

Cardiorespiratory arrest

Clive Weston

Abstract

During core medical training the trainee should acquire sufficient knowledge and skills, and demonstrate appropriate attitudes and behaviours to allow the competent assessment and resuscitation of patients who have suffered cardiorespiratory arrest. These attributes have been defined by the UK Resuscitation Council, whose most recent guidelines are based upon an extensive systematic review of evidence by teams of international experts.

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

Abrupt cardio-respiratory arrest is rapidly fatal without resuscitation. Outside hospital, the underlying cause is often transient cardiac ischaemia resulting in ventricular fibrillation (VF) in a heart that is deemed 'too good to die'. Untreated VF degenerates to asystole, but can be successfully managed with life supportive manoeuvres involving chest compressions, rescue breathing and defibrillation. Other causes of cardiac standstill (asystole and pulseless electrical activity (PEA) – formerly termed electromechanical dissociation) are more resistant to treatment, and recovery is unlikely unless an underlying cause such as profound hypoxia, hypovolaemia, cardiac tamponade or tension pneumothorax can be recognized and reversed. Techniques for cardiopulmonary resuscitation (CPR) are also useful in the management of arrest due to asphyxia, for example drowning. Long-term survival following pre-hospital cardio-respiratory arrest is unusual (< 10% of cases), but 30–40% in the subgroup whose arrests are witnessed, who then receive bystander CPR and are in VF when a defibrillator is available at the scene.

In hospital, sudden collapses are neither entirely unpredictable nor unpreventable, and while the victim has the advantage of rapid response from a 'cardiac arrest team', the outcome is poor because the arrest is frequently a terminal event due to severe co-morbidities. Save for the first few hours after hospitalization, the commonest rhythm confronting the hospital cardiac arrest team as they start resuscitation will be 'non-shockable' asystole/PEA. 'Pure' respiratory arrest – cessation of breathing

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What's new?

- New guidelines for the management of cardiorespiratory arrest were published in 2005.
- The importance of predicting and preventing cardiopulmonary arrest within hospital is leading to the development of critical care outreach teams in some hospitals.

with persisting effective cardiac activity – may be seen in acute respiratory diseases such as asthma or pneumonia, chronic lung disease, airways obstruction, neuromuscular disease and use of respiratory suppressant drugs such as morphine. Prognosis is reasonable because of the reversible nature of many of these causes. For cardiac arrests developing within hospital the rate of survival to discharge is about 20% overall – three-quarters with good neurological function – and 36% for those found in VF.¹ These disappointing figures emphasize the importance of prevention wherever possible.

Principles of treatment

The principles of resuscitation comprise:

- appropriate use of 'Do not resuscitate' orders
- rapid recognition that cardiorespiratory arrest has occurred
- a call for help to suitably trained personnel
- avoidance of danger to rescuers and victim
- assessment of the circumstances of the collapse
- maintenance of sufficient coronary perfusion (during the relaxation phase of chest compressions) to allow subsequent reversal of non-perfusing cardiac rhythms
- maintenance of sufficient cerebral perfusion (during the compression phase of chest compressions) to avoid permanent neurological damage following restoration of spontaneous circulation (ROSC)
- maintenance of oxygenation through positive-pressure ventilation
- correcting the immediate underlying problem with appropriate cardiac monitoring, defibrillation and drug use
- timely cessation of resuscitative attempts.

Seldom are single rescuers capable of all these steps, nor is all necessary equipment readily available. The response to cardiorespiratory arrest is characterized by the 'chain of survival', with early recognition and rapid delivery of basic life support (BLS), defibrillation and advanced management. All clinicians should possess BLS skills and all those completing specialist training in general internal medicine should participate in UK Resuscitation Council approved advanced life support courses, with re-certification every 3 years.

Guidelines for CPR

The International Liaison Committee on Resuscitation coordinated 281 individuals in an exhaustive evaluation of the evidence underpinning the management of cardiorespiratory arrest. The resulting consensus document has been incorporated into various national guidelines.^{2,3} A similar exercise is planned for 2010. Whilst the

basic principles governing CPR remain constant, these revisions contain important changes that have been implemented in practice.⁴ Changes were based upon the following observations:

- early after onset of VF, chest compressions appear more important than ventilation
- the combination of inadequate or interrupted chest compressions with excessive ventilation reduces coronary and cerebral perfusion during CPR
- interruptions in chest compressions were frequent and prolonged using previous guidelines
- when VF is prolonged, a period of BLS before defibrillation may increase survival
- initial biphasic defibrillation terminates VF in approximately 90% of cases but, even when organized cardiac rhythms ensue, immediate ROSC is rare.

Basic life support

Recommended adult BLS is shown in [Figure 1](#). Infrequent gasping ‘agonal’ breaths commonly occur early after collapse with cardiac arrest. If in doubt, rescuers should act as if breathing is not normal and start chest compressions. Health professionals should also check for a carotid pulse during the ‘breathing check’. Two initial rescue breaths are recommended only when there is obvious asphyxia, otherwise they delay the delivery of chest compressions and may deter a bystander from attempting resuscitation. Recent trials show no improvement in outcomes when combined compressions and ventilation are compared with chest compressions alone.⁵

The heel of the hand should be placed over the middle of the lower half of the sternum. The preferred compression rate is 100/min – faster rates are probably more effective (though more tiring) – with compression depth 4–5 cm and release of all pressure between compressions. The ratio of compressions to rescue breaths within one ‘cycle of CPR’ is 30:2 unless the airway has been secured with, for example, an endotracheal tube. Ventilation should include an ‘inspiratory’ phase lasting 1 second with enough volume (often only 500–600 ml) to make the chest

rise. Wherever possible, supplemental oxygen should be used. Excessive ventilation increases intrathoracic pressure, decreases venous return and reduces coronary and cerebral perfusion. At the recommended rate and ratio of compression and ventilation, 2 minutes of CPR will contain approximately five cycles. This rate of chest compression requires substantial effort⁶ and unless the rescuer is alone, someone else should take over compressions after 2 minutes or five cycles, if not before. A variety of mechanical devices have been developed to provide consistent and effective compressions, but their widespread use awaits evidence of improved outcomes from randomized trials.

Advanced life support

The algorithm for advanced life support (ALS) is shown in [Figure 2](#). If the collapse is witnessed and VF confirmed, a precordial thump is acceptable if a defibrillator is unavailable. Otherwise, CPR should start and, until the airway is secured, a compression to ventilation ratio of 30:2 should be used per cycle of CPR. Thereafter chest compressions (100/min) should continue uninterrupted while ventilation (10/min) should be sufficient to observe the chest rising. Ventilations and compressions may then be non-synchronized.

Chest compressions are stopped only to allow assessment of cardiac rhythm (as ‘shockable’ – VF or pulseless ventricular tachycardia – or ‘non-shockable’) and delivery of a shock. Increasingly, semi-automatic advisory defibrillators (AEDs) are used. Adhesive electrodes are placed without stopping CPR; ‘right’ electrode to the right of the sternum just below the clavicle, ‘left’ electrode as far into the left axilla as possible, not over breast tissue. It does not matter if the electrodes are reversed. An analysis of cardiac rhythm should be performed immediately and a single shock delivered if advised.

CPR should resume immediately after delivery, without a rhythm check and, unless signs of life become apparent, should continue for 2 min before reassessment of cardiac rhythm and, if organized electrical activity appears, a check for a palpable pulse. Repeated shocks without interposed cycles of CPR are not advised, so, following 2 min of CPR, if a shockable rhythm persists a second shock should be delivered (150–360 J biphasic) and CPR resumed again. This ‘single-shock sequence’ is a significant departure from previous guidelines with their emphasis on repeated shocks before restarting CPR. It significantly reduces time without chest compressions during resuscitation.⁷

Neither vasopressors nor antiarrhythmics have been proven to have long-term benefit during resuscitation, yet such drugs, delivered into a proximal vein, are included in the algorithm. Epinephrine (adrenaline) 1 mg is recommended just before the third shock in cases of resistant VF and every 3–5 min thereafter (i.e. before alternate shocks). Adrenaline is also recommended as soon as possible in cases of asystole or PEA and every 3–5 min thereafter (i.e. after every 2-minute cycle of CPR). Amiodarone 300 mg bolus injection should be given just before the fourth shock in cases of resistant VF, and further doses, including continuous infusion, may be of value. Lidocaine (1 mg/kg) may be used instead. Intravenous magnesium sulphate (1–2 g in 10 ml of 5% dextrose over 5 minutes) may also be of value, particularly in cases of polymorphic VT – ‘torsades de pointes’. In cases of asystole, or where the PEA is less than 60/min, a single injection of atropine 3 mg should be given.

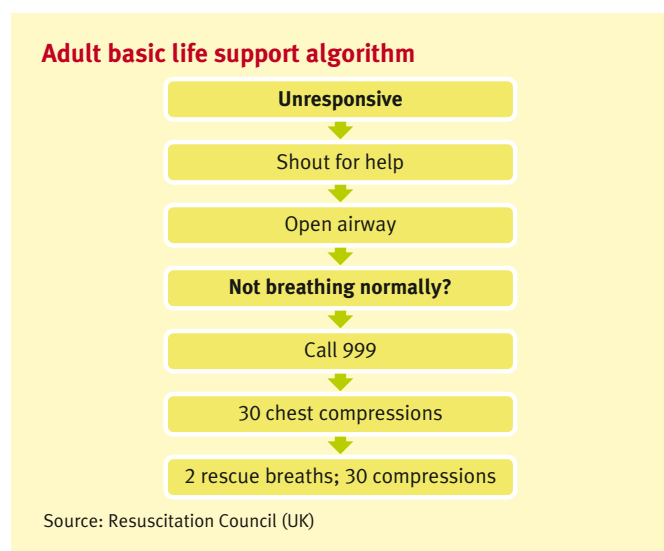


Figure 1

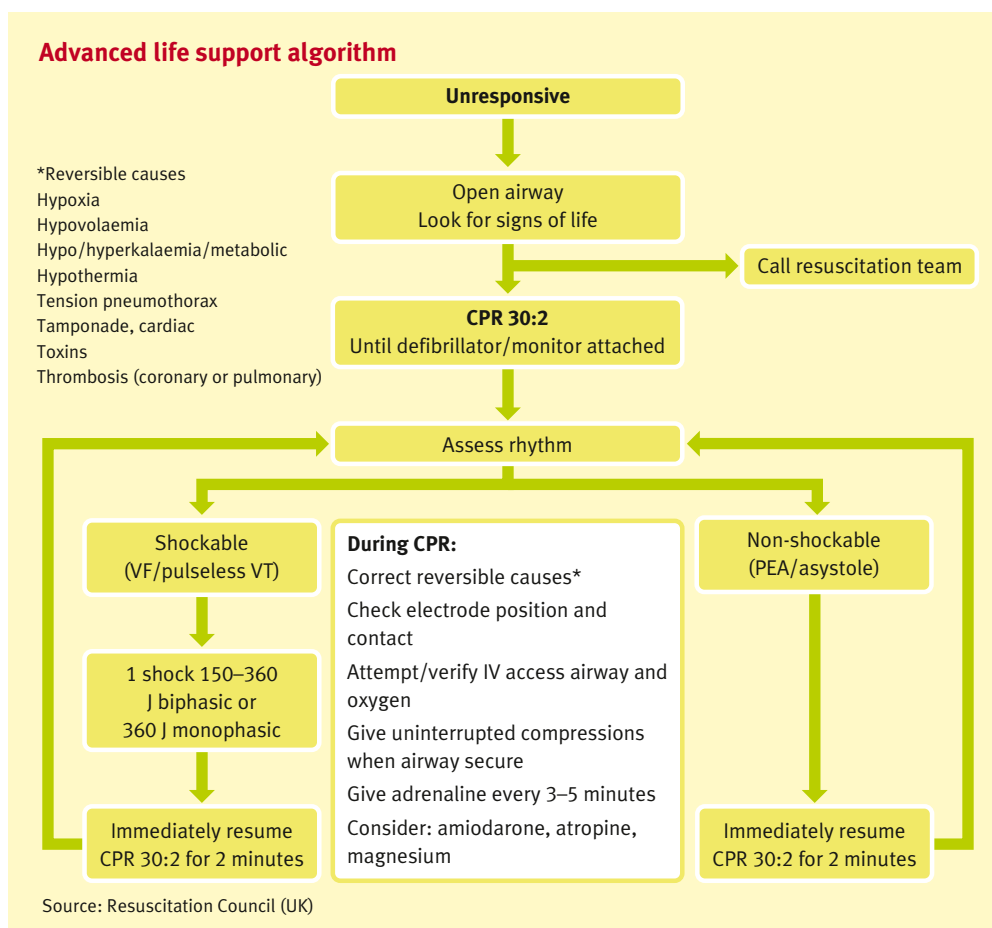


Figure 2

Defibrillation can be delayed if the amplitude of the VF is so low as to make distinction from asystole difficult and, outside hospital, when the response interval from call to arrival of the professional rescuers is greater than 4–5 minutes. In both circumstances, a period of BLS before defibrillation may be useful.⁸

During ALS a variety of potentially reversible causes of cardiopulmonary arrest should be considered. These include hypoxia, hypovolaemia, hyperkalaemia and other metabolic disturbances, hypothermia, tension pneumothorax, cardiac tamponade, toxic substances and thromboembolism.

Palpation for arterial pulses and estimation of blood gases are unreliable indicators of the efficacy of CPR. End-tidal carbon dioxide monitoring is a more effective non-invasive indicator of ‘cardiac output’ generated during CPR, but is not widely performed.

Leading the arrest team

CPR depends upon team-working. The leader of the team, frequently the senior attending physician, has responsibility for delivering resuscitation based upon the national guidelines. Lack of adequate leadership behaviour, characterized by a failure to calmly stand back and direct others to perform tasks (within their competence), a failure of information transfer and resolution of conflict, is associated with poorer performance.^{9,10} The leader also needs to liaise with senior anaesthetic and nursing staff and, having consulted with others, has the responsibility to

halt resuscitative efforts and ensure both adequate documentation of the various interventions and the sensitive ‘breaking of bad news’ to relatives of the deceased.

Prevention of cardiac arrest

Many unexpected in-hospital cardio-respiratory arrests are avoidable, particularly when patients are nursed in ‘inappropriate’ areas,¹¹ often preceded by slow progressive deterioration in physiological functioning. The immediate precursors of cardiac arrest in hospital are usually hypotension, acute respiratory insufficiency, metabolic or electrolyte disturbance, or cardiac ischaemia.¹ Early detection, using Patient At Risk (PAR) or Early Warning Scoring (EWS) systems, may allow activation of critical care outreach teams or medical emergency teams (MET) who may prevent subsequent arrest, or who may decide that CPR would be inappropriate. Using historical controls, such teams appear effective, but a randomized trial failed to show the expected reduction in unexpected death.¹² Regardless of this, all staff should be trained to recognize the ‘sick’ patient.

Predicting survival

Less than 20% of adults having an in-hospital cardiac arrest will survive to leave hospital; rates of survival for pre-hospital

arrest are substantially lower. A variety of factors have been associated with survival. The most important are the primary arrhythmia at onset, the rhythm present when CPR starts, provision of prompt, high-quality BLS, and early defibrillation when appropriate. Patient co-morbidities are also important. Even when adjusted for known predictors of outcome, cardiac arrests occurring at night and weekends carry a poorer prognosis, reflecting longer delays to treatment.^{13,14} Notwithstanding the need for efficient cardiac arrest management, the prevention or detection and reversal of the precursors of arrest (above) remains a high priority.

When resuscitation is successful and circulation is restored, the following predict death or a poor neurological outcome¹⁵:

- absent corneal reflexes at 24 hours
- absent pupillary responses at 24 hours
- absent withdrawal response to pain at 24 hours
- no motor response at 24 hours (and still absent at 72 hours).

When to stop

In the absence of a 'Do not attempt resuscitation' directive, the decision to start CPR is usually straightforward. Stopping resuscitation is more difficult. 'Termination of resuscitation' (TOR) outside

hospital should be considered when there has been no ROSC prior to transport, no shock was delivered, no bystander-initiated CPR, and the arrest was unwitnessed.¹⁶ When such patients are brought to hospital further resuscitative efforts are nearly always unsuccessful. Within hospital, there are no hard and fast rules for cessation. A survival prediction nomogram (Table 1) based upon a Resuscitation Predictor Scoring (RPS) scale has been developed and it is acceptable to consider stopping as early as 15 minutes after commencement in the absence of ROSC.¹⁷ Because of rare cases of ROSC after cessation of resuscitative efforts, it is advisable to passively monitor the patient for 10 minutes before confirming death.¹⁸ ♦

The resuscitation predictor score

Use this table 15 minutes into a resuscitation where the following are known:

- primary arrhythmia
- patient's age
- primary mode of arrest (respiratory or cardiac)*

Variable	Alive – 24 hrs
PEA or asystole + 70 years or older + primary cardiac	4%
PEA or asystole + 69 years or less + primary cardiac	6%
PEA or asystole + 70 years or older + primary respiratory	10%
PEA or asystole + 69 years or less + primary respiratory	17%
VT or VF + 70 years or older + primary cardiac	12%
VT or VF + 69 years or less + primary cardiac	28%
VT or VF + 70 years or older + primary respiratory	29%
VT or VF + 69 years or less + primary respiratory	**

Note: the RPS scale is based on the following premise:

- the shorter the resuscitation the greater the chance of survival (P = <0.001)
- primary arrhythmias of VT/VF have a significantly higher survival rate than PEA/asystole (P = <0.001)
- patients of 69 years or less are significantly more likely to survive (P = <0.001)
- where the primary mode of arrest is respiratory (as opposed to cardiac) the survival rate is significantly improved (P = 0.001)

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*Refers to the initial mode of arrest. Was it a primary respiratory arrest or a primary cardiac arrest?

**Not defined in scoring system due to insufficient data in original cohort.

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Table 1

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