:

- Open the mouth and look for any obvious object.
- If one is seen, make an attempt to remove it with a single finger sweep.

Do not attempt blind or repeated finger sweeps – these can impact the object more deeply into the pharynx and cause injury.

Rescue breaths:

- Open the airway and attempt 5 rescue breaths.
- Assess the effectiveness of each breath: if a breath does not make the chest rise, reposition the head before making the next attempt.

Chest compression and CPR:

- Attempt 5 rescue breaths and if there is no response, proceed immediately to chest compression regardless of whether the breaths are successful.
- Follow the sequence for single rescuer CPR (step 7B above) for approximately 1 min before summoning the EMS (if this has not already been done by someone else).
- When the airway is opened for attempted delivery of rescue breaths, look to see if the foreign body can be seen in the mouth.
- If an object is seen, attempt to remove it with a single finger sweep.
- If it appears that the obstruction has been relieved, open and check the airway as above. Deliver rescue breaths if the child is not breathing and then assess for signs of life. If there are none, commence chest compressions and perform CPR (step 7B above).
- If the child regains consciousness and is breathing effectively, place him in a safe side-lying (recovery) position and monitor breathing and conscious level whilst awaiting the arrival of the EMS.

Introduction

There is concern that resuscitation from cardiac arrest is not performed as well as it might because the variations in guidelines for different age groups cause confusion to providers, and therefore poor performance. As in 2005, most of the changes in paediatric guidelines for 2010 have been made for simplification and to minimise differences between adult and paediatric protocols. It is hoped that this will assist teaching and retention.

There remains a paucity of good quality evidence on which to base the resuscitation of infants and children. Most conclusions have had to be drawn from extrapolated adult studies and from experimental work.

In children, secondary cardiopulmonary arrests, caused by either respiratory or circulatory failure, are more frequent than primary arrests caused by arrhythmias.²⁷⁶ So-called asphyxial arrests or respiratory arrests are also more common in young adulthood (e.g. trauma, drowning, poisoning). The outcome from cardiopulmonary arrests in children is poor²¹ and identification of the antecedent stages of cardiac or respiratory failure is a priority, as effective early intervention may be life-saving.

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

- A** indicates airway (**A_c** for airway and cervical spine stabilisation for the injured child).
- B** indicates breathing.
- C** indicates circulation (with haemorrhage control in injured child).
- D** indicates disability (level of consciousness and neurological status).
- E** indicates exposure to ensure full examination (whilst respecting dignity and temperature conservation).

Interventions are made at each step of the assessment as abnormalities are identified. The next step of the assessment is not started until the preceding abnormality has been managed and corrected if possible. Summoning a paediatric rapid response team or medical emergency team may reduce the risk of respiratory and/or cardiac arrest in hospitalised children outside the intensive care setting.⁸⁴ This team should include at least one paediatric specialist and one specialised nurse and should be called to evaluate a potentially critically ill child who is not already in a paediatric intensive care unit (PICU) or paediatric emergency department (ED).

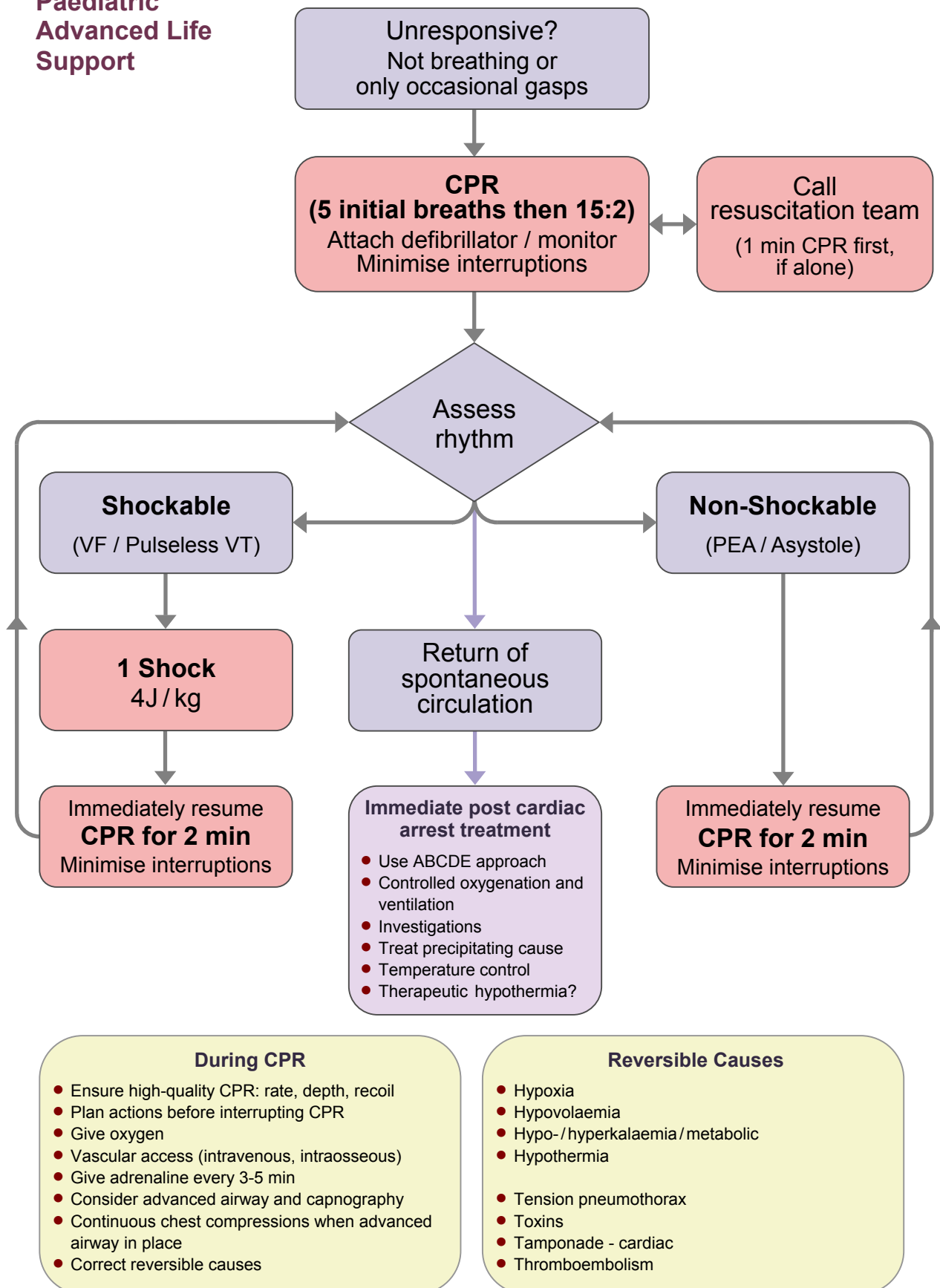
Guideline changes

- Adrenaline is given after the third shock for shockable rhythms and then during every alternate cycle (i.e. every 3-5 min during CPR). Adrenaline is still initially given as soon as vascular access is available in the non-shockable side of the algorithm.
- Amiodarone is given after the third shock for shockable rhythms. The dose is repeated after the fifth shock, if still in ventricular fibrillation/pulseless VT (VF/VT).
- Bag-mask ventilation remains the preferred method for achieving airway control and ventilation. If this fails, the laryngeal mask airway (or possibly other supraglottic airway device) is an acceptable alternative for suitably trained providers.
- Once spontaneous circulation has been restored, delivered oxygen should be titrated to limit the risk of hyperoxaemia.
- CO₂ detection (preferably with capnography) is even more strongly encouraged, not only to confirm placement of tracheal tubes but also to aid decision making during cardiopulmonary resuscitation (CPR) and management of ventilation after return of spontaneous circulation (ROSC).
- Post-resuscitation care should include consideration of induced hypothermia.

Sequence of actions

1. **Establish basic life support (see paediatric BLS chapter).**
2. **Oxygenate, ventilate, and start chest compression:**
 - Provide positive-pressure ventilation with high-concentration inspired oxygen.
 - Provide ventilation initially by bag and mask. Ensure a patent airway by using an airway manoeuvre as described in the paediatric basic life support chapter.
 - If it can be performed by a highly skilled operator with minimal interruption to chest compressions, the trachea should be intubated. This will both control the airway and enable chest compression to be given continuously, thus improving coronary perfusion pressure.
 - Take care to ensure that ventilation remains effective when continuous chest compressions are started.
 - Use a compression rate of 100 - 120 min⁻¹
 - Once the child has been intubated and compressions are uninterrupted, use a ventilation rate of approximately 10 - 12 min⁻¹.

**Paediatric
Advanced Life
Support**



Sequence of actions (continued)

3. Attach a defibrillator or monitor:

- Assess and monitor the cardiac rhythm.
- If using a defibrillator, place one defibrillator pad or paddle on the chest wall just below the right clavicle, and one in the mid-axillary line.
- Pads or paddles for children should be 8 - 12 cm in size, and 4.5 cm for infants. In infants and small children it may be best to apply the pads or paddles to the front and back of the chest if they cannot be adequately separated in the standard positions.
- If used, place monitoring electrodes in the conventional chest positions.

4. Assess rhythm and check for signs of life:

- Look for signs of life, which include responsiveness, coughing, and normal breathing.
- Assess the rhythm on the monitor:
 - Non-shockable (asystole or pulseless electrical activity (PEA)) **OR**
 - Shockable (VF/VT).

5A. Non-shockable (asystole or PEA):

This is the more common finding in children.

- **Perform continuous CPR:**
 - Continue to ventilate with high-concentration oxygen.
 - If ventilating with bag-mask give 15 chest compressions to 2 ventilations.
 - Use a compression rate of 100 - 120 min⁻¹.
 - If the patient is intubated, chest compressions can be continuous as long as this does not interfere with satisfactory ventilation.
 - Once the child has been intubated and compressions are uninterrupted use a ventilation rate of approximately 10 - 12 min⁻¹.

Note: Once there is ROSC, the ventilation rate should be 12 - 20 min⁻¹. Measure exhaled CO₂ to monitor ventilation and ensure correct tracheal tube placement.

- **Give adrenaline:**
 - If venous or intraosseous (IO) access has been established, give adrenaline 10 mcg kg⁻¹ (0.1 ml kg⁻¹ of 1 in 10,000 solution).
 - If there is no circulatory access, attempt to obtain IO access.
 - If circulatory access is not present, and cannot be obtained quickly, but the patient has a tracheal tube in place, consider giving adrenaline 100 mcg kg⁻¹ via the tracheal tube. This is the least satisfactory route (see routes of drug administration).
- **Continue CPR, only pausing briefly every 2 min to check for rhythm change.**

- Give adrenaline 10 mcg kg⁻¹ every 3 to 5 min (i.e. every other loop), while continuing to maintain effective chest compression and ventilation without interruption.
- **Consider and correct reversible causes:**
 - Hypoxia
 - Hypovolaemia
 - Hyper/hypokalaemia (electrolyte disturbances)
 - Hypothermia
 - Tension pneumothorax
 - Toxic/therapeutic disturbance
 - Tamponade (cardiac)
 - Thromboembolism
- **Consider the use of other medications such as alkalisating agents.**

5B. Shockable (VF/VT)

This is less common in paediatric practice but may occur as a secondary event and is likely when there has been a witnessed and sudden collapse. It is commoner in the intensive care unit and cardiac ward.

- **Continue CPR until a defibrillator is available.**
- **Defibrillate the heart:**
 - Charge the defibrillator while another rescuer continues chest compressions.
 - Once the defibrillator is charged, pause the chest compressions, quickly ensure that all rescuers are clear of the patient and then deliver the shock. This should be planned before stopping compressions.
 - Give 1 shock of 4 J kg⁻¹ if using a manual defibrillator.
 - If using an AED for a child of less than 8 years, deliver a paediatric-attenuated adult shock energy.
 - If using an AED for a child over 8 years, use the adult shock energy.
- **Resume CPR:**
 - Without reassessing the rhythm or feeling for a pulse, resume CPR **immediately**, starting with chest compression.
 - Consider and correct reversible causes (4Hs and 4Ts).

- **Continue CPR for 2 min, then pause briefly to check the monitor:**
 - If still VF/VT, **give a second shock** (with same energy level and strategy for delivery as the first shock).
- **Resume CPR:**
 - Without reassessing the rhythm or feeling for a pulse, resume CPR **immediately**, starting with chest compression.
- **Continue CPR for 2 min, then pause briefly to check the monitor:**
- **If still VF/VT, give a third shock** (with same energy level and strategy for delivery as the previous shock).
- **Resume CPR:**
 - Without reassessing the rhythm or feeling for a pulse, resume CPR **immediately**, starting with chest compression.
 - Give adrenaline 10 mcg kg⁻¹ and amiodarone 5 mg kg⁻¹ after the 3rd shock, once chest compressions have resumed.
 - Repeat adrenaline every alternate cycle (i.e. every 3-5 min) until ROSC.
 - Repeat amiodarone 5 mg kg⁻¹ one further time, after the 5th shock if still in a shockable rhythm.

Continue giving shocks every 2 min, continuing compressions during charging of the defibrillator and minimising the breaks in chest compression as much as possible.

Note: After each 2 min of uninterrupted CPR, pause briefly to assess the rhythm.

- **If still VF/VT:**
 - Continue CPR with the shockable (VF/VT) sequence.
- **If asystole:**
 - Continue CPR and switch to the non-shockable (asystole or PEA) sequence as above.
- **If organised electrical activity is seen**, check for signs of life and a pulse:
 - If there is ROSC, continue post-resuscitation care.
 - If there is **no** pulse (or a pulse rate of < 60 min⁻¹), and there are no other signs of life, continue CPR and continue as for the non-shockable sequence above.

If defibrillation was successful but VF/VT recurs, resume the CPR sequence and defibrillate. Give an amiodarone bolus (unless 2 doses have already been given) and start a continuous infusion.

Important note

Uninterrupted, good-quality CPR is vital. Chest compression and ventilation should be interrupted only for defibrillation. Chest compression is tiring for providers. The team leader should continuously assess and feed back on the quality of the compressions, and change the providers every 2 min.

Explanatory notes

Shockable rhythm sequence

The change in timing of administration of adrenaline and amiodarone has been in response to the change in the adult algorithm. There is no evidence that the treatment of VF should differ fundamentally from adult practice except that seeking and treating the reversible causes is particularly important in children because arrhythmias are unlikely to be due to coronary artery disease.

Shock energy level

The ideal energy level for safe and effective defibrillation in children is unknown. The recommendation of 2 - 4 J kg⁻¹ in Guidelines 2000 was based on a single historical study of effective outcomes. Extrapolation from adult data and experimental studies shows that biphasic shocks are at least as effective as monophasic shocks and produce less post-shock myocardial dysfunction. Clinical studies have shown that an initial monophasic or biphasic shock level of 2 J kg⁻¹ has a low success rate in paediatric VF.²⁷⁷⁻²⁷⁹ Paediatric case series have reported that shock levels of more than 4 J kg⁻¹ (up to 9 J kg⁻¹) have effectively defibrillated children less than 12 years of age with negligible adverse effects. In experimental studies, high energy levels cause less myocardial damage in young hearts than in adult hearts.

A single 4 J kg⁻¹ shock strategy improves first shock success rate and minimises interruption in chest compressions.

Tracheal tubes

Recent studies continue to show no greater risk of complications for children less than 8 years when cuffed, rather than uncuffed, tracheal tubes are used in the operating room and intensive care unit. Cuffed tracheal tubes are as safe as uncuffed tubes for infants (except neonates) and children if rescuers use the correct tube size and cuff inflation pressure, and verify tube position. The use of cuffed tubes increases the chance of selecting the correct size at the first attempt. Under certain circumstances (e.g. poor lung compliance, high airway resistance, and large glottic air leak) cuffed tracheal tubes may be preferable.

Alternative airways

Although bag-mask ventilation remains the recommended first line method for achieving airway control and ventilation in children, the LMA is an acceptable airway device for providers trained in its use. It is particularly helpful in airway obstruction caused by supraglottic airway abnormalities or if bag-mask ventilation is not possible. The LMA does not totally protect the airway from aspiration of secretions, blood or stomach contents, and therefore close observation is required. Use of the LMA is associated with a higher incidence of complications in small children compared with adults. Other supraglottic airway devices (e.g. laryngeal tube), which have been used successfully in children's anaesthesia, may also be useful, but there are few data on the use of these devices in paediatric emergencies.

Capnography

Monitoring end tidal CO_2 (ET CO_2) (preferably with capnography) reliably confirms tracheal tube placement in a child weighing more than 2 kg with a perfusing rhythm, and its use is strongly recommended after intubation, and during transport of an intubated child. The presence of a capnographic waveform for more than four ventilated breaths indicates that the tube is in the tracheobronchial tree, both in the presence of a perfusing rhythm and during cardiopulmonary arrest with CPR. Capnography does not rule out intubation of a bronchus. The absence of exhaled CO_2 during CPR does not guarantee tube misplacement because a low or absent end tidal CO_2 may reflect low or absent pulmonary blood flow.

Capnography may also provide information on the efficiency of chest compressions and a sudden rise in exhaled CO_2 can give an early indication of ROSC. Efforts should be made to improve chest compression quality if the ET CO_2 remains below 2 kPa as this may indicate low cardiac output and pulmonary blood flow. Care must be taken when interpreting ET CO_2 values after the administration of adrenaline or other vasoconstrictor drugs when there may be a transient decrease in values, or after the use of sodium bicarbonate when there may be a transient increase. Current evidence does not support the use of a threshold ET CO_2 value as an indicator for stopping the resuscitation attempt.

Routes of drug administration

Although atropine, adrenaline, naloxone, lidocaine and vasopressin are absorbed from the tracheobronchial tree, much lower blood concentrations result than if the same dose were given intravascularly. Conversely, good quality evidence in both adults and children show that intraosseous (IO) access is safe and effective and this route is therefore far preferable to tracheal administration, which should be used only if there is no alternative. Semi-automated devices for inserting IO needles are available. Although there are few data to support their use in children during CPR, reports of their use in other circumstances have shown them to be effective.

Drugs used in CPR

Adrenaline

This is an endogenous catecholamine with potent alpha, beta₁, and beta₂ adrenergic actions. Although it is central to the treatment algorithms both for non-shockable and shockable cardiac arrest rhythms, a prospective randomised adult study of the use of drugs (including adrenaline) in CPR showed an improvement in ROSC but not in long-term neurologically intact survival.¹⁴⁴ Adrenaline induces vasoconstriction, increases coronary perfusion pressure, enhances the contractile state of the heart, stimulates spontaneous contractions, and increases the intensity of VF so increasing the likelihood of successful defibrillation.

The recommended IV/IO dose of adrenaline in children is 10 mcg kg⁻¹. Subsequent doses of adrenaline should, if needed, be given every 3-5 min. Higher doses of intravascular adrenaline should not be used routinely in children because this may worsen outcome.

Amiodarone

Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Atrioventricular conduction is also slowed, and a similar effect is seen in accessory pathways. Amiodarone has a mild negative inotropic action and causes peripheral vasodilation through non-competitive alpha-blocking effects. The hypotension that occurs with IV amiodarone is related to the rate of delivery and is due more to the solvent (Polysorbate 80 and benzyl alcohol), which causes histamine release, than the drug itself.

In the treatment of shockable rhythms, give an initial IV bolus dose of amiodarone 5 mg kg⁻¹ after the third shock. Repeat the dose after the fifth shock if still in VF/VT. If defibrillation was successful but VF/VT recurs, amiodarone can be repeated (unless two doses have already been injected) and a continuous infusion started. Amiodarone can cause thrombophlebitis when injected into a peripheral vein and, ideally, should be administered via a central vein. If central venous access is unavailable (likely at the time of cardiac arrest) and it has to be given peripherally, it should be flushed liberally with 0.9% sodium chloride or 5% glucose.

Atropine

Atropine is effective in increasing heart rate when bradycardia is caused by excessive vagal tone (e.g. after insertion of nasogastric tube). The dose is 20 mcg kg⁻¹ and a minimum dose of 100 mcg should be given to avoid a paradoxical effect at low doses. There is no evidence that atropine has any benefit in asphyxial bradycardia or asystole and its routine use has been removed from the ALS algorithms.

Magnesium

This is a major intracellular cation and serves as a cofactor in many enzymatic reactions. Magnesium treatment is indicated in children with documented hypomagnesemia or with polymorphic VT (torsade de pointes), regardless of cause.

Calcium

Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction, but high plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may also impair cerebral recovery. The routine administration of calcium during cardiac arrest has been associated with increased mortality and it should be given only when specifically indicated, for example in hyperkalaemia, hypocalcaemia, and overdose of calcium-channel-blocking drugs.

Sodium bicarbonate

Cardiac arrest results in combined respiratory and metabolic acidosis, caused by cessation of pulmonary gas exchange, and the development of anaerobic cellular metabolism respectively. The best treatment for acidaemia in cardiac arrest is a combination of effective chest compression and ventilation (good quality CPR). Administration of sodium bicarbonate generates carbon dioxide, which diffuses rapidly into the cells, exacerbating intracellular acidosis if it is not rapidly cleared via the lungs. It also has the following detrimental effects:

- It produces a negative inotropic effect on an ischaemic myocardium.
- It presents a large, osmotically active, sodium load to an already compromised circulation and brain.
- It produces a shift to the left in the oxygen dissociation curve further inhibiting release of oxygen to the tissues.

The routine use of sodium bicarbonate in cardiac arrest is not recommended. It may be considered in prolonged arrest, and it has a specific role in hyperkalaemia and the arrhythmias associated with tricyclic antidepressant overdose.

Fluids for resuscitation

Hypovolaemia is a potentially reversible cause of cardiac arrest. If hypovolaemia is suspected, infuse intravenous or intraosseous fluids rapidly (20 ml kg⁻¹ boluses). In the initial stages of resuscitation there are no clear advantages in using colloid solutions, whatever the aetiology, so use isotonic saline solutions for initial volume resuscitation. Do not use dextrose-based solutions for volume replacement – these will be redistributed rapidly away from the intravascular space and will cause hyponatraemia and hyperglycaemia, which may worsen neurological outcome.^{222, 232, 280}

Post-resuscitation care

Oxygen

There is increasing evidence that hyperoxaemia can be detrimental and studies in neonates suggest some advantages in using room air during initial resuscitation (see Newborn Life Support).²⁷⁰ In the older child there is no evidence for any such advantages, so 100% oxygen should be used for initial resuscitation. After ROSC, inspired oxygen should be titrated, using pulse oximetry, to achieve an oxygen saturation of 94 - 98%.^{281, 282} In situations where dissolved oxygen plays an important role in oxygen transport such as smoke inhalation (carbon monoxide poisoning) and severe anaemia, maintain a high inspired oxygen (FiO₂).

Therapeutic hypothermia

Hypothermia is common in the child following cardiopulmonary resuscitation.²³¹ Central hypothermia (32-34°C) may be beneficial, whereas fever may be detrimental to the injured brain. Mild hypothermia has an acceptable safety profile in adults and neonates and, although it has been shown to improve neurological outcome in adults after VF arrest, an observational study neither supports nor refutes the use of therapeutic hypothermia in paediatric cardiac arrest.²⁸³

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

Complications of mild therapeutic hypothermia include increased risk of infection, cardiovascular instability, coagulopathy, hyperglycaemia, and electrolyte abnormalities such as hypophosphataemia and hypomagnesaemia.

Hyperthermia is associated with a poorer outcome,^{230, 284, 285} so infants and children with core temperatures over 37.5°C should be cooled actively to a normal level.

At the time of writing, there are ongoing, prospective, multicentre trials of therapeutic hypothermia in children following in and out-of-hospital cardiac arrest. (See the US [National Institutes of Health Clinical Trials](#) studies NCT00880087 and NCT00878644). The results from these may change this advice.

Blood glucose control

Neonatal, child and adult data show that both hyper- and hypo- glycaemia are associated with poor outcome after cardiopulmonary arrest but it is uncertain if this is causative or merely an association. Plasma glucose concentrations should be monitored closely in any ill or injured child, including after cardiac arrest. Do not give glucose-containing fluids during CPR except for treatment of hypoglycaemia.

Hyper- and hypo- glycaemia should be avoided following ROSC but tight glucose control has not shown survival benefits when compared with moderate glucose control in adults and increased the risk of hypoglycaemia in neonates, children and adults.

Parental presence

Many parents would like to be present during a resuscitation attempt; they can see that everything possible is being done for their child. Reports show that being at the side of the child is comforting to the parents or carers, and helps them to gain a realistic view of attempted resuscitation and death.²⁸⁶ Bereaved families who have been present in the resuscitation room show less anxiety and depression several months after the death.

Parental presence in the resuscitation room may also encourage healthcare providers' professional behaviour and facilitate their understanding of the child in the context of his family.

A dedicated staff member should be present with the parents at all times to explain the process in an empathetic and sympathetic manner. They can also ensure that the parents do not interfere with the resuscitation process or distract the resuscitation team. If the presence of the parents is impeding the progress of the resuscitation, they should be gently asked to leave. When appropriate, physical contact with the child should be allowed.

The resuscitation team leader, not the parents, will decide when to stop the resuscitation effort; this should be expressed with sensitivity and understanding. After the event, debriefing of the team should be conducted, to express any concerns and to allow the team to reflect on their clinical practice in a supportive environment.

Introduction

Passage through the birth canal is a hypoxic experience for the fetus, since significant respiratory exchange at the placenta is prevented for the 50-75 s duration of the average contraction. Though most babies tolerate this well, the few that do not may require help to establish normal breathing at delivery. Newborn life support (NLS) is intended to provide this help and comprises the following elements:

- drying and covering the newborn baby to conserve heat;
- assessing the need for any intervention;
- opening the airway;
- aerating the lung;
- rescue breathing;
- chest compression;
- administration of drugs (rarely).

Physiology

If subjected to sufficient hypoxia in utero, the fetus will attempt to breathe. If the hypoxic insult is continued the fetus will eventually lose consciousness. Shortly after this the neural centres controlling these breathing efforts will cease to function because of lack of oxygen. The fetus then enters a period known as primary apnoea.

Up to this point, the heart rate remains unchanged, but soon decreases to about half the normal rate as the myocardium reverts to anaerobic metabolism – a less fuel-efficient mechanism. The circulation to non-vital organs is reduced in an attempt to preserve perfusion of vital organs. The release of lactic acid, a by-product of anaerobic metabolism, causes deterioration of the biochemical milieu.

If the insult continues, shuddering (whole-body gasps at a rate of about 12 min⁻¹) are initiated by primitive spinal centres. If the fetus is still in utero, or if for some other reason these gasps fail to aerate the lungs, they fade away and the fetus enters a period known as secondary, or terminal, apnoea. Until now, the circulation has been maintained but, as terminal apnoea progresses, the rapidly deteriorating biochemical milieu begins to impair cardiac function. The heart eventually fails and, without effective intervention, the baby dies. The whole process probably takes almost 20 min in the term newborn human baby.

Thus, in the face of asphyxia, the baby can maintain an effective circulation throughout the period of primary apnoea, through the gasping phase, and even for a while after the onset of terminal apnoea. Thus, the most urgent requirement for any asphyxiated baby at birth is that the lungs be aerated effectively. Provided the baby's circulation is sufficient, oxygenated blood will then be conveyed from the aerated lungs to the heart. The heart rate will increase and the brain will be perfused with oxygenated blood. Following this, the neural centres responsible for normal breathing will, in many instances, function once again and the baby will recover.

Merely aerating the lungs is sufficient in the vast majority of cases. Although lung aeration is still vital, in a few cases cardiac function will have deteriorated to such an extent that the circulation is inadequate and cannot convey oxygenated blood from the aerated lungs to the heart. In this case, a brief period of chest compression may be needed. In a very few cases, lung aeration and chest compression will not be sufficient, and drugs may be required to restore the circulation. The outlook in this group of infants is poor.

Important guideline changes

The following are the main changes that have been made to the NLS guidelines in 2010:^{270, 287}

- For uncompromised babies, a delay in cord clamping of at least one minute from the complete delivery of the infant, is now recommended. As yet there is insufficient evidence to recommend an appropriate time for clamping the cord in babies who are severely compromised at birth. For babies requiring resuscitation, resuscitative intervention remains the priority.
- For term infants, air should be used for resuscitation at birth. If, despite effective ventilation, oxygenation (ideally guided by pulse oximetry) remains unacceptable, use of a higher concentration of oxygen should be considered.
- Preterm babies less than 32 weeks gestation may not reach the same arterial blood oxygen saturations in air as those achieved by term babies. Therefore blended oxygen and air should be given judiciously and its use guided by pulse oximetry. If a blend of oxygen and air is not available use what is available.
- Preterm babies of less than 28 weeks gestation should be completely covered up to their necks in a food-grade plastic wrap or bag, without drying, immediately after birth. They should then be nursed under a radiant heater and stabilised. They should remain wrapped until their temperature has been checked after admission. For these infants delivery room temperatures should be at least 26°C.
- The recommended compression:ventilation ratio for CPR remains at 3:1 for newborn resuscitation.
- Attempts to aspirate meconium from the nose and mouth of the unborn baby, while the head is still on the perineum, are not recommended. If

presented with a floppy, apnoeic baby born through meconium it is reasonable to inspect the oropharynx rapidly to remove potential obstructions. If appropriate expertise is available, tracheal intubation and suction may be useful. However, if attempted intubation is prolonged or unsuccessful, start mask ventilation, particularly if there is persistent bradycardia.

- If adrenaline is given then the intravenous route is recommended using a dose of 10-30 mcg kg⁻¹. If the tracheal route is used, it is likely that a dose of at least 50-100 mcg kg⁻¹ will be needed to achieve a similar effect to 10 mcg kg⁻¹ intravenously.
- Detection of exhaled carbon dioxide (capnography) in addition to clinical assessment is recommended as the most reliable method to confirm placement of a tracheal tube in neonates with a spontaneous circulation.
- Newly born infants born at term or near term with evolving moderate to severe hypoxic – ischaemic encephalopathy should, where possible, be treated with therapeutic hypothermia.

Suggested sequence of actions

Keep the baby warm and assess

Babies are born small and wet. They get cold very easily, especially if they remain wet and in a draught. For uncompromised babies, a delay in cord clamping of at least one minute from the complete delivery of the infant, is recommended.

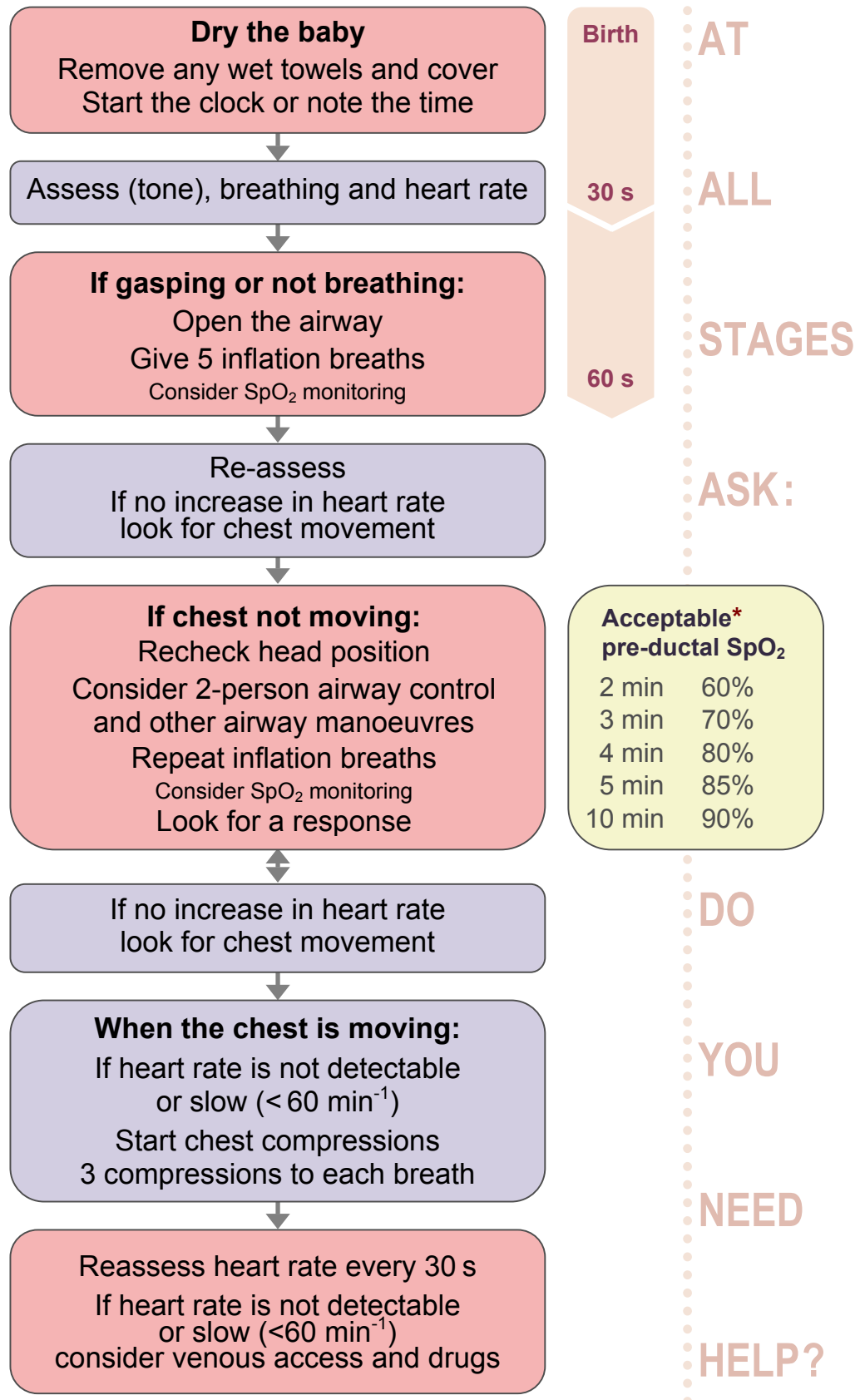
Whatever the situation it is important that the baby does not get cold at this stage. If intervention is required, in a term or near-term baby, dry the baby, remove the wet towels, and cover the baby with dry towels.

Significantly preterm babies are best placed, without drying, into food-grade plastic wrapping under a radiant heater. This process will provide significant stimulation and will allow time to assess tone, breathing, and heart rate.

Reassess these observations regularly every 30 s or so throughout the resuscitation process but it is the heart rate which is the key observation. The first sign of any improvement in the baby will be an increase in heart rate. Consider the need for help; if needed, ask for help immediately.

A healthy baby will be born blue but will have good tone, will cry within a few seconds of delivery and will have a good heart rate within a few minutes of birth (the heart rate of a healthy newborn baby is about 120-150 min⁻¹). A less healthy baby will be blue at birth, will have less good tone, may have a slow heart rate (less than 100 min⁻¹), and may not establish adequate breathing by 90-120 s. An ill baby will be born pale and floppy, not breathing and with a slow, very slow or undetectable heart rate.

Newborn Life Support



* See reference 297

Keep the baby warm and assess (continued)

The heart rate of a baby is judged best by listening with a stethoscope. It can also be felt by gently palpating the umbilical cord but a slow rate at the cord is not always indicative of a truly slow heart rate – feeling for peripheral pulses is not helpful.

A pulse oximeter is probably the best way of assessing heart rate and oxygenation in the delivery room. With practice it is possible to attach a pulse oximeter probe and to obtain a useful reading of heart rate and oxygen saturation about 90 s after delivery.²⁸⁸

Airway

Before the baby can breathe effectively the airway must be open. The best way to achieve this is to place the baby on his back with the head in the neutral position, i.e. with the neck neither flexed nor extended. Most newborn babies will have a relatively prominent occiput, which will tend to flex the neck if the baby is placed on his back on a flat surface. This can be avoided by placing some support under the shoulders of the baby, but be careful not to overextend the neck. If the baby is very floppy (i.e. has no or very little tone) it may also be necessary to apply chin lift or jaw thrust. These manoeuvres will be effective for the majority of babies requiring airway stabilisation at birth.

Airway suction immediately following birth should be reserved for babies who have obvious airway obstruction that cannot be rectified by appropriate positioning. Rarely, material may be blocking the oropharynx or trachea. In these situations, direct visualisation and suction of the oropharynx should be performed. For tracheal obstruction, intubation and suction on withdrawal of the endotracheal tube may be effective.

Breathing

Most babies have a good heart rate after birth and establish breathing by about 90 s. If the baby is not breathing adequately **give 5 inflation breaths**, preferably using air. Until now the baby's lungs will have been filled with fluid. Aeration of the lungs in these circumstances is likely to require sustained application of pressures of about 30 cm H₂O for 2-3 s – these are 'inflation breaths' (20-25 cm H₂O in preterm babies). If the heart rate was below 100 min⁻¹ initially then it should rapidly increase as oxygenated blood reaches the heart.

- If the heart rate does increase then you can assume that you have successfully aerated the lungs.
- If the heart rate increases but the baby does not start breathing for himself, then continue to provide regular breaths at a rate of about 30-40 min⁻¹ until the baby starts to breathe on his own.
- If the heart rate does not increase following inflation breaths, then either you have not aerated the lungs or the baby needs more than lung aeration alone. By far the most likely is that you have failed to aerate the lungs effectively.

- If the heart rate does not increase, *and* the chest does not passively move with each inflation breath, then you have not aerated the lungs.

If the lungs have not been aerated then consider:

- Is the baby's head in the neutral position?
- Do you need jaw thrust?
- Do you need a longer inflation time?
- Do you need a second person's help with the airway?
- Is there an obstruction in the oropharynx (laryngoscope and suction)?
- What about an oropharyngeal (Guedel) airway?
- Is there a tracheal obstruction?

Check that the baby's head and neck are in the neutral position; that your inflation breaths are at the correct pressure and applied for sufficient time (2-3 s inspiration); and that the chest moves with each breath. If the chest still does not move, ask for help in maintaining the airway and consider an obstruction in the oropharynx or trachea, which may be removable by suction under direct vision. An oropharyngeal (Guedel) airway may be helpful.

If the heart rate remains slow (less than 60 min^{-1}) or absent following 5 inflation breaths, despite good passive chest movement in response to your inflation efforts, start chest compression.

Chest compression

Almost all babies needing help at birth will respond to successful lung inflation with an increase in heart rate followed quickly by normal breathing. However, in some cases chest compression is necessary.

Chest compression should be started only when you are sure that the lungs have been aerated successfully.

In babies, the most efficient method of delivering chest compression is to grip the chest in both hands in such a way that the two thumbs can press on the lower third of the sternum, just below an imaginary line joining the nipples, with the fingers over the spine at the back.

Compress the chest quickly and firmly, reducing the antero-posterior diameter of the chest by about one third.²⁸⁹

The ratio of compressions to inflations in newborn resuscitation is 3:1.

Chest compressions move oxygenated blood from the lungs back to the heart. Allow enough time during the relaxation phase of each compression cycle for the heart to refill with blood. Ensure that the chest is inflating with each breath.

In a very few babies (less than one in every thousand births) inflation of the lungs and effective chest compression will not be sufficient to produce an effective circulation. In these circumstances drugs may be helpful.

Drugs

Drugs are needed rarely and only if there is no significant cardiac output despite effective lung inflation and chest compression.

The drugs used include adrenaline (1:10,000), occasionally sodium bicarbonate (ideally 4.2%), and dextrose (10%). They are best delivered via an umbilical venous catheter.

The recommended intravenous dose for adrenaline is 10 mcg kg^{-1} (0.1 ml kg^{-1} of 1:10,000 solution). If this is not effective, a dose of up to 30 mcg kg^{-1} (0.3 ml kg^{-1} of 1:10,000 solution) may be tried.

If the tracheal route is used, it must not interfere with ventilation or delay acquisition of intravenous access. The tracheal dose is thought to be between $50\text{-}100 \text{ mcg kg}^{-1}$.

The dose for sodium bicarbonate is between 1 and 2 mmol of bicarbonate kg^{-1} (2 to 4 ml of 4.2% bicarbonate solution).

The dose of dextrose recommended is 250 mg kg^{-1} (2.5 ml kg^{-1} of 10% dextrose).

Very rarely, the heart rate cannot increase because the baby has lost significant blood volume. If this is the case, there is often a clear history of blood loss from the baby, but not always. Use of isotonic crystalloid rather than albumin is preferred for emergency volume replacement. In the presence of hypovolaemia, a bolus of 10 ml kg^{-1} of 0.9% sodium chloride or similar given over 10 - 20 s will often produce a rapid response and can be repeated safely if needed.

Explanatory Notes

Resuscitation or stabilisation

Most babies born at term need no resuscitation and they can usually stabilise themselves during the transition from placental to pulmonary respiration very effectively. Provided attention is paid to preventing heat loss and a little patience is exhibited before cutting the umbilical cord, intervention is rarely necessary. However, as mentioned above, some babies will have suffered stresses or insults during labour. Help may then be required which is characterised by interventions designed to rescue a sick or very sick baby and this process can then reasonably be called resuscitation.

Significantly preterm babies, particularly those born below 30 weeks gestation, are a different matter. Most babies in this group are healthy at the time of delivery and yet all can be expected to benefit from help in making the transition. Intervention in this situation is usually limited to maintaining a baby healthy during this transition and is more appropriately called stabilisation.

In the past both situations have been referred to as resuscitation and this seems inappropriate and likely to cause confusion.

Umbilical cord clamping

For healthy term infants delaying cord clamping for at least one minute or until the cord stops pulsating following delivery improves iron status through early infancy.²⁹⁰ For preterm babies in good condition at delivery, delaying cord clamping for up to 3 min results in increased blood pressure during stabilisation, a lower incidence of intraventricular haemorrhage and fewer blood transfusions.²⁹¹ However, babies were more likely to receive phototherapy. There are limited data on the hazards or benefits of delayed cord clamping in the non-vigorous infant.^{292, 293}

Delaying cord clamping for at least one minute is recommended for newborn infants not requiring resuscitation.²⁸⁷ At present there is insufficient evidence to define an appropriate time to clamp the cord in babies apparently needing resuscitation. However, this may be because time is the wrong defining parameter and perhaps the cord should not be clamped until the baby has started breathing.

Oximetry and the use of supplemental oxygen

If resources are available, pulse oximetry should be used for all deliveries where it is anticipated that the infant may have problems with transition or need resuscitation. Oxygen saturation and heart rate can be measured reliably during the first minutes of life with a modern pulse oximeter.²⁸⁷

The sensor must be placed on the right hand or wrist to obtain an accurate reading of the preductal saturation.^{294, 295} Placement of the sensor on the baby before connecting to the instrument may result in faster acquisition of signal. In most cases a reliable reading can be obtained within 90 s of birth.²⁹⁶ Pulse oximetry can also provide an accurate display of heart rate during periods of good perfusion.

In healthy term babies, oxygen saturation increases gradually from approximately 60% soon after birth to over 90% at 10 min. In preterm infants hyperoxaemia is particularly damaging and if oxygen is used to achieve a saturation above 95% the risk of hyperoxaemia is high. Therefore the rate of rise in oxygen saturation after birth in preterm infants should not exceed that seen in term infants, although some supplemental oxygen may be required to achieve this.^{297, 298}

Colour

Using colour as a proxy for oxygen saturation is usually inaccurate.²⁹⁹ However, noting whether a baby is initially very pale and therefore either acidotic or anaemic at delivery may be useful as an indicator for later therapeutic intervention.

Airway suctioning with or without meconium

A multi-centre randomised controlled trial has shown that routine elective intubation and suctioning of vigorous infants at birth, did not reduce meconium aspiration syndrome (MAS).³⁰⁰ A further randomised study has shown that suctioning the nose and mouth of

such babies on the perineum and before delivery of the shoulders (intrapartum suctioning) is also ineffective.³⁰¹ Whilst non-vigorous infants born through meconium-stained amniotic fluid are at increased risk of MAS, tracheal suctioning has not been shown to improve the outcome.

There is no evidence to support or refute suctioning of the mouth and nose of babies born through clear amniotic fluid.

Recommendation

Routine *intrapartum* oropharyngeal and nasopharyngeal suctioning for infants born with clear and/or meconium-stained amniotic fluid is not recommended. In the absence of randomised, controlled trials, there is insufficient evidence to recommend a change in the current practice of performing direct oropharyngeal and tracheal suctioning of non-vigorous babies after birth with meconium-stained amniotic fluid if feasible. However, if attempted intubation is prolonged or unsuccessful, mask ventilation should be implemented, particularly if there is persistent bradycardia.

Laryngeal mask

Several studies have shown that laryngeal mask airways (LMAs) can be used effectively at birth to ventilate the lungs of babies weighing over 2000 g, greater than 33 weeks gestation and apparently needing resuscitation. Case reports suggest that LMAs have been used successfully when intubation has been tried and failed – and occasionally vice-versa. Data on smaller or less mature babies are scarce.

Recommendation

The LMA should be considered during resuscitation of the newborn if face mask ventilation is unsuccessful and tracheal intubation is unsuccessful or not feasible. The LMA may be considered as an alternative to a face mask for positive pressure ventilation among newborns weighing more than 2000 g or delivered ≥ 34 weeks gestation. There is limited evidence, however, to evaluate its use for newborns weighing < 2000 g or delivered < 34 weeks gestation. The LMA may be considered as an alternative to tracheal intubation as a secondary airway for resuscitation among newborns weighing more than 2000 g or delivered ≥ 34 weeks gestation. The LMA has not been evaluated in the setting of meconium stained fluid, during chest compressions, or for the administration of emergency intra-tracheal medications.

Exhaled carbon dioxide

Detection of exhaled carbon dioxide confirms tracheal intubation in neonates with a cardiac output more rapidly and more accurately than clinical assessment alone. False negative readings may occur in very low birth weight neonates and in infants during cardiac arrest. False positives may occur with colorimetric devices contaminated with adrenaline, surfactant and atropine.

Drugs in resuscitation at birth

Ventilation and chest compression may fail to resuscitate fewer than 1 in 1000 babies.³⁰² In this group, resuscitation drugs may be justified. Whilst there is evidence from animal studies for both adrenaline and sodium bicarbonate in increasing return of spontaneous circulation, there is no placebo-controlled evidence in human babies for the effectiveness of any drug intervention in this situation. Even for adults and children in cardiac arrest, there is insufficient evidence to suggest that vasopressors improve survival to discharge.

For this reason use of drugs before achieving lung aeration followed by chest compressions (known to be effective resuscitative interventions) can never be justified.²⁷⁰

Therapeutic hypothermia

Term or near-term infants, with evolving moderate to severe hypoxic-ischaemic encephalopathy, should be treated with therapeutic hypothermia.³⁰³⁻³⁰⁶ Whole body cooling and selective head cooling are both appropriate strategies.³⁰⁴⁻³⁰⁸ Cooling should be initiated and conducted under clearly-defined protocols with treatment in neonatal intensive care facilities and the capabilities for multidisciplinary care. Treatment should be consistent with the protocols used in the randomised clinical trials, i.e. commence within 6 h, continue for 72 h and re-warm over at least 4 h. All treated infants should be followed longitudinally and permission sought for their inclusion in the TOBY (Total Body Hypothermia for Neonatal Encephalopathy Trial) register (see the [University of Oxford National Perinatal Epidemiology Unit Toby Cooling Register](#)).

When to stop

In a newly-born baby with no detectable cardiac activity, and with cardiac activity that remains undetectable for 10 min, it is appropriate to consider stopping resuscitation. The decision to continue resuscitation efforts beyond 10 min with no cardiac activity is often complex and may be influenced by issues such as the presumed aetiology of the arrest, the gestation of the baby, the presence or absence of complications, and the parents' previous expressed feelings about acceptable risk of morbidity.

1. Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. *JAMA* 1960;173:1064-7.
2. Nolan JP, Nadkarni VM, Billi JE, et al. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 2: International Collaboration in Resuscitation Science. *Resuscitation* 2010;81:e26-e31.
3. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care---An International Consensus on Science. *Resuscitation* 2000;46:3-430.
4. Proceedings of the 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2005;67:157-341.
5. Nolan JP, Hazinski MF, Billi JE, et al. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 1: Executive Summary. *Resuscitation* 2010;81:e1-e25.
6. Nolan JP, Soar J, Zideman DA, et al. European Resuscitation Council Guidelines for Resuscitation 2010. Section 1. Executive Summary. *Resuscitation* 2010;81.
7. Shuster M, Billi JE, Bossaert L, et al. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. 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. *Resuscitation* 2010;81:e41-e47.
8. Nolan J, Soar J, Eikeland H. The chain of survival. *Resuscitation* 2006;71:270-1.
9. van Alem AP, Sanou BT, Koster RW. Interruption of cardiopulmonary resuscitation with the use of the automated external defibrillator in out-of-hospital cardiac arrest. *Ann Emerg Med* 2003;42:449-57.



10. Bobrow BJ, Zuercher M, Ewy GA, et al. Gasping during cardiac arrest in humans is frequent and associated with improved survival. *Circulation* 2008;118:2550-4.
11. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *JAMA* 2005;293:299-304.
12. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. *JAMA* 2005;293:305-10.
13. Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. *Resuscitation* 2006;71:137-45.
14. SOS-KANTO Study Group. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): an observational study. *Lancet* 2007;369:920-6.
15. Abella BS, Sandbo N, Vassilatos P, et al. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. *Circulation* 2005;111:428-34.
16. [Koster RW, Baubin MA, Caballero A, et al. European Resuscitation Council Guidelines for Resuscitation 2010. Section 2. Adult basic life support and use of automated external defibrillators. *Resuscitation* 2010;81.](#)
17. Handley AJ. Teaching hand placement for chest compression--a simpler technique. *Resuscitation* 2002;53:29-36.
18. Rea TD, Fahrenbruch C, Culley L, et al. CPR with chest compressions alone or with rescue breathing. *New England Journal of Medicine* 2010;363:423-33.
19. Svensson L, Bohm K, Castren M, et al. Compression-only CPR or standard CPR in out-of-hospital cardiac arrest. *New England Journal of Medicine* 2010;363:434-42.
- 19a. [Hüpfel M, Selig HF, Nagele P. Chest compression-only CPR: A meta-analysis. *Lancet* 2010;DOI:10.1016/S0140-6736\(10\)61454-7.](#)
20. Kitamura T, Iwami T, Kawamura T, Nagao K, Tanaka H, Hiraide A. Bystander-Initiated Rescue Breathing for Out-of-Hospital Cardiac Arrests of Noncardiac Origin. *Circulation* 2010;122:293-9.
21. Kitamura T, Iwami T, Kawamura T, et al. Conventional and chest-compression-only cardiopulmonary resuscitation by bystanders for children who have out-of-hospital cardiac arrests: a prospective, nationwide, population-based cohort study. *Lancet* 2010;375:1347-54.



22. Pell JP, Sirel JM, Marsden AK, Ford I, Walker NL, Cobbe SM. Presentation, management, and outcome of out of hospital cardiopulmonary arrest: comparison by underlying aetiology. *Heart* 2003;89:839-42.
23. Valenzuela TD, Roe DJ, Nichol G, Clark LL, Spaite DW, Hardman RG. Outcomes of rapid defibrillation by security officers after cardiac arrest in casinos. *N Engl J Med* 2000;343:1206-9.
24. Colquhoun MC, Chamberlain DA, Newcombe RG, et al. A national scheme for public access defibrillation in England and Wales: early results. *Resuscitation* 2008;78:275-80.
25. Valenzuela TD, Roe DJ, Cretin S, Spaite DW, Larsen MP. Estimating effectiveness of cardiac arrest interventions: a logistic regression survival model. *Circulation* 1997;96:3308-13.
26. [Soar J, Mancini ME, Bhanji F, et al. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 12: Education, Implementation, and Teams. *Resuscitation* 2010;81:e283-e325.](#)
27. Kitamura T, Iwami T, Kawamura T, Nagao K, Tanaka H, Hiraide A. Nationwide public-access defibrillation in Japan. *N Engl J Med* 2010;362:994-1004.
28. Hallstrom A, Cobb L, Johnson E, Copass M. Cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation. *N Engl J Med* 2000;342:1546-53.
29. Rea TD, Eisenberg MS, Culley LL, Becker L. Dispatcher-assisted cardiopulmonary resuscitation and survival in cardiac arrest. *Circulation* 2001;104:2513-6.
30. Rea TD, Stickney RE, Doherty A, Lank P. Performance of chest compressions by laypersons during the Public Access Defibrillation Trial. *Resuscitation* 2010;81:293-6.
31. O'Neill JF, Deakin CD. Evaluation of telephone CPR advice for adult cardiac arrest patients. *Resuscitation* 2007;74:63-7.
32. Bahr J, Klingler H, Panzer W, Rode H, Kettler D. Skills of lay people in checking the carotid pulse. *Resuscitation* 1997;35:23-6.
33. Clawson J, Olola C, Heward A, Patterson B. Cardiac arrest predictability in seizure patients based on emergency medical dispatcher identification of previous seizure or epilepsy history. *Resuscitation* 2007;75:298-304.



34. Sayre MR, Berg RA, Cave DM, Page RL, Potts J, White RD. Hands-only (compression-only) cardiopulmonary resuscitation: a call to action for bystander response to adults who experience out-of-hospital sudden cardiac arrest: a science advisory for the public from the American Heart Association Emergency Cardiovascular Care Committee. *Circulation* 2008;117:2162-7.
35. Heidenreich JW, Sanders AB, Higdon TA, Kern KB, Berg RA, Ewy GA. Uninterrupted chest compression CPR is easier to perform and remember than standard CPR. *Resuscitation* 2004;63:123-30.
36. Deakin CD, Nolan JP. European Resuscitation Council guidelines for resuscitation 2005. Section 3. Electrical therapies: automated external defibrillators, defibrillation, cardioversion and pacing. *Resuscitation* 2005;67 Suppl 1:S25-37.
37. Cobb LA, Fahrenbruch CE, Walsh TR, et al. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. *JAMA* 1999;281:1182-8.
38. Wik L, Hansen TB, Fylling F, et al. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. *JAMA* 2003;289:1389-95.
39. Baker PW, Conway J, Cotton C, et al. Defibrillation or cardiopulmonary resuscitation first for patients with out-of-hospital cardiac arrests found by paramedics to be in ventricular fibrillation? A randomised control trial. *Resuscitation* 2008;79:424-31.
40. Jacobs IG, Finn JC, Oxeer HF, Jelinek GA. CPR before defibrillation in out-of-hospital cardiac arrest: a randomized trial. *Emerg Med Australas* 2005;17:39-45.
41. Hayakawa M, Gando S, Okamoto H, Asai Y, Uegaki S, Makise H. Shortening of cardiopulmonary resuscitation time before the defibrillation worsens the outcome in out-of-hospital VF patients. *Am J Emerg Med* 2009;27:470-4.
42. Bradley SM, Gabriel EE, Aufderheide TP, et al. Survival Increases with CPR by Emergency Medical Services before defibrillation of out-of-hospital ventricular fibrillation or ventricular tachycardia: observations from the Resuscitation Outcomes Consortium. *Resuscitation* 2010;81:155-62.
43. Christenson J, Andrusiek D, Everson-Stewart S, et al. Chest compression fraction determines survival in patients with out-of-hospital ventricular fibrillation. *Circulation* 2009;120:1241-7.
44. Kramer-Johansen J, Wik L, Steen PA. Advanced cardiac life support before and after tracheal intubation--direct measurements of quality. *Resuscitation* 2006;68:61-9.
45. Grmec S. Comparison of three different methods to confirm tracheal tube placement in emergency intubation. *Intensive Care Med* 2002;28:701-4.



46. Lyon RM, Ferris JD, Young DM, McKeown DW, Oglesby AJ, Robertson C. Field intubation of cardiac arrest patients: a dying art? *Emerg Med J* 2010;27:321-3.
47. Jones JH, Murphy MP, Dickson RL, Somerville GG, Brizendine EJ. Emergency physician-verified out-of-hospital intubation: miss rates by paramedics. *Acad Emerg Med* 2004;11:707-9.
48. Pelucio M, Halligan L, Dhindsa H. Out-of-hospital experience with the syringe esophageal detector device. *Acad Emerg Med* 1997;4:563-8.
49. Jemmett ME, Kendal KM, Fourre MW, Burton JH. Unrecognized misplacement of endotracheal tubes in a mixed urban to rural emergency medical services setting. *Acad Emerg Med* 2003;10:961-5.
50. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. *Ann Emerg Med* 2001;37:32-7.
51. Wang HE, Simeone SJ, Weaver MD, Callaway CW. Interruptions in cardiopulmonary resuscitation from paramedic endotracheal intubation. *Ann Emerg Med* 2009;54:645-52 e1.
52. Garza AG, Gratton MC, Coontz D, Noble E, Ma OJ. Effect of paramedic experience on orotracheal intubation success rates. *J Emerg Med* 2003;25:251-6.
53. Bobrow BJ, Clark LL, Ewy GA, et al. Minimally interrupted cardiac resuscitation by emergency medical services for out-of-hospital cardiac arrest. *JAMA* 2008;299:1158-65.
54. Bobrow BJ, Ewy GA, Clark L, et al. Passive oxygen insufflation is superior to bag-valve-mask ventilation for witnessed ventricular fibrillation out-of-hospital cardiac arrest. *Ann Emerg Med* 2009;54:656-62 e1.
55. [Deakin CD, Nolan JP, Soar J, et al. European Resuscitation Council Guidelines for Resuscitation 2010. Section 4. Adult Advanced Life Support. Resuscitation 2010;81.](#)
56. Morrison LJ, Visentin LM, Kiss A, et al. Validation of a rule for termination of resuscitation in out-of-hospital cardiac arrest. *N Engl J Med* 2006;355:478-87.
57. Excellence NIfHaC. NICE clinical guideline 50 Acutely ill patients in hospital: recognition of and response to acute illness in adults in hospital. London: National Institute for Health and Clinical Excellence; 2007.
58. Muller D, Agrawal R, Arntz HR. How sudden is sudden cardiac death? *Circulation* 2006;114:1146-50.
59. Lowel H, Lewis M, Hormann A. [Prognostic significance of prehospital phase in acute myocardial infarct. Results of the Augsburg Myocardial Infarct Registry, 1985-1988]. *Dtsch Med Wochenschr* 1991;116:729-33.

60. Meaney PA, Nadkarni VM, Kern KB, Indik JH, Halperin HR, Berg RA. Rhythms and outcomes of adult in-hospital cardiac arrest. *Crit Care Med* 2010;38:101-8.
61. Smith GB. In-hospital cardiac arrest: Is it time for an in-hospital 'chain of prevention'? *Resuscitation* 2010.
62. National Confidential Enquiry into Patient Outcome and Death. *An acute problem?* London: NCEPOD; 2005.
63. Kause J, Smith G, Prytherch D, Parr M, Flabouris A, Hillman K. A comparison of antecedents to cardiac arrests, deaths and emergency intensive care admissions in Australia and New Zealand, and the United Kingdom--the ACADEMIA study. *Resuscitation* 2004;62:275-82.
64. Kenward G, Robinson A, Bradburn S, Steeds R. False cardiac arrests: the right time to turn away? *Postgrad Med J* 2007;83:344-7.
65. Beck DH, McQuillan P, Smith GB. Waiting for the break of dawn? The effects of discharge time, discharge TISS scores and discharge facility on hospital mortality after intensive care. *Intensive Care Med* 2002;28:1287-93.
66. Harrison GA, Jacques TC, Kilborn G, McLaws ML. The prevalence of recordings of the signs of critical conditions and emergency responses in hospital wards--the SOCCER study. *Resuscitation* 2005;65:149-57.
67. DeVita MA, Smith GB, Adam SK, et al. "Identifying the hospitalised patient in crisis"--a consensus conference on the afferent limb of rapid response systems. *Resuscitation* 2010;81:375-82.
68. *Critical care outreach 2003: progress in developing services. The National Outreach Report.* London, UK: Department of Health and National Health Service Modernisation Agency. London, UK: Department of Health and National Health Service Modernisation Agency; 2003.
69. Smith GB, Prytherch DR, Schmidt PE, Featherstone PI. Review and performance evaluation of aggregate weighted 'track and trigger' systems. *Resuscitation* 2008;77:170-9.
70. Smith GB, Prytherch DR, Schmidt PE, Featherstone PI, Higgins B. A review, and performance evaluation, of single-parameter "track and trigger" systems. *Resuscitation* 2008;79:11-21.
71. Day BA. Early warning system scores and response times: an audit. *Nurs Crit Care* 2003;8:156-64.
72. Soar J, McKay U. A revised role for the hospital cardiac arrest team? *Resuscitation* 1998;38:145-9.

73. Chan PS, Khalid A, Longmore LS, Berg RA, Kosiborod M, Spertus JA. Hospital-wide code rates and mortality before and after implementation of a rapid response team. *JAMA* 2008;300:2506-13.
74. Hillman K, Chen J, Cretikos M, et al. Introduction of the medical emergency team (MET) system: a cluster-randomised controlled trial. *Lancet* 2005;365:2091-7.
75. McDonnell A, Esmonde L, Morgan R, et al. The provision of critical care outreach services in England: findings from a national survey. *J Crit Care* 2007;22:212-8.
76. Harrison DA, Gao H, Welch CA, Rowan KM. The effects of critical care outreach services before and after critical care: a matched-cohort analysis. *J Crit Care* 2010;25:196-204.
77. Priestley G, Watson W, Rashidian A, et al. Introducing Critical Care Outreach: a ward-randomised trial of phased introduction in a general hospital. *Intensive Care Med* 2004;30:1398-404.
78. Smith GB, Osgood VM, Crane S. ALERT--a multiprofessional training course in the care of the acutely ill adult patient. *Resuscitation* 2002;52:281-6.
79. Spearpoint KG, Gruber PC, Brett SJ. Impact of the Immediate Life Support course on the incidence and outcome of in-hospital cardiac arrest calls: an observational study over 6 years. *Resuscitation* 2009;80:638-43.
80. Lewis G. The Confidential Enquiry into Maternal and Child Health (CEMACH). Saving Mothers' Lives: Reviewing maternal deaths to make motherhood safer – 2003-2005. The Seventh Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom. London: CEMACH; 2007 2007.
81. Barrett NA, Yentis SM. Outreach in obstetric critical care. *Best Pract Res Clin Obstet Gynaecol* 2008;22:885-98.
82. Tume L. A 3-year review of emergency PICU admissions from the ward in a specialist cardio-respiratory centre. *Care of the Critically Ill* 2005;21:4-7.
83. Sharek PJ, Parast LM, Leong K, et al. Effect of a rapid response team on hospital-wide mortality and code rates outside the ICU in a Children's Hospital. *JAMA* 2007;298:2267-74.
84. Tibballs J, Kinney S. Reduction of hospital mortality and of preventable cardiac arrest and death on introduction of a pediatric medical emergency team. *Pediatr Crit Care Med* 2009;10:306-12.
85. Smith GB. Increased do not attempt resuscitation decision making in hospitals with a medical emergency teams system-cause and effect? *Resuscitation* 2008;79:346-7.

86. Chen J, Flabouris A, Bellomo R, Hillman K, Finfer S. The Medical Emergency Team System and not-for-resuscitation orders: results from the MERIT study. *Resuscitation* 2008;79:391-7.
87. Jones DA, McIntyre T, Baldwin I, Mercer I, Kattula A, Bellomo R. The medical emergency team and end-of-life care: a pilot study. *Crit Care Resusc* 2007;9:151-6.
88. Agency NPS. Establishing a standard crash call telephone number in hospitals. Patient Safety Alert 02. London: National Patient Safety Agency; 2004.
89. O'Driscoll BR, Howard LS, Davison AG. BTS guideline for emergency oxygen use in adult patients. *Thorax* 2008;63 Suppl 6:vi1-68.
90. Marshall S, Harrison J, Flanagan B. The teaching of a structured tool improves the clarity and content of interprofessional clinical communication. *Qual Saf Health Care* 2009;18:137-40.
91. Featherstone P, Chalmers T, Smith GB. RSVP: a system for communication of deterioration in hospital patients. *Br J Nurs* 2008;17:860-4.
92. Perkins GD, Roberts C, Gao F. Delays in defibrillation: influence of different monitoring techniques. *Br J Anaesth* 2002;89:405-8.
93. Amir O, Schliamser JE, Nemer S, Arie M. Ineffectiveness of precordial thump for cardioversion of malignant ventricular tachyarrhythmias. *Pacing Clin Electrophysiol* 2007;30:153-6.
94. Haman L, Parizek P, Vojacek J. Precordial thump efficacy in termination of induced ventricular arrhythmias. *Resuscitation* 2009;80:14-6.
95. Pellis T, Kette F, Lovisa D, et al. Utility of pre-cordial thump for treatment of out of hospital cardiac arrest: a prospective study. *Resuscitation* 2009;80:17-23.
96. Kohl P, King AM, Boulin C. Antiarrhythmic effects of acute mechanical stimulation. In: Kohl P, Sachs F, Franz MR, eds. *Cardiac mechano-electric feedback and arrhythmias: from pipette to patient*. Philadelphia: Elsevier Saunders; 2005:304-14.
97. Chan PS, Krumholz HM, Nichol G, Nallamothu BK. Delayed time to defibrillation after in-hospital cardiac arrest. *N Engl J Med* 2008;358:9-17.
98. Soar J, Perkins GD, Harris S, et al. The immediate life support course. *Resuscitation* 2003;57:21-6.
99. Nolan J. Advanced life support training. *Resuscitation* 2001;50:9-11.
100. Perkins G, Lockey A. The advanced life support provider course. *BMJ* 2002;325:S81.

101. Flin R, Patey R, Glavin R, Maran N. Anaesthetists' non-technical skills. *Br J Anaesth* 2010.
102. Peberdy MA, Ornato JP, Larkin GL, et al. Survival from in-hospital cardiac arrest during nights and weekends. *JAMA* 2008;299:785-92.
103. Needleman J, Buerhaus P, Mattke S, Stewart M, Zelevinsky K. Nurse-staffing levels and the quality of care in hospitals. *N Engl J Med* 2002;346:1715-22.
104. Aiken LH, Clarke SP, Sloane DM, Sochalski J, Silber JH. Hospital nurse staffing and patient mortality, nurse burnout, and job dissatisfaction. *JAMA* 2002;288:1987-93.
105. Tourangeau AE, Cranley LA, Jeffs L. Impact of nursing on hospital patient mortality: a focused review and related policy implications. *Qual Saf Health Care* 2006;15:4-8.
106. Gabbott D, Smith G, Mitchell S, et al. Cardiopulmonary resuscitation standards for clinical practice and training in the UK. *Resuscitation* 2005;64:13-9.
107. Dyson E, Smith GB. Common faults in resuscitation equipment--guidelines for checking equipment and drugs used in adult cardiopulmonary resuscitation. *Resuscitation* 2002;55:137-49.
108. Pittman J, Turner B, Gabbott DA. Communication between members of the cardiac arrest team--a postal survey. *Resuscitation* 2001;49:175-7.
109. Morgan R, Westmoreland C. Survey of junior hospital doctors' attitudes to cardiopulmonary resuscitation. *Postgrad Med J* 2002;78:413-5.
110. Hayes CW, Rhee A, Detsky ME, Leblanc VR, Wax RS. Residents feel unprepared and unsupervised as leaders of cardiac arrest teams in teaching hospitals: a survey of internal medicine residents. *Crit Care Med* 2007;35:1668-72.
111. Edelson DP, Litzinger B, Arora V, et al. Improving in-hospital cardiac arrest process and outcomes with performance debriefing. *Arch Intern Med* 2008;168:1063-9.
112. Luettel D, Beaumont K, F. H. Recognising and responding appropriately to early signs of deterioration in hospitalised patients. London: National Patient Safety Agency; 2007.
113. Martin IC, Mason DG, Stewart J, Mason M, Smith NCE, Gill K. Emergency Admissions: A journey in the right direction? A report of the National Confidential Enquiry into Patient Outcome and Death. London: National Confidential Enquiry into Patient Outcome and Death; 2007.
114. Brennan RT, Braslow A. Skill mastery in public CPR classes. *Am J Emerg Med* 1998;16:653-7.

115. Chamberlain D, Smith A, Woollard M, et al. Trials of teaching methods in basic life support (3): comparison of simulated CPR performance after first training and at 6 months, with a note on the value of re-training. *Resuscitation* 2002;53:179-87.
116. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. *Resuscitation* 1996;33:107-16.
117. Lapostolle F, Le Toumelin P, Agostinucci JM, Catineau J, Adnet F. Basic cardiac life support providers checking the carotid pulse: performance, degree of conviction, and influencing factors. *Acad Emerg Med* 2004;11:878-80.
118. Liberman M, Lavoie A, Mulder D, Sampalis J. Cardiopulmonary resuscitation: errors made by pre-hospital emergency medical personnel. *Resuscitation* 1999;42:47-55.
119. Moule P. Checking the carotid pulse: diagnostic accuracy in students of the healthcare professions. *Resuscitation* 2000;44:195-201.
120. Nyman J, Sihvonen M. Cardiopulmonary resuscitation skills in nurses and nursing students. *Resuscitation* 2000;47:179-84.
121. Perkins GD, Stephenson B, Hulme J, Monsieurs KG. Birmingham assessment of breathing study (BABS). *Resuscitation* 2005;64:109-13.
122. Ruppert M, Reith MW, Widmann JH, et al. Checking for breathing: evaluation of the diagnostic capability of emergency medical services personnel, physicians, medical students, and medical laypersons. *Ann Emerg Med* 1999;34:720-9.
123. Tibballs J, Russell P. Reliability of pulse palpation by healthcare personnel to diagnose paediatric cardiac arrest. *Resuscitation* 2009;80:61-4.
124. Bang A, Herlitz J, Martinell S. Interaction between emergency medical dispatcher and caller in suspected out-of-hospital cardiac arrest calls with focus on agonal breathing. A review of 100 tape recordings of true cardiac arrest cases. *Resuscitation* 2003;56:25-34.
125. Bohm K, Rosenqvist M, Hollenberg J, Biber B, Engerstrom L, Svensson L. Dispatcher-assisted telephone-guided cardiopulmonary resuscitation: an underused lifesaving system. *Eur J Emerg Med* 2007;14:256-9.
126. Vaillancourt C, Verma A, Trickett J, et al. Evaluating the effectiveness of dispatch-assisted cardiopulmonary resuscitation instructions. *Acad Emerg Med* 2007;14:877-83.
127. White L, Rogers J, Bloomingdale M, et al. Dispatcher-assisted cardiopulmonary resuscitation: risks for patients not in cardiac arrest. *Circulation* 2010;121:91-7.

128. Lloyd MS, Heeke B, Walter PF, Langberg JJ. Hands-on defibrillation: an analysis of electrical current flow through rescuers in direct contact with patients during biphasic external defibrillation. *Circulation* 2008;117:2510-4.
129. Hollenberg J, Herlitz J, Lindqvist J, et al. Improved survival after out-of-hospital cardiac arrest is associated with an increase in proportion of emergency crew--witnessed cases and bystander cardiopulmonary resuscitation. *Circulation* 2008;118:389-96.
130. Iwami T, Nichol G, Hiraide A, et al. Continuous improvements in "chain of survival" increased survival after out-of-hospital cardiac arrests: a large-scale population-based study. *Circulation* 2009;119:728-34.
131. Krijne R. Rate acceleration of ventricular tachycardia after a precordial chest thump. *Am J Cardiol* 1984;53:964 - 5.
132. Berg RA, Sanders AB, Kern KB, et al. Adverse hemodynamic effects of interrupting chest compressions for rescue breathing during cardiopulmonary resuscitation for ventricular fibrillation cardiac arrest. *Circulation* 2001;104:2465-70.
133. Eftestol T, Sunde K, Steen PA. Effects of interrupting precordial compressions on the calculated probability of defibrillation success during out-of-hospital cardiac arrest. *Circulation* 2002;105:2270-3.
134. Olasveengen TM, Vik E, Kuzovlev A, Sunde K. Effect of implementation of new resuscitation guidelines on quality of cardiopulmonary resuscitation and survival. *Resuscitation* 2009;80:407-11.
135. Rea TD, Helbock M, Perry S, et al. Increasing use of cardiopulmonary resuscitation during out-of-hospital ventricular fibrillation arrest: survival implications of guideline changes. *Circulation* 2006;114:2760-5.
136. Steinmetz J, Barnung S, Nielsen SL, Risom M, Rasmussen LS. Improved survival after an out-of-hospital cardiac arrest using new guidelines. *Acta Anaesthesiol Scand* 2008;52:908-13.
137. Jost D, Degrange H, Verret C, et al. DEFI 2005: a randomized controlled trial of the effect of automated external defibrillator cardiopulmonary resuscitation protocol on outcome from out-of-hospital cardiac arrest. *Circulation* 2010;121:1614-22.
138. Soar J, Perkins GD, Abbas G, et al. [European Resuscitation Council Guidelines for Resuscitation 2010. Section 8. Cardiac arrest in special circumstances: electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution. *Resuscitation* 2010;81.](#)

139. Stiell IG, Walker RG, Nesbitt LP, et al. BIPHASIC Trial: a randomized comparison of fixed lower versus escalating higher energy levels for defibrillation in out-of-hospital cardiac arrest. *Circulation* 2007;115:1511-7.
140. Walsh SJ, McClelland AJ, Owens CG, et al. Efficacy of distinct energy delivery protocols comparing two biphasic defibrillators for cardiac arrest. *Am J Cardiol* 2004;94:378-80.
141. Koster RW, Walker RG, Chapman FW. Recurrent ventricular fibrillation during advanced life support care of patients with prehospital cardiac arrest. *Resuscitation* 2008;78:252-7.
142. Walker RG, Koster RW, Sun C, et al. Defibrillation probability and impedance change between shocks during resuscitation from out-of-hospital cardiac arrest. *Resuscitation* 2009;80:773-7.
143. Hess EP, Russell JK, Liu PY, White RD. A high peak current 150-J fixed-energy defibrillation protocol treats recurrent ventricular fibrillation (VF) as effectively as initial VF. *Resuscitation* 2008;79:28-33.
144. Olasveengen TM, Sunde K, Brunborg C, Thowsen J, Steen PA, Wik L. Intravenous drug administration during out-of-hospital cardiac arrest: a randomized trial. *JAMA* 2009;302:2222-9.
145. Herlitz J, Ekstrom L, Wennerblom B, Axelsson A, Bang A, Holmberg S. Adrenaline in out-of-hospital ventricular fibrillation. Does it make any difference? *Resuscitation* 1995;29:195-201.
146. Eftestol T, Wik L, Sunde K, Steen PA. Effects of cardiopulmonary resuscitation on predictors of ventricular fibrillation defibrillation success during out-of-hospital cardiac arrest. *Circulation* 2004;110:10-5.
147. Berg RA, Hilwig RW, Kern KB, Ewy GA. Precountershock cardiopulmonary resuscitation improves ventricular fibrillation median frequency and myocardial readiness for successful defibrillation from prolonged ventricular fibrillation: a randomized, controlled swine study. *Ann Emerg Med* 2002;40:563-70.
148. Achleitner U, Wenzel V, Strohmenger HU, et al. The beneficial effect of basic life support on ventricular fibrillation mean frequency and coronary perfusion pressure. *Resuscitation* 2001;51:151-8.
149. Fries M, Tang W, Chang YT, Wang J, Castillo C, Weil MH. Microvascular blood flow during cardiopulmonary resuscitation is predictive of outcome. *Resuscitation* 2006;71:248-53.
150. Ristagno G, Tang W, Huang L, et al. Epinephrine reduces cerebral perfusion during cardiopulmonary resuscitation. *Crit Care Med* 2009;37:1408-15.

151. Tang W, Weil MH, Sun S, Gazmuri RJ, Bisera J. Progressive myocardial dysfunction after cardiac resuscitation. *Crit Care Med* 1993;21:1046-50.
152. Angelos MG, Butke RL, Panchal AR, et al. Cardiovascular response to epinephrine varies with increasing duration of cardiac arrest. *Resuscitation* 2008;77:101-10.
153. Stiell IG, Wells GA, Hebert PC, Laupacis A, Weitzman BN. Association of drug therapy with survival in cardiac arrest: limited role of advanced cardiac life support drugs. *Acad Emerg Med* 1995;2:264-73.
154. Stiell IG, Wells GA, Field B, et al. Advanced cardiac life support in out-of-hospital cardiac arrest. *N Engl J Med* 2004;351:647-56.
155. Engdahl J, Bang A, Lindqvist J, Herlitz J. Can we define patients with no and those with some chance of survival when found in asystole out of hospital? *Am J Cardiol* 2000;86:610-4.
156. Engdahl J, Bang A, Lindqvist J, Herlitz J. Factors affecting short- and long-term prognosis among 1069 patients with out-of-hospital cardiac arrest and pulseless electrical activity. *Resuscitation* 2001;51:17-25.
157. Dumot JA, Burval DJ, Sprung J, et al. Outcome of adult cardiopulmonary resuscitations at a tertiary referral center including results of "limited" resuscitations. *Arch Intern Med* 2001;161:1751-8.
158. Tortolani AJ, Risucci DA, Powell SR, Dixon R. In-hospital cardiopulmonary resuscitation during asystole. Therapeutic factors associated with 24-hour survival. *Chest* 1989;96:622-6.
159. Coon GA, Clinton JE, Ruiz E. Use of atropine for brady-asystolic prehospital cardiac arrest. *Ann Emerg Med* 1981;10:462-7.
160. Price S, Uddin S, Quinn T. Echocardiography in cardiac arrest. *Curr Opin Crit Care* 2010;16:211-5.
161. Hernandez C, Shuler K, Hannan H, Sonyika C, Likourezos A, Marshall J. C.A.U.S.E.: Cardiac arrest ultra-sound exam--a better approach to managing patients in primary non-arrhythmogenic cardiac arrest. *Resuscitation* 2008;76:198-206.
162. Breitzkreutz R, Walcher F, Seeger FH. Focused echocardiographic evaluation in resuscitation management: concept of an advanced life support-conformed algorithm. *Crit Care Med* 2007;35:S150-61.
163. Soar J, Foster J, Breitzkreutz R. Fluid infusion during CPR and after ROSC--is it safe? *Resuscitation* 2009;80:1221-2.

164. Perkins GD, Davies RP, Soar J, Thickett DR. The impact of manual defibrillation technique on no-flow time during simulated cardiopulmonary resuscitation. *Resuscitation* 2007;73:109-14.
165. Stults KR, Brown DD, Cooley F, Kerber RE. Self-adhesive monitor/defibrillation pads improve prehospital defibrillation success. *Ann Emerg Med* 1987;16:872-7.
166. Deakin CD, McLaren RM, Petley GW, Clewlow F, Dalrymple-Hay MJ. A comparison of transthoracic impedance using standard defibrillation paddles and self-adhesive defibrillation pads. *Resuscitation* 1998;39:43-6.
167. Nolan JP, Soar J. Airway techniques and ventilation strategies. *Curr Opin Crit Care* 2008;14:279-86.
168. Gatward JJ, Thomas MJ, Nolan JP, Cook TM. Effect of chest compressions on the time taken to insert airway devices in a manikin. *Br J Anaesth* 2008;100:351-6.
169. Wiese CH, Semmel T, Muller JU, Bahr J, Ocker H, Graf BM. The use of the laryngeal tube disposable (LT-D) by paramedics during out-of-hospital resuscitation-an observational study concerning ERC guidelines 2005. *Resuscitation* 2009;80:194-8.
170. Li J. Capnography alone is imperfect for endotracheal tube placement confirmation during emergency intubation. *J Emerg Med* 2001;20:223-9.
171. Shavit I, Hoffmann Y, Galbraith R, Waisman Y. Comparison of two mechanical intraosseous infusion devices: a pilot, randomized crossover trial. *Resuscitation* 2009;80:1029-33.
172. Ong ME, Chan YH, Oh JJ, Ngo AS. An observational, prospective study comparing tibial and humeral intraosseous access using the EZ-IO. *Am J Emerg Med* 2009;27:8-15.
173. Gerritse BM, Scheffer GJ, Draaisma JM. Prehospital intraosseous access with the bone injection gun by a helicopter-transported emergency medical team. *J Trauma* 2009;66:1739-41.
174. Kudenchuk PJ, Cobb LA, Copass MK, et al. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. *N Engl J Med* 1999;341:871-8.
175. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. *N Engl J Med* 2002;346:884-90.
176. Masini E, Planchenault J, Pezziardi F, Gautier P, Gagnol JP. Histamine-releasing properties of Polysorbate 80 in vitro and in vivo: correlation with its hypotensive action in the dog. *Agents Actions* 1985;16:470-7.

177. Campbell S, Nolan PE, Jr., Bliss M, Wood R, Mayersohn M. Stability of amiodarone hydrochloride in admixtures with other injectable drugs. *Am J Hosp Pharm* 1986;43:917-21.
178. Thel MC, Armstrong AL, McNulty SE, Califf RM, O'Connor CM. Randomised trial of magnesium in in-hospital cardiac arrest. *Duke Internal Medicine Housestaff. Lancet* 1997;350:1272-6.
179. Allegra J, Lavery R, Cody R, et al. Magnesium sulfate in the treatment of refractory ventricular fibrillation in the prehospital setting. *Resuscitation* 2001;49:245-9.
180. Fatovich D, Prentice D, Dobb G. Magnesium in in-hospital cardiac arrest. *Lancet* 1998;351:446.
181. Hassan TB, Jagger C, Barnett DB. A randomised trial to investigate the efficacy of magnesium sulphate for refractory ventricular fibrillation. *Emerg Med J* 2002;19:57-62.
182. Miller B, Craddock L, Hoffenberg S, et al. Pilot study of intravenous magnesium sulfate in refractory cardiac arrest: safety data and recommendations for future studies. *Resuscitation* 1995;30:3-14.
183. Longstreth WT, Jr., Fahrenbruch CE, Olsufka M, Walsh TR, Copass MK, Cobb LA. Randomized clinical trial of magnesium, diazepam, or both after out-of-hospital cardiac arrest. *Neurology* 2002;59:506-14.
184. Weil MH, Rackow EC, Trevino R, Grundler W, Falk JL, Griffel MI. Difference in acid-base state between venous and arterial blood during cardiopulmonary resuscitation. *N Engl J Med* 1986;315:153-6.
185. Delguercio LR, Feins NR, Cohn JD, Coomaraswamy RP, Wollman SB, State D. Comparison of blood flow during external and internal cardiac massage in man. *Circulation* 1965;31:SUPPL 1:171-80.
186. Kramer-Johansen J, Myklebust H, Wik L, et al. Quality of out-of-hospital cardiopulmonary resuscitation with real time automated feedback: a prospective interventional study. *Resuscitation* 2006;71:283-92.
187. Sutton RM, Maltese MR, Niles D, et al. Quantitative analysis of chest compression interruptions during in-hospital resuscitation of older children and adolescents. *Resuscitation* 2009;80:1259-63.
188. Sutton RM, Niles D, Nysaether J, et al. Quantitative analysis of CPR quality during in-hospital resuscitation of older children and adolescents. *Pediatrics* 2009;124:494-9.

189. Cabrini L, Beccaria P, Landoni G, et al. Impact of impedance threshold devices on cardiopulmonary resuscitation: a systematic review and meta-analysis of randomized controlled studies. *Crit Care Med* 2008;36:1625-32.
190. Steen S, Liao Q, Pierre L, Paskevicius A, Sjoberg T. Evaluation of LUCAS, a new device for automatic mechanical compression and active decompression resuscitation. *Resuscitation* 2002;55:285-99.
191. Rubertsson S, Karlsten R. Increased cortical cerebral blood flow with LUCAS; a new device for mechanical chest compressions compared to standard external compressions during experimental cardiopulmonary resuscitation. *Resuscitation* 2005;65:357-63.
192. Timerman S, Cardoso LF, Ramires JA, Halperin H. Improved hemodynamic performance with a novel chest compression device during treatment of in-hospital cardiac arrest. *Resuscitation* 2004;61:273-80.
193. Halperin H, Berger R, Chandra N, et al. Cardiopulmonary resuscitation with a hydraulic-pneumatic band. *Crit Care Med* 2000;28:N203-N6.
194. Halperin HR, Paradis N, Ornato JP, et al. Cardiopulmonary resuscitation with a novel chest compression device in a porcine model of cardiac arrest: improved hemodynamics and mechanisms. *J Am Coll Cardiol* 2004;44:2214-20.
195. Hallstrom A, Rea TD, Sayre MR, et al. Manual chest compression vs use of an automated chest compression device during resuscitation following out-of-hospital cardiac arrest: a randomized trial. *JAMA* 2006;295:2620-8.
196. Ong ME, Ornato JP, Edwards DP, et al. Use of an automated, load-distributing band chest compression device for out-of-hospital cardiac arrest resuscitation. *JAMA* 2006;295:2629-37.
197. Larsen AI, Hjernevik AS, Ellingsen CL, Nilsen DW. Cardiac arrest with continuous mechanical chest compression during percutaneous coronary intervention. A report on the use of the LUCAS device. *Resuscitation* 2007;75:454-9.
198. Wagner H, Terkelsen CJ, Friberg H, et al. Cardiac arrest in the catheterisation laboratory: a 5-year experience of using mechanical chest compressions to facilitate PCI during prolonged resuscitation efforts. *Resuscitation* 2010;81:383-7.
199. Wirth S, Korner M, Treitl M, et al. Computed tomography during cardiopulmonary resuscitation using automated chest compression devices--an initial study. *Eur Radiol* 2009;19:1857-66.
200. Holmstrom P, Boyd J, Sorsa M, Kuisma M. A case of hypothermic cardiac arrest treated with an external chest compression device (LUCAS) during transport to re-warming. *Resuscitation* 2005;67:139-41.

201. Wik L, Kiil S. Use of an automatic mechanical chest compression device (LUCAS) as a bridge to establishing cardiopulmonary bypass for a patient with hypothermic cardiac arrest. *Resuscitation* 2005;66:391-4.
202. Nolan JP, Neumar RW, Adrie C, et al. Post-cardiac arrest syndrome: epidemiology, pathophysiology, treatment, and prognostication. A Scientific Statement from the International Liaison Committee on Resuscitation; the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiovascular Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative, and Critical Care; the Council on Clinical Cardiology; the Council on Stroke. *Resuscitation* 2008;79:350-79.
203. Laurent I, Monchi M, Chiche JD, et al. Reversible myocardial dysfunction in survivors of out-of-hospital cardiac arrest. *J Am Coll Cardiol* 2002;40:2110-6.
204. Ruiz-Bailen M, Aguayo de Hoyos E, Ruiz-Navarro S, et al. Reversible myocardial dysfunction after cardiopulmonary resuscitation. *Resuscitation* 2005;66:175-81.
205. Cerchiari EL, Safar P, Klein E, Diven W. Visceral, hematologic and bacteriologic changes and neurologic outcome after cardiac arrest in dogs. The visceral post-resuscitation syndrome. *Resuscitation* 1993;25:119-36.
206. Adrie C, Monchi M, Laurent I, et al. Coagulopathy after successful cardiopulmonary resuscitation following cardiac arrest: implication of the protein C anticoagulant pathway. *J Am Coll Cardiol* 2005;46:21-8.
207. Adrie C, Adib-Conquy M, Laurent I, et al. Successful cardiopulmonary resuscitation after cardiac arrest as a "sepsis-like" syndrome. *Circulation* 2002;106:562-8.
208. Adrie C, Laurent I, Monchi M, Cariou A, Dhainaou JF, Spaulding C. Postresuscitation disease after cardiac arrest: a sepsis-like syndrome? *Curr Opin Crit Care* 2004;10:208-12.
209. Balan IS, Fiskum G, Hazelton J, Cotto-Cumba C, Rosenthal RE. Oximetry-guided reoxygenation improves neurological outcome after experimental cardiac arrest. *Stroke* 2006;37:3008-13.
210. Kilgannon JH, Jones AE, Shapiro NI, et al. Association between arterial hyperoxia following resuscitation from cardiac arrest and in-hospital mortality. *JAMA* 2010;303:2165-71.
211. Spaulding CM, Joly LM, Rosenberg A, et al. Immediate coronary angiography in survivors of out-of-hospital cardiac arrest. *N Engl J Med* 1997;336:1629-33.
212. Sunde K, Pytte M, Jacobsen D, et al. Implementation of a standardised treatment protocol for post resuscitation care after out-of-hospital cardiac arrest. *Resuscitation* 2007;73:29-39.

213. Hovdenes J, Laake JH, Aaberge L, Haugaa H, Bugge JF. Therapeutic hypothermia after out-of-hospital cardiac arrest: experiences with patients treated with percutaneous coronary intervention and cardiogenic shock. *Acta Anaesthesiol Scand* 2007;51:137-42.
214. Knafelj R, Radsel P, Ploj T, Noc M. Primary percutaneous coronary intervention and mild induced hypothermia in comatose survivors of ventricular fibrillation with ST-elevation acute myocardial infarction. *Resuscitation* 2007;74:227-34.
215. Wolfrum S, Pierau C, Radke PW, Schunkert H, Kurowski V. Mild therapeutic hypothermia in patients after out-of-hospital cardiac arrest due to acute ST-segment elevation myocardial infarction undergoing immediate percutaneous coronary intervention. *Crit Care Med* 2008;36:1780-6.
216. Nielsen N, Hovdenes J, Nilsson F, et al. Outcome, timing and adverse events in therapeutic hypothermia after out-of-hospital cardiac arrest. *Acta Anaesthesiol Scand* 2009;53:926-34.
217. Snyder BD, Hauser WA, Loewenson RB, Leppik IE, Ramirez-Lassepas M, Gumnit RJ. Neurologic prognosis after cardiopulmonary arrest, III: seizure activity. *Neurology* 1980;30:1292-7.
218. Levy DE, Caronna JJ, Singer BH, Lapinski RH, Frydman H, Plum F. Predicting outcome from hypoxic-ischemic coma. *JAMA* 1985;253:1420-6.
219. Krumholz A, Stern BJ, Weiss HD. Outcome from coma after cardiopulmonary resuscitation: relation to seizures and myoclonus. *Neurology* 1988;38:401-5.
220. Zandbergen EG, Hijdra A, Koelman JH, et al. Prediction of poor outcome within the first 3 days of postanoxic coma. *Neurology* 2006;66:62-8.
221. Nolan JP, Laver SR, Welch CA, Harrison DA, Gupta V, Rowan K. Outcome following admission to UK intensive care units after cardiac arrest: a secondary analysis of the ICNARC Case Mix Programme Database. *Anaesthesia* 2007;62:1207-16.
222. Mullner M, Sterz F, Binder M, Schreiber W, Deimel A, Laggner AN. Blood glucose concentration after cardiopulmonary resuscitation influences functional neurological recovery in human cardiac arrest survivors. *J Cereb Blood Flow Metab* 1997;17:430-6.
223. Finfer S, Chittock DR, Su SY, et al. Intensive versus conventional glucose control in critically ill patients. *N Engl J Med* 2009;360:1283-97.
224. Preiser JC, Devos P, Ruiz-Santana S, et al. A prospective randomised multi-centre controlled trial on tight glucose control by intensive insulin therapy in adult intensive care units: the Glucontrol study. *Intensive Care Med* 2009;35:1738-48.

225. Griesdale DE, de Souza RJ, van Dam RM, et al. Intensive insulin therapy and mortality among critically ill patients: a meta-analysis including NICE-SUGAR study data. *CMAJ* 2009;180:821-7.
226. Wiener RS, Wiener DC, Larson RJ. Benefits and risks of tight glucose control in critically ill adults: a meta-analysis. *JAMA* 2008;300:933-44.
227. Krinsley JS, Grover A. Severe hypoglycemia in critically ill patients: risk factors and outcomes. *Crit Care Med* 2007;35:2262-7.
228. Padkin A. Glucose control after cardiac arrest. *Resuscitation* 2009;80:611-2.
229. Hickey RW, Kochanek PM, Ferimer H, Alexander HL, Garman RH, Graham SH. Induced hyperthermia exacerbates neurologic neuronal histologic damage after asphyxial cardiac arrest in rats. *Crit Care Med* 2003;31:531-5.
230. Zeiner A, Holzer M, Sterz F, et al. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. *Arch Intern Med* 2001;161:2007-12.
231. Hickey RW, Kochanek PM, Ferimer H, Graham SH, Safar P. Hypothermia and hyperthermia in children after resuscitation from cardiac arrest. *Pediatrics* 2000;106(pt 1):118-22.
232. Langhelle A, Tyvold SS, Lexow K, Hapnes SA, Sunde K, Steen PA. In-hospital factors associated with improved outcome after out-of-hospital cardiac arrest. A comparison between four regions in Norway. *Resuscitation* 2003;56:247-63.
233. Gunn AJ, Thoresen M. Hypothermic neuroprotection. *NeuroRx* 2006;3:154-69.
234. Froehler MT, Geocadin RG. Hypothermia for neuroprotection after cardiac arrest: mechanisms, clinical trials and patient care. *J Neurol Sci* 2007;261:118-26.
235. McCullough JN, Zhang N, Reich DL, et al. Cerebral metabolic suppression during hypothermic circulatory arrest in humans. *Ann Thorac Surg* 1999;67:1895-9; discussion 919-21.
236. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med* 2002;346:549-56.
237. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med* 2002;346:557-63.
238. Bernard SA, Jones BM, Horne MK. Clinical trial of induced hypothermia in comatose survivors of out-of-hospital cardiac arrest. *Ann Emerg Med* 1997;30:146-53.
239. Oddo M, Schaller MD, Feihl F, Ribordy V, Liaudet L. From evidence to clinical practice: effective implementation of therapeutic hypothermia to improve patient outcome after cardiac arrest. *Crit Care Med* 2006;34:1865-73.

240. Arrich J. Clinical application of mild therapeutic hypothermia after cardiac arrest. *Crit Care Med* 2007;35:1041-7.
241. Holzer M, Mullner M, Sterz F, et al. Efficacy and safety of endovascular cooling after cardiac arrest: cohort study and Bayesian approach. *Stroke* 2006;37:1792-7.
242. Polderman KH, Herold I. Therapeutic hypothermia and controlled normothermia in the intensive care unit: practical considerations, side effects, and cooling methods. *Crit Care Med* 2009;37:1101-20.
243. Kuboyama K, Safar P, Radovsky A, et al. Delay in cooling negates the beneficial effect of mild resuscitative cerebral hypothermia after cardiac arrest in dogs: a prospective, randomized study. *Crit Care Med* 1993;21:1348-58.
244. Laver SR, Padkin A, Atalla A, Nolan JP. Therapeutic hypothermia after cardiac arrest: a survey of practice in intensive care units in the United Kingdom. *Anaesthesia* 2006;61:873-7.
245. Edgren E, Hedstrand U, Nordin M, Rydin E, Ronquist G. Prediction of outcome after cardiac arrest. *Crit Care Med* 1987;15:820-5.
246. Young GB, Doig G, Ragazzoni A. Anoxic-ischemic encephalopathy: clinical and electrophysiological associations with outcome. *Neurocrit Care* 2005;2:159-64.
247. Al Thenayan E, Savard M, Sharpe M, Norton L, Young B. Predictors of poor neurologic outcome after induced mild hypothermia following cardiac arrest. *Neurology* 2008;71:1535-7.
248. Wijdicks EF, Parisi JE, Sharbrough FW. Prognostic value of myoclonus status in comatose survivors of cardiac arrest. *Ann Neurol* 1994;35:239-43.
249. Thomke F, Marx JJ, Sauer O, et al. Observations on comatose survivors of cardiopulmonary resuscitation with generalized myoclonus. *BMC Neurol* 2005;5:14.
250. Wijdicks EF, Hijdra A, Young GB, Bassetti CL, Wiebe S. Practice parameter: prediction of outcome in comatose survivors after cardiopulmonary resuscitation (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology* 2006;67:203-10.
251. [Deakin CD, Morrison LJ, Morley PT, et al. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 8: Advanced Life Support. *Resuscitation* 2010;81:e93-e169.](#)
252. Tiainen M, Kovala TT, Takkunen OS, Roine RO. Somatosensory and brainstem auditory evoked potentials in cardiac arrest patients treated with hypothermia. *Crit Care Med* 2005;33:1736-40.

253. Adrie C, Haouache H, Saleh M, et al. An underrecognized source of organ donors: patients with brain death after successfully resuscitated cardiac arrest. *Intensive Care Med* 2008;34:132-7.
254. Fieux F, Losser MR, Bourgeois E, et al. Kidney retrieval after sudden out of hospital refractory cardiac arrest: a cohort of uncontrolled non heart beating donors. *Crit Care* 2009;13:R141.
255. Carr BG, Goyal M, Band RA, et al. A national analysis of the relationship between hospital factors and post-cardiac arrest mortality. *Intensive Care Med* 2009;35:505-11.
256. Liu JM, Yang Q, Pirralo RG, Klein JP, Aufderheide TP. Hospital variability of out-of-hospital cardiac arrest survival. *Prehosp Emerg Care* 2008;12:339-46.
257. Carr BG, Kahn JM, Merchant RM, Kramer AA, Neumar RW. Inter-hospital variability in post-cardiac arrest mortality. *Resuscitation* 2009;80:30-4.
258. Keenan SP, Dodek P, Martin C, Priestap F, Norena M, Wong H. Variation in length of intensive care unit stay after cardiac arrest: where you are is as important as who you are. *Crit Care Med* 2007;35:836-41.
259. Nichol G, Aufderheide TP, Eigel B, et al. Regional systems of care for out-of-hospital cardiac arrest: A policy statement from the American Heart Association. *Circulation* 2010;121:709-29.
260. Nichol G, Soar J. Regional cardiac resuscitation systems of care. *Curr Opin Crit Care* 2010;16:223-30.
261. Soar J, Packham S. Cardiac arrest centres make sense. *Resuscitation* 2010;81:507-8.
262. Delacretaz E. Clinical practice. Supraventricular tachycardia. *N Engl J Med* 2006;354:1039-51.
263. Fuster V, Ryden LE, Cannom DS, et al. ACC/AHA/ESC 2006 Guidelines for the Management of Patients with Atrial Fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Circulation* 2006;114:e257-354.
264. Dauchot P, Gravenstein JS. Effects of atropine on the electrocardiogram in different age groups. *Clin Pharmacol Ther* 1971;12:274-80.
265. Chamberlain DA, Turner P, Sneddon JM. Effects of atropine on heart-rate in healthy man. *Lancet* 1967;2:12-5.



266. Bernheim A, Fatio R, Kiowski W, Weilenmann D, Rickli H, Rocca HP. Atropine often results in complete atrioventricular block or sinus arrest after cardiac transplantation: an unpredictable and dose-independent phenomenon. *Transplantation* 2004;77:1181-5.
267. Klumbies A, Paliege R, Volkmann H. [Mechanical emergency stimulation in asystole and extreme bradycardia]. *Z Gesamte Inn Med* 1988;43:348-52.
268. Zeh E, Rahner E. [The manual extrathoracal stimulation of the heart. Technique and effect of the precordial thump (author's transl)]. *Z Kardiol* 1978;67:299-304.
269. Chan L, Reid C, Taylor B. Effect of three emergency pacing modalities on cardiac output in cardiac arrest due to ventricular asystole. *Resuscitation* 2002;52:117-9.
270. [Wyllie J, Perlman JM, Kattwinkel J, et al. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 11: Neonatal Resuscitation. *Resuscitation* 2010;81:e255-e282.](#)
271. [de Caen AR, Kleinman ME, Chameides L, et al. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 10: Pediatric Basic and Advanced Life Support. *Resuscitation* 2010;81:e208-e254.](#)
272. [Sunde K, Jacobs I, Deakin CD, et al. 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 6: Defibrillation. *Resuscitation* 2010;81:e71-e85.](#)
273. Babbs CF, Nadkarni V. Optimizing chest compression to rescue ventilation ratios during one-rescuer CPR by professionals and lay persons: children are not just little adults. *Resuscitation* 2004;61:173-81.
274. Berg RA, Hilwig RW, Kern KB, Babar I, Ewy GA. Simulated mouth-to-mouth ventilation and chest compressions (bystander cardiopulmonary resuscitation) improves outcome in a swine model of prehospital pediatric asphyxial cardiac arrest. *Crit Care Med* 1999;27:1893-9.
275. Tang W, Weil MH, Jorgenson D, et al. Fixed-energy biphasic waveform defibrillation in a pediatric model of cardiac arrest and resuscitation. *Crit Care Med* 2002;30:2736-41.
276. Herlitz J, Svensson L, Engdahl J, et al. Characteristics of cardiac arrest and resuscitation by age group: an analysis from the Swedish Cardiac Arrest Registry. *Am J Emerg Med* 2007;25:1025-31.
277. Berg MD, Samson RA, Meyer RJ, Clark LL, Valenzuela TD, Berg RA. Pediatric defibrillation doses often fail to terminate prolonged out-of-hospital ventricular fibrillation in children. *Resuscitation* 2005;67:63-7.



278. Rodriguez-Nunez A, Lopez-Herce J, Garcia C, Dominguez P, Carrillo A, Bellon JM. Pediatric defibrillation after cardiac arrest: initial response and outcome. *Crit Care* 2006;10:R113.
279. Tibballs J, Carter B, Kiraly NJ, Ragg P, Clifford M. External and internal biphasic direct current shock doses for pediatric ventricular fibrillation and pulseless ventricular tachycardia. *Pediatr Crit Care Med* 2010.
280. Skrifvars MB, Pettila V, Rosenberg PH, Castren M. A multiple logistic regression analysis of in-hospital factors related to survival at six months in patients resuscitated from out-of-hospital ventricular fibrillation. *Resuscitation* 2003;59:319-28.
281. Van de Louw A, Cracco C, Cerf C, et al. Accuracy of pulse oximetry in the intensive care unit. *Intensive Care Med* 2001;27:1606-13.
282. Seguin P, Le Rouzo A, Tanguy M, Guillou YM, Feuillu A, Malledant Y. Evidence for the need of bedside accuracy of pulse oximetry in an intensive care unit. *Crit Care Med* 2000;28:703-6.
283. Doherty DR, Parshuram CS, Gaboury I, et al. Hypothermia therapy after pediatric cardiac arrest. *Circulation* 2009;119:1492-500.
284. Takino M, Okada Y. Hyperthermia following cardiopulmonary resuscitation. *Intensive Care Med* 1991;17:419-20.
285. Takasu A, Saitoh D, Kaneko N, Sakamoto T, Okada Y. Hyperthermia: is it an ominous sign after cardiac arrest? *Resuscitation* 2001;49:273-7.
286. [Biarent D, Bingham R, Eich C, et al. European Resuscitation Council Guidelines for Resuscitation 2010. Section 6. Paediatric Life Support. *Resuscitation* 2010;81.](#)
287. [Wyllie J, Richmond S. European Resuscitation Council Guidelines for Resuscitation 2010. Section 7. Resuscitation of babies at birth. *Resuscitation* 2010;81.](#)
288. O'Donnell CP, Kamlin CO, Davis PG, Morley CJ. Feasibility of and delay in obtaining pulse oximetry during neonatal resuscitation. *J Pediatr* 2005;147:698-9.
289. Meyer A, Nadkarni V, Pollock A, et al. Evaluation of the Neonatal Resuscitation Program's recommended chest compression depth using computerized tomography imaging. *Resuscitation* 2010;81:544-8.
290. McDonald SJ, Middleton P. Effect of timing of umbilical cord clamping of term infants on maternal and neonatal outcomes. *Cochrane Database Syst Rev* 2008:CD004074.
291. Rabe H, Reynolds G, Diaz-Rossello J. Early versus delayed umbilical cord clamping in preterm infants. *Cochrane Database Syst Rev* 2004:CD003248.



292. Aladangady N, McHugh S, Aitchison TC, Wardrop CA, Holland BM. Infants' blood volume in a controlled trial of placental transfusion at preterm delivery. *Pediatrics* 2006;117:93-8.
293. Mercer JS, Vohr BR, McGrath MM, Padbury JF, Wallach M, Oh W. Delayed cord clamping in very preterm infants reduces the incidence of intraventricular hemorrhage and late-onset sepsis: a randomized, controlled trial. *Pediatrics* 2006;117:1235-42.
294. Mariani G, Dik PB, Ezquer A, et al. Pre-ductal and post-ductal O₂ saturation in healthy term neonates after birth. *J Pediatr* 2007;150:418-21.
295. Toth B, Becker A, Seelbach-Gobel B. Oxygen saturation in healthy newborn infants immediately after birth measured by pulse oximetry. *Arch Gynecol Obstet* 2002;266:105-7.
296. O'Donnell CP, Kamlin CO, Davis PG, Morley CJ. Obtaining pulse oximetry data in neonates: a randomised crossover study of sensor application techniques. *Arch Dis Child Fetal Neonatal Ed* 2005;90:F84-5.
297. [Dawson JA, Kamlin CO, Vento M, et al. Defining the reference range for oxygen saturation for infants after birth. *Pediatrics* 2010;125:e1340-7.](#)
298. Wang CL, Anderson C, Leone TA, Rich W, Govindaswami B, Finer NN. Resuscitation of preterm neonates by using room air or 100% oxygen. *Pediatrics* 2008;121:1083-9.
299. O'Donnell CP, Kamlin CO, Davis PG, Carlin JB, Morley CJ. Clinical assessment of infant colour at delivery. *Arch Dis Child Fetal Neonatal Ed* 2007;92:F465-7.
300. Wiswell TE, Gannon CM, Jacob J, et al. Delivery room management of the apparently vigorous meconium-stained neonate: results of the multicenter, international collaborative trial. *Pediatrics* 2000;105:1-7.
301. Vain NE, Szyld EG, Prudent LM, Wiswell TE, Aguilar AM, Vivas NI. Oropharyngeal and nasopharyngeal suctioning of meconium-stained neonates before delivery of their shoulders: multicentre, randomised controlled trial. *Lancet* 2004;364:597-602.
302. Perlman JM, Risser R. Cardiopulmonary resuscitation in the delivery room: associated clinical events. *Arch Pediatr Adolesc Med* 1995;149:20-5.
303. Edwards AD, Brocklehurst P, Gunn AJ, et al. Neurological outcomes at 18 months of age after moderate hypothermia for perinatal hypoxic ischaemic encephalopathy: synthesis and meta-analysis of trial data. *BMJ* 2010;340:c363.
304. Gluckman PD, Wyatt JS, Azzopardi D, et al. Selective head cooling with mild systemic hypothermia after neonatal encephalopathy: multicentre randomised trial. *Lancet* 2005;365:663-70.

305. Shankaran S, Laptook AR, Ehrenkranz RA, et al. Whole-body hypothermia for neonates with hypoxic-ischemic encephalopathy. *N Engl J Med* 2005;353:1574-84.
306. Azzopardi DV, Strohm B, Edwards AD, et al. Moderate hypothermia to treat perinatal asphyxial encephalopathy. *N Engl J Med* 2009;361:1349-58.
307. Eicher DJ, Wagner CL, Katikaneni LP, et al. Moderate hypothermia in neonatal encephalopathy: efficacy outcomes. *Pediatr Neurol* 2005;32:11-7.
308. Lin ZL, Yu HM, Lin J, Chen SQ, Liang ZQ, Zhang ZY. Mild hypothermia via selective head cooling as neuroprotective therapy in term neonates with perinatal asphyxia: an experience from a single neonatal intensive care unit. *J Perinatol* 2006;26:180-4.

Appendix: Conflict of interest declaration

Name and position	Affiliation	Declared interest
Jerry Nolan Editor, RC(UK) Executive member	Consultant in Anaesthesia and Intensive Care Medicine Royal United Hospital, Combe Park Bath BA1 3NG	Editor-in-Chief, <i>Resuscitation</i> . Board Member ERC. Co-Chair ILCOR. Chair, National Cardiac Arrest Audit.
Jasmeet Soar Co-Editor RC(UK) Chairman, ILS Subcommittee Chairman	Consultant in Anaesthesia and Intensive Care Medicine Southmead Hospital North Bristol NHS Trust Bristol BS10 5NB	Editor, <i>Resuscitation</i> . Chairman ERC ILS course committee. Co-chair ILCOR education implementation and teams taskforce. National Cardiac Arrest Audit steering group.
Robert Bingham RC(UK) Paediatric Subcommittee Chairman	Consultant Paediatric Anaesthetist Great Ormond St Hospital for Children NHS Trust London WC1N 3JH	ERC and ILCOR Vice Chair ERC ICC. Association of Paediatric Anaesthetists Council member.
Mick Colquhoun RC(UK) BLS/AED Subcommittee Deputy Chairman	General Practitioner (retired) c/o Resuscitation Council (UK) Tavistock House North, Tavistock Square London WC1H 9HR	Medical director of a company producing and marketing on-line training materials.
Robin Davies	Senior Resuscitation Officer Heart of England NHS Foundation Trust Birmingham B9 5SS	NIL
Charles Deakin RC(UK) Executive member	Consultant Anaesthetist Southampton General Hospital SO16 6YD	Chair/Co-Chair of ALS Committee of ERC and ILCOR respectively. Editorial board of <i>Resuscitation</i> . Occasional consulting work for Smiths Medical.



Name and position	Affiliation	Declared interest
Anthony Handley RC(UK) BLS/AED Subcommittee Chairman	Honorary Consultant Physician c/o Resuscitation Council (UK) Tavistock House North, Tavistock Square London WC1H 9HR	Consultant Adviser on CPR/AED Provision and Training: <ul style="list-style-type: none">• British Airways;• Virgin Atlantic Airways;• DC Leisure Management. Adviser to ERC board. Member ERC BLS/AED Subcommittee. Chairman ERC BLS/AED International Course Committee. Chairman ERC E-learning taskforce. Chief Medical Adviser, Royal Life Saving Society UK; Honorary Medical Officer, Irish Water Safety; Chairman, Medical Committee, International Life Saving Federation. Honorary Medical Adviser, International Life Saving Federation of Europe. Member BLS/AED Task Force ILCOR.
Fiona Jewkes RC(UK) Executive member	Clinical Author, NHS Pathways, RBDT Connecting for Health, Vantage House 40 Aire Street Leeds LS1 4HT	Clinical Author, NHS Pathways. Chair, Prehospital Paediatric Life Support. Hon Co Secretary, Joint Royal Colleges Ambulance Liaison Committee. Board Member, Faculty of Prehospital Care. Examiner for the Diploma in Immediate Medical Care. BASICS Education Committee For RCPCH; Member of the intercollegiate fever study Member of the project board and asthma and anaphylaxis working group for pathways of care for children with allergy project. For RCGP: Member of urgent care audit toolkit working group Representative to the Intercollegiate Board for prehospital emergency medicine. Medical advisor to a small company producing major incident equipment. Lecturer for the military PGMO course.
Andrew Lockey RC(UK) Honorary Secretary, ALS Subcommittee Chairman	Consultant Emergency Medicine Calderdale and Huddersfield NHS Trust Halifax HX3 0PW	Medical Advisor to 'First on Scene' First Aid company. ERC Executive Committee – Representative for RC(UK) and ERC Educational Advisory Group. Vice Chair – ERC Educational Advisory Group. Member of the ERC ALS and GIC ICCs.

Name and position	Affiliation	Declared interest
<p>Ian Maconochie RC(UK) EPLS Subcommittee Chairman</p>	<p>Consultant Paediatric Emergency Medicine St. Mary's Hospital London W2 1NY</p>	<p>Royal College of Paediatrics:</p> <ul style="list-style-type: none"> • Officer for Clinical Standards • Member of the Clinical Standards Committee • Member of the Intercollegiate Fever DH study <p>College of Emergency Medicine:</p> <ul style="list-style-type: none"> • Member of the Clinical Standards Committee <p>European Resuscitation Council:</p> <ul style="list-style-type: none"> • National director for EPLS • EPLS International Course Committee member. <p>NICE:</p> <ul style="list-style-type: none"> • Finished membership of the meningitis and meningococcaemia guideline – to be published in full in 2010. <p>Trustee Children Action Prevention Trust. Trustee Traumacare Charity. Member of Emergency Planning Clinical Advisory Group (DH). Medical advisor to Therakind. Assistance in running a study to look at intranasal irritation with diamorphine administration via that route. Medical advisor to a small company dealing with major incident equipment. Medical advisor to a newly founded small company to produce media material on national guidelines.</p>
<p>Gavin Perkins RC(UK) ALS Subcommittee Deputy Chairman</p>	<p>Associate Clinical Professor Critical Care and Resuscitation University of Warwick Warwick Medical School CV4 7AL</p>	<p>Employer, University of Warwick. Editor, <i>Resuscitation</i>. Grant recipient from NIHR for studies on quality of CPR and mechanical chest compression devices. Co-Director Research ICS. Medical Advisor – ‘First on scene’. Medical Advisor – RLSS (UK). ERC course committees.</p>
<p>David Pitcher RC(UK) Vice Chairman</p>	<p>Consultant Cardiologist Worcestershire Royal Hospital, Worcester, WR5 1DD</p>	<p>Member of the NICE guidelines development group on transient loss of consciousness.</p>
<p>Sam Richmond RC(UK) NLS Subcommittee past- Chairman</p>	<p>Consultant Neonatologist Sunderland District General Hospital Sunderland SR4 7TP</p>	<p>NIL</p>



Name and position	Affiliation	Declared interest
Sheila Simpson RC(UK) EPLS Subcommittee Deputy Chairman	Senior Resuscitation Officer Great Ormond St Hospital for Children NHS Trust London WC1N 3JH	NIL
Gary Smith	Consultant in Critical Care Portsmouth Hospitals NHS Trust Portsmouth PO6 3LY	Employed, and paid, by Portsmouth Hospitals NHS Trust, which owns and runs the Acute Life-Threatening Events – recognition and Treatment (ALERT) course and the Bedside Emergency Assessment Course for HCAs (BEACH) and which receives payment for sales of the courses and course material to other healthcare institutions. Employed, and paid, by Portsmouth Hospitals NHS Trust, which is co-developer of the VitalPAC electronic data collection and handling system and which receives royalties from the sales of the system to other healthcare institutions. My wife is a share-holder in the Learning Clinic, the organisation which co-developed and markets VitalPAC (see above). Employers, Portsmouth Hospitals NHS Trust, have received several honoraria payments in recent years as a result of lectures/presentations given by me. I have not received these honoraria. Lead a research group that a) provides clinical advice and b) is involved in the development of novel patient monitoring devices with 1) Laerdal Medical and 2) The McLaren Group. Member of Royal College of Physicians of London's National Early Warning Score Development and Implementation Group.
Jonathan Wyllie RC(UK) NLS Subcommittee Chairman	Consultant Neonatologist The James Cook University Hospital Middlesbrough TS4 3BW	Chair NLS for ERC. Co-Chair ILCOR Neonatal Task Force. Vice-ICC for ERC NLS. Non-voting member of the ERC board. Member of Clinical advisory group for NEAS (ambulance service). Member of Clinical Advisory Group for HEMS Member of the APLS international working group (ALSG). Author of the NLS manual. Author of APLS neonatal chapter.