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CLINICAL REVIEW

Cardiopulmonary resuscitation

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Cardiorespiratory arrest is the most extreme medical emergency—death or permanent brain injury will ensue unless cardiopulmonary resuscitation (CPR) is started within minutes. Four key interventions—known collectively as the chain of survival and comprising early recognition of cardiac arrest, high quality CPR, prompt defibrillation, and effective post-resuscitation care—improve outcomes.[w1] This review covers recent developments in CPR and the evidence supporting them.

What is the incidence and outcome of sudden cardiac arrest?

The global incidence of out of hospital cardiac arrest in adults treated by emergency medical services is 62 cases per 100 000 person years; 75-85% of these arrests have a primary cardiac cause.[1] The reported incidence of out of hospital cardiac arrest and its outcome vary considerably. In Europe, the estimated survival to hospital discharge for such cardiac arrests is 8%.[1] Evidence suggests that survival rates are increasing,[w2-w4] mainly because CPR is being attempted more often. The improvement is modest, however, because of the decreasing incidence of ventricular fibrillation and pulseless ventricular tachycardia (25-30% of out of hospital cardiac arrests), which have a better prognosis. A recent high quality observational study from the Netherlands has shown that implantable cardioverter defibrillators account for about a third of this decline.[2] The decline in these rhythms has also been attributed to increased use of β blockers.[w5]

Patients having an in hospital cardiac arrest often have multiple comorbidities and the cause of the cardiac arrest is often multifactorial (table 1⇓). According to American registry data, about 200 000 cardiac arrests occur in hospital each year (67 cases per 100 000 person years), with 17.6% of patients surviving to hospital discharge.[3 4] A quarter of patients present with ventricular fibrillation or pulseless ventricular tachycardia. These patients have better survival to discharge (37.2%) than those with pulseless electrical activity or asystole (survival 11.3%). Early data (unpublished) from the UK National Cardiac Arrest Audit show similar outcomes.[w6] Outcomes of in hospital cardiac arrest are probably better than those of out of hospital arrests because the arrests are witnessed and CPR and advanced life support are started promptly.

How are CPR guidelines developed?

Since the 1990s, the International Liaison Committee on Resuscitation has facilitated systematic reviews of CPR science. In 2005 and 2010, the committee published an International Consensus on CPR Science with Treatment Recommendations.[5 6] The 2010 recommendations involved experts from more than 30 countries who evaluated the findings from 277 systematic reviews based on PICO (population, intervention, comparison, outcome) format questions that had been undertaken over three years. The summary treatment recommendations that came from this rigorous process were used to formulate more detailed practical resuscitation guidelines globally.[7 8] It has not been possible to produce a single set of global guidelines because of regional variations driven by cultural and economic differences. Nevertheless, the guidelines are sufficiently similar to enable international application of the core interventions. The CPR science review and guideline cycle occurs every five years. The remainder of this review will cover key aspects of the 2010 CPR guidelines and will also highlight new research that may influence future guidelines.

Is cardiac arrest preventable?

The vital signs of patients about to have a cardiac arrest in hospital often deteriorate in the hours preceding the event. A meta-analysis concluded that rapid response systems (that incorporate "tracking" of vital signs and criteria for "triggering" a clinical response) reduce the incidence of cardiac arrests outside of the intensive care unit but do not reduce hospital mortality rates.[9 w7] This might be explained by an increase in the number of do not attempt CPR (DNACPR) decisions.

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Web references
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Summary points

- About 8% of resuscitation attempts after out of hospital cardiac arrest result in survival to hospital discharge
- Give chest compressions to a depth of 5-6 cm at 100-120 per minute; fully release between each compression and minimise interruptions; untrained bystanders should use compression only cardiopulmonary resuscitation (CPR)
- CPR prompt and feedback devices improve the quality of CPR but have yet to be shown to improve survival
- Undertake defibrillation with minimal interruption in chest compressions
- The optimal method for managing the airway during cardiac arrest is unknown
- Mechanical CPR devices may have a role during transport and in the cardiac catheterisation laboratory
- Although adrenaline is recommended and used routinely, its effect on long term neurological outcome is unclear

Sources and selection criteria

The 2010 Consensus on Cardiopulmonary Resuscitation Science with Treatment recommendations published by the International Liaison Committee on Resuscitation summarised the findings from 277 systematic reviews based on PICO (population, intervention, comparison, outcome) format questions. We continuously screen the scientific literature for resuscitation studies and have supplemented our findings and the contents of the 2010 recommendations with PubMed searches to identify other relevant resuscitation studies published since 2010.

When should a do not attempt CPR (DNACPR) decision be considered?

CPR has undoubtedly saved many thousands of lives over the years. Successful CPR was first described in groups of patients considered to have "hearts too young to die." However cardiac arrest (cessation of heart beat) is also part of the natural dying process. Different countries' laws and ethics relating to resuscitation, end of life care, and advance decisions (living wills) vary greatly. In the United Kingdom, CPR may be withheld when clinical judgment concludes that CPR will not be able to restart the patient's heart and breathing and restore circulation; the benefits of CPR are agreed to be outweighed by the burdens and risks after careful discussion with the patient (or those close to the patient); a patient has an advanced decision (living will) or makes an informed decision to refuse CPR.[w8]

A recent National Confidential Enquiry into Patient Outcome and Death reviewed more than 700 in hospital cardiac arrests in the UK. The report found that most patients receiving CPR were elderly, frail, and had multiple comorbidities.[w9] Of the 230 cases for which assessors could form an opinion, they considered resuscitation on clinical grounds would be futile in 196 (85%). The report rightly calls for increased involvement of consultants in decisions on CPR among patients admitted to hospital. Better linkage with community DNACPR decisions is also needed.

How is cardiac arrest recognised?

People with cardiac arrest are unconscious, unresponsive, and not breathing or breathing abnormally—laypeople and healthcare professionals are taught to recognise these signs of cardiac arrest (fig 1⇓). A short seizure or gasping (agonal breathing) is common immediately after cardiac arrest.[w10] Agonal breathing is a sign of cardiac arrest and should prompt initiation of CPR.[10] Palpating for a carotid pulse is an unreliable and time consuming method to detect cardiac arrest and should be attempted only by those who are experienced in clinical assessment. Electrocardiography or other advanced monitoring, if available, may confirm the diagnosis, but its absence should not delay treatment.

How is CPR started?

In adults, CPR is started with 30 chest compressions followed by two ventilations (see table 2⇓ for summary of resuscitation interventions based on age). Continue to alternate chest compressions and ventilations (30:2) until a tracheal tube or a supraglottic airway device (see below) has been inserted, then continue ventilation at 10 breaths per minute while compressing the chest continually.

Animal data and evidence from observational clinical studies indicate that the quality of CPR strongly influences blood flow and outcome. The European Resuscitation Council currently recommends 100-120 chest compressions of 5-6 cm depth per minute ("push hard and fast"); pressure should be fully released between chest compressions and interruptions minimised.[11]

A recent observational clinical study found that the highest rates of return of spontaneous circulation were associated with chest compression rates of about 125 per minute[12]; however, other studies have shown that compression depth becomes too shallow at rates of more than 120 per minute.[13 w11] On the basis of all the available data, the optimal compression rate is 120 per minute.[w12]

The proportion of time during which chest compressions are performed in each minute of CPR (the chest compression fraction) is independently associated with better survival after out of hospital cardiac arrest caused by ventricular fibrillation: in one observational study a chest compression fraction of 61-80% was associated with the highest survival rate.[14]

Does compression only cardiopulmonary resuscitation have a role?

Standard CPR, which includes mouth to mouth ventilation, is more difficult to learn and remember than chest compression only CPR,[w13] and it interrupts chest compressions.[w14]

At the onset of sudden (non-asphyxial) cardiac arrest, the patient's lungs and great vessels contain enough oxygen to supply the tissues for several minutes if an adequate amount of blood can be circulated. A meta-analysis of several observational studies reported similar survival rates in people who received bystander compression only CPR compared with those resuscitated with standard CPR.[15 w15-w21] After introducing a programme of bystander compression only CPR throughout Arizona, lay rescuer CPR increased from 28% to 40% (P<0.001) and overall survival increased from 3.7% to 9.8% (P<0.001).[16] By contrast, the most recent and largest observational study showed that, compared with compression only CPR, standard CPR was associated with increased neurologically favourable survival at one month. This was particularly true for young people with a non-cardiac cause of cardiac arrest or if the start of CPR was delayed in witnessed cases of cardiac arrest of a non-cardiac cause.[17] Most (about 70%) out of hospital cardiac arrests in children have a non-cardiac cause, and, in a Japanese

nationwide prospective observational study, outcome in children was better when laypeople performed standard CPR rather than compression only CPR.[18]

Reliable data support dispatcher assisted bystander CPR, which occurs when the dispatcher instructs the caller to perform CPR while awaiting arrival of the emergency medical services. A meta-analysis of three prospective randomised trials found a 22% increase in the rate of survival to hospital discharge when the dispatcher gave telephone instructions for compression only CPR instead of standard CPR.[15 w22-w24]

The 2010 European Resuscitation Council (ERC) guidelines recommend teaching standard CPR to laypeople and healthcare providers, but compression only CPR is encouraged for those who are untrained or unable or unwilling to perform mouth to mouth ventilation.[11] Compression only CPR is clearly better than no CPR, and this was the primary message in high profile media campaigns in the UK and the United States that target people untrained in CPR.[w25 w26]

Can the quality of CPR be improved?

The quality of CPR can be improved with the use of CPR prompt and feedback devices. These vary in sophistication—from simple metronomes that guide compression rate, to modified defibrillators that monitor compression depth and rate from an accelerometer placed on the chest and ventilation volume and rate by measuring changes in transthoracic impedance. Audio and visual feedback to rescuers are given in real time and data can be stored for later review during debriefing.[w27] A systematic review of prompt and feedback devices concluded that during training they improve the acquisition and retention of skills, and that they improve the quality of CPR in clinical practice, but there is no evidence that they improve patient outcomes.[19] A prospective cluster randomised study showed that real time visual and audible feedback resulted in CPR that more closely matched the guidelines, but patient outcomes were not affected.[20]

Mechanical chest compression devices deliver consistent high quality CPR, but meta-analyses have failed to show that they improve patient outcomes.[w28 w29] The two main devices are the load distributing band (ZOLL Medical Corporation), which comprises a backboard and a disposable chest band that is tightened and loosened 80 times a minute, and a mechanical compression-decompression device (Physio-Control), which incorporates a suction cup that is pushed up and down on the chest by a battery powered piston.

A large multicentre randomised study of the load distributing band (Circulation Improving Resuscitation Care (CIRC) trial) showed that it did not improve survival to hospital discharge compared with high quality manual CPR (presented by L Wik at the Resuscitation Science Symposium, Orlando, Florida, 12-13 Nov 2011). Two ongoing large randomised studies (LINC and PARAMEDIC trials) are evaluating the prehospital use of the mechanical compression-decompression device.

Although existing data do not support the routine use of mechanical devices, they are used in many parts of the world and may have a role in specific circumstances, such as during transport or when access to the patient is limited, such as CPR during percutaneous coronary intervention.[21]

What is the role of impedance threshold devices in CPR?

The impedance threshold device augments the negative intrathoracic pressure generated during the decompression phase of chest compression; this increases the return of venous blood, thereby increasing blood flow with the subsequent chest compression. Animal and human studies show that use of these devices improves haemodynamic values compared with standard CPR.[w30] The devices have a greater haemodynamic effect when used together with active compression-decompression CPR. In an unblinded randomised trial in out of hospital cardiac arrests, the impedance threshold device combined with active compression-decompression CPR improved survival to hospital discharge with good neurological function compared with conventional CPR.[w31] When compared with a sham valve during standard CPR, use of the impedance threshold device produced no benefit.[w32] Given these conflicting data and the cost of the single use valve, routine use of these devices is not recommended.

Are manual or automated defibrillators more effective?

Defibrillation using a manual defibrillator or an automated external defibrillator is the only effective treatment for ventricular fibrillation or pulseless ventricular tachycardia cardiac arrest. Current guidelines recommend an energy level of 150 J for the first shock, with subsequent shocks at the same or higher values (up to 360 J) depending on the specific defibrillator. Use of an automated defibrillator does not require specific training (the rescuer simply follows the audiovisual instructions when the device is switched on). Two high quality population based cohort studies show that use of these devices by bystanders doubles survival after out of hospital cardiac arrest.[w33 w34]

Despite one study showing that shocks from a defibrillator can be delivered without injury to a gloved rescuer who maintains contact with the patient's chest,[22] expert consensus and current guidelines recommend that manual chest compressions are interrupted to enable safe defibrillation.[w35] When chest compressions are paused the right ventricle dilates and encroaches on the left ventricle—this may prevent the myocardium from contracting effectively even if defibrillation restores coordinated electrical activity.[w36] One observational study of in hospital cardiac arrest found increased shock success with shorter pre-shock pauses (the interval between stopping compressions and shock delivery; adjusted odds ratio 1.86 for every five second decrease in pre-shock pause).[23] In an observational out of hospital cardiac arrest study, survival to hospital discharge decreased by 18% for every five second increase in pre-shock pause up to 40 seconds.[24] It should be possible to deliver a shock with a manual defibrillator with no more than a five second interruption to chest compression.[25] A strategy to do this safely includes the continuation of chest compressions while the defibrillator is charging, and this is now taught in advanced life support courses (fig 2⇓).

What is the best way to manage the airway during CPR?

The optimal method for managing the airway during CPR is unknown. Attempting to intubate the trachea can cause serious interruption to chest compressions —longer than one minute in 30% of cases in one high quality observational study of intubation by paramedics.[26] Attempted intubation has a high failure rate unless undertaken by highly experienced workers, and it may result in unrecognised oesophageal intubation.[w37] The routine use of waveform capnography (a monitor that displays exhaled carbon dioxide as a waveform and not just a

numerical value or colour) reduces the incidence of unrecognised oesophageal intubation (pulmonary blood flow during CPR is usually sufficient to generate detectable exhaled carbon dioxide). It also provides an early indication of return of spontaneous circulation (sudden increase in end tidal carbon dioxide).[27] Some observational studies have documented an association between tracheal intubation and worse survival after out of hospital cardiac arrest.[w38] Supraglottic airway devices (such as the laryngeal mask airway, i-gel (airway device with a non-inflatable laryngeal cuff, integral bite block, and gastric drain tube, made by Intersurgical), and laryngeal tube) are increasingly being used for resuscitation. Although they are easier to insert, no prospective controlled trials are available to provide data on clinical outcomes compared with intubation. Two recent observational studies found worse survival rates with the use of such devices than with tracheal intubation during resuscitation after out of hospital cardiac arrest.[28 29] Until data from prospective controlled trials are available, European Resuscitation Council consensus guidelines recommend that tracheal intubation is attempted only by highly skilled people and that other rescuers manage the airway with a facemask or a supraglottic airway device.

What is the role of adrenaline during resuscitation?

When injected during cardiac arrest, adrenaline (epinephrine) increases aortic relaxation (diastolic) pressure and, in animal studies, thereby augments coronary and cerebral blood flow. Two randomised controlled trials evaluated the use of 1 mg doses of adrenaline in out of hospital cardiac arrest.[30 31] The first trial randomised patients to intravenous cannulation with injection of drugs (including adrenaline) versus neither until return of spontaneous circulation.[30] The other study compared adrenaline with placebo.[31] Both studies found increased rates of return of spontaneous circulation with adrenaline but no difference in survival to hospital discharge. Post hoc analysis of one of these trials showed that patients who received adrenaline had better short term outcomes but an overall decrease in survival to discharge (odds ratio 0.52, 95% confidence interval 0.29 to 0.92; P=0.024) and worse neurological outcomes.[w39] In an observational study of 417 188 out of hospital cardiac arrests in Japan, after adjustment for potential confounders, use of adrenaline was associated with a return of spontaneous circulation rate 2.5 times higher but a one month survival rate roughly half of that in those not given adrenaline.[32] Similar findings have been seen in North American and Swedish registry data.[w40 w41] Animal data indicate that, although adrenaline improves global cerebral blood flow during CPR, flow in the microcirculation is reduced.[w42] This might account for the failure of adrenaline to convert the higher return of spontaneous circulation rates into better long term survival. The uncertainty should be resolved through a large randomised placebo controlled trial. Until such a trial is completed, current guidelines recommend that adrenaline is given every three to five minutes during cardiac arrest (adults 1 mg; children 10 µg/kg; fig 3⇓).

Does echocardiography have a role during CPR?

Focused echocardiography used during the brief pause for a rhythm check may enable identification of potentially reversible causes of cardiac arrest: pericardial tamponade, pulmonary embolism, and hypovolaemia.[w43] A prospective observational study found that the finding of pseudo-pulseless electrical activity (cardiac wall motion seen on echocardiography in a pulseless patient) alters management and is associated with increased survival.[w44]

What happens after successful CPR?

Once return of spontaneous circulation is achieved, unless the duration of cardiac arrest has been very short, patients will be comatose for variable periods and most will develop the post-cardiac arrest syndrome, which comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia-reperfusion response, and any persistent precipitating pathology.[33 w45] Coronary artery disease is the most common cause of out of hospital cardiac arrest, and many of these patients will require urgent coronary angiography and percutaneous coronary intervention.[34] The increasing use of urgent percutaneous coronary intervention in this situation is driving the treatment of these patients in regional cardiac arrest centres.[35] Table 3⇓ describes interventions to optimise outcome.

A systematic review of nine prospective studies, three follow-up of untreated control groups in randomised controlled trials, 11 retrospective cohort studies, and 47 cases series concluded that the quality of life in survivors who leave hospital is generally good, although they may have psychological and cognitive problems.[36] The wide range in the type and timing of the neurological assessments used in the studies make it impossible to provide specific summary data. It is generally accepted that long term assessments should not be made until at least six months, and preferably one year, after cardiac arrest. Studies documenting these long term neurological outcomes generally report that more than 85% of patients have a "good" outcome defined by a cerebral performance category of 1 or 2 (online appendix).[w46 w47] Category 2 is described broadly as "disabled but independent" and includes patients with hemiplegia, seizures, and permanent memory changes. This system provides only a crude measurement of neurological outcome; studies that use much more sensitive tests of memory and cognition generally show subtle cognitive deficits in most survivors of cardiac arrest.[w48 w49]

How should we teach CPR?

The ability to recognise cardiac arrest and deliver CPR is an essential skill for all healthcare professionals. Knowledge and skills in this area can deteriorate within three to six months after training. Frequent assessments and, when needed, refresher training, are recommended to maintain knowledge and skills. Properly validated short video and online self instruction courses with hands on practice are an effective alternative to instructor led basic life support skills.[w50] A large randomised controlled trial showed that the costs of training can be reduced if courses that combine e-learning and face to face training are used.[37]

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Questions for future research

Key research studies currently recruiting

- Continuous chest compressions (CCC; ClinicalTrials.gov NCT01372748)
- Randomised comparison of the effectiveness of the Laryngeal Mask Airway Supreme, i-gel, and current practice in the initial airway management of prehospital cardiac arrest: a feasibility study (REVIVE-Airways; ISRCTN18528625)
- Comparison of conventional adult out of hospital cardiopulmonary resuscitation against a concept with mechanical chest compressions and simultaneous defibrillation—LINC study (ClinicalTrials.gov NCT00609778)
- Prehospital randomised assessment of a mechanical compression device in cardiac arrest (PaRAMeDIC; ISRCTN08233942) www.warwick.ac.uk/go/paramedic
- Amiodarone, lidocaine, or neither for out of hospital cardiac arrest caused by ventricular fibrillation or tachycardia (ALPS; ClinicalTrial.gov NCT01401647)
- Therapeutic hypothermia to improve survival after cardiac arrest in paediatric patients—THAPCA-OH (out of hospital) trial (ClinicalTrials.gov NCT00878644)
- Therapeutic hypothermia to improve survival after cardiac arrest in paediatric patients—THAPCA-IH (in hospital) trial (ClinicalTrials.gov NCT00880087)
- Target temperature management after cardiac arrest (TTM; ClinicalTrials.gov NCT01020916)
- Prehospital resuscitation intranasal cooling effectiveness survival study (PRINCESS; ClinicalTrials.gov NCT01400373)

Other areas for future research

- What are the optimal methods for acquisition and retention of cardiopulmonary resuscitation skills?
- Does the use of adrenaline in cardiac arrest improve long term neurological outcome? The definitive answer will come only with a large placebo controlled randomised trial
- After the return of spontaneous circulation, does the use of controlled reoxygenation (targeting a specific arterial blood oxygen saturation) improve neurological outcome?

Tips for non-specialists

If someone collapses, is unconscious, unresponsive, and not breathing (or taking occasional gasps)*:

1. Call for help—ensure that an ambulance is coming, and if available an automated external defibrillator
2. Start chest compressions—push hard (5-6 cm in an adult) and fast (100-120 times per minute)
3. If you are trained, willing, and able to do so, give two ventilations after every 30 compressions
4. Do not stop unless the person shows signs of regaining consciousness—such as coughing, opening of the eyes, speaking, or moving purposefully—and starts to breathe normally
5. If an automated external defibrillator is available switch on and follow the audiovisual prompts

*Do not check for a pulse because this sign is unreliable for confirming cardiac arrest

Additional educational resources

Resources for healthcare professionals

- Resuscitation Council (UK) (www.resus.org.uk)—Information on courses, guidelines, and conferences
- National Cardiac Arrest Audit (https://ncaa.icnarc.org)—Information about the audit, including instructions on how to enrol a hospital in the scheme
- International Liaison Committee on Resuscitation (www.ilcor.org)—Provides access to the International Consensus on Cardiopulmonary Resuscitation (CPR) Science
- European Resuscitation Council (ERC) (www.erc.edu)—Information on CPR courses and training opportunities in Europe; includes access to the full ERC guidelines
- American Heart Association CPR and Emergency Cardiovascular Care (http://www.heart.org/HEARTORG/CPRAndECC/CPR_UCM_001118_SubHomePage.jsp)—Information for healthcare professionals and lay people on all aspects of CPR

Resources for patients

- British Heart Foundation (www.bhf.org.uk/heart-health/life-saving-skills/hands-only-cpr.aspx)—Information on a hands only CPR campaign
- Citizen CPR Foundation (www.citizencpr.org/)—Information for lay people interested in CPR
- American Heart Association (http://handsonlycpr.org)—Information on a hands only CPR campaign
- Resuscitation Council (UK) (www.resus.org.uk/pages/pub_AED.htm)—CPR training material
- Resuscitation Council (UK) (www.youtube.com/user/ResusCouncilUK)—Link to public information videos on CPR

vice chair of the Resuscitation Council (UK) (unpaid), chair of the advanced life support working group of the European Resuscitation Council (unpaid), co-chair of the advanced life support task force of the International Liaison Committee on Resuscitation (unpaid), and an editor of *Resuscitation* (honorarium received); GDP is chair of the Resuscitation Council (UK) advanced life support committee (unpaid), chair of the basic life support and automated external defibrillation working group of the European Resuscitation Council (unpaid), co-chair of the basic life support and automated external defibrillation task force of the International Liaison Committee on Resuscitation (unpaid), and an editor of *Resuscitation* (honorarium received); GDP holds research funding from the National Institute for Health Research to investigate mechanical chest compression and CPR feedback and prompt devices. All authors have been involved in local, national, and international resuscitation guideline development processes and in producing learning materials.

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Tables

Table 1| Immediate factors (present within 1 hour) related to 51 919 in hospital cardiac arrests*

Factor	No (%)
Acute respiratory insufficiency	18 948 (37)
Hypotension	20 410 (39)
Acute myocardial infarction or ischaemia	4989 (10)
Metabolic or electrolyte disturbance	5240 (10)
Acute pulmonary oedema	991 (2)
Acute pulmonary embolism	978 (2)

*Data from the United States National Registry of Cardiopulmonary Resuscitation.[4]

Table 2| Summary of resuscitation interventions by patient age group

	Age group			
Intervention	**Adults**	**Children (1 year to puberty)**	**Infants (<1 year)**	**Newborns**
Compression: ventilation ratio (compressions at 100-120/min)	30:2	15:2	15:2	3:1
Starting with	Compressions first	5 breaths first		
Chest compression landmark	Middle of lower half sternum	1 finger breadth above xiphisternum (lower third of chest)		Just below inter-nipple line (lower third of chest)
Compression depth	5-6 cm	At least 1/3 anterioposterior diameter of chest (4 cm infants, 5 cm children)		
Defibrillation energy	According to manufacturer or maximum setting	4 J/kg		
Adrenaline dose (intravenous or intraosseous)	1 mg	10 µg/kg		
Amiodarone dose (intravenous or intraosseous)	300 mg	5 mg/kg		

Adapted from Nolan and Soar.[w80]

Table 3| Post-cardiac arrest care

Problem	Intervention	Rationale
Post-cardiac arrest brain injury, which is the cause of death in two thirds of patients with out of hospital cardiac arrest and in a quarter of those with in hospital cardiac arrest admitted to the intensive care unit[w51]	Controlled reoxygenation aimed at 94-98% oxygen saturation of arterial blood	Animal studies show worse neurological outcome with hyperoxaemia during the first hour after return of spontaneous circulation.[w52] Two adult observational studies based on the same dataset and a paediatric observational study show worse outcomes associated with hyperoxaemia.[w53- w55] However, another observational study in adults failed to show an association between hyperoxaemia and death once severity of illness had been included in the analysis[w56]
	In patients who need mechanical ventilation, control arterial carbon dioxide—aim for a normal value	Hypocapnia is harmful to the injured brain because of cerebral vasoconstriction and decreased cerebral blood flow.[w57] The resultant cerebrospinal fluid alkalosis is neurotoxic because of increased release of excitatory amino acids
	Blood glucose control—aim for blood glucose concentration of 4-10 mmol/L*	Both hypoglycaemia and hyperglycaemia are associated with worse outcome.[w58-w60] Therapeutic hypothermia increases insulin requirements and blood glucose variability[w61]
	Control seizures—treat with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate; clonazepam may be the most effective treatment for myoclonus[w62]	Seizures occur in about a quarter of comatose patients after return of spontaneous circulation and increase mortality fourfold, but 17% of those developing seizures can still achieve a good neurological outcome.[w63] In a sedated mechanically ventilated patient who is receiving neuromuscular drugs, electroencephalographic monitoring is needed to recognise seizure activity
	Targeted temperature management (therapeutic hypothermia)—cool patients to 32-34 °C, maintain for 24 hours, and rewarm at 0.25 °C/h	Animal data indicate that hyperthermia worsens outcome.[w64] Two randomised controlled trials also showed better neurological outcome in comatose patients who had experienced out of hospital ventricular fibrillation cardiac arrest and were cooled to 32-34 °C for 12-24 hours.[w65 w66] Observational data indicate that mild hypothermia may also improve neurological outcome after non-ventricular fibrillation or pulseless ventricular tachycardia cardiac arrest[w67]
	Optimise sedation with short acting drugs (such as propofol, alfentanil, remifentanil)	Sedation helps facilitate cooling during treatment with therapeutic hypothermia. Short acting drugs enable earlier neurological assessment. Sedation reduces oxygen consumption and helps prevent shivering.[w68] Hypothermia reduces clearance of many drugs by at least a third.[w69] Ensure that drugs have been cleared before considering withdrawal in a comatose patient[w70]
	Control shivering with adequate sedation, bolus doses of a neuromuscular blocker, and magnesium[w62]	Shivering will increase oxygen consumption and reduce cooling rate[w71]
Post-cardiac arrest myocardial dysfunction; this problem is common and usually resolves after 72 hours[w72]	Fluid resuscitation	This can be guided by blood pressure, heart rate, urine output, and rate of plasma lactate clearance, central venous oxygen saturation or cardiac output monitoring[w62]
	Inotropes and vasopressors	
	Mechanical circulatory support	An intra-aortic balloon pump or extracorporeal support may be needed in those with severe cardiogenic shock[w73]
Ischaemia-reperfusion injury	Supportive treatments in the intensive care unit	Ischaemia-reperfusion may be associated with a marked systemic inflammatory response[w74]
Persistent precipitating pathology	Coronary reperfusion after myocardial infarction	Consider urgent coronary angiography and percutaneous coronary intervention in all those with a primary out of hospital cardiac arrest, especially, but not exclusively, if the 12 lead electrocardiograph shows ST elevation myocardial infarction[w75]
Prognostication	Identify comatose patients who have no chance of a neurological recovery	It is not in the patient's best interests to continue active treatment after resuscitation if the patient is comatose and there is no chance of a good neurological outcome. Use of therapeutic hypothermia (along with the additional sedation usually needed) makes existing guidelines[w76] on prognostication unreliable.[w77 w78] Outcome is most reliably predicted by clinical examination 3 days after return to normothermia combined, if possible, with electrophysiological testing[w79]

*1 mmol/L=18 mg/dL.

Figures

Fig 1 Adult basic life support algorithm; reproduced with permission from the Resuscitation Council (UK)

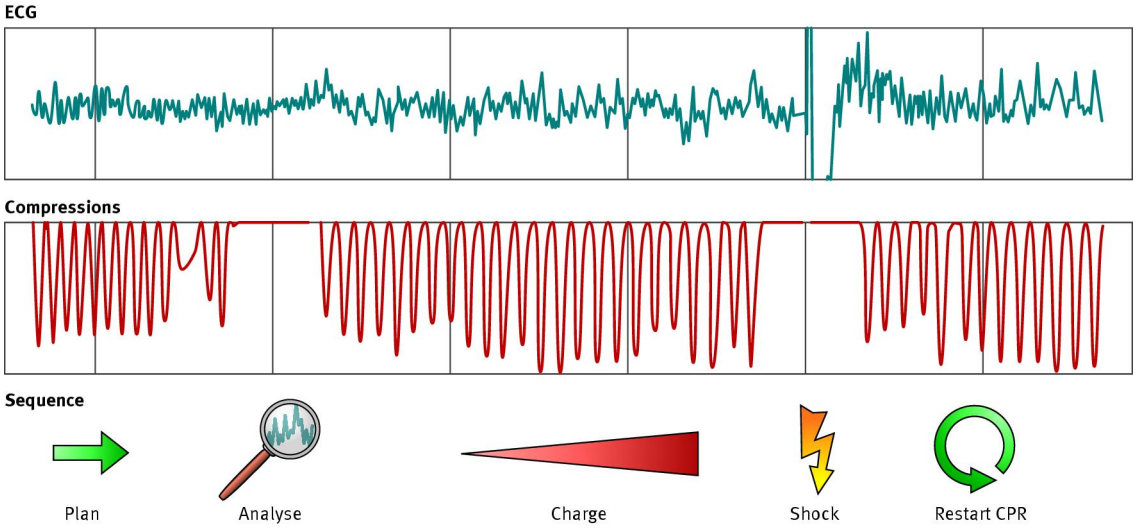

Fig 2 Defibrillation sequence: plan; pause chest compressions briefly; check rhythm, and confirm shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia). Restart chest compressions. Charge defibrillator while chest compressions are ongoing; once defibrillator is charged stop chest compressions; give shock—no one should touch the patient during shock delivery; resume chest compressions immediately after shock delivery. CPR=cardiopulmonary resuscitation; ECG=electrocardiograph

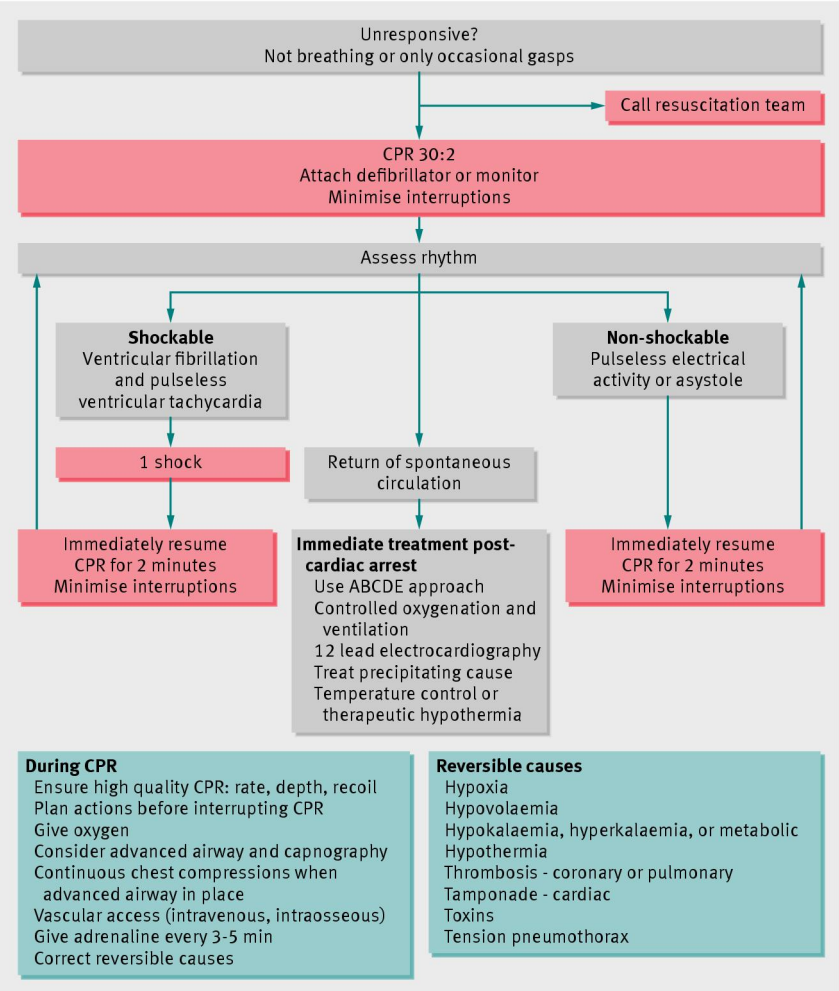

Fig 3 Advanced life support algorithm; reproduced with permission of the Resuscitation Council (UK). CPR=cardiopulmonary resuscitation