Cardiopulmonary resuscitation

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Abstract
Advanced cardiopulmonary resuscitation is a specific 'Capability in Practice' in the new Curriculum for Internal Medicine (Stage 1) in the UK, successful completion of which requires a doctor to understand issues regarding consent to resuscitation, possess relevant procedural skills and have demonstrated the ability to lead a team in providing effective resuscitation. The Resuscitation Council (UK) has published quality standards for practice and training, based on its 2015 guidelines. These guidelines align with similar international initiatives, taking account of new evidence from clinical trials of cardiac arrest management. In an effort to improve long-term survival after cardiac arrest in the community, national strategies confirm the importance of the 'chain of survival' concept that summarizes important components of successful resuscitation. They also promote the establishment of out-of-hospital cardiac arrest centres – hospitals that can provide optimal post-resuscitation interventions to patients showing a return of spontaneous circulation.

Keywords
Advanced life support; asystole; cardiac arrest; cardiopulmonary resuscitation; defibrillation; pulseless electrical activity; ventricular fibrillation

Key points
- Within hospital, the use of early warning scoring systems allows identification of those at risk of arrest and the instigation of preventive measures
- Basic life support begins with rapid recognition of cardiac arrest, a call for help and prompt provision of high-quality chest compressions with minimal interruption
- Evidence that drugs improve long-term survival is lacking, although they make return of spontaneous circulation (ROSC) more likely
- Monitoring end-tidal CO_2 allows assessment of the quality of cardiopulmonary resuscitation
- Patients achieving ROSC after out-of-hospital cardiac arrest should be admitted to a designated cardiac arrest centre where they can benefit from multidisciplinary interventions to mitigate the 'post-resuscitation syndrome'

Introduction
Outside hospital, most unexpected cardiac arrests occur in older people with heart diseases such as coronary artery disease, valve disease or cardiomyopathy. Rare cases of structural or primary arrhythmic disorders predominate in the young. Untreated, resultant pulseless ventricular tachycardia/fibrillation (pVT/VF) soon degenerates to asystole. Additionally, various potentially reversible conditions, including profound hypoxia, hypovolaemia, pulmonary embolism, cardiac tamponade and pneumothorax, can present with pulseless electrical activity (PEA) and asystole.

Ambulance personnel attempt resuscitation in 50–60 cases of out-of-hospital cardiac arrest per 100,000 population per year. Although return of spontaneous circulation (ROSC) occurs in a quarter of patients arriving at hospital, <10% survive to discharge. Survivals of 20–50% are reported in individual whose arrests are witnessed, receive early cardiopulmonary resuscitation (CPR) from bystanders, and are in VF when a defibrillator is applied. However, the initial rhythm is 'shockable' in only 20% of resuscitation attempts by ambulance personnel.

Within hospital, 60% of arrests occur in general wards, 20% in emergency departments or admission units, and 10% in cardiac care units. Few patients experience arrest after admission with uncomplicated acute myocardial infarction. The most common (70%) presenting rhythms are 'non-shockable' asystole/PEA. Outcomes are poor because cardiac arrest often happens in frail patients with severe co-morbidities. ROSC is detected in about 40% of patients; just under half of these survive to discharge. Risk-adjusted outcomes are substantially worse for arrests during the night (28% lower ROSC, 52% lower survival) and at weekends (12% lower ROSC, 28% lower survival) compared with arrests during weekday daytimes (Monday to Friday, 08:00 until 19:59 hours).[1]

'Primary' respiratory arrest can result from respiratory diseases, such as asthma or pneumonia, chronic lung disease, airway obstruction, neuromuscular disease or use of respiratory suppressant drugs. Prognosis is favourable because of the reversible nature of many of these causes.

Principles of treatment

The response to cardiorespiratory arrest has been likened to a 'chain of survival' – effective performance of each component is necessary for the overall 'strength of the chain'. The principles of resuscitation comprise:

- appropriate use of 'Do Not Attempt Resuscitation' (DNAR) orders
- risk assessment and prevention
- rapid recognition of arrest, with alert of suitably trained personnel
- assessment of the circumstances of the collapse
- maintenance of coronary perfusion to allow subsequent reversal of non-perfusing cardiac rhythms
- maintenance of cerebral perfusion to avoid permanent neurological damage
- adequate oxygenation
- restoration of a perfusing cardiac rhythm with defibrillation and drugs
- careful post-resuscitation care to mitigate post-arrest cerebral and cardiac dysfunction
- timely cessation of resuscitative attempts.

All clinicians should possess basic life support (BLS) skills. Those in core and specialty training for general internal medicine should demonstrate appropriate knowledge and competence through passing professional postgraduate examinations, holding a valid advanced life support (ALS) certificate, and participating in various workplace-based assessments, including reflecting on practice.

Guidelines and standards for cardiopulmonary resuscitation

The International Liaison Committee on Resuscitation (www.ilcor.org/home), an non-governmental transnational scientific collaboration, performs 5-yearly cyclical rigorous evaluations, and commissions systematic reviews, aiming to achieve near-continuous updating of resuscitation science with which to inform practice guidelines.

The Resuscitation Council (UK) has produced Quality Standards for the management cardiorespiratory arrest, whether in or outside hospital. These include instructions regarding training clinical staff, organizing 'resuscitation teams' within hospitals, use of policies to guide ethical decisions about resuscitation, and promoting audit and research. One such standard is participation in the National Cardiac Arrest Audit of in-hospital CPR. As of March 2018, 80% of acute hospitals in England were participating in this audit.

Recent iterations of guidelines contain important changes based upon these observations:[2]

- Soon after onset of VF, when oxygenation is usually adequate, chest compressions are more important than ventilation.
- Interrupting chest compressions (e.g. pulse-checking, ventilating) is associated with poorer outcomes than continuous compressions; quality of compressions matters.
- Initial defibrillation terminates VF in 90% of cases, but immediate ROSC is rare even when organized cardiac rhythms ensue.

- Cardiac ultrasound during resuscitation provides insights into causes of arrest and aids decision-making with respect to stopping resuscitation.
- End-tidal carbon dioxide ($EtCO_2$), measured by waveform capnography, is a guide to the quality of CPR; significant increases occur rapidly on ROSC.
- After ROSC, survival may be improved by a package of interventions, including coronary angiography and percutaneous coronary intervention.

Basic life support

The emphasis is on rapid recognition of cardiac arrest, a call for help and prompt provision of high-quality chest compressions with minimal interruption.

Recommended adult BLS is shown in Figure 1. Infrequent gasping breaths commonly occur early after cardiac arrest. If in doubt, rescuers should act as if breathing is *not* normal, call the resuscitation team (or ambulance service if outside hospital) and *start chest compressions*. Two initial 'rescue breaths' are *no longer recommended* to lay-rescuers as this delays chest compressions and deters bystanders from beginning resuscitation. Trained health professionals may check for a carotid pulse during a 'breathing check' of <10 seconds, using techniques to open the airway, such as a 'jaw thrust', chin lift and head tilt if there is evidence of airway obstruction, or while providing two rescue breaths after every 30 compressions. When multiple rescuers are available, an airway adjunct such as an oropharyngeal airway can be inserted before initial bag-and-mask ventilation.

pVT is occasionally reversed by *precordial thump* – the delivery of a blow to the lower half of the sternum with the ulnar edge of a clenched fist from a distance of about 20 cm. This is rarely successful and is only justified when a defibrillator is not yet available and a cardiac monitor shows pVT/VF.

Chest compression comprises rhythmic downward pressure with the heel of the hand applied over the middle of the lower half of the sternum at 100–120 per minute to a depth 5–6 cm, with release of all pressure between compressions. A rate *above* 120 per minute leads to poor-quality CPR. Delivering compressions is tiring. If possible, the rescuer performing compressions should be changed every 2 minutes. Various mechanical devices have been developed to deliver more effective and reliable chest compressions, particularly in situations where manual CPR is difficult (e.g. the cardiac catheter laboratory). However, they have not consistently been shown to be superior to manual CPR, requiring interruption of compressions as they are applied. Manual CPR remains the standard of care.

The ratio of compressions to breaths within one 'cycle of CPR' is 30:2 unless the airway has been secured with, for example, an (endo)tracheal tube or supraglottic device. Tracheal intubation often causes prolonged interruption of chest compressions and should only be attempted by skilled individuals. Ventilation should include an 'inspiratory' phase lasting 1 second with enough volume (about 500–600 ml) to make the chest rise. Wherever possible, supplemental oxygen should be used. Excessive rate and depth of ventilation increases intrathoracic pressure, decreases venous return and reduces coronary and cerebral perfusion.

Compression-only cardiopulmonary resuscitation

Compression-only CPR is easier to describe and teach; lay-responders are more likely to attempt it than standard CPR. Resuscitation outside hospital with bystander-administered chest compressions alone, prompted by telephone instruction, leads to better outcomes than conventional chest compressions interrupted by rescue breathing. However, there was no benefit shown when an ambulance crew provided continuous compressions and asynchronous positive-pressure ventilations via bag-and-mask ventilation compared with standard 30:2 CPR. Existing guidelines continue to recommend both compressions *and* rescue breathing if the attending rescuer is trained to provide both.[3]

Advanced life support

The algorithm for ALS is shown in Figure 2. A compression-to-ventilation ratio of 30:2 during each cycle of CPR should continue until the airway is secured. Thereafter, chest compressions (100–120 per minute) should continue uninterrupted and need not be synchronized with (10 per minute) ventilations.

Adhesive defibrillator electrodes are placed without stopping compressions – the 'right' electrode to the right of the sternum just below the clavicle, the 'left' electrode as far into the left axilla as possible (not over breast tissue). Defibrillator electrodes should not be placed directly over implantable cardiac devices (such as pacemakers). In such cases, an alternative position should be chosen (e.g. antero-posterior), and device function checked soon after successful resuscitation. Chest compressions are interrupted only to allow a brief assessment of cardiac rhythm (either as 'shockable' – pVT/VF – or 'non-shockable'). When a shock is indicated, compressions are resumed as the defibrillator charges and then stopped just before the first shock (at least 150 J), aiming for a 'pre-shock pause' of as few as 5 seconds. Oxygen delivery systems are removed to allow safe defibrillation. CPR restarts immediately, without a rhythm check and, unless signs of life become apparent, should continue for 2 minutes before further reassessment of cardiac rhythm and (if organized electrical activity appears) a check for a palpable pulse. After 2 minutes of CPR, if a shockable rhythm persists, a second shock (150–360 J) should be delivered and CPR resumed. A 'Stand clear' instruction should be given, and its implementation assured, before each shock. This 'single-shock sequence' significantly reduces time without chest compressions during resuscitation.

Repeated shocks without interposed CPR are justified in specific circumstances (e.g. during a coronary intervention or when a conscious patient develops witnessed VF and a defibrillator is immediately available). Up to three shocks can be delivered in rapid succession, checking for ROSC after each. If this three-shock strategy is unsuccessful, those three shocks are considered equivalent to the first shock of the ALS algorithm.

Defibrillation can be delayed when the amplitude of VF is low enough to make distinction from asystole difficult and, outside hospital, when the response interval from call to arrival of the professional rescuers is >4–5 minutes. In both circumstances, a period of BLS before defibrillation may be useful. However, defibrillation should generally occur as rapidly as possible.

During ALS, a variety of potentially reversible causes of arrest should be considered. These have been grouped into 'the 4 Hs' – hypoxia, hypovolaemia, hyperkalaemia (and other metabolic disturbances) and hypothermia – and 'the 4 Ts' – tension pneumothorax, cardiac tamponade, ingestion of toxic substances and thromboembolism (including pulmonary embolism and coronary artery thrombosis).

Drugs during resuscitation

Although vasopressors and antiarrhythmic drugs given during resuscitation are associated with increased ROSC, long-term survival is *not* improved. Such drugs, delivered into a proximal vein, continue to be used. Their inclusion will be reassessed upon imminent publication of large randomized trials.[2,3]

Adrenaline/epinephrine 1 mg intravenously (or intraosseously) should be given when compressions restart after the third shock in cases of resistant VF, and every 3–5 minutes thereafter. Adrenaline is also recommended as soon as possible in cases of asystole/PEA, and every 3–5 minutes thereafter unless ROSC is achieved.

Amiodarone 300 mg bolus as an intravenous injection should be given after the third shock in cases of resistant VF, and further doses (e.g. 150 mg after the fifth shock), including continuous infusion, may be of value.

Lidocaine (1.0–1.5 mg/kg or 100 mg by bolus injection) can be used *instead* of (but not in addition to) amiodarone.

Magnesium sulfate (1–2 g intravenously in 10 ml of glucose 5% over 5 minutes) can be given in cases of polymorphic VT (torsade de pointes) or in hypokalaemia-induced arrhythmias. The routine use of intravenous calcium chloride and sodium bicarbonate is not recommended, but can be considered in life-threatening hyperkalaemia.

Atropine is *no longer recommended* in cases of asystole or PEA, but can be used for reversal of severe bradycardia, when isoprenaline, adrenaline and aminophylline can also be helpful. External pacing is not

of use in asystole, but can be f value in ventricular standstill, when isolated P waves are seen during cardiac monitoring.

Ventilation and monitoring during resuscitation

To prevent aspiration and provide optimal ventilation, the airway is best secured by placement of a tracheal tube. Substantial skill and confidence is needed to reliably and rapidly insert the tube, without causing prolonged interruptions in chest compressions or incorrectly intubating the oesophagus. Alternative supraglottic airway devices are likely to be safer and more effective when used by non-experts, but their use has not been associated with better outcomes.

Monitoring of exhaled CO_2 is now recommended during resuscitation. 'Sidestream' devices can be used in both intubated and non-intubated patients to display $EtCO_2$ values and waveforms. Complete absence of expired CO_2 suggests that the airway is entirely obstructed or that a tracheal tube is misplaced (or that there might be massive pulmonary embolism). Normal $EtCO_2$ (4.0–5.7 KPa) is attenuated during CPR, and acts as a guide to the quality of CPR. Good-quality CPR can result in $EtCO_2$ values of 2.0–2.5 kPa, and ROSC (which may not be apparent on cardiac monitoring during chest compressions) is associated with sudden increases to normal values.[2] Monitoring can also inform decisions to stop resuscitation; $EtCO_2$ <1.3 kPa after 20 minutes of resuscitation is predictive of poor outcome.

Transthoracic echocardiography appears in the ALS algorithm. Brief ultrasound scanning can identify some of the potentially reversible causes of cardiac arrest (e.g. cardiac tamponade), while complete lack of cardiac movement after prolonged resuscitation carries a poor prognosis. In thin patients, sub-xiphoid probe placement may provide satisfactory images without interrupting compressions; otherwise orthodox left parasternal positions are required, with consequent cessation of compression. Apical views can be possible if defibrillator pads have not been placed over the apex.

Post-resuscitation care – 'cardiac arrest centres'

Immediately after ROSC, intravenous access should be confirmed, and ventilation should aim to achieve normal oxygen and CO_2 levels. Blood pressure should be supported if necessary (with fluid replacement and inotropic drugs) to a systolic pressure of >100 mmHg (or a mean pressure of 65–80 mmHg). Intra-arterial pressure monitoring should be considered. A 12-lead electrocardiograph (ECG) should be performed.

When the duration of circulatory arrest is brief, recovery of consciousness can be rapid with no obvious adverse consequences. In other cases, however, a 'post-resuscitation syndrome' ensues; this is characterized by clinical, laboratory, electrophysiological and radiological evidence of brain injury, global myocardial dysfunction, and a systemic response with effects similar to sepsis, coupled with the continuing effects of the underlying precipitating pathology.

A detailed discussion of the management of this complex combination of issues is beyond the scope of this article.[4] Briefly, neuroprotection is promoted by sedation and ventilation to maintain oxygen saturation at 94–98%, treatment of seizures, arrhythmias and hypotension (sufficient for adequate urine production and tissue perfusion), and careful correction of hyperglycaemia (treating when blood glucose >10 mmol/litre). Targeted temperature management coupled with prevention of fever (using antipyretics) is recommended for at least 24 hours in resuscitated patients who remain unresponsive after ROSC. There appears to be no benefit in striving for therapeutic hypothermia to 32–34°C compared with maintaining temperature at 36°C.

ST segment elevation on ECG soon after ROSC is highly predictive of acute coronary artery occlusion, and thus the potential that urgent reperfusion might improve cardiac function. However, the absence of ST elevation does not reliably exclude coronary occlusion. Observational studies support urgent coronary angiography and percutaneous coronary intervention in individuals with ST segment elevation, *even* if they remain comatose and ventilated. The benefit of early angiography in cardiac arrest survivors who do not have ST elevation is contestable, and requires careful consideration rather than directly transfer to the catheter laboratory.

National policies increasingly promote systems of care whereby all individuals who achieve ROSC after out-of-hospital cardiac arrest are admitted to a designated cardiac arrest centre.[5] These hospitals possess facilities and expertise for all aspects of post-resuscitation care, including round-the-clock access to angiography, advanced circulatory support devices, intensive care services capable of targeted temperature management, and ready access to radiological and neurological services. It remains unclear whether such cardiac arrest centres will offer a receiving service for survivors of cardiac arrests occurring in neighbouring hospitals.

Leading the arrest team

CPR depends upon teamworking. Lack of adequate leadership, characterized by a failure to stand back calmly and direct others to perform tasks, and to transfer information and resolve conflict, is associated with poorer performance. In simulated situations, previous training in leadership is associated with better leadership behaviour and the provision of higher quality CPR by the whole team. The leader should liaise with senior anaesthetic and nursing staff and, after consultation, be responsible for stopping resuscitation, ensuring adequate documentation, debriefing the resuscitation team if time permits, and sensitively 'breaking bad news' to relatives of the deceased.

Prevention of cardiac arrest

An assessment should be made of/with each hospital patient with respect to the appropriateness of CPR in case of cardiac arrest, and the conclusion clearly recorded. The National Early Warning Score (NEWS), based on routine clinical observations, may detect changes that predict imminent collapse; it can also allow activation of medical emergency teams that may prevent subsequent arrest or decide that CPR would be inappropriate. The association between better hospital survival rates and a lower incidence of reported cardiac arrest has been used to suggest that hospitals with better outcomes are also those able to prevent cardiac arrest in severely ill inpatients, and those that are more active in determining resuscitation status.

When to stop

Without a DNAR directive, the decision to start CPR is usually straightforward. Stopping resuscitation is more complex. 'Termination of resuscitation' outside hospital is reasonable when, despite provision of ALS, all the following criteria are met: no ROSC; no shock delivered; cardiac arrest unwitnessed; and no bystander-initiated CPR. Within hospital, there are no hard and fast rules for cessation. Decision-making should take into account the patient's functional state before the arrest, co-morbidities, likely cause of the arrest, and responses to interventions during resuscitation. As mentioned above, low $EtCO_2$ levels during CPR and the absence of cardiac movement on echocardiography can inform decisions to stop.

Legends to figures

Figure 1 BLS algorithm. AED, Reproduced with the kind permission of the Resuscitation Council (UK).

Figure 2 ALS algorithm. ABCDE, xxxxx. Reproduced with the kind permission of the Resuscitation Council (UK).

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 64-year-old inpatient abruptly became breathlessness.
On clinical examination, the woman was anxious and confused with cold hands and prolonged capillary refill time. Heart rate was 98 beats/minute, irregular and of low volume. Blood pressure was unrecordable. Respiratory rate was 26/minute. Jugular venous pressure was elevated, heart sounds difficult to distinguish, and breath sounds were normal. Oxygen saturations were 92% on 35% oxygen.

Investigation

•ECG showed atrial fibrillation with a ventricular rate of 130/minute, right bundle branch block with T wave inversion in V1–V3 but no ST elevation

Her pulse then became impalpable without a change in cardiac rhythm, and she became unconscious.

What is the most likely cause of the cardiorespiratory arrest?

A. Acute myocardial infarction
B. Brugada syndrome
C. Cardiac tamponade
D. Hypovolaemia
E. Pulmonary embolism

Correct answer: E. This patient had pulseless electrical activity (PEA) after abrupt-onset hypotension and hypoxia. Of the possible options listed here, pulmonary embolism would best fit the clinical picture. Hospital patients are likely to experience venous thromboembolism as a result of reduced mobility, surgical treatments and/or co-morbidities. Hypovolaemia (D) (e.g. from significant gastrointestinal bleeding) is not usually associated with hypoxaemia. Cardiac tamponade (C) presents with hypotension and elevated venous pressure, but is unlikely to occur so abruptly in the absence of obvious trauma (e.g. stabbing), aortic dissection or myocardial rupture – the latter two being rare. Echocardiography performed during resuscitation would be of value in this case. Although acute myocardial infarction (A) might present with PEA if a sufficiently extensive amount of myocardium was affected, it is unlikely to be the cause here because of the absence of chest discomfort, absence of significant ST segment elevation and absence of clinical evidence of pulmonary oedema (no crackles on auscultation) despite severe hypoxia. Brugada syndrome (B), which can be associated with a right bundle branch block appearance on ECG, usually presents with abrupt onset of ventricular tachycardia.

Question 2

A 56-year-old man developed acute severe central chest pain and was attended by ambulance paramedics.
On clinical examination, his heart rate was 96 beats/minute, and blood pressure 148/88 mmHg. A 12-lead ECG showed ST segment elevation and tall T waves in V2–V4.

A diagnosis of acute ST elevation myocardial infarction was made and it was decided to transport the patient to the nearest interventional cardiac centre. A cannula was placed in a peripheral vein before transport, and the patient's heart was monitored en route via defibrillator electrodes.

When the ambulance was 5 miles away from the hospital, the patient developed ventricular fibrillation, losing consciousness. The driver stopped the vehicle at the request of the paramedic.

What is the best course of action?

A Commence chest compressions at a rate of 100/minute with asynchronous ventilation (10/minute)
B Defibrillate immediately
C Give two rescue breaths and then commence chest compressions
D Perform a precordial thump
E Turn on the sirens and blue lights and proceed to the hospital as quickly as possible

Correct answer: B. This is a patient who has had a cardiac arrest witnessed by a highly trained healthcare professional and already has a defibrillator attached. Ventricular fibrillation occurring in the early stages of myocardial infarction is readily reversible by defibrillation. The priority is to defibrillate as soon as possible (up to three shocks without interposed chest compressions in such circumstances) (not E). Although in practice a precordial thump (D) and a few chest compressions (A, C) could be performed as the defibrillator is charging, this is not advised as it might distract from immediate defibrillation.

Question 3

A 78-year-old man collapsed on a medical ward. The resuscitation team found ward nurses delivering chest compressions and intermittent rescue breathing using a bag-and-mask with oxygen. They applied defibrillator electrodes and found asystole. Cardiopulmonary resuscitation (CPR) was immediately restarted, and within 2 minutes venous access was established and a tracheal tube inserted. End-tidal carbon dioxide ($EtCO_2$) waveform capnography suggested good-quality CPR. Adrenaline (epinephrine) 1 mg was given intravenously. Within 1 minute of giving adrenaline, $EtCO_2$ abruptly rose to normal values.

What should the resuscitation team leader now instruct the team to do?

A Continue CPR until the end of the 2-minute cycle and then check for a pulse
B Give a second dose of adrenaline intravenously
C Reduce the supplemental oxygen concentration to avoid carbon dioxide retention
D Stop ventilating but carry on with chest compressions
E Stop chest compressions and check for a pulse

Correct answer: A. End-tidal carbon dioxide ($EtCO_2$) is the partial pressure of CO_2 at the end of exhalation and reflects cardiac output and pulmonary blood flow. $EtCO_2$ is attenuated during cardiopulmonary resuscitation (CPR), and a rapid rise in $EtCO_2$ during CPR may indicate return of spontaneous circulation (ROSC). In these circumstances, it is best *not* to stop compressions (D, E) and ventilation, but to continue until the end of the planned cycle of CPR. If ROSC is then confirmed, further doses of adrenaline (epinephrine; B) will be at best unnecessary and at worst harmful. $EtCO_2$ in this setting should not be used as a guide to the need for oxygen supplementation (C) or to the development of spontaneous respiration.