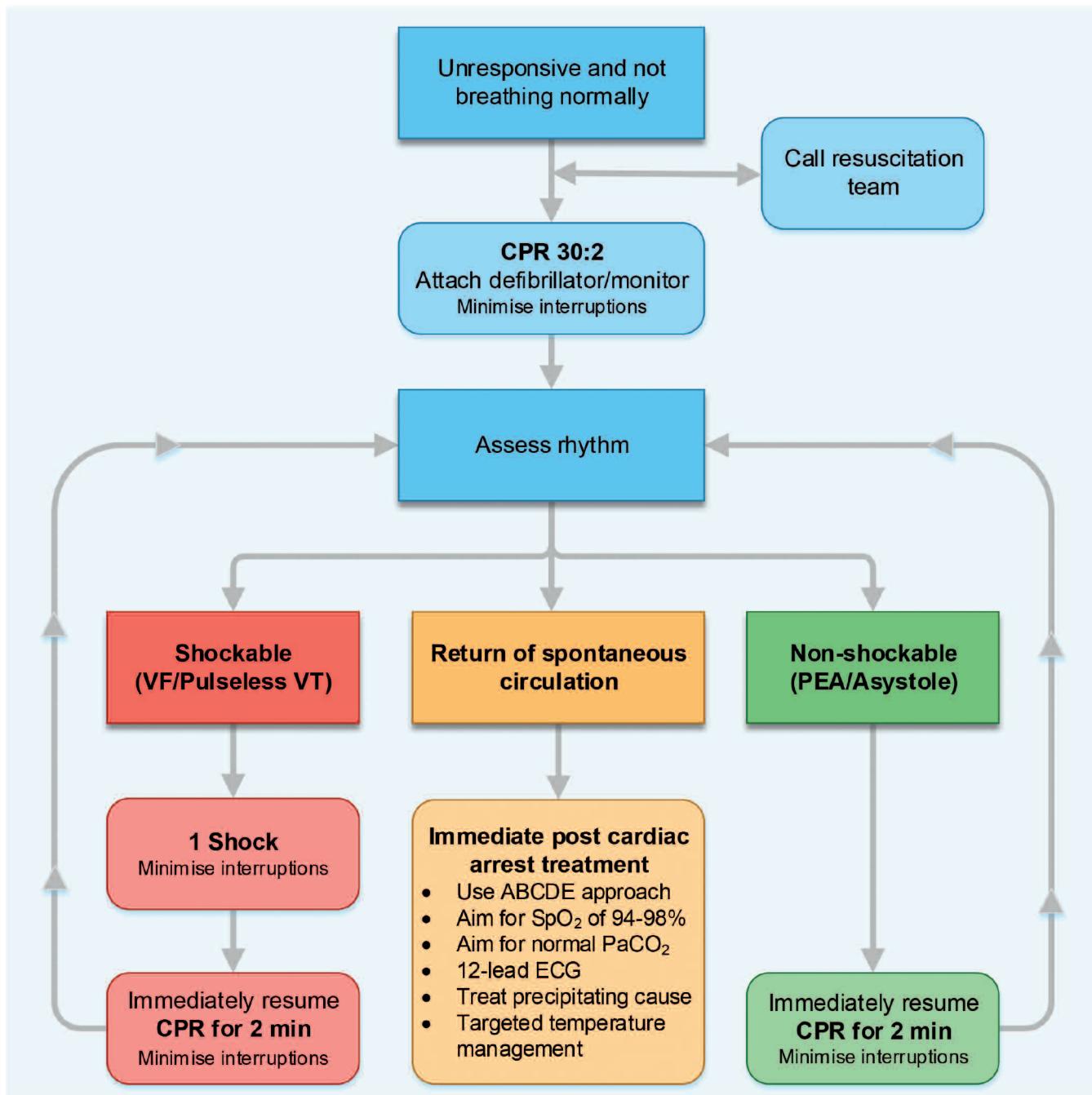


Vogel

Advanced Life Support

7th Edition January 2016

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During CPR

- Ensure high quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Continuous compressions when advanced airway in place
- Vascular access (intravenous or intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks

Treat Reversible Causes

- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypothermia
- Thrombosis - coronary or pulmonary
- Tension pneumothorax
- Tamponade – cardiac
- Toxins

Consider

- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR

Figure 1.2 Adult Advanced Life Support algorithm

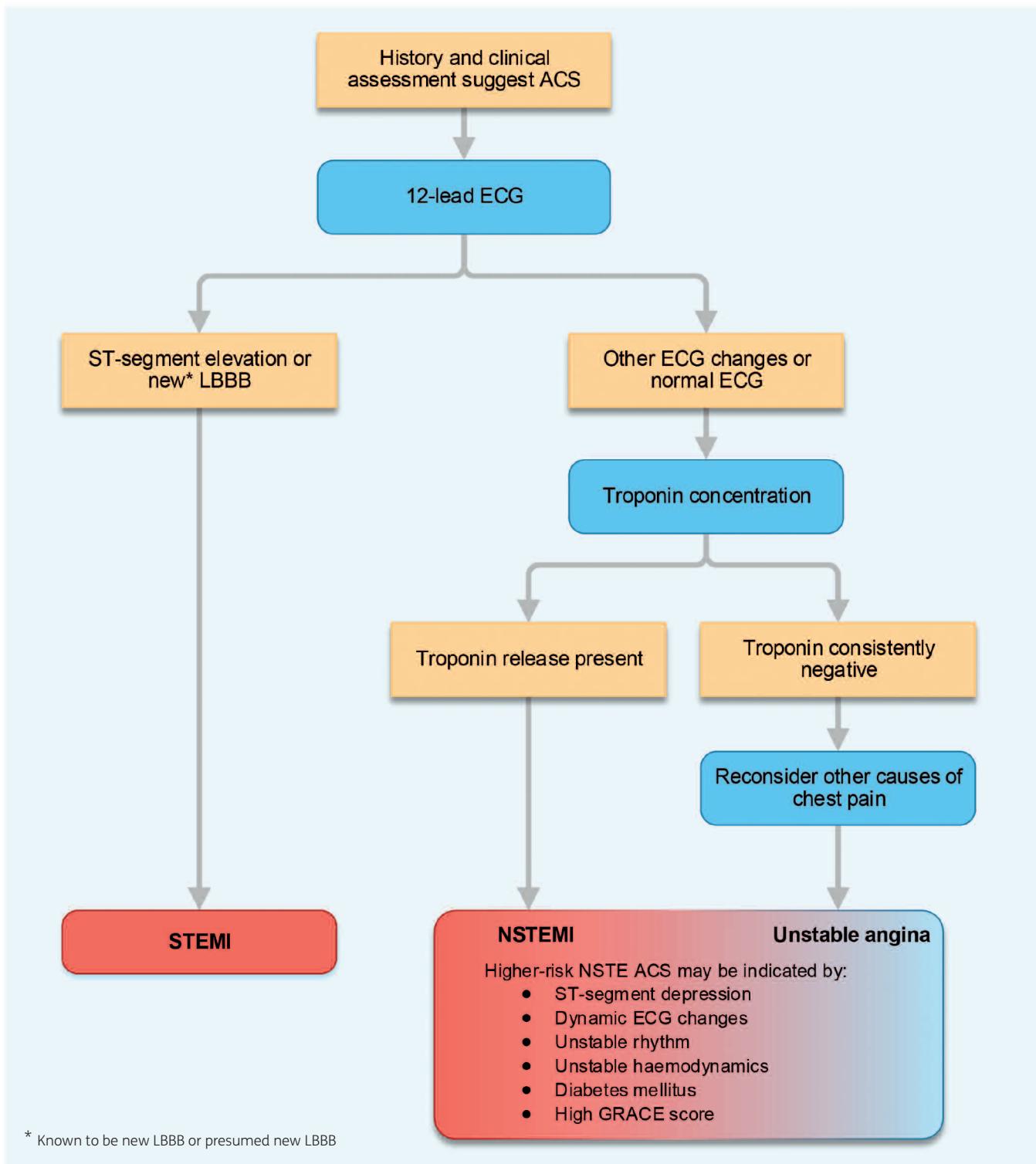


Figure 4.1 Algorithm for identifying the type of acute coronary syndrome

STEMI – ST-segment-elevation myocardial infarction; **NSTE ACS** – Non-ST-segment-elevation acute coronary syndromes; **NSTEMI** – Non-ST-segment-elevation myocardial infarction; **UA** – Unstable angina; **GRACE** – Global Registry of Acute Coronary Events

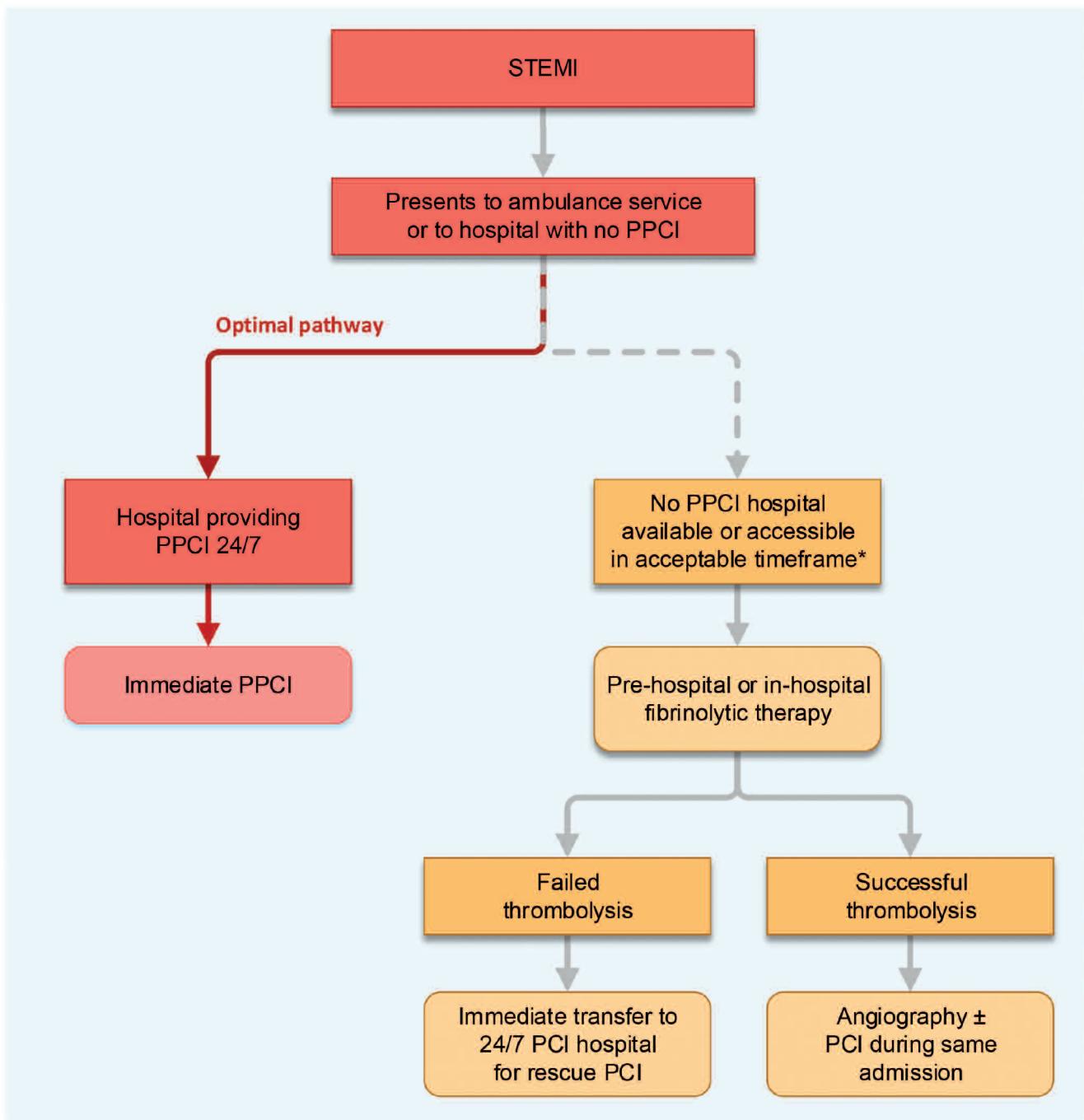


Figure 4.10 Algorithm showing options for access to reperfusion therapy for STEMI

*see NICE CG 167 and ESC guidelines for STEMI

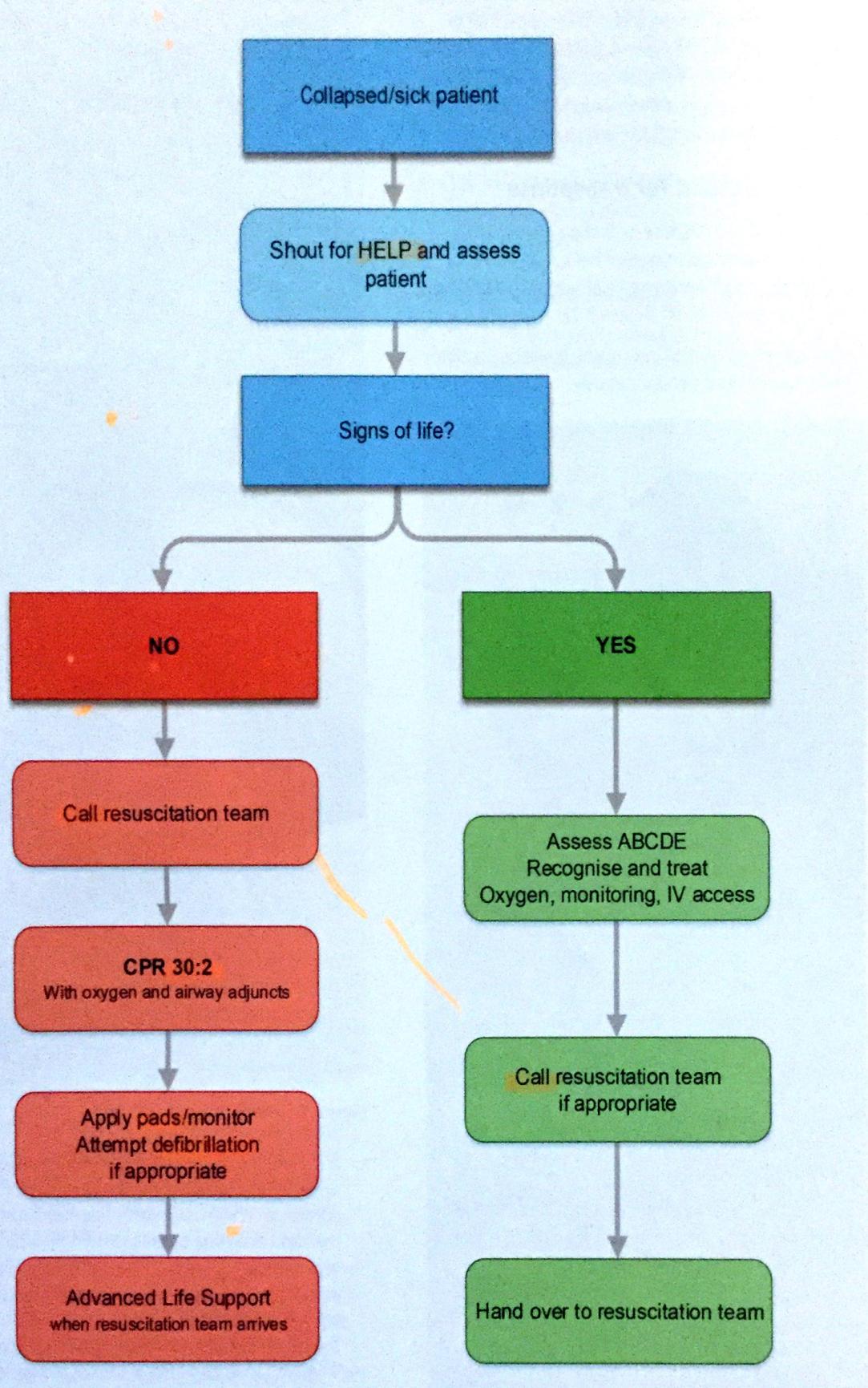


Figure 5.1 In-hospital resuscitation algorithm

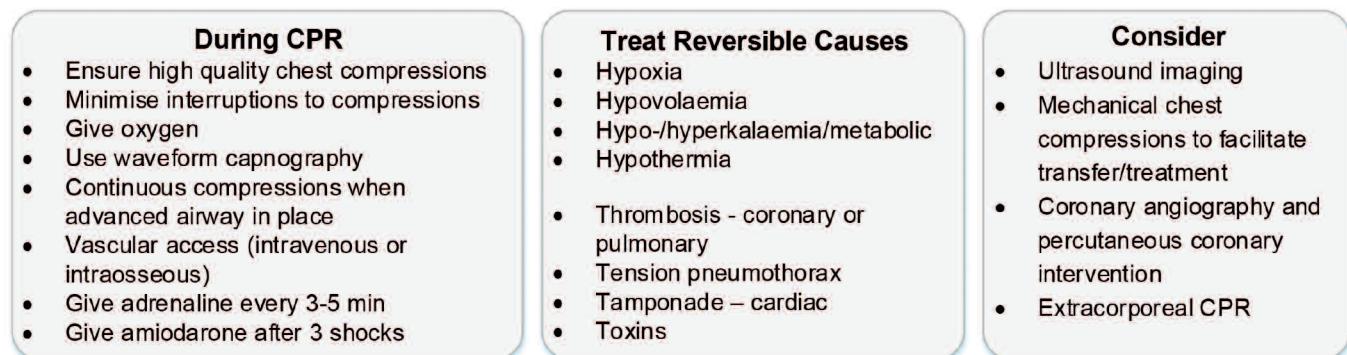
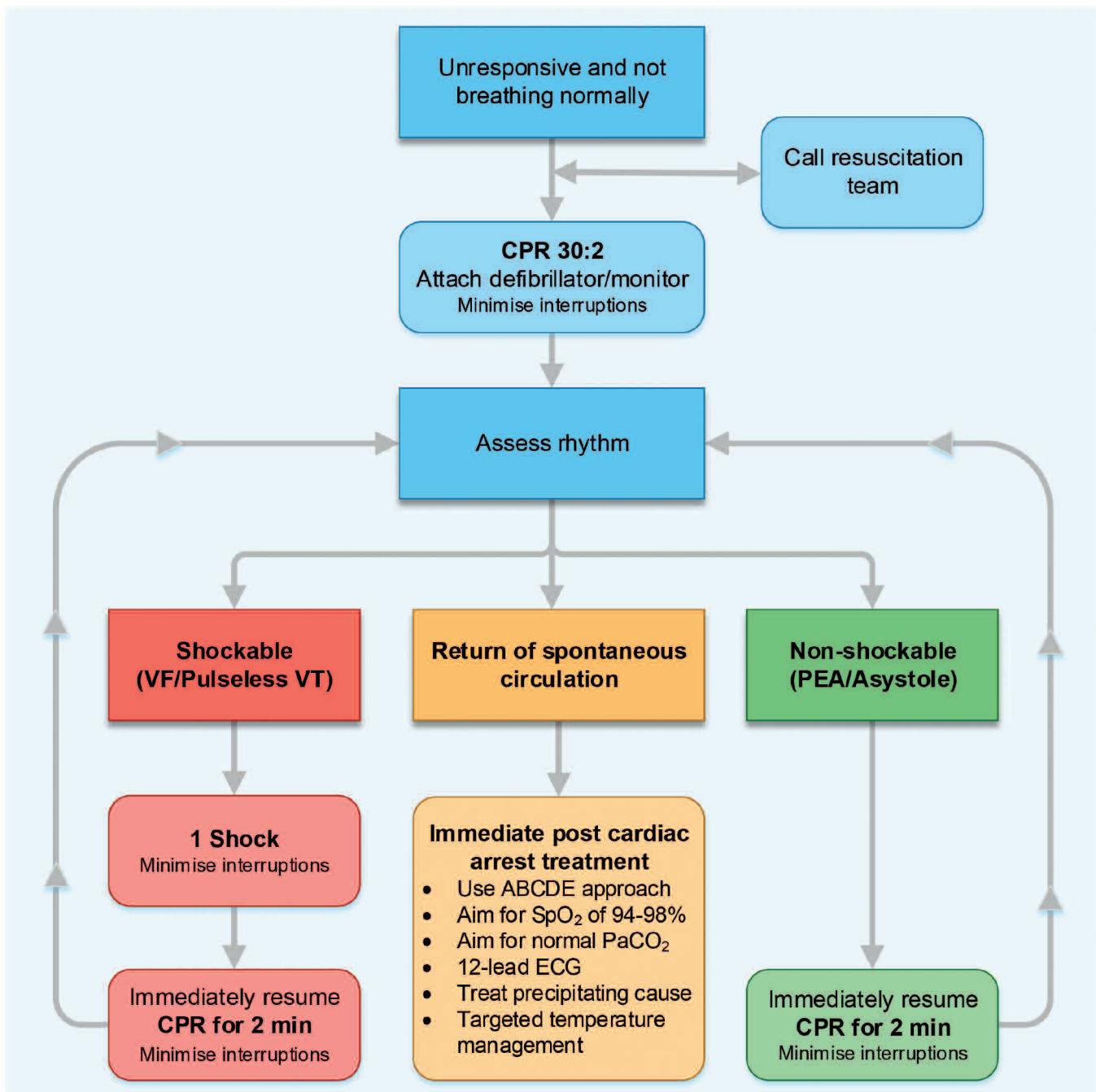


Figure 6.1 Adult Advanced Life Support algorithm

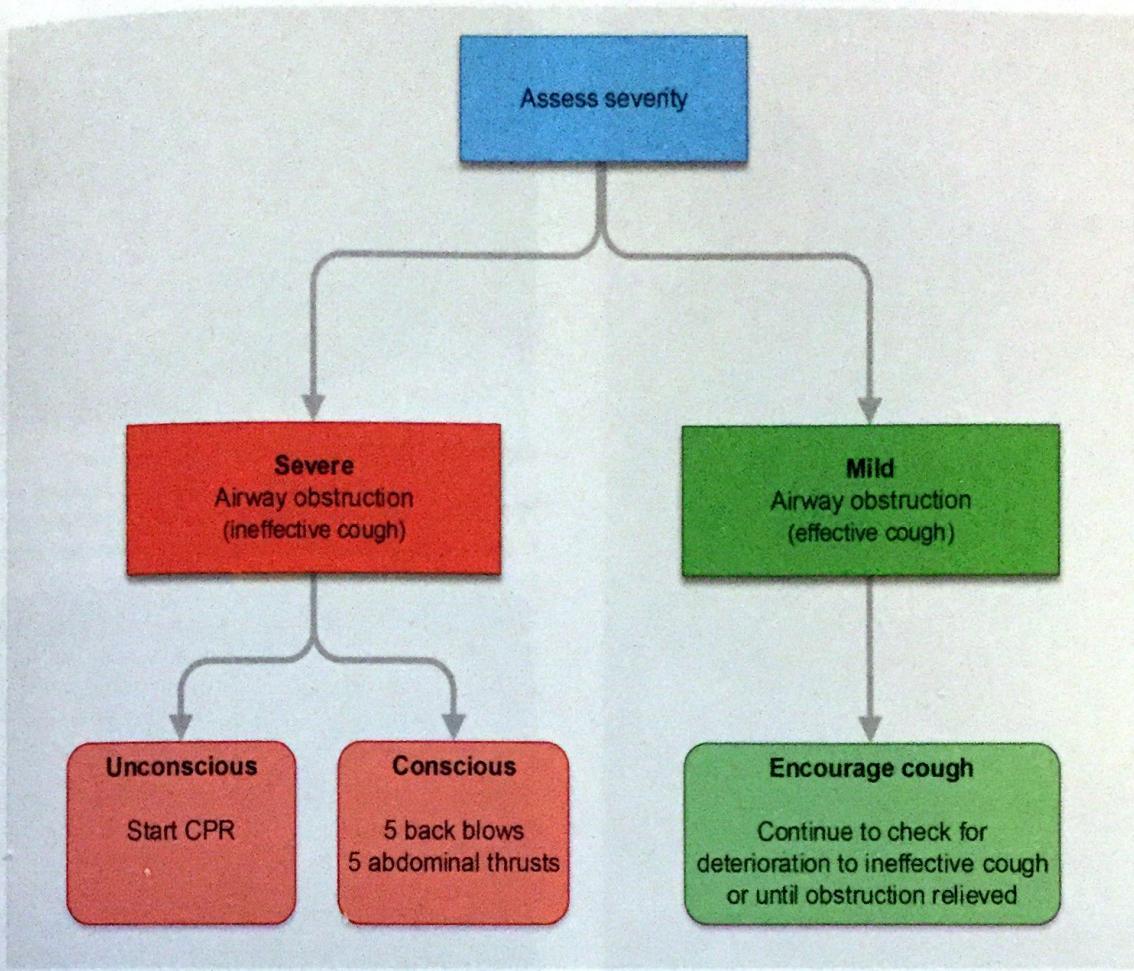


Figure 7.1 Adult choking algorithm

2. If the patient shows signs of **severe airway obstruction** and is conscious:
 - Give up to **5 back blows**.
 - Stand to the side and slightly behind the patient.
 - **Support the chest with one hand and lean the patient well forwards.**
 - Give up to **5 sharp blows between the scapulae** with the heel of the other hand.
 - Check to see if each back blow has relieved the airway obstruction.
 - If 5 back blows fail to relieve the airway obstruction give up to **5 abdominal thrusts**.
 - Stand behind the patient and put both arms round the **upper part of his abdomen**.
 - Place a **clenched fist** just under the **xiphisternum**; grasp this hand with your other hand and pull sharply **inwards and upwards**.
 - Repeat up to 5 times.
 - If the obstruction is still not relieved, continue alternating **5 back blows with abdominal thrusts**.
3. If the patient becomes unconscious, call the resuscitation team and start CPR.
4. As soon as an individual with appropriate skills is

present, undertake **laryngoscopy** and attempt to remove any foreign body with Magill's forceps.

Basic techniques for opening the airway

Once airway obstruction is recognised, take immediate action to relieve the obstruction and maintain a clear airway. Three manoeuvres that can be used to relieve upper airway obstruction are:

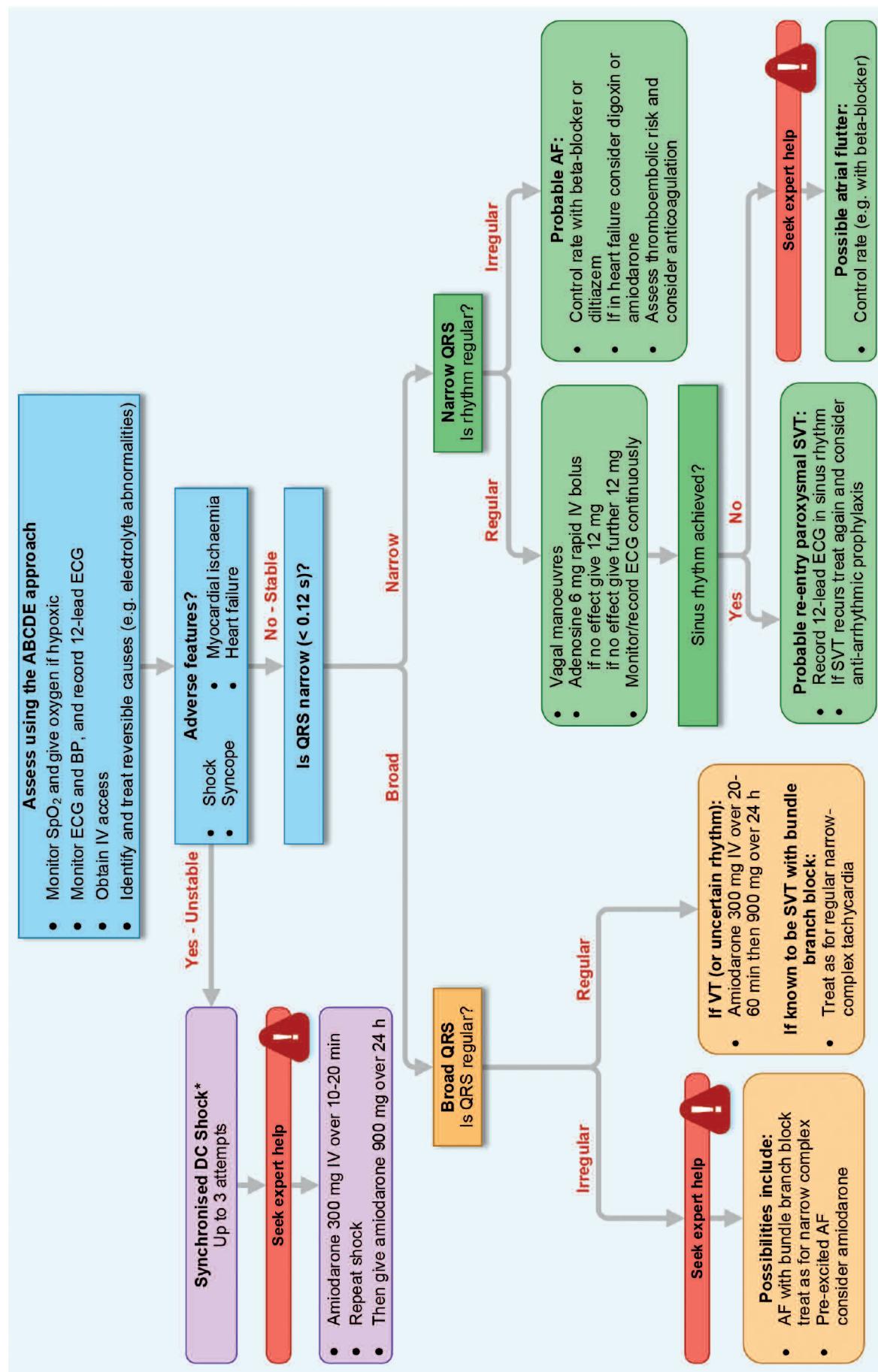
- **head tilt**
- **chin lift**
- **jaw thrust**.

Head tilt and chin lift

Place one hand on the patient's forehead and tilt the head back gently; place the fingertips of the other hand under the point of the patient's chin, and gently lift to stretch the anterior neck structures (Figure 7.2).

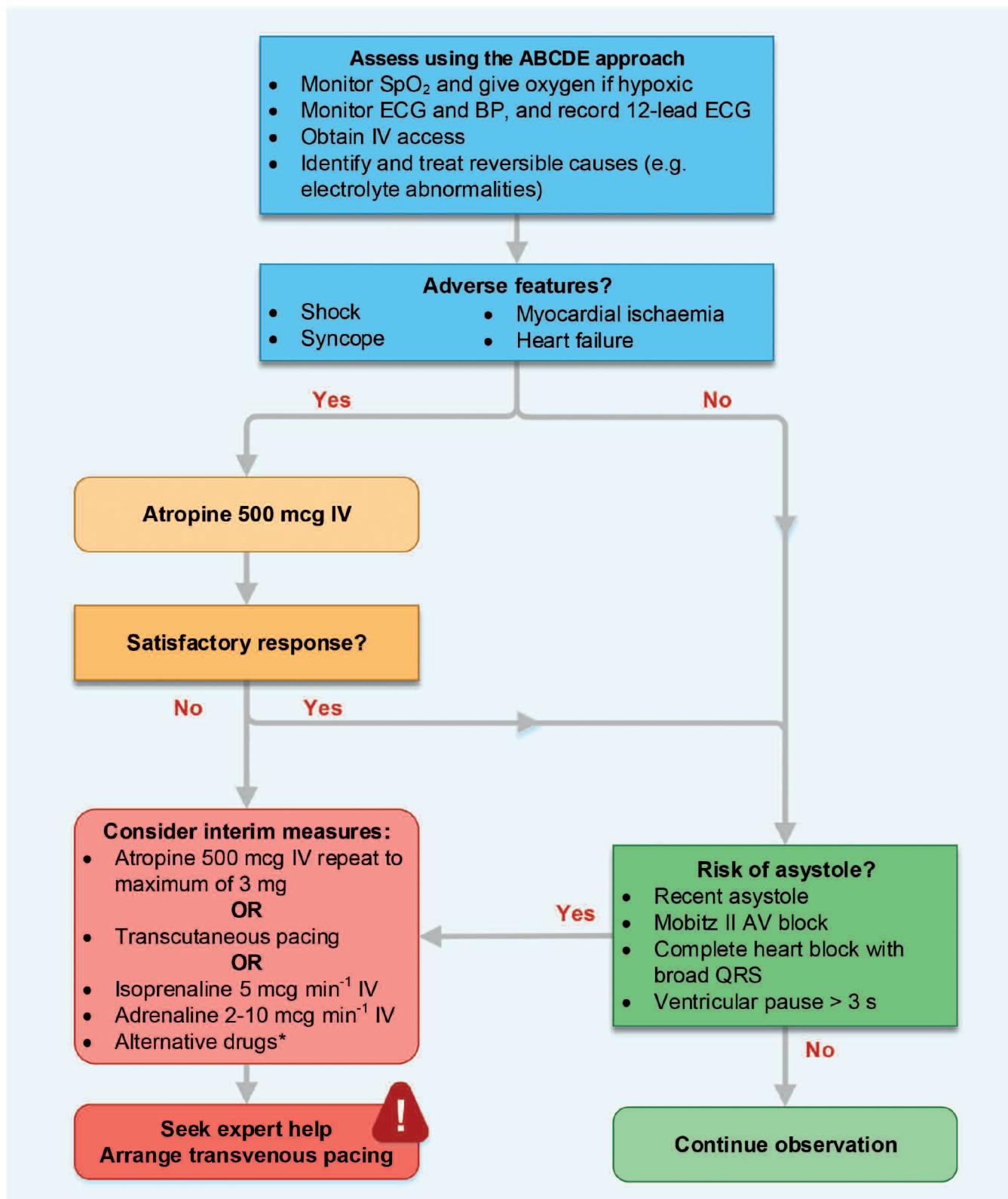
Jaw thrust

Jaw thrust is an alternative manoeuvre for bringing the mandible forward and relieving obstruction by the tongue, soft palate and epiglottis (Figure 7.3). It is most successful when applied with a head tilt.



*Conscious patients require sedation or general anaesthesia for cardioversion

Figure 11.1 Tachycardia algorithm



* Alternatives include:

- Aminophylline
- Dopamine
- Glucagon (if bradycardia is caused by beta-blocker or calcium channel blocker)
- Glycopyrrrolate (may be used instead of atropine)

Figure 11.2 Bradycardia algorithm

AAGBI Safety Guideline

Management of Severe Local Anaesthetic Toxicity



1 Recognition

Signs of severe toxicity:

- Sudden alteration in mental status, severe agitation or loss of consciousness, with or without tonic-clonic convulsions
- Cardiovascular collapse: sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias may all occur
- Local anaesthetic (LA) toxicity may occur some time after an initial injection

2 Immediate management

- Stop injecting the LA
- Call for help
- Maintain the airway and, if necessary, secure it with a tracheal tube
- Give 100% oxygen and ensure adequate lung ventilation (hyperventilation may help by increasing plasma pH in the presence of metabolic acidosis)
- Confirm or establish intravenous access
- Control seizures: give a benzodiazepine, thiopental or propofol in small incremental doses
- Assess cardiovascular status throughout
- Consider drawing blood for analysis, but do not delay definitive treatment to do this

3 Treatment

IN CIRCULATORY ARREST

- Start cardiopulmonary resuscitation (CPR) using standard protocols
- Manage arrhythmias using the same protocols, recognising that arrhythmias may be very refractory to treatment
- Consider the use of cardiopulmonary bypass if available

GIVE INTRAVENOUS LIPID EMULSION

(following the regimen overleaf)

- Continue CPR throughout treatment with lipid emulsion
- Recovery from LA-induced cardiac arrest may take >1 h
- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

WITHOUT CIRCULATORY ARREST

Use conventional therapies to treat:

- hypotension,
- bradycardia,
- tachyarrhythmia

CONSIDER INTRAVENOUS LIPID EMULSION

(following the regimen overleaf)

- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

4 Follow-up

- Arrange safe transfer to a clinical area with appropriate equipment and suitable staff until sustained recovery is achieved
- Exclude pancreatitis by regular clinical review, including daily amylase or lipase assays for two days
- Report cases as follows:
 - in the United Kingdom to the National Patient Safety Agency (via www.npsa.nhs.uk)
 - in the Republic of Ireland to the Irish Medicines Board (via www.imb.ie)

If Lipid has been given, please also report its use to the international registry at www.lipidregistry.org. Details may also be posted at www.lipidrescue.org

Your nearest bag of Lipid Emulsion is kept.....

This guideline is not a standard of medical care. The ultimate judgement with regard to a particular clinical procedure or treatment plan must be made by the clinician in the light of the clinical data presented and the diagnostic and treatment options available.

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IMMEDIATELY

Give an initial intravenous bolus injection of 20% lipid emulsion $1.5 \text{ ml}.\text{kg}^{-1}$ over 1 min

AND

Start an intravenous infusion of 20% lipid emulsion at $15 \text{ ml}.\text{kg}^{-1}.\text{h}^{-1}$

AFTER 5 MIN

Give a **maximum of two** repeat boluses (same dose) if:

- cardiovascular stability has not been restored **or**
- an adequate circulation deteriorates

Leave 5 min between boluses

A maximum of **three** boluses can be given (including the initial bolus)

AND

Continue infusion at same rate, but:

- Double the rate to $30 \text{ ml}.\text{kg}^{-1}.\text{h}^{-1}$ at any time after 5 min, if:
- cardiovascular stability has not been restored
 - an adequate circulation deteriorates
- Continue infusion until stable and adequate circulation restored or maximum dose of lipid emulsion given

Do not exceed a maximum cumulative dose of $12 \text{ ml}.\text{kg}^{-1}$

An approximate dose regimen for a 70-kg patient would be as follows:

IMMEDIATELY

Give an initial intravenous bolus injection of 20% lipid emulsion 100 ml over 1 min

AND

Start an intravenous infusion of 20% lipid emulsion at $1000 \text{ ml}.\text{h}^{-1}$

AFTER 5 MIN

Give a **maximum of two** repeat boluses of 100 ml

AND

Continue infusion at same rate but **double** rate to $2000 \text{ ml}.\text{h}^{-1}$ if indicated at any time

Do not exceed a maximum cumulative dose of 840 ml



This AAGBI Safety Guideline was produced by a Working Party that comprised:
Grant Cave, Will Harrop-Griffiths (Chair), Martyn Harvey, Tim Meek, John Picard, Tim Short and Guy Weinberg.

This Safety Guideline is endorsed by the Australian and New Zealand College of Anaesthetists (ANZCA).

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Figure 12.3 Management of severe local anaesthetic toxicity

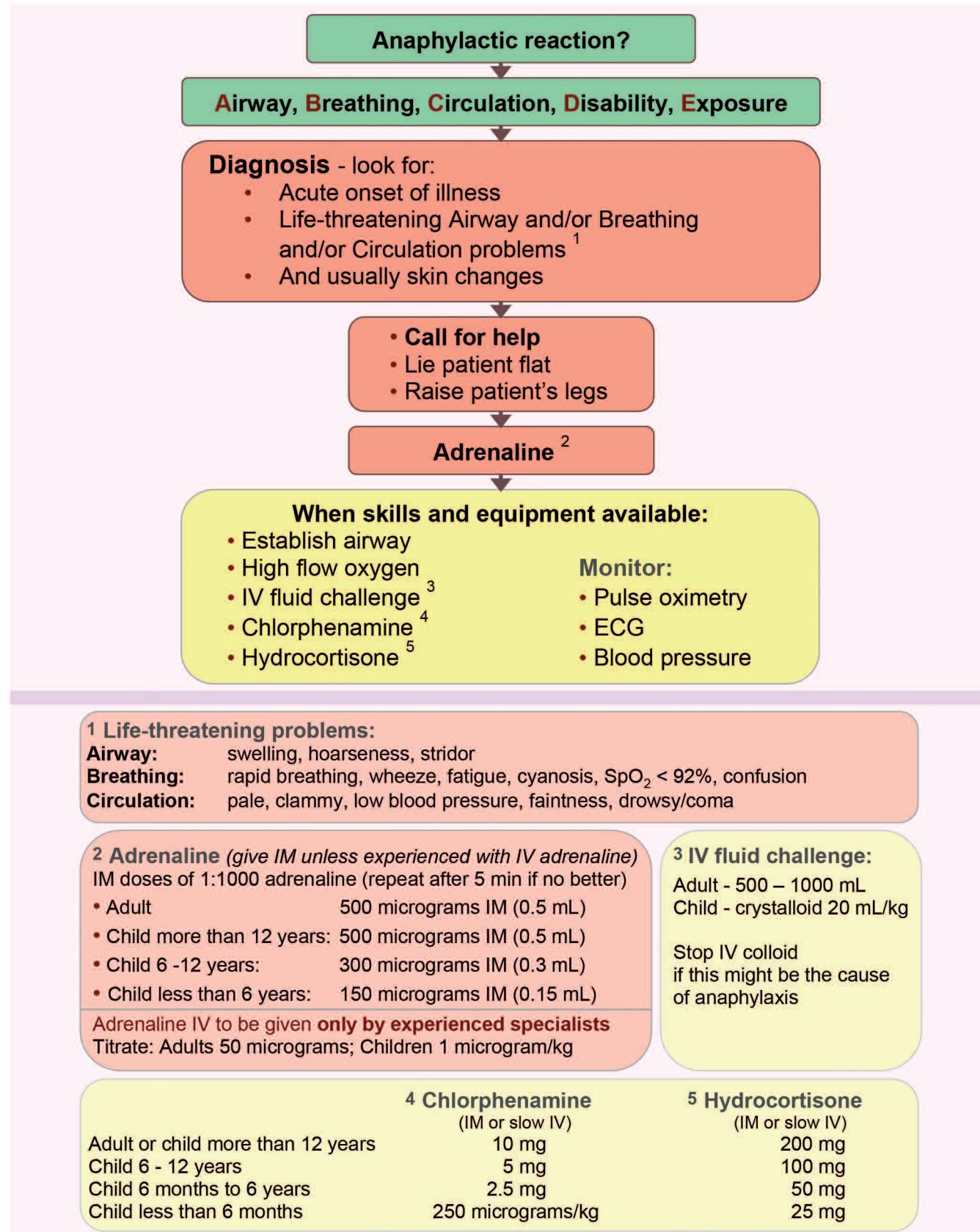


Figure 12.4 Anaphylaxis algorithm

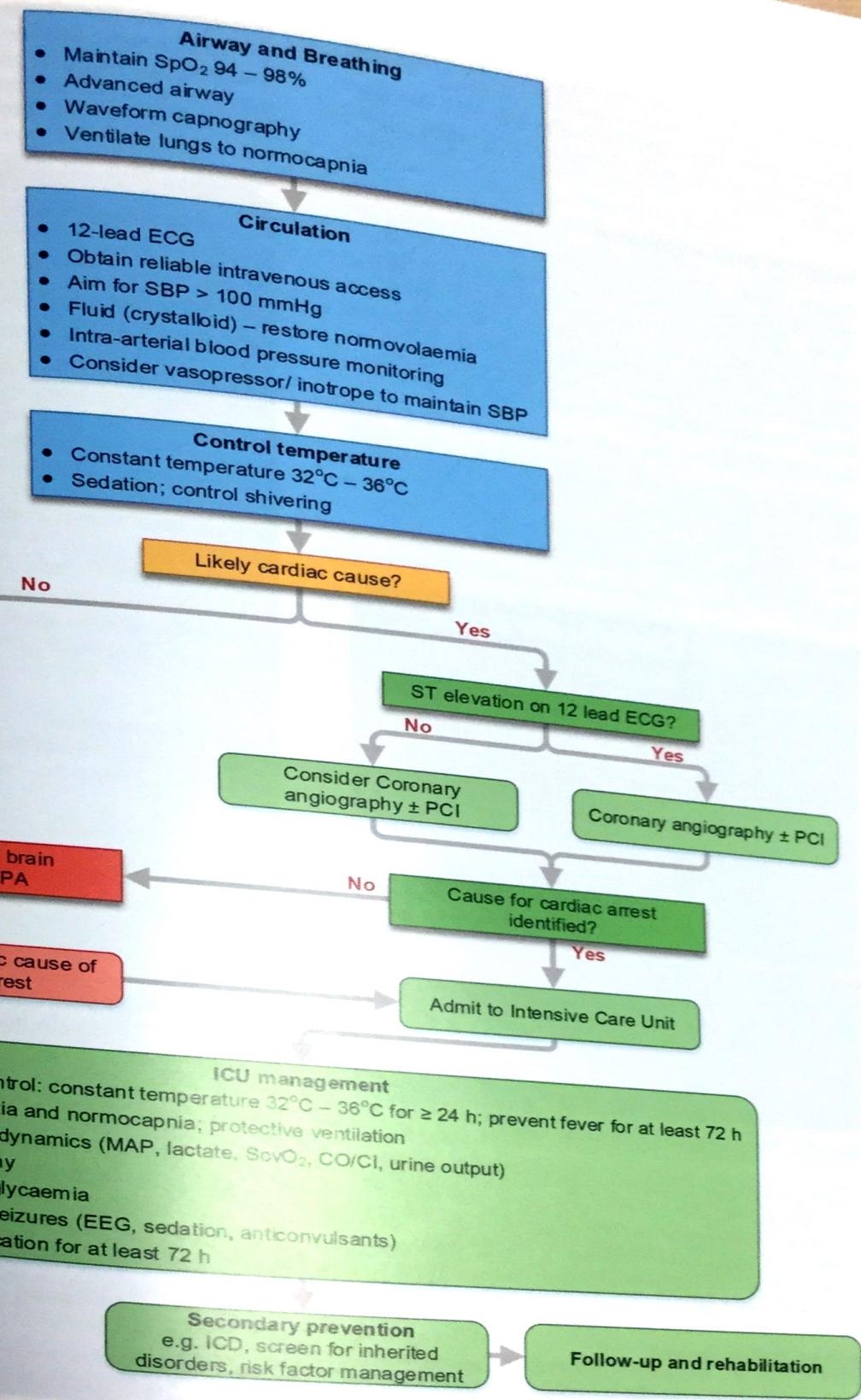


Figure 13.1 Post-resuscitation care algorithm

SBP systolic blood pressure; PCI percutaneous coronary intervention; CTPA computed tomography pulmonary angiogram; MAP mean arterial pressure; ScvO₂ central venous oxygenation; CO/CI cardiac output/cardiac index; EEG electroencephalography; ICD implanted cardioverter defibrillator.

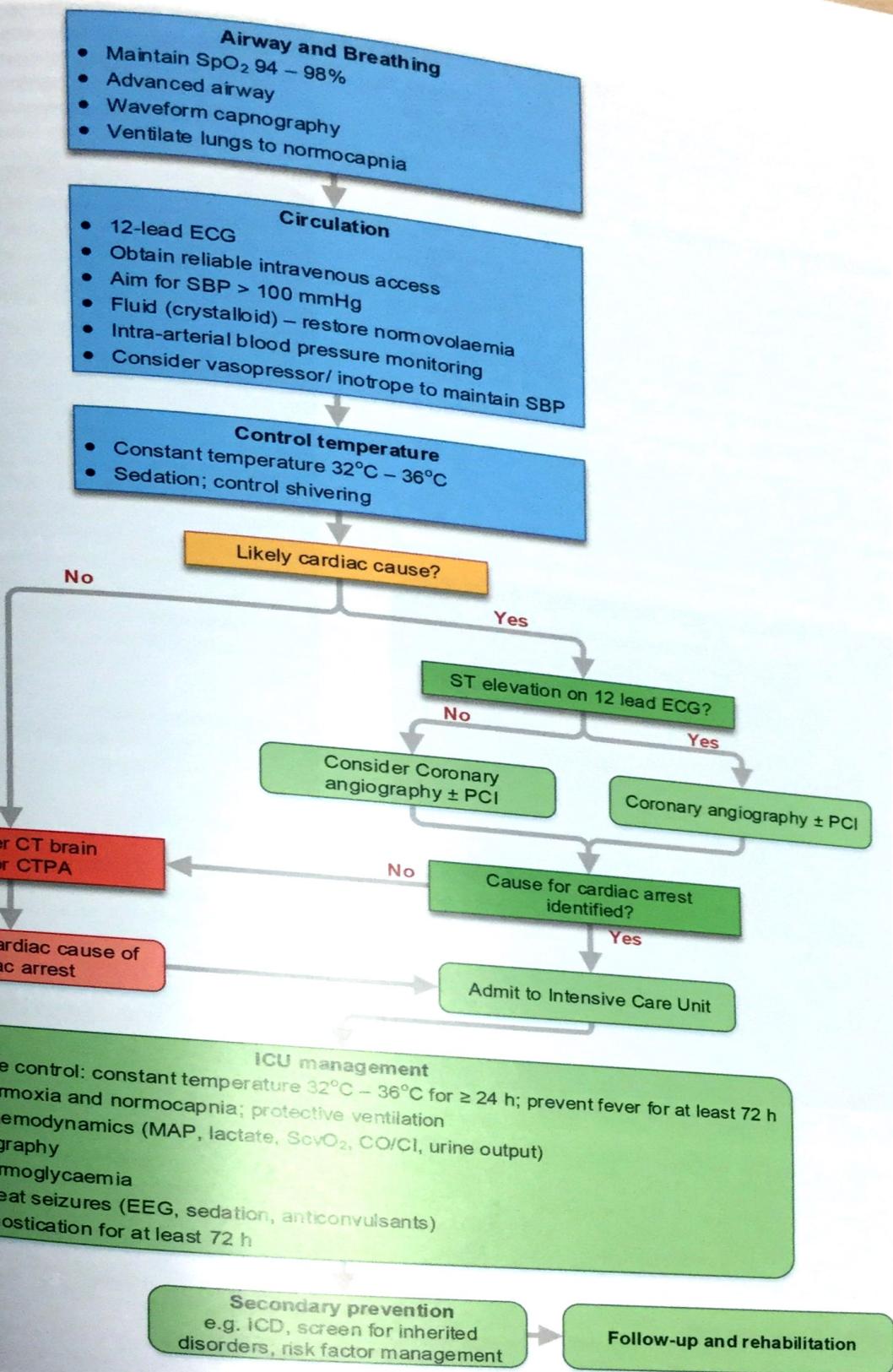


Figure 13.1 Post-resuscitation care algorithm

SBP systolic blood pressure; PCI percutaneous coronary intervention; CTPA computed tomography pulmonary angiogram; MAP mean arterial pressure; ScvO_2 central venous oxygenation; CO/CI cardiac output/cardiac index; EEG electroencephalography; ICD implanted cardioverter defibrillator.

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Advanced Life Support in perspective

Chapter 1

The problem

Coronary heart disease (CHD) is the biggest single cause of death in the UK. In 2012, 16% of male deaths and 10% of female deaths were from CHD, a total of around 74,000 deaths. There are nearly half a million hospital admissions associated with CHD in the UK each year.

English ambulance services initiate resuscitation on about 28,000 patients who sustain an out-of-hospital cardiac arrest each year (52 cases per 100,000 inhabitants). Most cardiac arrests occur in the home and are presumed to have a cardiac cause. Around two-thirds of cardiac arrests are witnessed by either a bystander or ambulance staff. The presenting rhythm is shockable (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)) in about a quarter of cases and non-shockable in the remainder – asystole in about 50% and pulseless electrical activity (PEA) in about 25% of cases. Amongst those in whom resuscitation is attempted, 25–30% initially achieve a return of spontaneous circulation (ROSC) but only approximately 8% survive to go home from hospital.

The incidence of in-hospital cardiac arrest is difficult to assess because it is influenced heavily by factors such as the criteria for hospital admission and implementation of do not attempt cardiopulmonary resuscitation (DNACPR) decisions. Data from 144 hospitals contributing data to the UK National Cardiac Arrest Audit showed that during 2011–2013 the overall incidence of adult in-hospital cardiac arrest was 1.6 per 1000 hospital admissions. The incidence varied seasonally, peaking in winter. The overall survival to hospital discharge was 18.4%. The presenting rhythm was shockable (VF/pVT) in 16.9% and non-shockable (asystole or pulseless electrical activity) in 72.3% of cases, and rates of survival to hospital discharge associated with these rhythms were 49.0% and 10.5%, respectively, but varied substantially across hospitals. All these individuals received chest compressions and/or defibrillation and were attended by the hospital resuscitation team in response to a 2222 call. Many patients who have an in-hospital cardiac arrest have significant comorbidity, which influences the initial rhythm and, in these cases, strategies to prevent cardiac arrest are particularly important.

The Chain of Survival

The interventions that contribute to a successful outcome after a cardiac arrest can be conceptualised as a chain - the Chain of Survival (Figure 1.1). The chain is only as strong as its weakest link; all four links of the Chain of Survival must be strong. They are:

- Early recognition and call for help
- Early cardiopulmonary resuscitation (CPR)



Figure 1.1 Chain of Survival

- Early defibrillation
- Post-resuscitation care.

Early recognition and call for help

Out of hospital, early recognition of the importance of chest pain will enable the victim or a bystander to call the ambulance service so that the victim can receive treatment that may prevent cardiac arrest. Once cardiac arrest has occurred, early recognition is critical to enable rapid activation of the ambulance service and prompt initiation of bystander CPR. In the UK, the ambulance service can be contacted by dialing 999.

In hospital, early recognition of the critically ill patient who is at risk of cardiac arrest and a call for the resuscitation team or medical emergency team (MET) will enable treatment to prevent cardiac arrest (Chapter 3). A universal number for calling the resuscitation team or MET should be adopted in all hospitals - in the UK this number is 2222. If cardiac arrest occurs, do not delay defibrillation until arrival of the resuscitation team – clinical staff should be trained to use a defibrillator.

Early CPR

Chest compressions and ventilation of the victim's lungs will slow down the rate of deterioration of the brain and heart. After out-of-hospital cardiac arrest, bystander CPR extends the period for successful resuscitation and at least doubles to quadruples the chance of survival after VF/pVT cardiac arrest. Performing chest-compression-only CPR is better than giving no CPR at all. Despite the well-accepted importance of CPR, bystander CPR rates are only approximately 40%. After in-hospital cardiac arrest, chest compressions and ventilation must be undertaken immediately, but should not delay attempts to defibrillate those patients in VF/pVT. Interruptions to chest compressions must be minimised and should occur only very briefly during defibrillation attempts and rhythm checks.

Early defibrillation

Defibrillation within 3–5 min of collapse can produce survival rates as high as 50–70%. This can be achieved through public access defibrillation, when a bystander uses a nearby AED to deliver the first shock. Each minute of delay to defibrillation reduces the probability of survival to hospital discharge by 10–12%. In the UK, an AED is deployed to fewer than 2% of out-of-hospital cardiac arrests (OHCA) before ambulance arrival. In hospitals, sufficient healthcare personnel should be trained and authorised to use a defibrillator to enable the first responder to a cardiac arrest to attempt defibrillation as rapidly as possible and certainly within 3 min.

Post-resuscitation care

Return of a spontaneous circulation is an important phase in the continuum of resuscitation; however, the ultimate goal is to return the patient to a state of normal cerebral function, a stable cardiac rhythm, and normal haemodynamic function, so that they can leave hospital in

reasonable health at minimum risk of a further cardiac arrest. The quality of treatment in the post-resuscitation period influences the patient's ultimate outcome. The post-resuscitation phase starts at the location where ROSC is achieved. The ALS provider must be capable of providing high quality post-resuscitation care until the patient is transferred to an appropriate high-care area.

ALS algorithm

The ALS algorithm (Figure 1.2) is the centre point of the ALS course and is applicable to most CPR situations. Some modifications may be required when managing cardiac arrest in special circumstances (Chapter 12).

Improving outcomes from cardiac arrest

High quality care is safe, effective, patient-centred, timely, efficient and equitable. Hospitals, resuscitation teams and ALS providers should ensure they deliver these aspects of quality to improve the care of the deteriorating patient and patients in cardiac arrest. We describe below some of the strategies to improve outcomes.

Evidence based guidelines

Improving outcomes from cardiac arrest depends on the implementation of evidence based guidelines. The contents of this ALS provider manual are consistent with the Resuscitation Council (UK) Guidelines. The process used to produce the Resuscitation Council (UK) Guidelines has been accredited by the National Institute for Health and Care Excellence. The guidelines process includes:

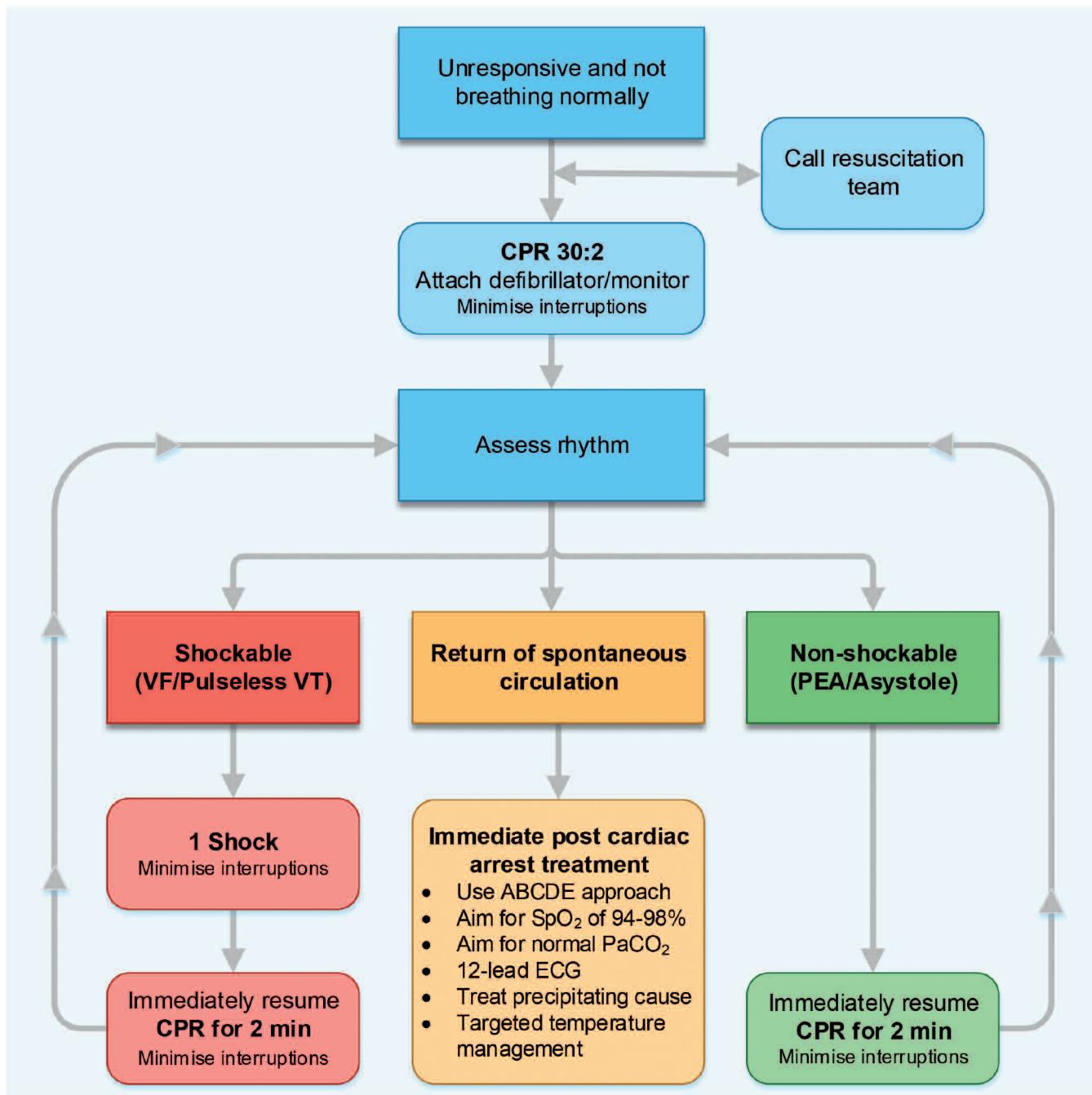
- Systematic reviews with grading of the quality of evidence and strength of recommendations. This led to the 2015 International Liaison Committee on Resuscitation (ILCOR) Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations.
- The involvement of stakeholders from around the world including members of the public and cardiac arrest survivors.
- Collaboration with the European Resuscitation Council, and adapting its guidelines for use in the UK.

The current Resuscitation Council (UK) Guidelines for cardiopulmonary resuscitation can be found at www.resus.org.uk

Quality standards

Healthcare organisations have an obligation to provide a high quality resuscitation service, and to ensure that staff are trained and updated regularly and with appropriate frequency to a level of proficiency appropriate to each individual's expected role. The same core standards apply to all health care settings:

- The deteriorating patient is recognised early and there is an effective system to summon help in order to prevent cardiorespiratory arrest.



- During CPR**
- Ensure high quality chest compressions
 - Minimise interruptions to compressions
 - Give oxygen
 - Use waveform capnography
 - Continuous compressions when advanced airway in place
 - Vascular access (intravenous or intraosseous)
 - Give adrenaline every 3-5 min
 - Give amiodarone after 3 shocks

- Treat Reversible Causes**
- Hypoxia
 - Hypovolaemia
 - Hypo-/hyperkalaemia/metabolic
 - Hypothermia
 - Thrombosis - coronary or pulmonary
 - Tension pneumothorax
 - Tamponade – cardiac
 - Toxins

- Consider**
- Ultrasound imaging
 - Mechanical chest compressions to facilitate transfer/treatment
 - Coronary angiography and percutaneous coronary intervention
 - Extracorporeal CPR

Figure 1.2 Adult Advanced Life Support algorithm

- Cardiorespiratory arrest is recognised early and CPR is started immediately.
- Emergency assistance is summoned immediately, as soon as cardiorespiratory arrest is recognised, if help has not been summoned already.
- Defibrillation, if appropriate, is attempted within 3 min of identifying cardiorespiratory arrest.
- Appropriate post-cardiorespiratory-arrest care is received by those who are resuscitated successfully. This includes safe transfer.
- Implementation of standards is measured continually and processes are in place to deal with any problems identified.
- Staff receive at least annual training and updates in CPR, based on their expected roles.
- Staff have an understanding of decisions relating to CPR.
- Appropriate equipment is immediately available for resuscitation.

The Resuscitation Council (UK) *Quality standards for cardiopulmonary resuscitation practice and training* provide further detailed information.

Measuring patient outcomes

Continuous measurement of compliance with processes, and patient outcomes at a national and local level provides information on the impact of changes in practice, identifies areas for improvement, and also enables comparison in outcomes between different organisations. Uniform definitions for collecting data for cardiac arrest exist.

The National Out of Hospital Cardiac Arrest Outcomes project measures patient, process and outcome variables from out-of-hospital-cardiac arrest in the UK. The project is run in collaboration with the National Ambulance Service Medical Directors Group with support from the British Heart Foundation, Resuscitation Council (UK) and University of Warwick. The project is designed to measure the epidemiology, and outcomes of cardiac arrest and to serve as a national resource for continuous quality improvement initiatives for cardiac arrest.

The National Cardiac Arrest Audit (NCAA) is an ongoing, national, comparative outcome audit of in-hospital cardiac arrests. It is a joint initiative between the Resuscitation Council (UK) and the Intensive Care National Audit & Research Centre (ICNARC) and is open to all acute hospitals in the UK and Ireland. The audit monitors and reports on the incidence of, and outcome from, in-hospital cardiac arrest in order to inform practice and policy. It aims to identify and foster improvements in the prevention, care delivery and outcomes from cardiac arrest.

Data are collected according to standardised definitions and entered onto the NCAA secure web-based system. Once data are validated, hospitals are provided with activity reports and risk-adjusted comparative reports,

allowing a comparison of to be made not only within, but also between, hospitals locally, nationally and internationally.

Safety incident reporting

In England and Wales, hospitals can report patient safety incidents to the National Reporting and Learning System (NRLS) (<http://www.nrls.npsa.nhs.uk/report-a-patient-safety-incident/>). A patient safety incident is defined as 'any unintended or unexpected incident that could have harmed or did lead to harm for one or more patients being cared for by the National Health Service (NHS)'. Resuscitation related incidents are associated with equipment problems, communication, delays in the resuscitation team attending, and failure to escalate treatment.

The ALS course

The Resuscitation Council (UK) ALS course provides a standardised approach to cardiopulmonary resuscitation in adults. The course is targeted at doctors, nurses, and other healthcare professionals who are expected to provide ALS in and out of hospital. The multidisciplinary nature of the course encourages efficient teamwork. By training together, all ALS providers are given the opportunity to gain experience as both resuscitation team members and team leaders. The ALS course teaches the knowledge and skills required to:

- recognise and treat the deteriorating patient using a structured ABCDE approach
- deliver standardised CPR in adults
- manage a cardiac arrest by working with a multidisciplinary team in an emergency situation
- use non-technical skills to facilitate strong team leadership and effective team membership.

The Resuscitation Council (UK) offers the option for a two-day face-to-face ALS course, or the eALS course that consists of one day of e-learning that can be done in the participants own time and one day of face-to-face teaching at an ALS course centre. A large multi-centre randomised-controlled trial showed that the learning outcomes were similar for these two courses. The face-to-face component of both courses includes workshops, skill stations, cardiac arrest simulation (CAS) training, and lectures. Candidates' knowledge is assessed by means of a multiple choice question paper. Practical skills in airway management and the initial approach to a collapsed patient (including defibrillation where appropriate) are assessed continuously. There is also assessment of a simulated cardiac arrest (CASTest). Candidates reaching the required standard receive a Resuscitation Council (UK) ALS Provider certificate.

Resuscitation knowledge and skills deteriorate with time and therefore recertification is required for those who have not recently undertaken the course to maintain ALS Provider status. Recertification provides the opportunity to refresh resuscitation skills and to be updated on

resuscitation guidelines, and can be undertaken by attending a provider course or an accredited recertification course. All ALS providers have a responsibility to maintain their skills in resuscitation and to keep up to date with changes in guidelines and practice, and the requirement for recertification should be seen as an absolute minimum frequency of refreshing skills and knowledge.

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Non-technical skills in resuscitation

Chapter
2

Contents

- Non-technical skills
- Situational awareness
- Decision making
- Team working, including team leadership
- Task management
- The importance of communication
- Resuscitation teams

Learning outcomes

To enable you to:

- Be an effective team leader and team member
- Consider the role of non-technical skills during resuscitation
- Effectively use structured communication tools such as SBAR and RSVP

Non-technical skills

The skills of chest compressions, defibrillation, and rhythm recognition are considered typically to be important aspects of cardiac arrest management. These are all technical skills that are learnt from books, lectures, courses and peers. Although they are important for the successful resuscitation of a patient, there is another group of skills that is becoming increasingly recognised in medicine – non-technical skills. Non-technical skills can be defined as the cognitive, social and personal resource skills that complement technical skills and contribute to safe and efficient task performance. More simply, they are the things that affect our personal performance. Non-technical skills of leadership and teamwork have been identified as important contributory factors to technical skill performance in both simulated settings and poor clinical outcomes in acute medical settings.

The importance of non-technical skills in emergencies is now widely accepted across many acute medical specialties including surgery, anaesthesia, critical care and acute medicine. Examples of poor non-technical skills include unwillingness to help, poor communication, poor leadership, poor decision-making, and no clear roles, all of which can lead to system errors. Episodes of cardiac arrest with documented system errors are associated with poor clinical outcomes such as decreased rates of return of spontaneous circulation and worse survival. Previous research has demonstrated that leadership behaviour is correlated with quality of CPR, with shorter hands-off time, pre-shock pauses and time to first shock. Understanding and improving non-technical skills may help to reduce human errors, creating more effective teams and improve patient safety. An effective team leader can help focus the team members, improve team commitment and act as the role model for the others. The key non-technical skills are situational awareness, decision making, team working and leadership, and task management.

Situational awareness

This can be described as an individual's awareness of the environment at the moment of an event and the analysis of this to understand how an individual's actions may impact on future events. This becomes particularly important when many events are happening simultaneously, for example, at a cardiac arrest. High information input with poor situational awareness may lead to poor decision making and serious consequences. At a cardiac arrest, all those participating will have varying degrees of situational awareness. In a well-functioning team, all members will have a common understanding of current events, or shared situational awareness. It is important that only the relevant information is shared otherwise there is too much distraction or noise.

At a cardiac arrest, important situational awareness factors include:

- consideration of the location of the arrest, which can give clues to the cause
- obtaining information from staff about the events leading up to the arrest
- confirmation of the diagnosis
- determining who is present – including names, roles, and who is leading
- noting the actions already initiated (e.g. chest compressions)
- checking that a monitor has been attached and interpreting what it shows
- communicating with the team, gathering information
- implementing any immediate action necessary
- consideration of the likely impact of interventions
- determining the immediate needs.

Decision making

This is defined as the cognitive process of choosing a specific course of action from several alternatives. At a cardiac arrest, the many decisions to be made usually fall to the team leader. The leader will assimilate information from the team members and from personal observation and will use this to determine appropriate interventions. Typical decisions made at a cardiac arrest include:

- diagnosis of the cardiac arrest rhythm
- choice of shock energy to be used for defibrillation
- likely reversible causes of the cardiac arrest
- how long to continue resuscitation.

Once a decision has been made, clear unambiguous communication with the team members is essential to ensure that it is implemented.

Team working, including team leadership

This is one of the most important non-technical skills that contribute to successful management of critical situations. A team is a group of individuals working together with a common goal or purpose. In a team, the members usually have complementary skills and, through coordination of effort, work synergistically. Teams work best when everyone knows each other's name, when they are doing something they perceive to be important, and when their role is within their experience and competence. Optimal team function mandates a team leader. There are several characteristics of a good resuscitation team member:

- Competence – has the skills required at a cardiac arrest and performs them to the best of their ability.
- Commitment – strives to achieve the best outcome for the patient.
- Communicates openly – being able to articulate their findings and actions taken, raise concerns about clinical or safety issues, and listens to briefings and instructions.
- Supportive – enables others to achieve their best.
- Accountable – for their own and the team's actions.
- Prepared to admit when help is needed.
- Creative – suggests different ways of interpreting the situation.
- Participates in providing feedback.

Team leadership

A team leader provides guidance, direction and instruction to the team members to enable successful completion of their stated objective (Figure 2.1). They lead by example and integrity. Team leaders need experience not simply seniority. Team leadership can be considered a process; it is available to everyone with training and not restricted to those with leadership traits.



Figure 2.1 Team leader

There are several attributes recognisable in a good team leader:

- Knows everyone in the team by name and knows their capability.
- Accepts the leadership role.
- Is able to delegate tasks appropriately.
- Is knowledgeable and has sufficient credibility to influence the team through role modelling and professionalism.
- Stays calm, keeps everyone focused and controls distractions.
- Is a good communicator – not just good at giving instructions, but also a good listener and decisive in action.
- Is empathetic towards the whole team.
- Is assertive and authoritative when required.
- Shows tolerance towards hesitancy or nervousness in the emergency setting.
- Has good situational awareness; has the ability to constantly monitor the situation, with an up-to-date overview, listening and deciding on a course of action.

During a cardiac arrest, the role of team leader is not always immediately obvious. The leader should state early on that they are assuming the role of team leader. Specifically, at a cardiac arrest the leader should:

- Follow current resuscitation guidelines or explain a reason for any significant deviation from standard protocols.
- Consult with the team or call for senior advice and assistance if unsure about an intervention.
- Play to the strengths of team members and allow them some autonomy if their skills are adequate.
- Allocate roles and tasks throughout the resuscitation and be specific. This avoids several people or nobody attempting the task!
- Use the two-minute periods of chest compressions to plan tasks and safety aspects of the resuscitation attempt with the team.
- Thank the team at the end of the resuscitation attempt and ensure that staff and relatives are being supported.
- Complete all documentation and ensure an adequate handover.

Task management

During the resuscitation of a patient, either in full cardiac arrest or peri-arrest, there are numerous tasks to be carried out by the team members, either sequentially or simultaneously. The coordination and control, or management, of these tasks is the responsibility of the team leader (Figure 2.2). They include:

- Planning and, where appropriate, briefing of the team (e.g. prior to arrival of the patient in the emergency department).
- Being inclusive of all team members.
- Being prepared for both the expected and the unexpected.



Figure 2.2 Task management

- Identifying the resources required - ensuring that equipment is checked and specifics organised and delegated.
- Prioritising actions of the team.
- Watching out for fatigue, stress and distress amongst the team.
- Managing conflict.
- Communicating with relatives.
- Communicating with experts for safe handover both by telephone and in person.
- Debriefing the team.
- Reporting untoward incidents, particularly equipment or system failures.
- Participation in audit (Chapter 1).

To illustrate the importance of non-technical skills, a modified team performance tool, the Team Emergency Assessment Measure (TEAM) tool, is used during scenario teaching on the ALS Course. This tool is used as a teaching instrument and discussion point for both instructors and candidates throughout the face-to-face teaching and during teaching scenarios. (Figure 2.3).

The importance of communication when managing a sick patient

Communication problems are a factor in up to 80% of adverse incidents or near miss reports in hospitals. This failure of communication is also evident when a medical emergency occurs on a ward and a doctor or nurse summons senior help. The call for help is often suboptimal, with failure by the caller to communicate the seriousness of the situation and to convey information in a way that informs the recipient of the urgency of the situation. The poor quality information heightens the anxiety of the person responding to the call, who is then uncertain of the nature of the problem they are about to face. A well-structured process that is simple, reliable and dependable, will enable the caller to convey the important facts and urgency, and will help the recipient to plan ahead. It was for similar reasons that the ABCDE approach was developed as an aide memoire of the key technical skills required to manage a cardiac arrest.

Team Emergency Assessment Measure (TEAM)

Adapted and modified from Cooper et al (2010) to be used in ALS training courses

Leadership		Not seen (✓)	Observed (✓)
1	The team leader let the team know what was expected of them through direction and command. Examples: Uses members' names, allocates tasks, makes clear decisions.		
2	The team leader maintained a global perspective. Examples: Monitors clinical procedures, checks safety, plans ahead, remains 'hands off'.		
Teamwork			
3	The team communicated effectively, using both verbal and non-verbal communication. Examples: relay findings, raise concerns, use names, appropriate body language.		
4	The team worked together to complete tasks in a timely manner. Examples: coordination of defibrillation, maintain chest compressions, assist each other.		
5	The team acted with composure and control. Examples: performed allocated roles, accept criticism.		
6	The team adapted to changing situations. Examples: Adapt to rhythm changes, patient deterioration, change of roles.		
7	The team monitored and reassessed the situation Examples: rhythm changes, ROSC, when to terminate resuscitation.		
8	The team anticipated potential actions. Examples: defibrillation, airway management, drug delivery.		
Task management			
9	The team prioritised tasks. Examples: continuous chest compressions, defibrillation, airway management, drug delivery.		
10	The team followed approved standards/guidelines.		
Comments			
Examples: What area was good? What area needs improvement?			

Figure 2.3 Team Emergency Assessment Measure (TEAM) tool

SBAR	RSVP	Content	Example
SITUATION	REASON	<ul style="list-style-type: none"> Introduce yourself and check you are speaking to the correct person Identify the patient you are calling about (who and where) Say what you think the current problem is, or appears to be State what you need advice about Useful phrases: <ul style="list-style-type: none"> The problem appears to be cardiac/respiratory/neurological/sepsis I'm not sure what the problem is but the patient is deteriorating The patient is unstable, getting worse and I need help 	<ul style="list-style-type: none"> Hi, I'm Dr Smith the medical F2 I am calling about Mr Brown on acute medical admissions who I think has a severe pneumonia and is septic He has an oxygen saturation of 90% despite high-flow oxygen and I am very worried about him
BACKGROUND	STORY	<ul style="list-style-type: none"> Background information about the patient Reason for admission Relevant past medical history 	<ul style="list-style-type: none"> He is 55 and previously fit and well He has had fever and a cough for 2 days He arrived 15 minutes ago by ambulance
ASSESSMENT	VITAL SIGNS	<ul style="list-style-type: none"> Include specific observations and vital sign values based on ABCDE approach Airway Breathing Circulation Disability Exposure The early warning score is... 	<ul style="list-style-type: none"> He looks very unwell and is tiring Airway - he can say a few words Breathing - his respiratory rate is 24, he has bronchial breathing on the left side. His oxygen saturation is 90% on high-flow oxygen. I am getting a blood gas and chest X-ray Circulation - his pulse is 110, his blood pressure is 110/60 Disability - he is drowsy but can say a few words Exposure - he has no rashes
RECOMMENDATION	PLAN	<ul style="list-style-type: none"> State explicitly what you want the person you are calling to do What by when? Useful phrases: <ul style="list-style-type: none"> I am going to start the following treatment; is there anything else you can suggest? I am going to do the following investigations; is there anything else you can suggest? If they do not improve; when would you like to be called? I don't think I can do any more; I would like you to see the patient urgently 	<ul style="list-style-type: none"> I am getting antibiotics ready and he is on IV fluids I need help - please can you come and see him straight away

Table 2.1 SBAR (Situation, Background, Assessment, Recommendation) and RSVP (Reason, Story, Vital signs, Plan)

The use of the SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital signs, Plan) tool enables effective, timely communication between individuals from different clinical backgrounds and hierarchies (Table 2.1).

Resuscitation teams

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 to summon a team (e.g. medical emergency team) before cardiac arrest occurs (Chapter 3). The term resuscitation team reflects the range of response teams. As the team may change daily or more frequently, as shift pattern working is introduced, members may not know each other or the skill mix of the team members. The team should therefore meet at the beginning of their period on duty to:

- Introduce themselves; communication is much easier and more effective if people can be referred to by their name.
- Identify everyone's skills and experience.
- Allocate the team leader. Skill and experience takes precedence over seniority.
- Allocate responsibilities; if key skills are lacking (e.g. nobody skilled in tracheal intubation), work out how this deficit can be managed (Figure 2.4).
- Review any patients who have been identified as 'at risk' during the previous duty period.

Finally, every effort should be made to enable the team members to meet to debrief (e.g. difficulties or concerns about their performance, problems or concerns with equipment and submit incident reports). It may also be possible to carry out a formal handover to the incoming team.



Figure 2.4 Team briefing

Summary learning

- Non-technical skills are important during resuscitation.
- Use SBAR or RSVP for effective communication.

My key take-home messages from this chapter

Further reading

Andersen PO, Jensen MK, Lippert A, et al: Identifying non-technical skills and barriers for improvement of teamwork in cardiac arrest teams. Resuscitation 2010; 81:695–702.

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Acknowledgment

TEAM tool adapted and modified from Cooper et al (2010) to be used in ALS training courses. See <http://medicalemergencyteam.com/> for full details.

Recognising deterioration and preventing cardiorespiratory arrest

Chapter
3

Contents

- Prevention of in-hospital cardiac arrest: the Chain of Prevention
- Recognising the deteriorating patient
- Response to critical illness
- Causes of deterioration and cardiorespiratory arrest
- The ABCDE approach

Learning outcomes

To enable you to:

- Understand the importance of early recognition of the deteriorating patient
- Consider the relevant causes of cardiorespiratory arrest in adults
- Identify and treat patients at risk of cardiorespiratory arrest using the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach

Introduction

Early recognition of the deteriorating patient and prevention of cardiac arrest is the first link in the Chain of Survival. Once cardiac arrest occurs, about 20% of patients having an in-hospital cardiac arrest will survive to go home. 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.

Survival after in-hospital cardiac arrest is more likely if the arrest is witnessed and monitored, the rhythm is ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT), the primary cause is myocardial ischaemia, and the patient is successfully defibrillated immediately.

Most cardiorespiratory arrests in hospital are not sudden or unpredictable events: in approximately 80% of cases there is deterioration in clinical signs during the few hours before cardiac arrest. These patients often have slow and progressive physiological deterioration, particularly hypoxia and hypotension (i.e. Airway, Breathing, Circulation problems) that is unnoticed by staff, or is recognised but treated poorly. The cardiac arrest rhythm in this group is usually non-shockable (pulseless electrical activity (PEA) or asystole) and the survival rate to hospital discharge is very low.

Early recognition and effective treatment of the deteriorating patient might prevent cardiac arrest, death or an unanticipated intensive care unit (ICU) admission. Hospitals with the lowest incidence of in-hospital cardiac arrest have the highest cardiac arrest survival, perhaps suggesting better selection of patients suitable for cardiopulmonary resuscitation (CPR) or better prevention of cardiac arrest. Closer attention to patients who have a false cardiac arrest (i.e. a cardiac arrest team call when the patient has not had a cardiac arrest) may also improve outcome, because up to one-third of these patients die during their in-hospital stay. Early recognition will also help to

identify individuals for whom CPR is not appropriate or who do not wish to be resuscitated.

Prevention of in-hospital cardiac arrest: the Chain of Prevention

The Chain of Prevention can assist hospitals in structuring care processes to prevent and detect patient deterioration and cardiac arrest. The five rings of the chain represent: staff education; the monitoring of patients; the recognition of patient deterioration; a system to call for help; and an effective response (Figure 3.1):

- **Education:** how to observe patients; interpretation of observed signs; the recognition of signs of deterioration; and the use of the ABCDE approach; simple skills to stabilise the patient pending the arrival of more experienced help; knowledge of rationale for and role in the rapid response system in use in the hospital.
- **Monitoring:** patient assessment and the measurement and recording of vital signs, which may include the use of electronic monitoring and/or documentation devices.
- **Recognition** encompasses the tools available to identify patients in need of additional monitoring or intervention, including suitably designed vital signs charts and sets of predetermined ‘calling criteria’ to ‘flag’ the need to escalate monitoring or to call for more expert help.
- **Call for help** protocols for summoning a response to a deteriorating patient should be universally known and understood, unambiguous and mandated. Doctors and nurses often find it difficult to ask for help or escalate treatment as they feel their clinical judgement may be criticised. Hospitals should ensure all staff are empowered to call for help. A structured communication tool such as SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital signs, Plan) should be used to call for help.
- **Response** to a deteriorating patient must be assured, of specified speed and by staff with appropriate acute or critical care skills, and experience.

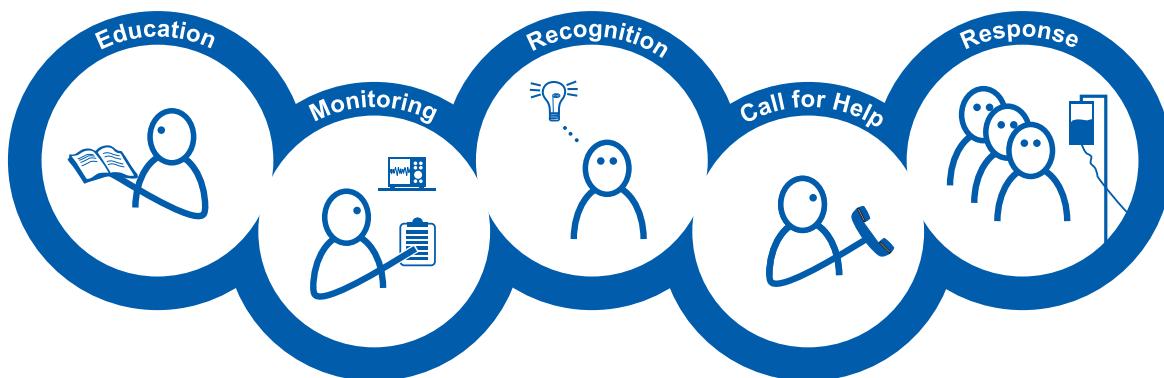


Figure 3.1 The Chain of Prevention

Recognising the deteriorating patient

In general, the clinical signs of critical illness are similar whatever the underlying process because they reflect failing respiratory, cardiovascular, and neurological systems (i.e. ABCDE problems) (see below). Abnormal physiology is common on general wards, yet the measurement and recording of important physiological observations of acutely ill patients occurs less frequently than is desirable, especially at night. The assessment of very simple vital signs, such as respiratory rate, may help to predict cardiorespiratory arrest. To help early detection of critical illness, many hospitals use early warning scores (EWS) or calling criteria. Early warning scoring systems allocate points to measurements of routine vital signs on the basis of their derangement from an arbitrarily agreed ‘normal’ range. The weighted score of one or more vital sign observations, or the total EWS, indicates the level of intervention required (e.g. increased frequency of vital signs monitoring, or the need to call ward doctors or resuscitation teams to the patient). In the UK, the National Early Warning Score is recommended (Table 3.1).

Early warning scores are dynamic and change over time and the frequency of observations should be increased to track improvement or deterioration in a patient’s condition. If it is clear a patient is deteriorating call for help early rather than waiting for the patient to reach a specific score.

The patient’s EWS is calculated based on Table 3.1. An increased score indicates an increased risk of deterioration and death. There should be a graded response to scores according to local hospital protocols (Table 3.2).

Alternatively, systems incorporating calling criteria are based on routine observations, which activate a response when one or more variables reach an extremely abnormal value. Recent research suggests that EWS may be better discriminators of outcomes than calling criteria. Some hospitals combine elements of both systems.

PHYSIOLOGICAL PARAMETERS	3	2	1	0	1	2	3
Respiration Rate	≤8		9 - 11	12 - 20		21 - 24	≥25
Oxygen Saturation	≤91	92 - 93	94 - 95	≥96			
Any Supplemental Oxygen		Yes		No			
Temperature	≤35.0		35.1 - 36.0	36.1 - 38.0	38.1 - 39.0	≥39.1	
Systolic BP	≤90	91 - 100	101 - 110	111 - 219			≥220
Heart Rate	≤40		41 - 50	51 - 90	91 - 110	111 - 130	≥131
Level of Consciousness				A			V, P, or U

Table 3.1 National Early Warning Score (NEWS)

Nurse concern may also be an important predictor of patient deterioration. Even when doctors are alerted to a patient's abnormal physiology, there is often delay in attending to the patient or referring to higher levels of care.

Response to critical illness

The traditional response to cardiac arrest is reactive: the name 'cardiac arrest team' implies that it will be called only after cardiac arrest has occurred. In many hospitals the role of the cardiac arrest team is incorporated into that of other teams (e.g. rapid response team, critical care outreach team, medical emergency team (MET)). These teams are usually activated according to the patient's EWS (see above) or according to specific calling criteria. For

example, the MET usually comprises medical and nursing staff from intensive care and acute medicine and responds to specific calling criteria (Table 3.3). Any member of the healthcare team, and in some cases the patient or their relatives, can initiate a MET call. Early involvement of the MET may reduce cardiac arrests, deaths and unanticipated ICU admissions, and may facilitate decisions about limitation of treatment (e.g. do not attempt cardiopulmonary resuscitation (DNACPR) decisions). Medical emergency team interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids. The benefits of the MET system remain to be proved, but evidence is increasing of their worth.

NEWS score	Minimum observation frequency	Clinical response
0	12 hourly	<ul style="list-style-type: none"> Continue routine NEWS monitoring with every set of observations.
1–4	4–6 hourly	<ul style="list-style-type: none"> Inform registered nurse, who must assess the patient. Registered nurse to decide if increased frequency of monitoring and/or escalation of clinical care is required.
5,6 or a score of 3 in any single variable	1 hourly	<ul style="list-style-type: none"> Registered nurse to urgently inform the medical team caring for the patient. Urgent assessment by a clinician with core competencies to assess acutely ill patients. Clinical care in a monitored setting.
≥7	Continuous monitoring of vital signs	<ul style="list-style-type: none"> Registered nurse to immediately inform the medical team caring for the patient – this should be at least at Specialist Registrar level. Emergency assessment by a clinical team with critical care competencies, which also includes a practitioner/s with advanced airway skills. Consider transfer of clinical care to a level 2 or 3 care facility (i.e. high dependency or critical care unit).

Table 3.2 Example escalation protocol based on early warning score (EWS)

Call MET if	
Acute change in:	Physiology:
Airway	Threatened
Breathing	All respiratory arrests Respiratory rate < 5 min ⁻¹ Respiratory rate > 36 min ⁻¹
Circulation	All cardiac arrests Pulse rate < 40 min ⁻¹ Pulse rate > 140 min ⁻¹ Systolic blood pressure < 90 mmHg
Neurology	Sudden decrease in level of consciousness Decrease in GCS of > 2 points Repeated or prolonged seizures
Other	Any patient causing concern who does not fit the above criteria

Table 3.3 Medical emergency team (MET) calling criteria

In the UK, a system of pre-emptive ward care known as critical care outreach, has developed. Outreach services exist in many forms ranging from a single nurse to a 24-hour, seven days per week multiprofessional team. An outreach team or system may reduce ward deaths, postoperative adverse events, ICU admissions and readmissions, and increase survival. Rapid response teams, such as outreach teams or METs, play a role in educating and improving acute care skills of ward personnel.

All critically ill patients should be admitted to an area that can provide the greatest supervision and the highest level of organ support and nursing care. This is usually in a critical care area (e.g. ICU, high dependency unit (HDU), or resuscitation room). These areas should be staffed by doctors and nurses experienced in advanced resuscitation and critical care skills.

Hospital staffing tends to be at its lowest during the night and at weekends. This influences patient monitoring, treatment and outcomes. Admission to general wards in the evening, or to hospital at weekends is associated with increased mortality. Studies have shown that in-hospital cardiac arrests occurring in the late afternoon, at night or at weekends are more often non-witnessed and have a lower survival rate. Patients discharged at night from ICUs to general wards have an increased risk of ICU readmission and in-hospital death compared with those discharged during the day and those discharged to HDUs.

Causes of deterioration and cardiorespiratory arrest

Deterioration and cardiorespiratory arrest can be caused by primary airway and/or breathing and/or cardiovascular problems.

Airway obstruction

For a detailed review of airway management see Chapter 7.

Causes

Airway obstruction can be complete or partial. Complete airway obstruction rapidly causes cardiac arrest. Partial obstruction often precedes complete obstruction. Partial airway obstruction can cause cerebral or pulmonary oedema, exhaustion, secondary apnoea, and hypoxic brain injury, and eventually cardiac arrest.

Central nervous system depression may cause loss of airway patency and protective reflexes. Causes include head injury and intracerebral disease, hypercarbia, the depressant effect of metabolic disorders (e.g. diabetes mellitus), and drugs, including alcohol, opioids and general anaesthetics. Laryngospasm can occur with upper airway stimulation in a semi-conscious patient whose airway reflexes remain intact.

In some people, the upper airway can become obstructed when asleep (obstructive sleep apnoea). This is more common in obese patients and obstruction can be worsened in the presence of other factors (e.g. sedative drugs).

Causes of airway obstruction

- Central nervous system depression
- Blood
- Vomitus
- Foreign body (e.g. tooth, food)
- Direct trauma to face or throat
- Epiglottitis
- Pharyngeal swelling (e.g. infection, oedema)
- Laryngospasm
- Bronchospasm – causes narrowing of the small airways in the lung
- Bronchial secretions
- Blocked tracheostomy

Recognition

Assess the patency of the airway in anyone at risk of obstruction. A conscious patient will complain of difficulty in breathing, may be choking, and will be distressed. With partial airway obstruction, efforts at breathing will be noisy. Complete airway obstruction is silent and there is no air movement at the patient's mouth. Any respiratory movements are usually strenuous. The accessory muscles of respiration will be involved, causing a 'see-saw' or 'rocking-horse' pattern of chest and abdominal movement: the chest is drawn in and the abdomen expands on inspiration, and the opposite occurs on expiration.

Treatment

The priority is to ensure that the airway remains patent. Treat any problem that places the airway at risk; for example, suck blood and gastric contents from the airway and, unless contraindicated, turn the patient on their side. Give oxygen as soon as possible to achieve an arterial blood oxygen saturation by pulse oximetry (SpO_2) in the range of 94–98%. Assume actual or impending airway obstruction in anyone with a depressed level of consciousness, regardless of cause. Take steps to safeguard the airway and prevent further complications such as aspiration of gastric contents. This may involve nursing the patient on their side or with a head-up tilt. Simple airway opening manoeuvres (head tilt/chin lift or jaw thrust), insertion of an oropharyngeal or nasal airway, elective tracheal intubation or tracheostomy may be required. Consider insertion of a nasogastric tube to empty the stomach.

Breathing problems

Causes

Breathing inadequacy may be acute or chronic. It may be continuous or intermittent, and severe enough to cause apnoea (respiratory arrest), which will rapidly cause cardiac arrest. Respiratory arrest often occurs because of a combination of factors; for example, in a patient with chronic respiratory inadequacy, a chest infection, muscle weakness, or fractured ribs can lead to exhaustion, further depressing respiratory function. If breathing is insufficient to oxygenate the blood adequately (hypoxaemia), a cardiac arrest will occur eventually.

Respiratory drive

Central nervous system depression may decrease or abolish respiratory drive. The causes are the same as those for airway obstruction from central nervous system depression.

Respiratory effort

The main respiratory muscles are the diaphragm and intercostal muscles. The latter are innervated at the level of their respective ribs and may be paralysed by a spinal cord lesion above this level. The innervation of the diaphragm is at the level of the third, fourth and fifth segment of the cervical cord. Spontaneous breathing cannot occur with severe cervical cord damage above this level.

Inadequate respiratory effort, caused by muscle weakness or nerve damage, occurs with many diseases (e.g. myasthenia gravis, Guillain-Barré syndrome, and multiple sclerosis). Chronic malnourishment and severe long-term illness may also contribute to generalised weakness.

Breathing can also be impaired with restrictive chest wall abnormalities such as kyphoscoliosis. Pain from fractured ribs or sternum will prevent deep breaths and coughing.

Lung disorders

Lung function is impaired by a pneumothorax or haemothorax. A tension pneumothorax causes a rapid

failure of gas exchange, a reduction of venous return to the heart, and a fall in cardiac output. Severe lung disease will impair gas exchange. Causes include infection, aspiration, exacerbation of chronic obstructive pulmonary disease (COPD), asthma, pulmonary embolus, lung contusion, acute respiratory distress syndrome (ARDS) and pulmonary oedema.

Recognition

A conscious patient will complain of shortness of breath and be distressed. The history and examination will usually indicate the underlying cause. Hypoxaemia and hypercarbia can cause irritability, confusion, lethargy and a decrease in the level of consciousness. Cyanosis may be visible but is a late sign. A fast respiratory rate ($> 25 \text{ min}^{-1}$) is a useful, simple indicator of breathing problems. Pulse oximetry is an easy, non-invasive measure of the adequacy of oxygenation (Chapter 15). However, it is not a reliable indicator of ventilation and an arterial blood gas sample is necessary to obtain values for arterial carbon dioxide tension (PaCO_2) and pH. A rising PaCO_2 and a decrease in pH are often late signs in a patient with severe respiratory problems.

Treatment

Give oxygen to all acutely ill hypoxaemic patients and treat the underlying cause. Give oxygen at 15 L min^{-1} using a high-concentration reservoir mask. Once the patient is stable, change the oxygen mask and aim for a SpO_2 in the range of 94–98%. For example, suspect a tension pneumothorax from a history of chest trauma and confirm by clinical signs and symptoms. Early needle decompression (thoracocentesis), or thoracostomy, followed by chest drain insertion is needed (Chapter 12).

Patients who are having difficulty breathing or are becoming tired will need respiratory support. Non-invasive ventilation using a face mask or a helmet can be useful and prevent the need for tracheal intubation and ventilation. For patients who cannot breathe adequately, sedation, tracheal intubation and controlled ventilation is needed.

Circulation problems

Causes

For a detailed review of cardiac causes of cardiac arrest see Chapter 4.

Circulation problems may be caused by primary heart disease or by heart abnormalities secondary to other problems. Most often, circulation problems in acutely ill patients are due to hypovolaemia. The heart may stop suddenly or may produce an inadequate cardiac output for a period of time before stopping.

Primary heart problems

The commonest cause of sudden cardiac death (SCD) is an arrhythmia caused by either ischaemia or myocardial infarction. Cardiac arrest can also be caused by an arrhythmia due to other forms of heart disease, by heart block, electrocution and some drugs.

Sudden cardiac arrest may also occur with cardiac failure, cardiac tamponade, cardiac rupture, myocarditis and hypertrophic cardiomyopathy.

Causes of ventricular fibrillation

- Acute coronary syndromes (Chapter 4)
- Hypertensive heart disease
- Valve disease
- Drugs (e.g. antiarrhythmic drugs, tricyclic antidepressants, digoxin)
- Inherited cardiac diseases (e.g. long QT syndromes)
- Acidosis
- Abnormal electrolyte concentration (e.g. potassium, magnesium, calcium)
- Hypothermia
- Electrocution

Secondary heart problems

The heart is affected by changes elsewhere in the body. For example, cardiac arrest will occur rapidly following asphyxia from airway obstruction or apnoea, tension pneumothorax, or acute severe blood loss. Severe hypoxia and anaemia, hypothermia, oligaemia and severe septic shock will also impair cardiac function and this may lead to cardiac arrest.

Recognition

The signs and symptoms of cardiac disease include chest pain, shortness of breath, syncope, tachycardia, bradycardia, tachypnoea, hypotension, poor peripheral perfusion (prolonged capillary refill time), altered mental state, and oliguria.

Although the risk of SCD is greater for patients with known severe cardiac disease, most SCJs occur in people with unrecognised disease. Asymptomatic or silent cardiac disease may include hypertensive heart disease, aortic valve disease, cardiomyopathy, myocarditis, and coronary disease.

Recognition of risk 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 SCJs 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 as an independent risk factor for increased risk of death. Apparently healthy children and young adults who have 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 (<http://guidance.nice.org.uk/CG109>).

Treatment

Treat the underlying cause of circulatory failure. In many sick patients, this means giving intravenous fluids to treat hypovolaemia. Assess patients with chest pain for an acute coronary syndrome (ACS). A comprehensive description of the management of ACS is given in Chapter 4. Most patients with cardiac ischaemic pain will be more comfortable sitting up. In some instances lying flat may provoke or worsen the pain. Consider using an anti-emetic, especially if nausea is present.

Survivors of an episode of VF are likely to have a further episode unless preventative treatment is given. These patients may need percutaneous coronary intervention, coronary artery bypass grafting, or an implantable defibrillator.

Treating the underlying cause should prevent many secondary cardiac arrests; for example, early goal-directed therapy to optimise vital organ perfusion decreases the risk of death in severe sepsis. Cardiovascular support includes correction of underlying electrolyte or acid-base disturbances, and treatment to achieve a desirable cardiac rate, rhythm and output. Advanced cardiovascular monitoring and echocardiography may be indicated. Appropriate manipulation of cardiac filling may require fluid therapy and vasoactive drugs. Inotropic drugs and vasoconstrictors may be indicated to support cardiac output and blood pressure.

The ABCDE approach

Underlying principles

The approach to all deteriorating or critically ill patients is the same. The underlying principles are:

1. Use the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach to assess and treat the patient.
2. Do a complete initial assessment and re-assess regularly.
3. Treat life-threatening problems before moving to the next part of assessment.
4. Assess the effects of treatment.
5. Recognise when you will need extra help. Call for appropriate help early.
6. Use all members of the team. This enables interventions (e.g. assessment, attaching monitors, intravenous access), to be undertaken simultaneously.
7. Communicate effectively - use the SBAR or RSVP approach (Chapter 2).
8. The aim of the initial treatment is to keep the patient alive, and achieve some clinical improvement. This will buy time for further treatment and making a diagnosis.
9. Remember – it can take a few minutes for treatments to work, so wait a short while before reassessing the patient after an intervention.

First steps

1. Ensure personal safety. Wear apron and gloves as appropriate.
2. First look at the patient in general to see if the patient appears unwell.
3. If the patient is awake, ask “How are you?”. If the patient appears unconscious or has collapsed, shake him and ask “Are you alright?” If he responds normally he has a patent airway, is breathing and has brain perfusion. If he speaks only in short sentences, he may have breathing problems. Failure of the patient to respond is a clear marker of critical illness.
4. This first rapid ‘Look, Listen and Feel’ of the patient should take about 30 s and will often indicate a patient is critically ill and there is a need for urgent help. Ask a colleague to ensure appropriate help is coming.
5. If the patient is unconscious, unresponsive, and is not breathing normally (occasional gasps are not normal) start CPR according to the guidance in Chapter 5. If you are confident and trained to do so, feel for a pulse to determine if the patient has a respiratory arrest. If there are any doubts about the presence of a pulse start CPR.
6. Monitor the vital signs early. Attach a pulse oximeter, ECG monitor and a non-invasive blood pressure monitor to all critically ill patients, as soon as possible.
7. Insert an intravenous cannula as soon as possible. Take bloods for investigation when inserting the intravenous cannula.

Airway (A)

Airway obstruction is an emergency. Get expert help immediately. Untreated, airway obstruction causes hypoxia and risks damage to the brain, kidneys and heart, cardiac arrest, and death.

1. Look for the signs of airway obstruction:
 - Airway obstruction causes paradoxical chest and abdominal movements ('see-saw' respirations) and the use of the accessory muscles of respiration. Central cyanosis is a late sign of airway obstruction. In complete airway obstruction, there are no breath sounds at the mouth or nose. In partial obstruction, air entry is diminished and often noisy.
 - In the critically ill patient, depressed consciousness often leads to airway obstruction.
2. Treat airway obstruction as a medical emergency:
 - Obtain expert help immediately. Untreated, airway obstruction causes hypoxaemia (low PaO_2) with the risk of hypoxic injury to the brain, kidneys and heart, cardiac arrest, and even death.
 - In most cases, only simple methods of airway clearance are required (e.g. airway opening manoeuvres, airways suction, insertion of an oropharyngeal or nasopharyngeal airway). Tracheal intubation may be required when these fail.
3. Give oxygen at high concentration:
 - Provide high-concentration oxygen using a mask with oxygen reservoir. Ensure that the oxygen flow is sufficient (usually 15 L min^{-1}) to prevent collapse of the reservoir during inspiration. If the patient's trachea is intubated, give high concentration oxygen with a self-inflating bag.
 - In acute respiratory failure, aim to maintain an oxygen saturation of 94–98%. In patients at risk of hypercapnic respiratory failure (see below) aim for an oxygen saturation of 88–92%.

Breathing (B)

During the immediate assessment of breathing, it is vital to diagnose and treat immediately life-threatening conditions (e.g. acute severe asthma, pulmonary oedema, tension pneumothorax, and massive haemothorax).

1. Look, listen and feel for the general signs of respiratory distress: sweating, central cyanosis, use of the accessory muscles of respiration, and abdominal breathing.
2. Count the respiratory rate. The normal rate is $12\text{--}20 \text{ breaths min}^{-1}$. A high ($> 25 \text{ min}^{-1}$) or increasing respiratory rate is a marker of illness and a warning that the patient may deteriorate suddenly.
3. Assess the depth of each breath, the pattern (rhythm) of respiration and whether chest expansion is equal on both sides.

4. Note any chest deformity (this may increase the risk of deterioration in the ability to breathe normally); look for a raised jugular venous pulse (JVP) (e.g. in acute severe asthma or a tension pneumothorax); note the presence and patency of any chest drains; remember that abdominal distension may limit diaphragmatic movement, thereby worsening respiratory distress.
5. Record the inspired oxygen concentration (%) and the SpO₂ reading of the pulse oximeter. The pulse oximeter does not detect hypercapnia. If the patient is receiving supplemental oxygen, the SpO₂ may be normal in the presence of a very high PaCO₂.
6. Listen to the patient's breath sounds a short distance from his face: rattling airway noises indicate the presence of airway secretions, usually caused by the inability of the patient to cough sufficiently or to take a deep breath. Stridor or wheeze suggests partial, but significant, airway obstruction.
7. Percuss the chest: hyper-resonance may suggest a pneumothorax; dullness usually indicates consolidation or pleural fluid.
8. Auscultate the chest: bronchial breathing indicates lung consolidation with patent airways; absent or reduced sounds suggest a pneumothorax or pleural fluid or lung consolidation caused by complete obstruction.
9. Check the position of the trachea in the suprasternal notch: deviation to one side indicates mediastinal shift (e.g. pneumothorax, lung fibrosis or pleural fluid).
10. Feel the chest wall to detect surgical emphysema or crepitus (suggesting a pneumothorax until proven otherwise).
11. The specific treatment of respiratory disorders depends upon the cause. Nevertheless, all critically ill patients should be given oxygen. In a subgroup of patients with COPD, high concentrations of oxygen may depress breathing (i.e. they are at risk of hypercapnic respiratory failure - often referred to as type 2 respiratory failure). Nevertheless, these patients will also sustain end-organ damage or cardiac arrest if their blood oxygen tensions are allowed to decrease. In this group, aim for a lower than normal PaO₂ and oxygen saturation. Give oxygen via a Venturi 28% mask (4 L min⁻¹) or a 24% Venturi mask (4 L min⁻¹) initially and reassess. Aim for target SpO₂ range of 88–92% in most COPD patients, but evaluate the target for each patient based on the patient's arterial blood gas measurements during previous exacerbations (if available). Some patients with chronic lung disease carry an oxygen alert card (that documents their target saturation) and their own appropriate Venturi mask.
12. If the patient's depth or rate of breathing is judged to be inadequate, or absent, use bag-mask or pocket mask ventilation to improve oxygenation and ventilation, whilst calling immediately for expert help. In cooperative patients who do not have airway

obstruction consider the use of non-invasive ventilation (NIV). In patients with an acute exacerbation of COPD, the use of NIV is often helpful and prevents the need for tracheal intubation and invasive ventilation.

Circulation (C)

In almost all medical and surgical emergencies, consider hypovolaemia to be the primary cause of shock, until proven otherwise. Unless there are obvious signs of a cardiac cause, give intravenous fluid to any patient with cool peripheries and a fast heart rate. In surgical patients, rapidly exclude haemorrhage (overt or hidden). Remember that breathing problems, such as a tension pneumothorax, can also compromise a patient's circulatory state. This should have been treated earlier on in the assessment.

1. Look at the colour of the hands and digits: are they blue, pink, pale or mottled?
2. Assess the limb temperature by feeling the patient's hands: are they cool or warm?
3. Measure the capillary refill time (CRT). Apply cutaneous pressure for 5 s on a fingertip held at heart level (or just above) with enough pressure to cause blanching. Time how long it takes for the skin to return to the colour of the surrounding skin after releasing the pressure. The normal value for CRT is usually < 2 s. A prolonged CRT suggests poor peripheral perfusion. Other factors (e.g. cold surroundings, poor lighting, old age) can prolong CRT.
4. Assess the state of the veins: they may be under-filled or collapsed when hypovolaemia is present.
5. Count the patient's pulse rate (or preferably heart rate by listening to the heart with a stethoscope).
6. Palpate peripheral and central pulses, assessing for presence, rate, quality, regularity and equality. Barely palpable central pulses suggest a poor cardiac output, whilst a bounding pulse may indicate sepsis.
7. Measure the patient's blood pressure. Even in shock, the blood pressure may be normal, because compensatory mechanisms increase peripheral resistance in response to reduced cardiac output. A low diastolic blood pressure suggests arterial vasodilation (as in anaphylaxis or sepsis). A narrowed pulse pressure (difference between systolic and diastolic pressures; normally 35–45 mmHg) suggests arterial vasoconstriction (cardiogenic shock or hypovolaemia) and may occur with rapid tachyarrhythmia.
8. Auscultate the heart. Is there a murmur or pericardial rub? Are the heart sounds difficult to hear? Does the audible heart rate correspond to the pulse rate?
9. Look for other signs of a poor cardiac output, such as reduced conscious level and, if the patient has a urinary catheter, oliguria (urine volume < 0.5 mL kg⁻¹ h⁻¹).

10. Look thoroughly for external haemorrhage from wounds or drains or evidence of concealed haemorrhage (e.g. thoracic, intra-peritoneal, retroperitoneal or into gut). Intra-thoracic, intra-abdominal or pelvic blood loss may be significant, even if drains are empty.
11. The specific treatment of cardiovascular collapse depends on the cause, but should be directed at fluid replacement, haemorrhage control and restoration of tissue perfusion. Seek the signs of conditions that are immediately life threatening (e.g. cardiac tamponade, massive or continuing haemorrhage, septicaemic shock), and treat them urgently.
12. Insert one or more large (14 or 16 G) intravenous cannulae. Use short, wide-bore cannulae, because they enable the highest flow.
13. Take blood from the cannula for routine haematological, biochemical, coagulation and microbiological investigations, and cross-matching, before infusing intravenous fluid.
14. Give a bolus of 500 mL of warmed crystalloid solution (e.g. Hartmann's solution or 0.9% sodium chloride) over less than 15 min if the patient is hypotensive. Use smaller volumes (e.g. 250 mL) for patients with known cardiac failure or trauma and use closer monitoring (listen to the chest for crackles after each bolus).
15. Reassess the heart rate and BP regularly (every 5 min), aiming for the patient's normal BP or, if this is unknown, a target > 100 mmHg systolic.
16. If the patient does not improve, repeat the fluid challenge. Seek expert help if there is a lack of response to repeated fluid boluses.
17. If symptoms and signs of cardiac failure (dyspnoea, increased heart rate, raised JVP, a third heart sound and pulmonary crackles on auscultation) occur, decrease the fluid infusion rate or stop the fluids altogether. Seek alternative means of improving tissue perfusion (e.g. inotropes or vasopressors).
18. If the patient has primary chest pain and a suspected ACS, record a 12-lead ECG early.
19. Immediate general treatment for ACS includes:
 - Aspirin 300 mg, orally, crushed or chewed, as soon as possible.
 - Nitroglycerine, as sublingual glyceryl trinitrate (tablet or spray).
 - Oxygen: only give oxygen if the patient's SpO₂ is less than 94% breathing air alone.
 - Morphine (or diamorphine) titrated intravenously to avoid sedation and respiratory depression.

Disability (D)

Common causes of unconsciousness include profound hypoxia, hypercapnia, cerebral hypoperfusion, or the recent administration of sedatives or analgesic drugs.

1. Review and treat the ABCs: exclude or treat hypoxia and hypotension.
2. Check the patient's drug chart for reversible drug-induced causes of depressed consciousness. Give an antagonist where appropriate (e.g. naloxone for opioid toxicity).
3. Examine the pupils (size, equality and reaction to light).
4. Make a rapid initial assessment of the patient's conscious level using the AVPU method: Alert, responds to Vocal stimuli, responds to Painful stimuli or Unresponsive to all stimuli. Alternatively, use the Glasgow Coma Scale score. A painful stimuli can be given by applying supra-orbital pressure (at the supraorbital notch).
5. Measure the blood glucose to exclude hypoglycaemia using a rapid finger-prick bedside testing method. In a peri-arrest patient use a venous or arterial blood sample for glucose measurement as finger prick sample glucose measurements can be unreliable in sick patients. Follow local protocols for management of hypoglycaemia. For example, if the blood sugar is less than 4.0 mmol L⁻¹ in an unconscious patient, give an initial dose of 50 mL of 10% glucose solution intravenously. If necessary, give further doses of intravenous 10% glucose every minute until the patient has fully regained consciousness, or a total of 250 mL of 10% glucose has been given. Repeat blood glucose measurements to monitor the effects of treatment. If there is no improvement consider further doses of 10% glucose. Specific national guidance exists for the management of hypoglycaemia in adults with diabetes mellitus.
6. Nurse unconscious patients in the lateral position if their airway is not protected.

Exposure (E)

To examine the patient properly full exposure of the body may be necessary. Respect the patient's dignity and minimise heat loss.

Additional information

1. Take a full clinical history from the patient, any relatives or friends, and other staff.
2. Review the patient's notes and charts:
 - Study both absolute and trended values of vital signs.
 - Check that important routine medications are prescribed and being given.
3. Review the results of laboratory or radiological investigations.
4. Consider which level of care is required by the patient (e.g. ward, HDU, ICU).
5. Make complete entries in the patient's notes of your findings, assessment and treatment. Where necessary, hand over the patient to your colleagues.
6. Record the patient's response to therapy.
7. Consider definitive treatment of the patient's underlying condition.

Summary learning

- Most patients who have an in-hospital cardiac arrest have warning signs and symptoms before the arrest.
- Early recognition and treatment of the deteriorating patient will prevent some cardiorespiratory arrests.
- Use strategies such as early warning scoring (EWS) systems to identify patients at risk of cardiorespiratory arrest.
- Airway, breathing and circulation problems can cause cardiorespiratory arrest.
- Use the ABCDE approach to assess and treat critically ill patients.

My key take-home messages from this chapter

Further reading

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Cardiac causes of cardiac arrest

Chapter
4

Contents

- Recognition and treatment of acute coronary syndromes and the other conditions that may lead to cardiac arrest
- Recognition of the features and events that may indicate high risk and interventions to prevent progression to cardiac arrest

Learning outcomes

To enable you to:

- Recognise and assess an acute coronary syndrome
- Understand the disease process which causes acute coronary syndromes
- Consider the immediate treatment of each type of acute coronary syndrome
- Consider the treatment needed after recovery from an acute coronary syndrome
- Recognise and respond to other cardiac conditions that may cause cardiac arrest

Introduction

Whilst rapid resuscitation offers the best chance of recovery from cardiac arrest, it is clearly better to prevent cardiac arrest whenever possible. Many cardiac arrests are caused by coronary artery disease (CAD) and occur most commonly in the context of an acute coronary syndrome (ACS). It is therefore important that you:

- understand how to recognise an ACS
- understand how to assess a patient with an ACS
- know what treatments may reduce the risk of cardiac arrest and death
- know what actions are needed to achieve the best outcome when a person has a cardiac arrest as the presenting feature of a suspected ACS
- are aware of some of the other cardiac conditions that may cause cardiac arrest, and how to recognise and treat them to reduce the risk of cardiac arrest and death.

Acute coronary syndromes

Definitions and pathogenesis

The term acute coronary syndromes describes a group of clinical conditions, all of which usually present with chest pain or discomfort resulting from myocardial ischaemia. The distinct categories of ACS are distinguished initially by the presence or absence of ST-segment elevation on a 12-lead ECG and, in those without ST-segment elevation, by the presence or absence of a raised blood troponin concentration suggesting myocardial injury (Figure 4.1):

- ST-segment-elevation myocardial infarction (STEMI)
- Non-ST-segment-elevation acute coronary syndromes (NSTE ACS)
 - Non-ST-segment-elevation myocardial infarction (NSTEMI)
 - Unstable angina (UA).

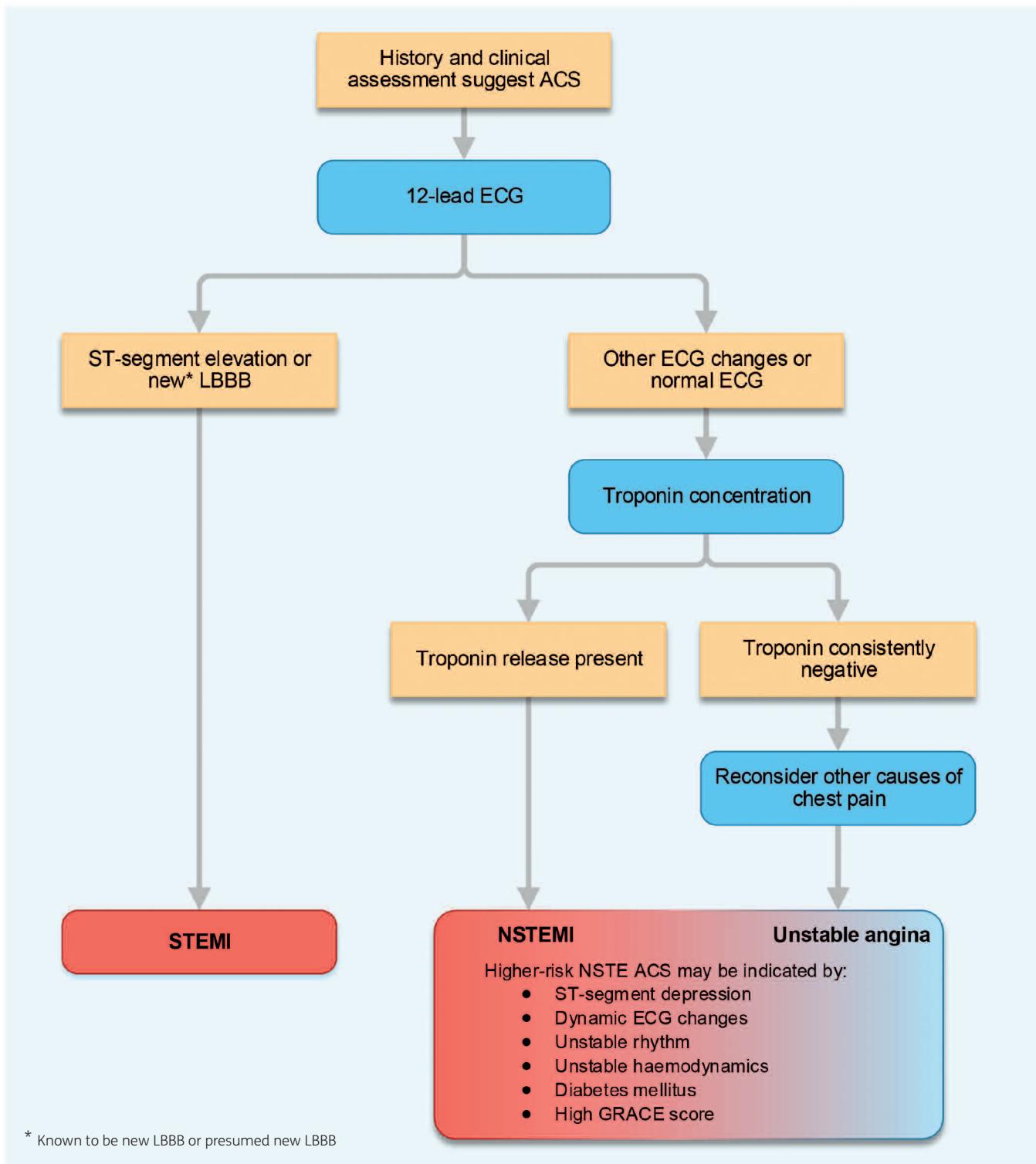


Figure 4.1 Algorithm for identifying the type of acute coronary syndrome

STEMI – ST-segment-elevation myocardial infarction; **NSTE ACS** – Non-ST-segment-elevation acute coronary syndromes; **NSTEMI** – Non-ST-segment-elevation myocardial infarction; **UA** – Unstable angina; **GRACE** – Global Registry of Acute Coronary Events

These syndromes form part of a spectrum of the same disease process. In most cases this results from coronary artery disease and is initiated by the rupture or erosion of an atherosclerotic plaque within a coronary artery causing:

- acute thrombosis within the vessel lumen, often with haemorrhagic extension into the atherosclerotic plaque
- contraction of smooth muscle cells within the artery wall resulting in vasoconstriction that reduces the lumen of the artery
- associated partial or complete obstruction of the lumen, often with embolism of thrombus into the distal part of the vessel.

This process results in a sudden and critical reduction in blood flow to the myocardium. The degree and extent of this reduction in blood flow to the myocardium largely determines the clinical presentation and type of ACS.

Coronary artery disease

Coronary artery disease (CAD) is one of the leading causes of premature death in the world. It is characterised by accumulation of atherosclerotic plaques within the coronary arteries. CAD is a chronic process that can begin during adolescence and progress slowly throughout life. Risk factors for CAD include a family history of atherosclerosis, smoking, diabetes mellitus, hypertension, hyperlipidemia, sedentary lifestyle and obesity. CAD is believed to be an abnormal response to vessel injury and these risk factors are thought to accelerate the inflammatory process that results in atherosclerosis. Over time atherosclerotic plaques can narrow the coronary artery lumen progressively. For many years such developing atherosclerosis may cause no symptoms. However, if the plaque starts to impair the blood flow to the myocardium angina can occur. If a thrombus (blood clot) forms at the site of atherosclerosis myocardial infarction may result.

Angina (stable and unstable)

Typical stable angina is a constricting pain, tightness or discomfort in the front of the chest, and/or in the neck, shoulders, jaw, or arms that is provoked by physical exertion and relieved by stopping that exertion. It is usually caused by myocardial ischaemia, most commonly due to obstructive coronary artery disease.

Recognising stable angina is not always straightforward as some patients present with less typical symptoms. Patients may present with clear central chest pain or tightness but they may also report less typical discomfort, including symptoms that feel like indigestion. As with acute myocardial infarction (AMI), the pain/discomfort may radiate to the throat, into one or both arms, and into the back or upper abdomen. Some patients experience angina predominantly in one or more of these areas without chest pain. Some groups of people (including females, diabetics and the elderly) are more likely to present with atypical or less-specific symptoms such as breathlessness. Ischaemic chest pain (due to angina or AMI) may be misdiagnosed as

indigestion or non-cardiac chest pain. Symptoms of this nature, which are provoked only by exercise, which settle promptly when exercise ceases and do not occur at rest are referred to as stable angina. Although stable angina needs correct recognition and appropriate management it is not a medical emergency.

In contrast, unstable angina is defined by one or more of:

1. Angina on exertion, occurring with increasing frequency over a few days, provoked by progressively less exertion. This is sometimes referred to as 'crescendo angina'.
2. Episodes of angina-like pain occurring recurrently and unpredictably, without specific provocation by exercise. These episodes may be relatively short-lived (e.g. a few minutes) and may settle spontaneously or be relieved temporarily by sublingual glyceryl trinitrate, before recurring again.
3. An unprovoked and prolonged episode of chest pain, raising suspicion of AMI, but without definite ECG changes or laboratory evidence of AMI (see below).

In unstable angina (UA) the ECG may:

- a) be normal
- b) show evidence of acute myocardial ischaemia (usually ST-segment depression)
- c) show non-specific abnormalities (e.g. T-wave inversion).

In UA, by definition, biomarkers for myocardial necrosis (i.e. troponin release) are not present. The use of high-sensitivity troponin assays has resulted in a diagnosis of NSTEMI in many patients who would previously have been considered to have UA. Although the ECG may be normal or show non-specific features, ECG abnormalities (such as ST-segment depression) are a marker of increased risk. However, a normal ECG and an undetectable troponin level do not necessarily mean that an individual patient with UA is at low risk. Therefore all patients with UA should have their risk assessed using an established risk calculator, such as the Global Registry of Acute Coronary Events (GRACE) score (see below).

If the ECG and troponin concentration are normal and the estimated risk is low, patients can be considered for further risk assessment with non-invasive tests (e.g. exercise testing or non-invasive imaging) and other possible causes for their chest pain can be considered. In some patients who present with angina-like symptoms coronary artery disease may later be excluded as the cause of those symptoms.

Non-ST-segment-elevation myocardial infarction (NSTEMI)

Acute myocardial infarction presents typically with chest pain that is felt as a heaviness or tightness or indigestion-like discomfort in the chest or upper abdomen, usually sustained for at least 20 min. The chest pain/discomfort

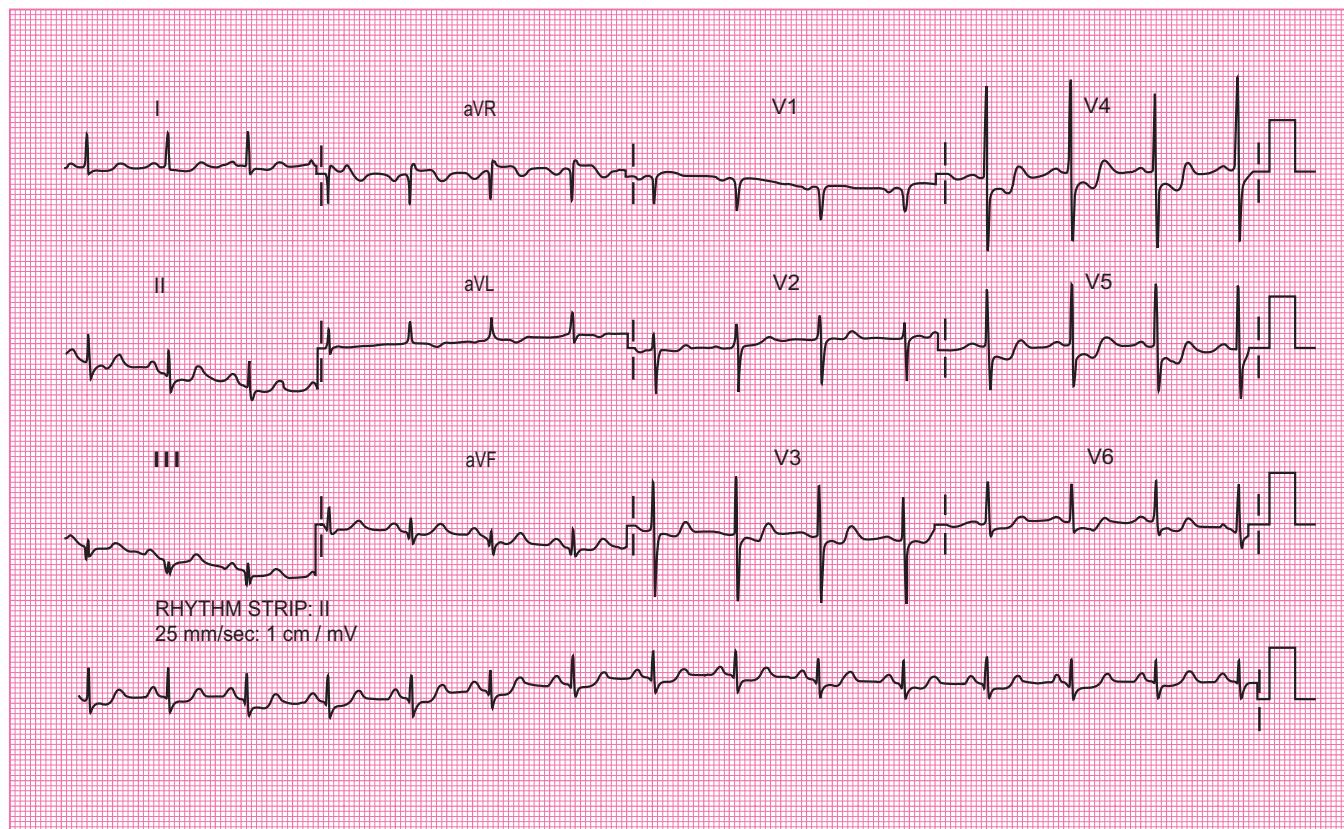


Figure 4.2 ECG showing acute ST-segment depression caused by myocardial ischaemia in a patient with a non-ST-segment-elevation ACS

often radiates into the throat, into one or both arms, into the back or into the upper abdomen. Some patients experience the discomfort predominantly in one or more of these other areas rather than in the chest. The pain is usually accompanied by sweating; belching, nausea or vomiting may occur also. As with angina, some people with AMI experience less typical symptoms, such as acute breathlessness. A small proportion of people with AMI experience no symptoms.

When a patient presents with chest pain suggestive of AMI and has raised biomarkers (e.g. troponin) but has no ST segment elevation on the ECG this is referred to as NSTEMI. The 12-lead ECG may be normal, show ST-segment depression or non-specific ECG abnormalities such as T-wave inversion (Figures 4.2 and 4.3). ECG changes are associated with increased risk. The confirmation of the release of cardiac-specific troponin indicates that myocardial damage has occurred (in contrast to unstable angina which is not associated with troponin release). In this situation it is likely that there has been partial or intermittent occlusion of the ‘culprit’ coronary artery.

The amount of troponin released reflects the amount of myocardium damaged. Some patients with NSTEMI will be at high risk of progression to complete coronary occlusion, further myocardial infarction and sudden arrhythmic death. The risk of this is highest in the first few hours, days

and months after the index event and diminishes progressively with time. The need for further treatment is largely determined by assessment of risk.

NSTEMI and unstable angina are classified together as ‘non-ST-segment-elevation ACS (NSTE ACS)’ because the treatment of the two is essentially the same. This is in contrast to the treatment of STEMI where immediate reperfusion is a major priority.

ST-segment-elevation myocardial infarction (STEMI)

A history of sustained acute chest pain typical of AMI, accompanied by acute ST-segment elevation (Figures 4.4, 4.5, 4.6) or new left bundle branch block (LBBB) on a 12-lead ECG, is the basis for the diagnosis of STEMI. These findings indicate on-going myocardial damage, typically caused by acute, complete, thrombotic occlusion of the ‘culprit’ coronary artery. Left untreated there will be further myocardial damage in the territory of the occluded artery. This may be evidenced by the development of Q waves on the ECG and by impairment of left ventricular function (seen on echocardiography). During the acute phase of STEMI there is a substantial risk of ventricular tachycardia (VT), ventricular fibrillation (VF) (Figure 4.7) and sudden death. Confirmation of an elevated troponin is not required to make the diagnosis of STEMI or to ensure that the patient receives appropriate treatment without delay.

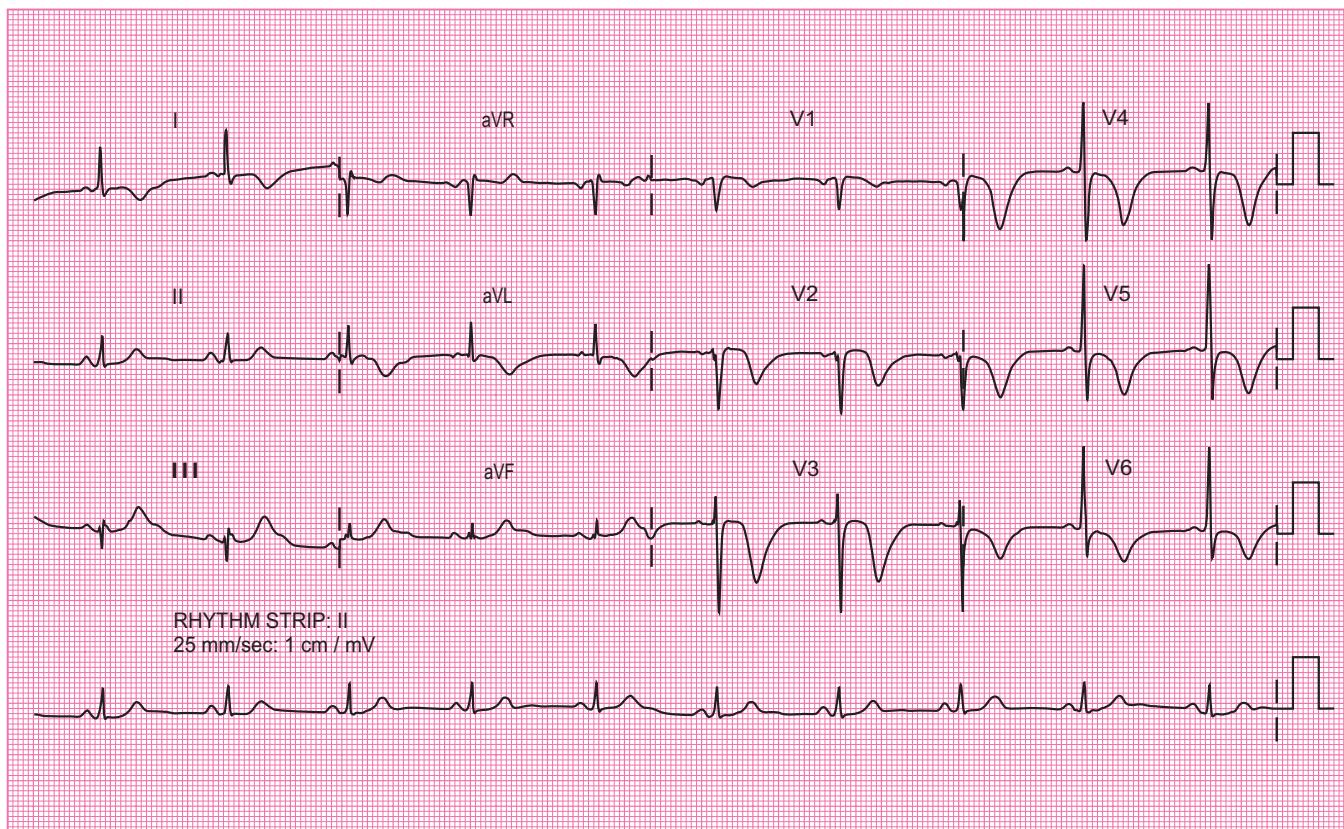


Figure 4.3 ECG showing T wave inversion in a patient with NSTEMI

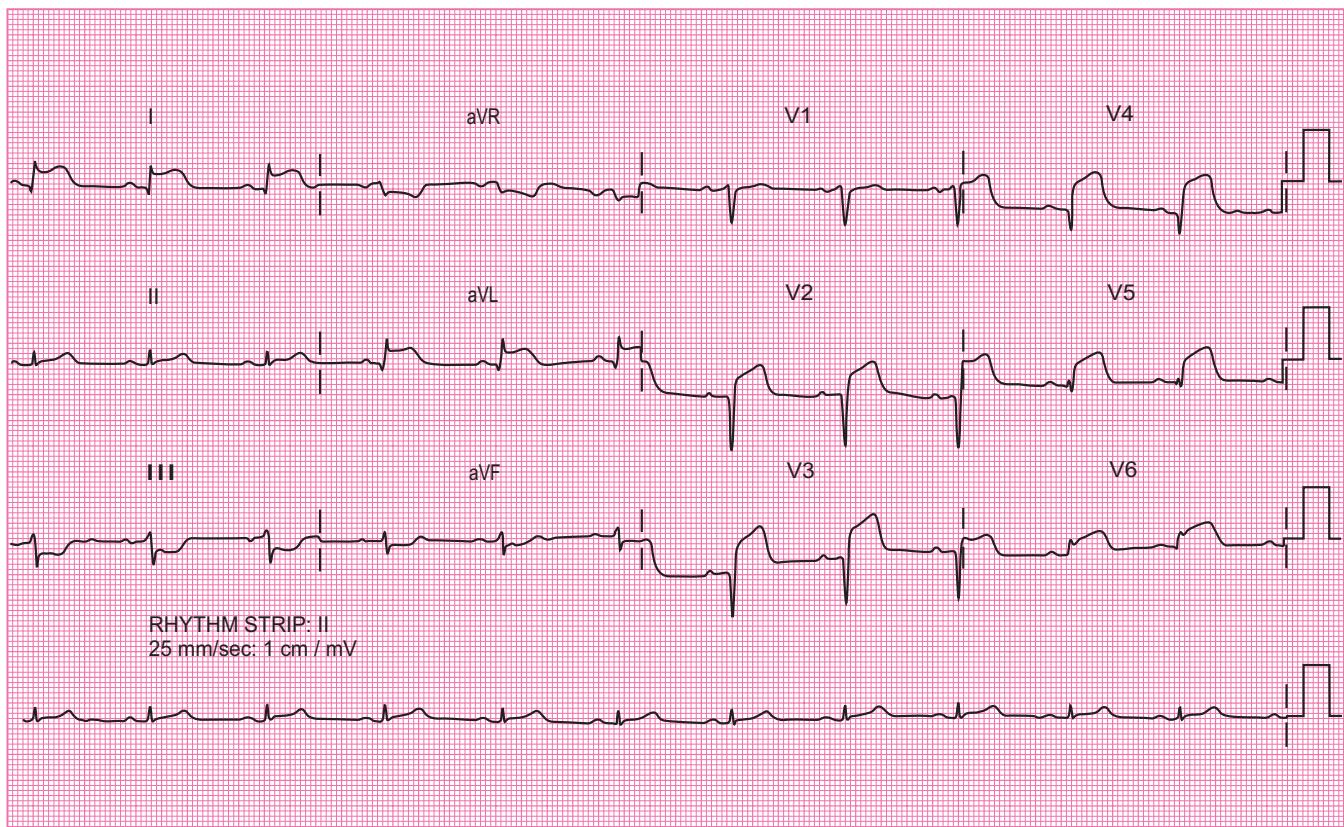


Figure 4.4 ECG showing an anterolateral STEMIs

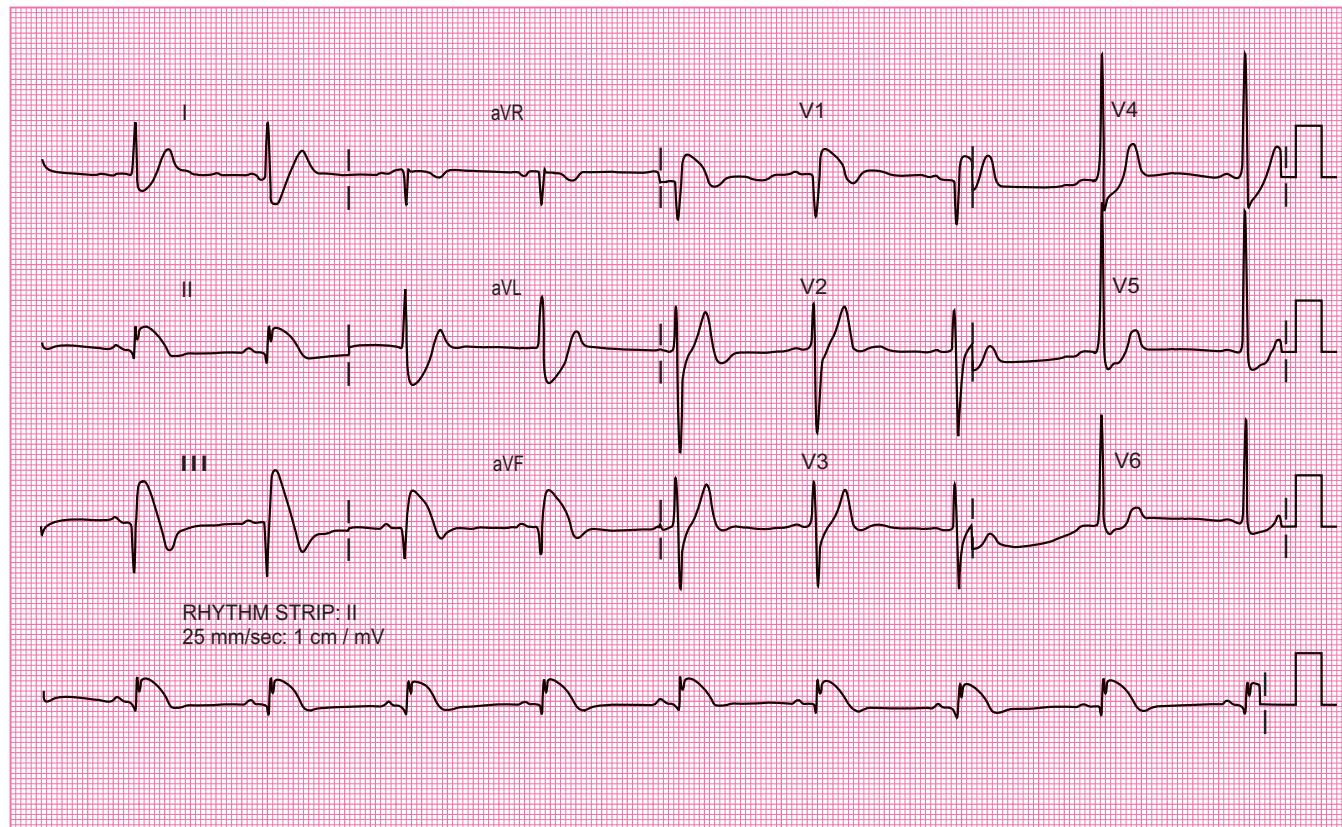


Figure 4.5 ECG showing an inferior STEMI

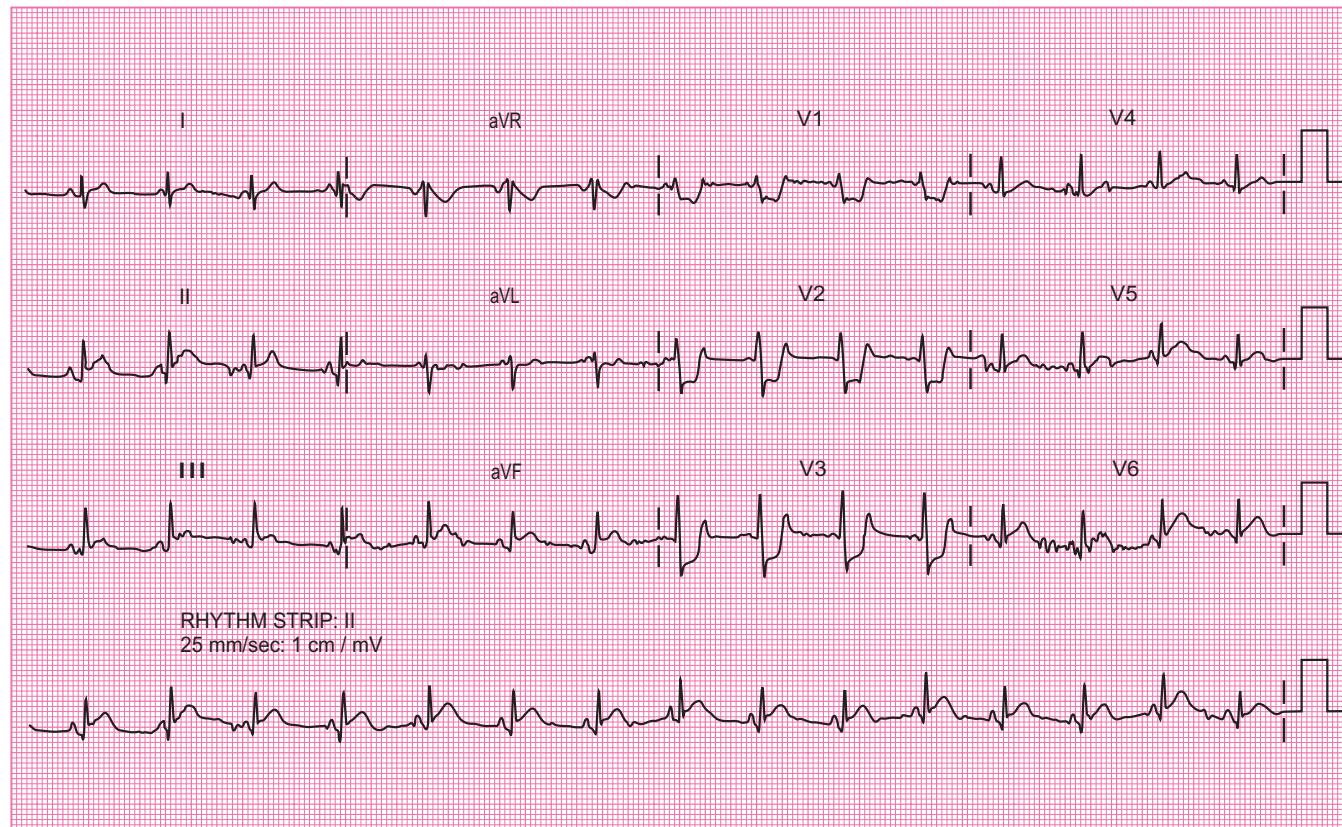


Figure 4.6 ECG showing a posterior STEMl

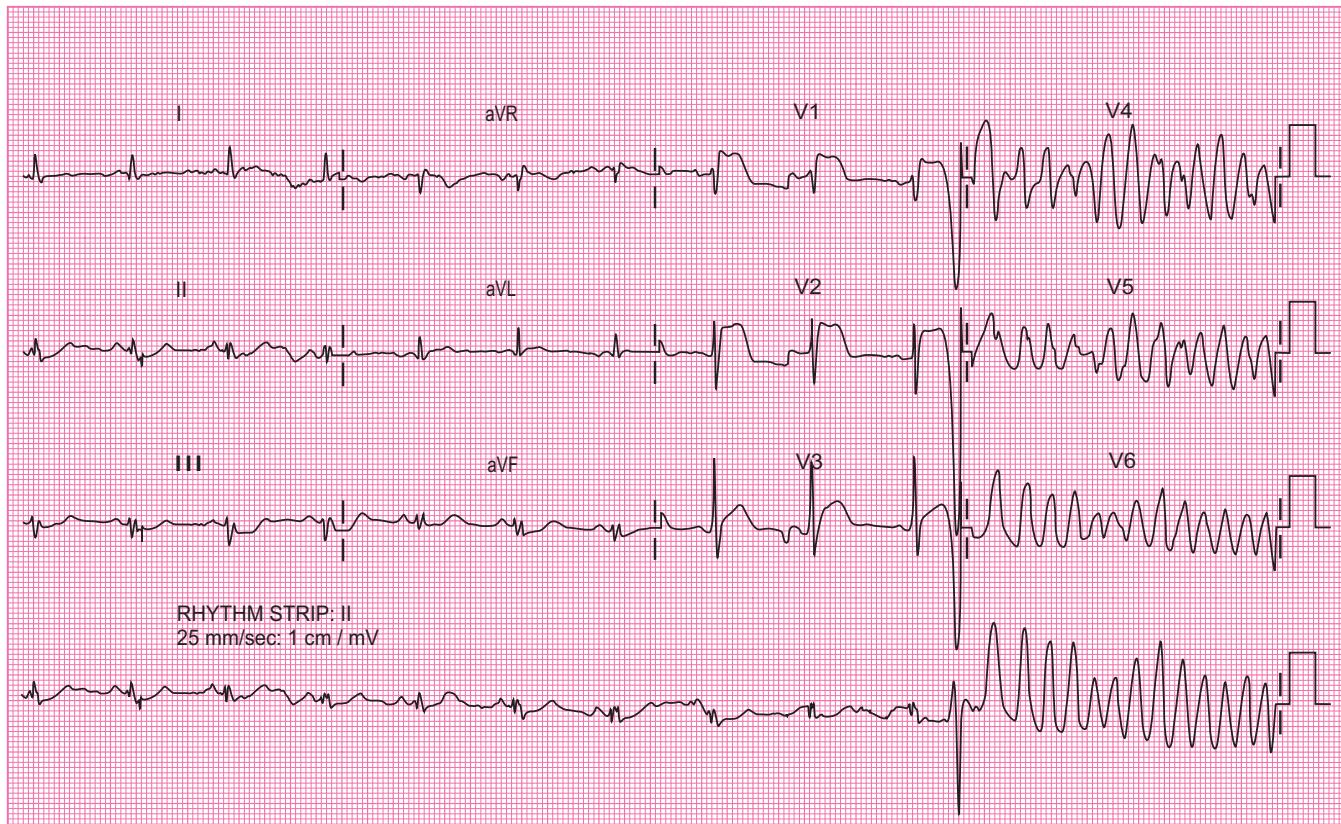


Figure 4.7 ECG showing onset of VF in a patient with acute anteroseptal STEMI

Diagnosis of acute coronary syndromes

History

An accurate history is the crucial first step in establishing a diagnosis, although it is important to realise that patients often present atypically. Some patient groups (e.g. elderly people, diabetics, females, people with renal disease and people during a peri-operative period) are more likely than others to develop an ACS with little or no chest discomfort. In such people, particularly diabetics, the dominant symptom may be breathlessness. The pain of angina or myocardial infarction may be mistaken for indigestion, both by patients and by healthcare professionals. Symptoms such as belching, nausea or vomiting are not helpful in distinguishing cardiac pain from indigestion; they may accompany angina and myocardial infarction.

In addition to the history of chest pain, the presence of certain clinical findings and medical problems increases the probability of CAD and AMI. These include older age, male gender, a positive family history and known atherosclerosis (including in non-coronary territories, such as peripheral or carotid artery disease). The presence of risk factors such as hypertension, smoking, hypercholesterolemia and, in particular, diabetes mellitus and renal insufficiency all increase the likelihood of an ACS.

Clinical examination

Clinical examination is often of limited benefit in making a positive diagnosis of ACS. Severe pain of any source may provoke sweating, pallor and tachycardia, which

commonly accompany ACS. However, a careful history and examination are essential in order to recognise other causes of chest pain and to aid the diagnosis of other life-threatening conditions.

Examination may identify other important abnormalities (e.g. a cardiac murmur or signs of heart failure) that will influence choices of investigation and treatment. In patients with acute chest pain remember to consider other important and potentially life-threatening differential diagnoses such as aortic dissection and pulmonary embolism. Major pulmonary embolism presents typically with unexplained breathlessness and hypoxia, sometimes with sudden collapse, and may cause ECG abnormalities (see below). The presence of aortic dissection may be suggested by acute, severe chest pain radiating to the back, which may be accompanied by other clinical signs. These include hypotension, loss of a peripheral pulse or asymmetry of pulses in the upper limbs, aortic regurgitation or signs of stroke from carotid artery involvement. Suspect aortic dissection in any patient who has acute chest pain and marked hypotension but no obvious ECG evidence of AMI. However, in a patient with a good history and typical ECG evidence of STEMI do not delay reperfusion therapy without strong clinical evidence to justify prior investigation for possible aortic dissection or pulmonary embolism.

Initial examination also serves as an important baseline to detect changes due to progression of the underlying condition or in response to treatment. Signs of shock and heart failure are especially important as they indicate a substantially increased risk of death.

The 12-lead ECG

Record a 12-lead ECG as soon as possible (within 10 min) during the initial assessment and repeat recordings subsequently to assess progression of the ACS and the response to treatment. For many patients an initial 12-lead ECG should form part of pre-hospital assessment by the ambulance service. Pre-hospital ECG recording in patients with STEMI expedites reperfusion therapy (see below), leading to a reduction in mortality.

The presence of ECG abnormalities on the initial recording may support the clinical suspicion of ACS and indicate the appropriate treatment. However, a single normal 12-lead ECG does not exclude an ACS and the ECG should be repeated at intervals to increase the chances of detecting an abnormality.

The ECG is the initial component of risk assessment and planning of treatment. Acute ST-segment elevation or new LBBB in a patient with a typical history of AMI establishes the diagnosis of STEMI. This diagnosis mandates immediate treatment to re-open the occluded coronary artery (reperfusion therapy), preferably by emergency primary percutaneous coronary intervention (PPCI). If this cannot be achieved within 120 min of the onset of chest pain, fibrinolytic therapy (given pre-hospital if appropriate) should be considered as an alternative. Reperfusion therapy should not be delayed whilst awaiting the result of troponin assays in patients with STEMI.

The ECG provides some information about the site and extent of myocardial damage in AMI, particularly in STEMI. This is important since the site and extent of myocardial ischaemia or damage influences prognosis and, in some cases, the appropriate choice of treatment:

- Anterior or anteroseptal infarction is seen in leads V1 – V4 and is typically caused by a lesion in the left anterior descending (LAD) coronary artery. Extension to involve leads V5 – V6, I and aVL indicates an anterolateral infarct (Figure 4.4). An anterior infarct on average is more likely to cause substantial impairment of left ventricular function and has a worse prognosis than an inferior or lateral infarct.
- Inferior infarction is seen in leads II, III, and aVF (Figure 4.5), and is caused often by a lesion in the right coronary artery or, less commonly, the circumflex artery.
- Lateral infarction is seen in leads V5 – V6 and/or leads I and aVL (sometimes aVL alone) and is caused usually by a lesion in the circumflex artery or diagonal branch of the LAD artery.
- Posterior myocardial infarction can be recognised when there is a reciprocal ST-segment depression in the anterior chest leads (Figure 4.6). ST-segment depression in these leads may reflect posterior ST-segment elevation, and development of a dominant R wave in V1 and V2 reflects posterior Q wave development. This is most commonly due to a right

coronary artery occlusion but may be caused by a dominant circumflex artery lesion in individuals in whom this artery provides the main blood supply to the posterior part of the left ventricle and septum. Suspicion of posterior infarction can be confirmed by repeating the ECG using posterior leads. These leads (V8, V9 and V10) are placed in a horizontal line around the chest, continuing from V6 (mid-axillary line) and V7 (posterior axillary line). V9 is placed to the left of the spine, V8 half way between V7 and V9 and V10 to the right of the spine.

- Right ventricular (RV) infarction may be present in up to one third of patients with inferior and posterior STEMI. Extensive RV infarction may be seen on a conventional 12-lead ECG when ST-segment elevation in lead V1 accompanies an inferior (Figure 4.5) or posterior STEMI (Figure 4.6); use of right-sided precordial leads, especially V4R can also be useful in detecting RV infarction. In this case right-sided precordial leads, particularly V4R, may reveal RV infarction. Two-dimensional echocardiography is also very useful. A diagnosis of extensive RV infarction is suggested by fluid-responsive hypotension and signs of high systemic venous pressure (manifest as jugular venous distension) without pulmonary congestion.
- The ST-segment depression and T-wave inversion that may occur in NSTEMI are less-clearly related to the site of myocardial damage than the changes in STEMI.

Acute ECG abnormalities may be caused by conditions other than an ACS. The ECGs of some people with subarachnoid haemorrhage or traumatic brain injury may show acute changes including ST segment depression or elevation, or T wave inversion (Figure 4.8). The ECGs of some people with major pulmonary embolism may show acute changes that include T wave inversion in leads V1–V4, overlying an acutely dilated right ventricle. (Figure 4.9).

Laboratory tests

Laboratory tests are also an important component of diagnosis and risk assessment.

Cardiac-specific troponins (T and I) are two proteins that are components of the contractile structure of myocardial cells (the sarcomere). Because concentrations of troponin in the blood of healthy individuals are undetectably low, and cardiac-specific troponins measured by current assays do not detect troponin from other muscle types (e.g. skeletal muscle), troponin assays are very sensitive and specific markers of cardiac injury. In the context of a typical clinical presentation of an ACS, troponin release provides evidence of myocardial damage and therefore indicates that myocardial infarction has occurred (rather than unstable angina, which will be troponin negative). Troponin values provide an important marker of risk. The greater the troponin concentration, the greater is the risk of a further event or death. A combination of ST-segment depression on the ECG and raised troponin identifies a particularly high-risk group for subsequent AMI and cardiac death.

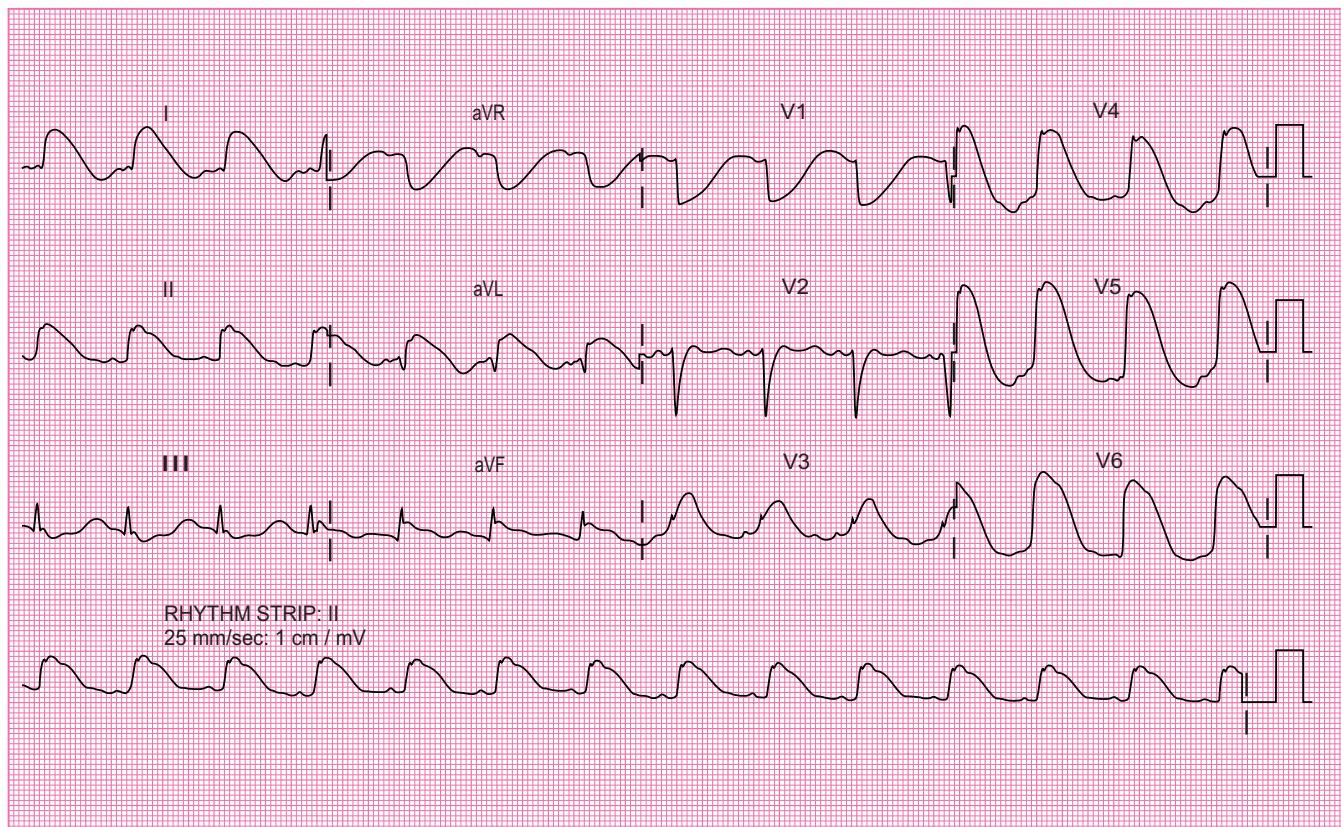


Figure 4.8 Extreme example of an ECG abnormality due to traumatic brain injury. This shows marked ST-segment elevation in the anterolateral leads.

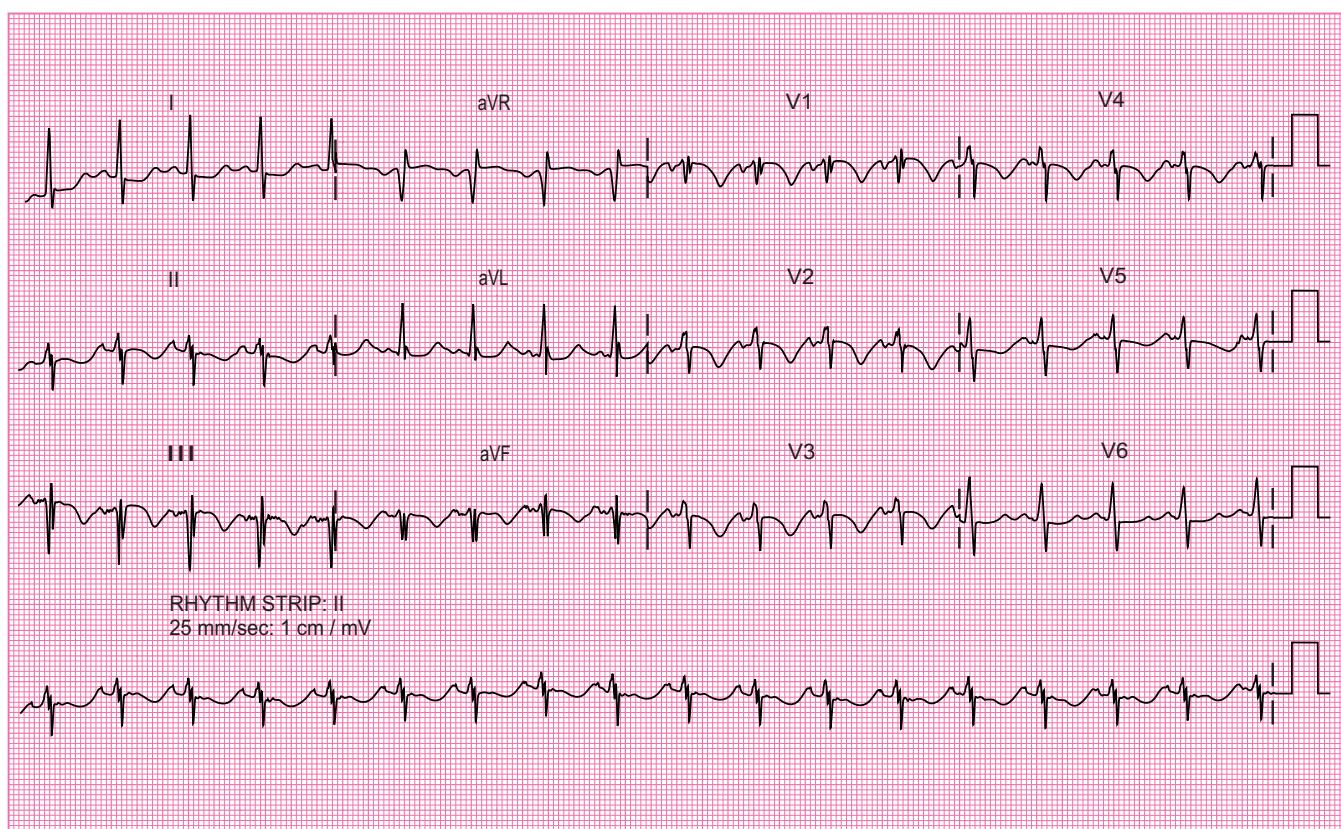


Figure 4.9 ECG showing evidence of acute right ventricular pressure overload due to major pulmonary embolism. An S wave in lead I and a deep Q wave and T wave inversion in lead III are accompanied by T wave inversion in the anterior chest leads (over the right ventricle). In this case the ECG changes were misinterpreted as evidence of an acute coronary syndrome, despite the history and clinical features suggesting pulmonary embolism.

The release of troponin does not in itself indicate a diagnosis of ACS. Troponin release aids diagnosis and is a marker of risk when the history indicates a high probability of AMI. Troponin may be released in other life-threatening conditions presenting with chest pain, such as pulmonary embolism, aortic dissection and myocarditis. Troponin values may also be elevated in acute or chronic heart failure, arrhythmias, chronic renal failure and acute sepsis. Troponin results must be interpreted in the context of the clinical history and presentation and the reference values of the local laboratory.

Echocardiography

Echocardiography is the most important non-invasive imaging modality in the acute setting because it is rapidly and widely available. Left ventricular (LV) systolic function is directly related to prognosis and, in a person with acute chest pain, regional wall motion abnormalities increase the likelihood of an ACS (but are not diagnostic).

Echocardiography may also allow prompt diagnosis of other conditions such as cardiomyopathies, valve disease, pericardial disease, aortic dissection and pulmonary embolism. Echocardiography can also confirm or exclude right ventricular (RV) dilatation and impairment when extensive RV infarction is suspected. In addition it can be used to diagnose complications of AMI such as a ventricular septal defect and severe mitral regurgitation, both of which may require urgent surgical correction.

Quantitative risk assessment scores: GRACE and CRUSADE

An ACS is a potentially unstable condition that is prone to further ischaemia, which may lead to death or AMI in the short or long term. The management of ACS includes anti-ischaemic and antithrombotic drug treatments in addition to coronary revascularisation. This is to prevent or reduce complications and improve outcomes but some of these treatments may also cause bleeding, which worsens prognosis. The timing and intensity of each of these interventions should be tailored to an individual patient's risk and be balanced between the risk of an adverse cardiac outcome and the risk of bleeding due to the treatment given.

Quantitative assessment of risk is useful for clinical decision-making as it is not uncommon for risk to be either over or under-estimated. The Global Registry of Acute Coronary Events (GRACE) risk score provides the most accurate stratification of risk, both on admission and over 6 months. The admission GRACE score is based on eight variables:

- age
- signs of heart failure
- heart rate at presentation
- blood pressure at presentation
- serum creatinine concentration

- ECG changes
- troponin concentration
- cardiac arrest at presentation.

The GRACE score has good discriminative power for predicting risk but because of its complexity is best calculated using an online calculator or app (www.outcomes.umassmed.org/grace/).

Bleeding is associated with an adverse prognosis in ACS, and all efforts should be made to avoid or reduce bleeding whenever possible. A few variables can help to classify patients into different levels of risk for major bleeding during hospital admission. Bleeding risk scores such as CRUSADE (www.crusadebleedingscore.org/) – for use in NSTEMI ACS – have therefore been developed to assess the risk of bleeding and their use is now recommended in guidelines.

Immediate treatment

General measures in all acute coronary syndromes

Patients presenting with a suspected ACS should have a rapid clinical assessment and a 12-lead ECG recorded. If the diagnosis of ACS is confirmed or likely, treat immediately to relieve symptoms, limit myocardial damage and reduce the risk of cardiac arrest. Connect the patient to a cardiac monitor. Immediate treatment for ACS comprises:

- aspirin 300 mg orally, crushed or chewed, as soon as possible
- sublingual glyceryl trinitrate (spray or tablet) unless the patient is hypotensive
- oxygen if the patient is hypoxic (saturation < 94% on air):
 - aim for saturation of 94–98%
 - in the presence of chronic obstructive pulmonary disease aim for 88–92%
- pain relief with IV opiate analgesia (morphine):
 - titrated to control symptoms whilst avoiding sedation and respiratory depression
 - given with an anti-emetic.

Treatment of STEMI (or AMI with new LBBB)

For patients presenting with STEMI within 12 h of symptom onset, mechanical or pharmacological reperfusion must be achieved without delay. The risk/benefit ratio for reperfusion therapy favours this treatment for those who are at highest risk of immediate major myocardial damage and death. In STEMI, coronary reperfusion may be achieved in one of two ways:

- Percutaneous coronary intervention (PCI) may be used to re-open the occluded artery. This is referred to as primary PCI (PPCI).
- Fibrinolytic therapy may be given in an attempt to dissolve the occluding thrombus that caused the AMI.

Primary percutaneous coronary intervention

Primary PCI is the preferred method of reperfusion. The aim is to restore the blood supply at the earliest possible time to myocardium that has not yet been damaged irreversibly. Clinical trials have confirmed the effectiveness of early reperfusion therapy in reducing infarct size, complications, and mortality from AMI. Reperfusion therapy is most effective when undertaken very soon after the onset of myocardial infarction and the benefit diminishes progressively with delay. Beyond 12 h from the onset of chest pain, there may be little or no benefit from reperfusion therapy in many patients, but emergency PCI should be considered in this situation if there is clinical or ECG evidence of on-going ischaemia.

PPCI is the preferred treatment for STEMI provided it can be performed by an experienced team in a timely manner. Coronary angiography is used to identify the occluded coronary artery, following which a guidewire is passed through the occluding thrombus. Passage of the guidewire may restore blood flow but use of a balloon or aspiration devices may be required to restore flow within the vessel. In some settings glycoprotein IIb/IIIa inhibitors may be injected IV or directly into the coronary artery as adjunctive anti-thrombotic therapy. A stent is then inserted into the previously occluded segment of the artery, to reduce the risk of re-occlusion. PPCI is the most reliable method of re-opening and maintaining the patency of the culprit artery in the majority of patients. There is a lower risk of major bleeding, particularly intracerebral, with PPCI than with fibrinolytic therapy.

For PPCI to provide reliable, timely reperfusion a fully-equipped catheter laboratory, staffed by an experienced team, must be available 24 h per day. A fail-safe pathway of communication and care must be in place to ensure that patients in whom STEMI is diagnosed can access the service, ideally by direct transfer to this facility (Figure 4.10). Services should aim to achieve a ‘call-to-balloon’ time (i.e. time from call for help to attempted re-opening of the culprit artery) of < 120 min whenever possible. Delays to treatment are associated with higher mortality. Where PPCI is not available immediately, the need to achieve reperfusion as early as possible remains a high priority and for those patients initial treatment with fibrinolytic therapy may offer the best chance of achieving early reperfusion.

Effective PPCI requires the use of appropriate anti-thrombotic therapy. In addition to aspirin, all patients should also be given one of the platelet ADP receptor blockers prior to PPCI, using one of the following loading doses:

- clopidogrel 600 mg
- prasugrel 60 mg (not if > 75 years, < 60 kg, history of bleeding or stroke)
- ticagrelor 180 mg.

Anticoagulation with unfractionated or low molecular weight heparin is given in the catheter laboratory and in high-risk cases a glycoprotein IIb/IIIa inhibitor may also be

given. Bivalirudin, a direct thrombin inhibitor may be chosen as an alternative to heparin.

Fibrinolytic therapy

Fibrinolytic therapy substantially reduces mortality from AMI when given during the first few hours after the onset of chest pain, but is less effective than PPCI. The advantage of fibrinolytic therapy is that it does not require a cardiac catheter laboratory or skilled angioplasty team. Early reperfusion may be achieved by pre-hospital fibrinolytic therapy with resulting clinical benefit, particularly when transport times to hospital are very long. Early treatment may also be achieved by minimising door-to-needle time (time from arrival at hospital to administration of fibrinolytic therapy).

Fibrinolytic therapy carries a risk of bleeding, including cerebral haemorrhage, and not all patients can be given this treatment safely. Table 4.1 lists typical indications for reperfusion therapy and the typical contraindications to fibrinolytic drugs are shown in Table 4.2. Most of these contra-indications are relative; experienced clinicians should decide whether the benefit from fibrinolytic therapy outweighs the risk to the individual patient or whether emergency angiography with a view to primary PCI would be more appropriate.

The algorithm in Figure 4.10 provides a guide to choosing reperfusion therapy for STEMI.

Typical indications for immediate reperfusion therapy for AMI

Presentation within 12 hours of onset of chest pain suggestive of AMI and:

- ST segment elevation > 0.2 mV in 2 adjacent chest leads, or > 0.1 mV in 2 or more ‘adjacent’ limb leads or
- Dominant R waves and ST depression in V1-V3 (posterior infarction) or
- New-onset (or presumed new-onset) LBBB

Table 4.1 Typical indications for immediate reperfusion therapy for acute myocardial infarction.

Whilst fibrinolytic therapy may reopen an occluded artery, additional antithrombotic therapy must be given to minimise the risk of further thrombotic occlusion.

Give all patients receiving a fibrinolytic agent for STEMI:

- aspirin as a 300 mg loading dose and
- clopidogrel as a 300 mg loading dose and
- antithrombin therapy:
 - low molecular weight heparin (IV bolus then SC) or
 - unfractionated heparin (full dose) or
 - fondaparinux.

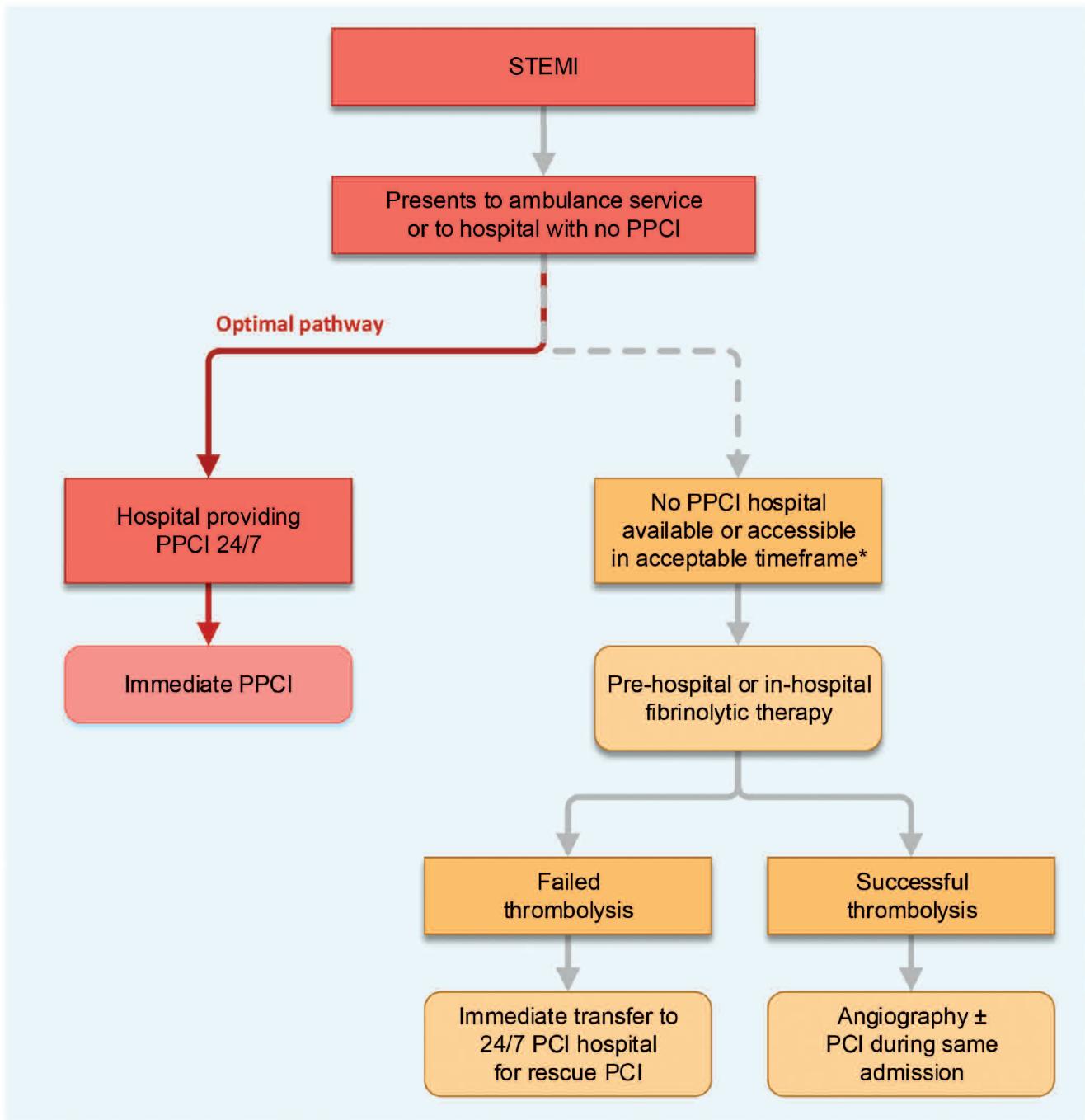


Figure 4.10 Algorithm showing options for access to reperfusion therapy for STEMI

*see NICE CG 167 and ESC guidelines for STEMI

Typical contraindications to fibrinolytic therapy

Absolute

- Previous haemorrhagic stroke
- Ischaemic stroke during the previous 6 months
- Central nervous system damage or neoplasm
- Recent (within 3 weeks) major surgery, head injury or other major trauma
- Active internal bleeding (menses excluded) or gastrointestinal bleeding within the past month
- Known or suspected aortic dissection
- Known bleeding disorder

Relative

- Refractory hypertension (systolic blood pressure > 180 mm Hg)
- Transient ischaemic attack in preceding 6 months
- Oral anticoagulant treatment
- Pregnancy or less than 1 week post-partum
- Traumatic CPR
- Non-compressible vascular puncture
- Active peptic ulcer disease
- Advanced liver disease
- Infective endocarditis
- Previous allergic reaction to the fibrinolytic drug to be used

Table 4.2 Typical contraindications to fibrinolytic therapy

Rescue angioplasty

In 20–30% of patients receiving a fibrinolytic drug for STEMI, reperfusion is not achieved. Observe patients closely with cardiac monitoring during and after administration of a fibrinolytic. Record a 12-lead ECG at 60–90 min after giving fibrinolytic therapy. Failure of ST-segment elevation to resolve by more than 50% compared with the pre-treatment ECG suggests that fibrinolytic therapy has failed to re-open the culprit artery. In some people, the transient, often brief occurrence of an accelerated idioventricular rhythm occurs as a ‘reperfusion arrhythmia’ (Figure 4.11), indicating that the artery has reopened, but this does not occur in all cases and may not always be witnessed or its significance recognised. Symptoms are not a reliable guide to reperfusion, partly because many patients will have received opiate analgesia. Even after initially successful thrombolysis there is a significant risk of re-occlusion and patients should be admitted to a cardiac care unit with continuous ECG monitoring.

In cases of failed reperfusion (or re-occlusion and further infarction with recurrence of ST-segment elevation)

transfer the patient without delay to a cardiac catheter laboratory for PCI. This is referred to as ‘rescue PCI’ and has been shown to improve event-free survival and reduce heart failure when compared to more conservative therapy or repeat fibrinolytic therapy. Like PPCI, rescue PCI must be performed without any time delay in order to be effective.

Wherever possible, following initiation of fibrinolytic therapy, patients should be transferred to a PCI centre for early angiography (and PCI if indicated). Studies have shown that early routine angiography following thrombolysis, with PCI if required, reduced the rates of further infarction and recurrent ischaemia.

Treatment of NSTE ACS (unstable angina and NSTEMI)

In contrast to STEMI, there is no evidence of benefit from immediate reperfusion therapy in most patients with NSTE ACS (with ST-segment depression, T-wave changes or normal ECGs), regardless of whether the ultimate diagnosis is UA or NSTEMI. There is no role for fibrinolytic therapy in these patients.

For patients with NSTE ACS, risk stratification to assess the likelihood of future events is required to guide further treatment. Patients with NSTEMI (positive biomarkers/troponin) and patients with unstable angina (negative troponin) but other high-risk features have an increased risk of further coronary events and death. Patients who have been resuscitated from cardiac arrest due to suspected NSTE ACS are an especially high-risk group. All these higher-risk patients require immediate careful assessment (in most cases including coronary angiography to assess the need for revascularisation by PCI or coronary artery bypass surgery) and medical treatment (see below).

The immediate treatment objectives for all patients with these syndromes are:

- To prevent new thrombus formation, which may occlude an artery and lead to, or extend, myocardial damage.
- To reduce myocardial oxygen demand, providing myocardial cells with a better chance of survival in the presence of a limited supply of oxygen and glucose.

To prevent further thrombus formation:

- Give subcutaneous low molecular weight heparin in therapeutic doses (weight-adjusted, 12-hourly, dose reduction in renal impairment), or fondaparinux (once daily, contraindicated in severe renal impairment).
- Give aspirin 75 mg daily after the initial 300 mg loading dose.

In addition to aspirin, patients who have an elevated blood troponin (NSTEMI) or are planned for angiography +/- revascularisation should be given one of the platelet ADP receptor blockers in the following doses:

- clopidogrel 300 mg (consider 600 mg for rapid loading before urgent angiography), then 75 mg daily maintenance dose or
- prasugrel 60 mg (contraindicated if > 75 years, < 60 kg, history of bleeding or stroke), then 10 mg daily maintenance dose or
- ticagrelor 180 mg, then 90 mg twice daily maintenance dose.

In certain high-risk patients, if early PCI is planned, a glycoprotein IIb/IIIa inhibitor may be considered (tirofiban or eptifibatide). However it is not clear if they offer any additional benefit in patients on dual anti-platelet therapy and they are associated with increased bleeding.

To reduce myocardial oxygen demand:

- start beta-adrenoceptor blockade (unless contraindicated)
- consider diltiazem if beta blockade is contraindicated
- avoid dihydropyridine calcium channel blockers (e.g. nifedipine)
- consider intravenous nitrate infusion if angina persists or recurs after sublingual nitrate
- consider early introduction of an angiotensin converting enzyme inhibitor (ACEI), especially if there is left ventricular systolic impairment or heart failure
- treat complications such as heart failure or tachyarrhythmia promptly and effectively.

Subsequent management of patients with acute coronary syndromes

Suspected unstable angina – low risk score

Patients with suspected unstable angina without high-risk features at presentation (ECG and troponin levels normal, low GRACE score) should undergo early further risk assessment either by exercise testing or non-invasive imaging.

NSTEMI and high-risk unstable angina

Patients with unstable angina and high-risk features (e.g. resting ST-segment depression, high-risk features on exercise test or non-invasive imaging or high GRACE score) should be considered for early investigation by percutaneous coronary angiography.

Patients with NSTEMI should be regarded as a high-risk group, requiring early assessment by coronary angiography within 72 h of presentation.

Many patients with high-risk NSTE ACS will benefit from revascularisation by PCI and some with complex coronary anatomy will require coronary artery bypass graft surgery. Use formal risk-scoring systems such as GRACE to guide clinical management. Those patients at the highest risk derive the greatest benefit from early intervention, in terms of reducing further major cardiac events.

STEMI

If fibrinolytic therapy has been used, many patients will be left with a severe stenosis or unstable plaque in the culprit coronary artery. PCI can stabilise this situation and reduce the risk of re-occlusion of the artery and resulting further myocardial infarction, cardiac arrest or sudden death. Coronary angiography and, if indicated, PCI should be undertaken early during the same hospital admission. Patients who have had successful thrombolysis but are not in a PCI-capable hospital should be transferred for angiography and possible PCI.

In patients with completed STEMI who have not been treated with reperfusion therapy (e.g. because of late presentation) it is usually recommended that coronary angiography is undertaken during the same hospital admission. Although the benefits of re-opening an occluded culprit artery late after STEMI are uncertain, there is often disease in other coronary vessels that can give rise to further major coronary events over subsequent months. Defining the severity and anatomy of such disease can help to identify those at highest risk, in whom early intervention may reduce that risk.

Cardiac rehabilitation

In all patients after an ACS, an effective programme of cardiac rehabilitation can speed the return to normal activity and encourage measures that will reduce future risk (see below). There is evidence that effective cardiac rehabilitation reduces readmission to hospital and improves overall prognosis and survival. Cardiac rehabilitation is a continuous process, beginning in the cardiac care unit and progressing through to a community-based approach to lifestyle modification and secondary prevention. Cardiac rehabilitation should include a supervised exercise programme.

Secondary prevention

In patients with established coronary disease, general measures to reduce cardiovascular risk ('secondary prevention') can reduce the likelihood of future coronary events (including sudden cardiac death) and stroke.

Anti-thrombotic therapy

Continued platelet inhibition is appropriate in all patients. They should receive low-dose aspirin (75 mg daily) for life, unless contraindicated. Give clopidogrel 75 mg daily/ or prasugrel 10 mg daily/ or ticagrelor 90 mg twice daily) to patients with high-risk ACS and all those undergoing PCI; current guidelines recommend treatment for a minimum of one year. Clopidogrel alone may be used in patients who are intolerant of aspirin. In patients who develop atrial fibrillation as a complication of ischaemic heart disease, there is an additional risk of thromboembolism from the left atrium. Treatment with warfarin or a novel oral anticoagulant is more effective than aspirin or clopidogrel in preventing intra-cardiac thrombus formation, and should be considered in addition to, or instead of platelet inhibition.

Preservation of left ventricular function

Prognosis after AMI is partly determined by the severity of left ventricular impairment. Treatment after AMI with an ACEI can reduce the re-modelling that contributes to left ventricular dilatation and impairment, and where there is left ventricular systolic impairment, the use of ACEI therapy in adequate dose can reduce the risk and severity of subsequent heart failure, and the risk of future AMI and death. Echocardiographic examination of left ventricular function is appropriate during the first few days after an ACS to assess risk and identify those patients likely to benefit most from this treatment. The majority of patients should be considered for ACEI treatment during the first few days after AMI.

Beta-adrenoceptor blockade

Treatment with a beta-blocker, started early after AMI and continued, has been shown to reduce mortality. In the absence of a contraindication, a beta-blocker should be started in the acute phase of treatment and continued indefinitely. Careful dose adjustment is needed in each individual patient to achieve adequate beta blockade whilst avoiding adverse effects. There is evidence that prior treatment with beta blockade may reduce the size of subsequent myocardial infarction, so in patients with coronary disease this treatment may have a ‘cardioprotective’ effect, and it may help to protect against other complications such as arrhythmia. In patients with chronic heart failure and left ventricular systolic impairment there is evidence of symptomatic and prognostic benefit from some beta blocking drugs.

Reduction of cholesterol

Further reduction in risk can be achieved by effective suppression of cholesterol concentration in the blood, specifically suppression of LDL cholesterol. Statins reduce the risk of future coronary events by at least 30%. A low-fat, high-fibre diet and regular exercise will complement cholesterol suppression by drugs. High-dose statin treatment is often advised after an ACS (e.g. atorvastatin 80 mg daily).

Stopping smoking

At least as important in reducing risk, is the removal of other avoidable risk factors such as smoking. Advise all people who smoke to stop and offer assistance from a smoking cessation service. Information, encouragement and support for patients to help them to stop smoking should begin at an early stage after presentation with an ACS.

Anti-hypertensive treatment

Effective control of raised blood pressure, using drugs as well as non-pharmacological methods, reduces the risk of stroke and of heart failure and contributes to some reduction in the risk of future coronary events.

Cardiac arrhythmia complicating acute coronary syndromes

Ventricular arrhythmia in ACS

In some people, cardiac arrest in VF or pulseless VT (pVT) may be the presenting feature of an ACS. If return of spontaneous circulation (ROSC) is achieved in a person whose cardiac arrest is likely to be due to an ACS, record a 12-lead ECG as soon as possible. If this shows evidence of STEMI, emergency reperfusion therapy is needed, whenever possible by PPCI, together with all other treatment for STEMI. This also applies to those patients with STEMI who are unconscious and ventilated after ROSC (See Chapter 13). If NSTEMI is suspected as the cause of the arrest consider urgent coronary angiography with a view to PCI if necessary, together with all other treatment for a high-risk NSTE ACS. Where cardiac arrest is very likely to be due to an ACS but usual advanced life support measures fail to achieve ROSC, in some hospitals it is possible to consider the use of a mechanical chest compression device or extracorporeal CPR to enable coronary angiography and PCI to be performed as part of the on-going resuscitation attempt.

When ventricular arrhythmia complicates an acute coronary syndrome (Figure 4.7), interpret its significance in the context of the clinical setting and the time of onset of the arrhythmia relative to the onset of the ACS. When VF/pVT cardiac arrest occurs within the first 24–48 h after STEMI, and subsequent recovery is uncomplicated, the risk of another ventricular arrhythmia is relatively low but may be determined by other factors, such as the severity of left ventricular impairment. If VF/pVT cardiac arrest occurs in the context of non-ST-segment-elevation ACS, there may be a continuing risk of further ventricular arrhythmia. If the arrhythmia has been caused by severe myocardial ischaemia, urgent revascularisation is needed to prevent recurrence of the ischaemia and arrhythmia. If this is not achievable or if the arrhythmia has occurred without evidence of severe ischaemia, the patient will be at risk of recurrent ventricular arrhythmia and should be referred to a heart rhythm specialist with a view to insertion of an implantable cardioverter-defibrillator (ICD) before discharge from hospital.

Patients who have a VF/pVT cardiac arrest as a late complication after myocardial infarction, or outside the context of an ACS, are at risk of recurrent cardiac arrest and should be seen by a heart rhythm specialist with a view to ICD implantation before discharge from hospital.

When an arrhythmia occurs in the setting of an ACS it is important to check for and treat other factors that may have predisposed to the arrhythmia (e.g. hypokalaemia, heart failure).

It is important to recognise an accelerated idioventricular rhythm that is seen commonly and transiently after reperfusion of a previously occluded coronary artery, whether

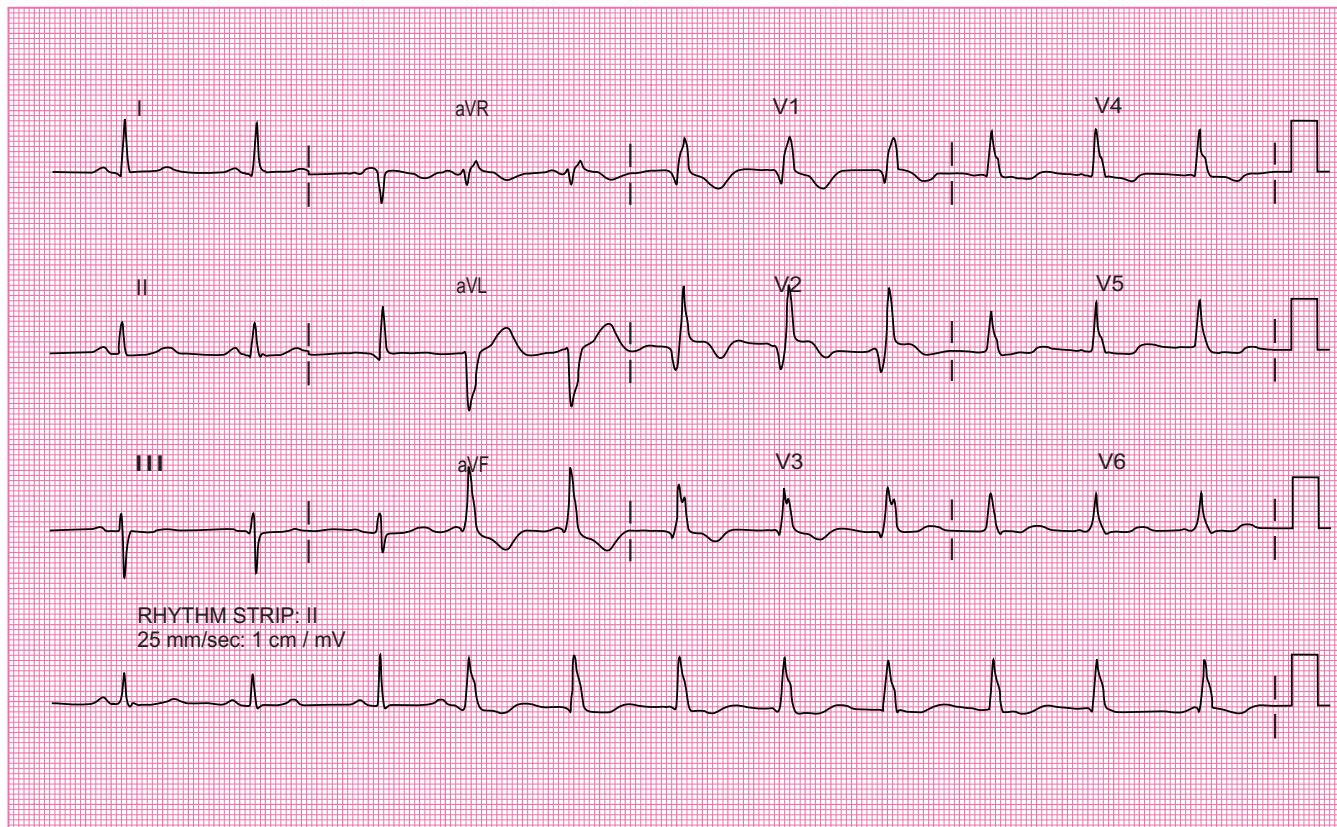


Figure 4.11 ECG showing development of an accelerated idioventricular rhythm at a slightly faster rate than the preceding sinus rhythm, in a patient who had received fibrinolytic therapy for an anterior STEMI. This rhythm heralded the success of reperfusion therapy, caused no haemodynamic compromise and resolved spontaneously.

following PCI or after fibrinolysis. An example is shown in Figure 4.11. After fibrinolytic therapy this may provide strong indication that thrombolysis has been achieved. These rhythms are usually transient and do not require treatment.

Other cardiac arrhythmia

The treatment of other cardiac arrhythmia will be covered in more detail in Chapter 11.

When atrial fibrillation occurs in the context of an ACS it may indicate left ventricular failure: treatment should address that as well as focusing on control of heart rate or rhythm.

When AV block occurs in the context of an inferior AMI there is often transient dysfunction of the conducting system and excessive vagal activity. QRS complexes are often narrow and the heart rate may not be especially slow. Treat symptomatic bradycardia in this setting with atropine; consider temporary cardiac pacing only if bradycardia and hypotension persist after atropine. Complete AV block in this setting is usually transient and permanent cardiac pacing is rarely necessary. The heart block often resolves after PPCI, so do not delay arrangements for PPCI because of AV block in this setting.

When AV block occurs in the context of acute anterior myocardial infarction this usually implies extensive myocardial injury and a poor prognosis. The QRS complexes are usually broad and the heart rate is usually

slow and resistant to atropine. Temporary cardiac pacing is usually needed and should not be delayed. Patients in this situation will often require a permanent pacemaker but on average have a poor prognosis.

Other complications of ACS

Heart failure

Patients with heart failure complicating AMI or other ACS are at increased risk of deterioration, cardiac arrest and death: prompt, effective treatment of the heart failure is required to reduce risk. Give a loop diuretic (e.g. furosemide) and/or glyceryl trinitrate (sublingual and/or intravenous) for immediate symptomatic treatment. Give regular loop diuretic to maintain symptom control. Ensure that ACEI treatment has been started and increase the dose as tolerated, until the target dose is achieved. In patients intolerant of ACEI, consider an angiotensin receptor blocker. Maintain beta blockade and increase the dose as tolerated, unless contraindicated. For patients with heart failure or significant LV systolic impairment (ejection fraction 40% or less) start an aldosterone antagonist (e.g. eplerenone or spironolactone).

Cardiogenic shock

This consists of hypotension with poor peripheral perfusion, often accompanied by pulmonary oedema, drowsiness or mental confusion due to poor cerebral perfusion and oliguria caused by poor renal perfusion. The mortality is very high, but can be reduced by early revascularisation.

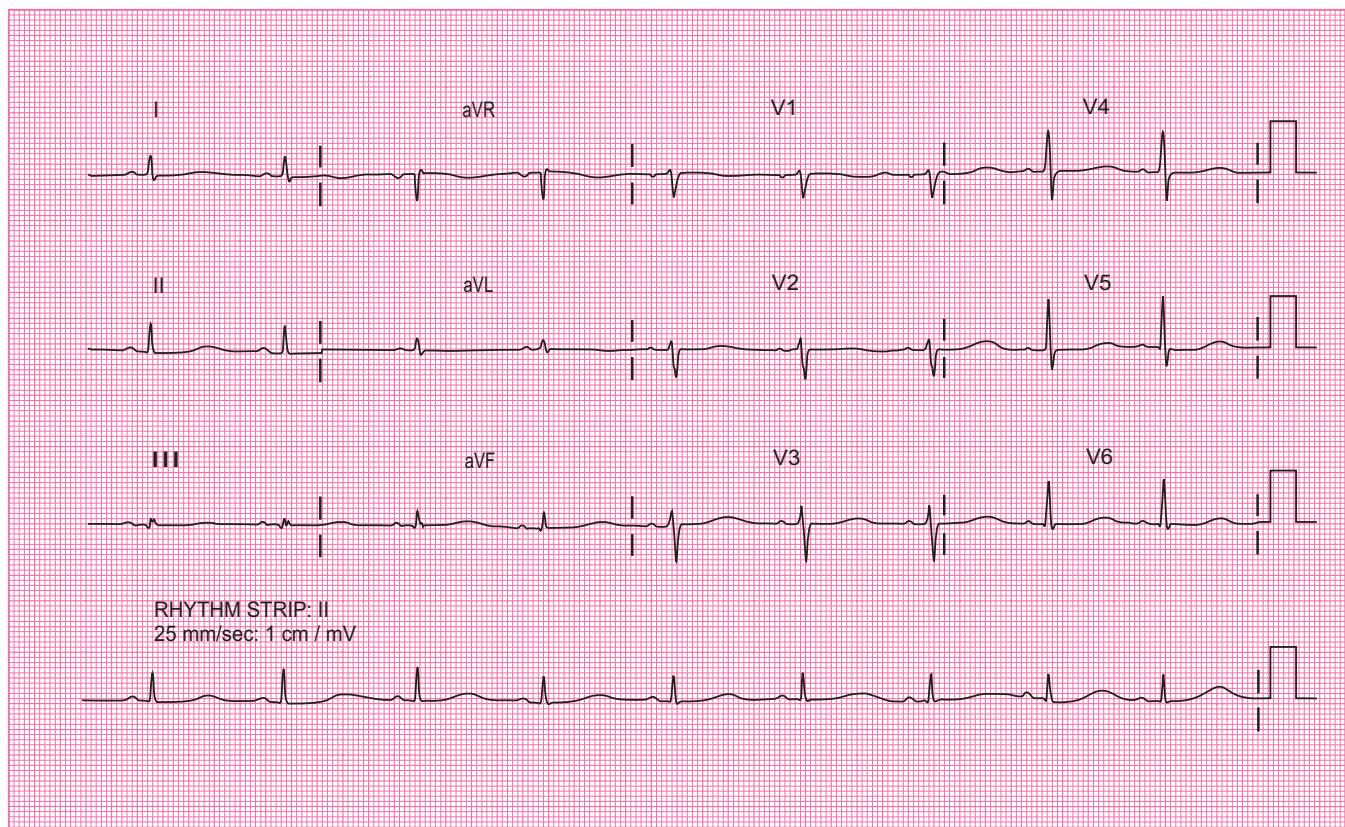


Figure 4.12 ECG recording in a patient who presented with episodes of syncope. It shows marked QT prolongation due to an inherited long QT syndrome.

Some patients may improve with inotropic therapy, but this requires initiation and supervision by those experienced in its use. Other treatments such as intra-aortic balloon pumping, other forms of mechanical circulatory support and ventilatory support may be considered in selected patients. These require expert assessment and management in an ICU by a specialist multidisciplinary team.

When cardiogenic shock develops in a patient after STEMI, seek early expert assessment with a view to emergency PCI as this may be life-saving.

Other cardiac causes of cardiac arrest and sudden cardiac death

Whilst coronary artery disease is the commonest cause of cardiac arrest and sudden cardiac death (SCD) in adults over the age of 35 years, other (mostly acquired) conditions can cause SCD in this age group. Inherited conditions are a less common cause in older adults but predominate as the causes of SCD in people under the age of 35. Table 4.3 lists some of the important causes.

Identifying the cause of a cardiac arrest or sudden death

When someone has a cardiac arrest or dies suddenly, it is important to obtain as much detail as possible of the circumstances of the event in order to assist in identifying the cause. Following SCD the autopsy is crucial, as it may identify some inherited conditions (e.g. hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy) and enable screening of family members

to identify others at risk in whom treatment may prevent SCD. Detailed examination of hearts of SCD victims by an expert cardiac pathologist is recommended as these conditions may not always be easy to confirm or exclude at a routine autopsy. If no abnormality is found at autopsy to explain SCD, this is termed sudden arrhythmic death syndrome (SADS). This should always raise suspicion of an 'electrical' inherited cardiac condition that may affect other family members and place them at similar risk.

When a person suffers cardiac arrest it is important that those attempting resuscitation consider from the outset the likely cause of the arrest. This may identify a reversible cause, but may also lead to early recognition of a cause that will guide management of the resuscitation effort and/or post-resuscitation care. This is equally important in out-of-hospital and in-hospital cardiac arrest. A detailed history of the event, clinical examination following ROSC, a 12-lead ECG recorded as soon as possible after ROSC and echocardiography as soon as practicable may each provide evidence that allows early recognition of one of the conditions listed in Table 4.3.

Identifying people at risk

Whilst it is important to consider the cause in every instance of cardiac arrest and sudden death, you should be alert also for warning symptoms that may allow recognition and treatment of a life-threatening condition, so preventing cardiac arrest or SCD. For example, in people with severe aortic stenosis those symptoms might be angina, breathlessness or syncope. People at risk of sudden death from cardiac arrhythmia may experience syncope (Figure 4.12). Assess people who experience one or

Condition	Causes	Further detail
Long QT syndromes (LQTS)	Inherited (autosomal dominant) ion channel disorders. Many different genotypes but types 1–3 are most common.	Predispose to torsade de pointes VT and VF
Acquired QT interval prolongation	Drug therapy. Ischaemic heart disease. Myocarditis.	Predisposes to torsade de pointes VT and VF
Brugada syndrome	Inherited (autosomal dominant) ion channel disorder	Occurs worldwide but more common in SE Asia. Risk of SCD higher in young males.
Short QT syndrome (SQTS)	Rare, inherited (autosomal dominant) ion channel disorder	Predisposes to torsade de pointes VT and VF
Catecholaminergic polymorphic ventricular tachycardia (CPVT)	Rare, inherited (autosomal dominant) ion channel disorder	Predisposes to torsade de pointes VT and VF, especially on exercise
Arrhythmogenic right ventricular cardiomyopathy (ARVC)	Inherited (autosomal dominant)	Predisposes to VT and VF
Hypertrophic cardiomyopathy (HCM)	Inherited (autosomal dominant). Several different genotypes	SCD risk is due to VT and VF. Risk varies with genotype and with individual factors
Wolff-Parkinson-White (WPW) syndrome	Mostly sporadic. Infrequent familial incidence.	Not all WPW patients are at risk of SCD. Risk is due to rapid transmission of AF to the ventricles, triggering VT or VF
High-grade atrioventricular block	Conducting system fibrosis. Calcific aortic stenosis. Myocardial diseases including ischaemic heart disease. Cardiac surgery. Drug therapy. Occasionally congenital.	Predisposes to ventricular standstill (asystole). Some people with extreme bradycardia develop torsade de pointes VT and VF
Severe aortic stenosis	Congenital bicuspid valve (becomes severe at age 50–70 or younger) Degenerative (becomes severe in elderly patients)	If untreated may progress to heart failure or SCD, probably mostly due to VT or VF
Dilated cardiomyopathy	Probably multiple causes. Familial in a minority of cases.	Many develop progressive heart failure but there is risk of SCD due to VT or VF
Ischaemic heart disease due to coronary atheroma	Partly genetic, partly acquired	SCD risk is mainly due to VT or VF, which may be in response to acute ischaemia or infarction or may be due to previous myocardial scarring
Other myocardial diseases	Hypertensive heart disease, sarcoid heart disease etc	May predispose to ventricular arrhythmia or AVB in some patients
Anomalous coronary artery anatomy	Congenital	Rare cause of SCD in young people, often on exercise. Risk varies with the anomalous anatomical pattern

Table 4.3 Important causes of sudden cardiac death.

Reproduced from: Pitcher D. Sudden Cardiac Death. In ABC of Resuscitation, Sixth Edition 2012 Soar J, Perkins G, Nolan J eds. Wiley-Blackwell; Oxford.

more episodes of syncope to identify features of common problems that do not carry a significant risk of death (such as uncomplicated faints) and to identify the small minority of people in whom syncope may be the only warning of a preventable risk of SCD. Syncope that occurs during exertion should raise particular concern and lead to urgent, expert assessment.

Increasingly, screening of asymptomatic people may lead to identification of clinical signs, or of ECG or echocardiographic abnormalities that lead to diagnosis of a condition predisposing to cardiac arrest and SCD. There is international debate about the cost-effectiveness of screening low-risk groups and the screening techniques that are appropriate. Various groups of people may undergo screening including sportspeople, pilots and family members of people with inherited cardiac conditions (ICCs) or suspected ICCs. In the latter group screening is best co-ordinated through a dedicated cardiology-genetics clinic dealing specifically with ICCs.

Avoidance of cardiac arrest in people at risk

The treatment needed to minimise the risk of cardiac arrest and SCD depends largely on the underlying condition, but also varies according to individual circumstances in people with the same condition. In some people, reducing risk of SCD requires avoidance of specific activities or circumstances that predispose to SCD in their specific situation. In many people with a long QT syndrome beta blockade may be the only treatment needed to reduce risk to a minimum, but in others beta blockade may provide insufficient protection and an ICD may be needed. In severe aortic stenosis, aortic valve replacement is likely to offer the only opportunity for risk reduction. In complete AV block pacemaker implantation restores average life expectancy to normal for the person's age. These examples are not exhaustive and are mentioned to illustrate the range of conditions and treatments that must be considered. Urgent, detailed specialist assessment is needed. Refer anyone thought to be at risk to the most appropriate specialist without delay.

Summary learning

- The acute coronary syndromes comprise unstable angina, NSTEMI and STEMI.
- Rapid initial assessment by history, examination and 12-lead ECG will help to determine the diagnosis and immediate risk.
- Give aspirin to patients presenting with ACS.
- Give nitroglycerine and, if required, morphine (with an anti-emetic) to relieve pain.
- Give oxygen to hypoxaemic patients to achieve SpO₂ 94–98% (88–92% in COPD).
- Consider immediate reperfusion therapy (preferably by PPCI) in people with STEMI or AMI with new LBBB.
- Consider early coronary angiography (+/- revascularisation) in people with NSTEMI or high-risk unstable angina.
- Effective assessment and immediate treatment of patients with ACS will reduce the risk of cardiac arrest and death.
- A range of other cardiac conditions can cause cardiac arrest and sudden death.
- When a person has a cardiac arrest or SCD obtain detailed information about the event and consider the most likely cause.
- Assess people with syncope to identify those in whom it may be a warning symptom of a risk of SCD that may be prevented.
- Refer people who may be at risk (and/or their families where appropriate) for appropriate expert assessment.

My key take-home messages from this chapter

Further reading

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In-hospital resuscitation

Chapter
5

Contents

- Features specific to in-hospital resuscitation
- Sequence for the collapsed patient in hospital

Learning outcomes

To enable you to:

- Start resuscitation in hospital
- Maintain high quality cardiopulmonary resuscitation (CPR) with minimal interruption
- Prioritise your actions according to the in-hospital algorithm

Introduction

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. The public expect that all clinical staff can undertake CPR. For every in-hospital cardiac arrest, ensure that:

- cardiorespiratory arrest is recognised immediately
- help is summoned using a standard telephone number ([2222 in the UK](#))
- CPR is started immediately and, if indicated, [defibrillation](#) is attempted as soon as possible ([within 3 min](#)).

This chapter is primarily for healthcare professionals who are first to respond to an in-hospital cardiac arrest, but may also be relevant to healthcare professionals working in other clinical settings.

Features specific to in-hospital resuscitation

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

- location (clinical/non-clinical area; monitored/unmonitored area)
- skills of the first responders
- number of responders
- equipment available
- hospital response system to cardiac arrest and medical emergencies (e.g. medical emergency team (MET), resuscitation team).

Location

In patients who are being monitored closely, clinical deterioration and cardiorespiratory arrest is usually identified rapidly. Patients in areas without facilities for close monitoring can have unobserved deterioration and an unwitnessed cardiorespiratory arrest. 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).

Cardiac arrest patients may have to be moved to enable effective resuscitation. The Resuscitation Council (UK) has published guidance for safer handling during resuscitation in healthcare settings:
<https://www.resus.org.uk/publications/guidance-for-safer-handling-during-cpr-in-healthcare-settings/>

Training of first responders

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 and greater experience in resuscitation than those who are not involved regularly in resuscitation in their normal clinical role. Hospital staff responding to a cardiac arrest may have different levels of skill to manage the airway, breathing and circulation. Rescuers must undertake tasks and procedures in which they are trained and proficient or with appropriate clinical supervision.

Number of responders

The single responder must first ensure help is coming. Usually, other staff are nearby and the resuscitation team called whilst other staff start CPR simultaneously. Hospital staff numbers tend to be fewer at night and weekends. This may influence patient monitoring, recognition, treatment and outcomes; studies show that survival rates from in-hospital cardiac arrest are lower during nights and weekends.

Equipment available

Staff in all clinical areas should have immediate access to resuscitation equipment and drugs to facilitate rapid resuscitation of the patient in cardiorespiratory arrest. The equipment used for CPR (including defibrillators) and the layout of equipment and drugs should be standardised throughout the hospital. You should be familiar with the resuscitation equipment used in your clinical area.

A review by the Resuscitation Council (UK) of serious patient safety incidents associated with CPR and patient deterioration showed that equipment problems during resuscitation (e.g. equipment missing or not working) is common. All resuscitation equipment needs to be checked on a regular basis to ensure it is ready for use. Specially designed trolleys or sealed tray systems can improve speed of access to equipment and reduce adverse incidents.

Defibrillators with automated rhythm recognition should be considered for use in clinical and non-clinical areas where staff do not have rhythm recognition skills or rarely need to use a defibrillator. However, manual defibrillators used by trained staff are associated with increased survival after in-hospital cardiac arrest compared with when automated external defibrillators (AEDs) are used. Use an AED when someone trained to use a manual defibrillator is not immediately available, or when only an AED is available.

ALS

Waveform capnography must be used to confirm correct tracheal tube placement during resuscitation and can also help guide resuscitation interventions (Chapter 6). Waveform capnography monitoring is available on newer defibrillators as part of portable monitors or as a hand held device.

After successful resuscitation, patients often need transferring to other clinical areas (e.g. intensive care unit) or other hospitals. Transfer equipment and drugs should be available to enable this.

Resuscitation team

Hospitals should have a system to recognise patients at risk of cardiac arrest and allow staff to call a resuscitation team before cardiac arrest occurs. For example, the medical emergency team (MET) deals with patients who are deteriorating and at risk of cardiac arrest. 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 prevent futile resuscitation attempts in those who are unlikely to benefit from CPR (Chapter 3).

Resuscitation teams should have formal briefings for introductions and to plan their roles and responsibilities during actual resuscitation events. Team members should also participate in a debriefing after each event, to allow performance and concerns to be discussed openly. This has most benefit when discussions are based on data collected during the event.

Sequence for a collapsed patient in hospital

An algorithm for the initial management of in-hospital cardiac arrest is shown in Figure 5.1.

1. Ensure personal safety

- There are very few reports of harm to rescuers during resuscitation.
- Your personal safety and that of resuscitation team members is the first priority during any resuscitation attempt.
- Check that the patient's surroundings are safe.
- Put on gloves as soon as possible. Other personal protective equipment (PPE) (eye protection, face mask, aprons, gowns) may be necessary especially when the patient has a serious infection such as tuberculosis. Follow local infection control measures to minimise risk.
- The actual risk of infection to rescuers during CPR is much lower than perceived. There are isolated reports of infections such as tuberculosis (TB), and severe acute respiratory distress syndrome (SARS). Transmission of HIV during CPR has never been reported.
- Be careful with sharps; a sharps box must be available.
- Use safe handling techniques for moving victims during resuscitation.

Call resu

C
With oxygenApply i
Attempt
if apAdvanced
when resu

Figure 5.1 In-hospital resuscitation algorithm

ALS

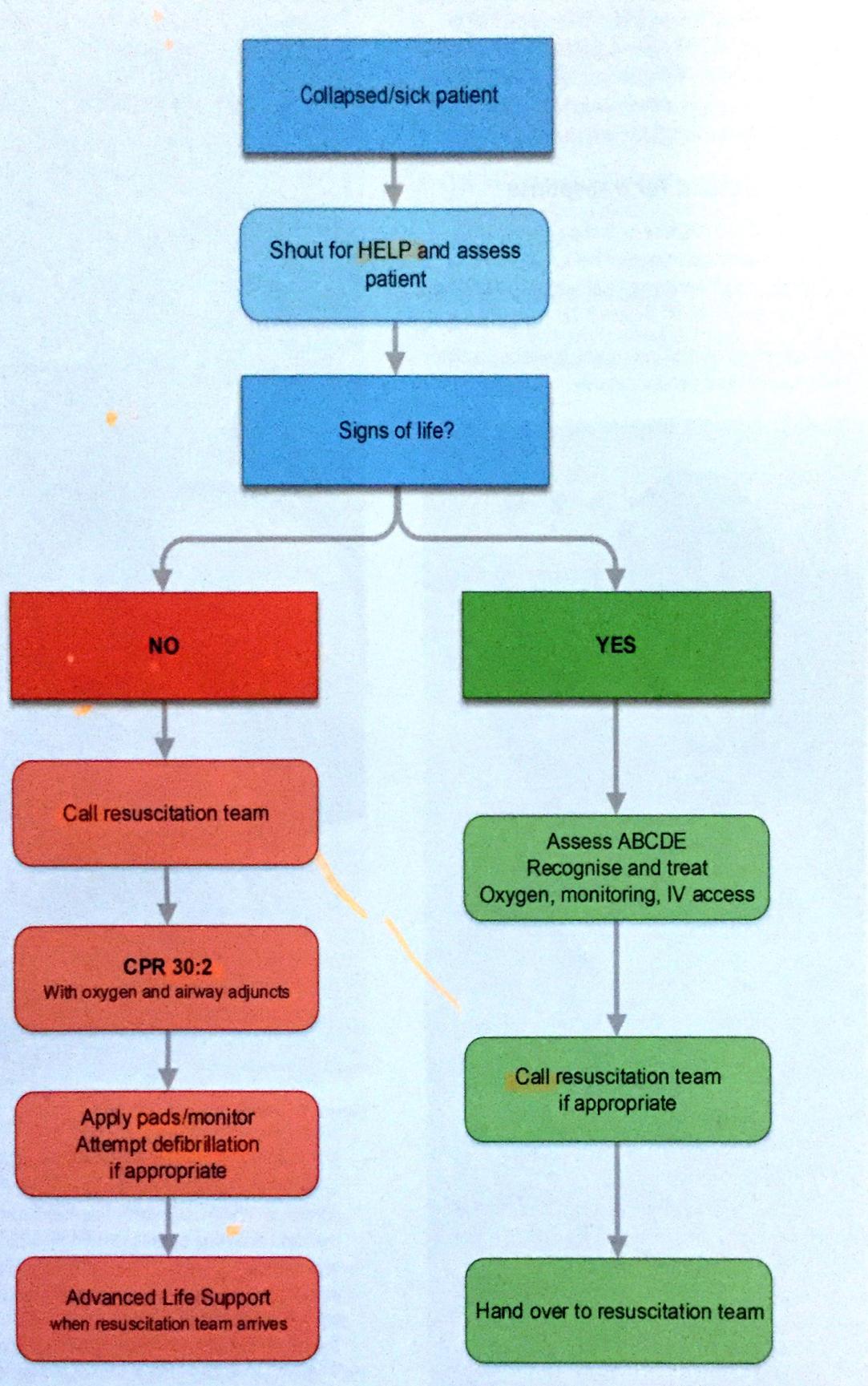
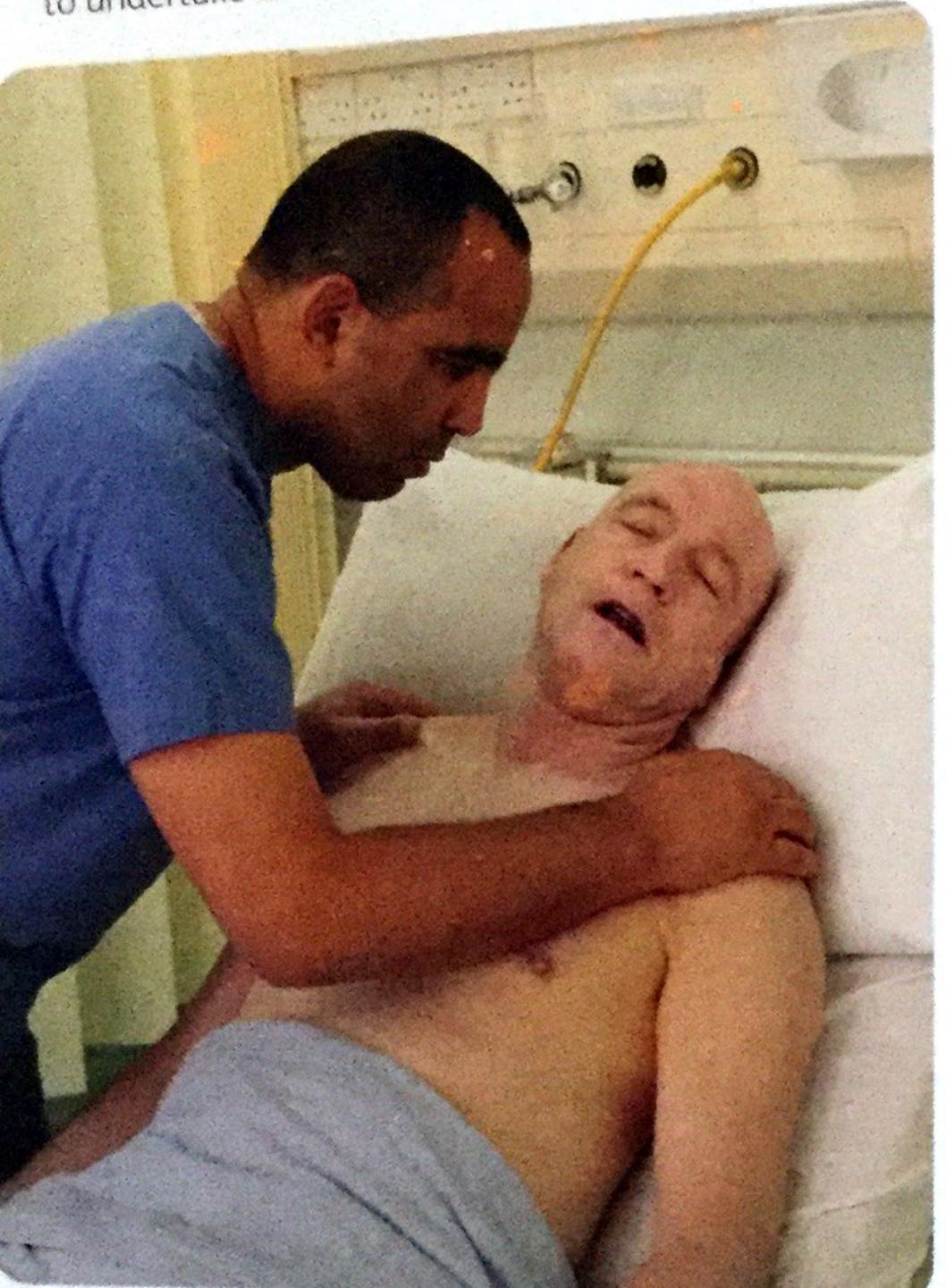


Figure 5.1 In-hospital resuscitation algorithm

loudly: "Are you alright?"

If other members of staff are nearby it will be possible to undertake actions simultaneously.



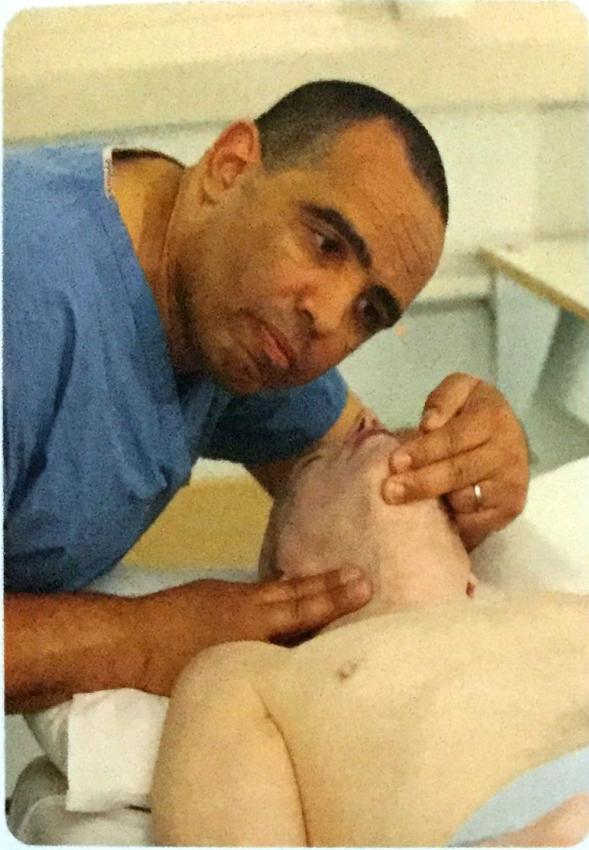


Figure 5.4 Simultaneous check for breathing and carotid pulse

- Listen at the victim's mouth for breath sounds
- Feel for air on your cheek.
- If trained and experienced in the assessment of sick patients, check for breathing and assess the carotid pulse at the same time (Figure 5.4).
- Agonal breathing (occasional, irregular gasps) 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. Agonal breathing and limb movement can also occur during chest compressions as cerebral perfusion improves, but is not indicative of a return of spontaneous circulation.
- Starting CPR on a very sick patient with a low cardiac output rarely causes significant damage and can be beneficial. However, a delay in diagnosis of cardiac arrest and starting CPR will effect survival adversely and must be avoided.
- If there is any doubt about the presence or absence of signs of life or a pulse, start CPR immediately (step 4B). Delays in diagnosis of cardiac arrest and starting CPR will affect survival adversely and must be avoided. Several studies show that even trained healthcare staff cannot assess the breathing and pulse sufficiently reliably to confirm cardiac arrest. If the patient is already attached to monitoring in a critical care area this will add to, rather than replace, the assessment for signs of life.

4A If there are signs of life or a pulse

- Urgent medical assessment is required. Depending on

the local protocols, this may take the form of a resuscitation team. While awaiting the team, assess and treat the patient using the ABCDE approach, give oxygen, attach monitoring and insert an intravenous cannula.

- 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 are no signs of life and no pulse

- Start CPR and get a colleague to call the resuscitation team and collect the resuscitation equipment and a defibrillator.
- If alone, leave the patient to get help and equipment.
- Give 30 chest compressions followed by 2 ventilations.
- The correct hand position for chest compression is the middle of the lower half of the sternum.
- This hand position can be found quickly if you have been taught to 'place the heel of one hand in the centre of the chest with the other hand on top' and your teaching included a demonstration of placing hands in the middle of the lower half of the sternum (Figure 5.5)
- Ensure high quality chest compressions:
 - Depth of 5–6 cm
 - Rate of 100–120 compressions min⁻¹



Figure 5.5 Hand position for chest compressions

- Allow the chest to recoil completely after each compression
- Take approximately the same amount of time for compression and relaxation
- Minimise any interruptions to chest compression (hands-off time).
- If available, use a prompt and/or feedback device to help ensure high quality chest compressions. Do not rely on a palpable carotid or femoral pulse to assess effective arterial flow.
- Each time compressions are resumed, place your hands without delay in the centre of the chest.
- The quality of chest compressions delivered by a single rescuer can decay over time, often even before they feel tired. If there are enough team members, this person should change about every 2 min or sooner if they are unable to maintain high quality chest compressions. This change should be done with minimal interruption to compressions. This should be done during planned pauses in chest compression such as during rhythm assessment.
- Use whatever equipment is available immediately for airway and ventilation; for example, a pocket mask with an oral airway and oxygen, or a self-inflating bag-mask (Figure 5.6), or a supraglottic airway (e.g. laryngeal mask airway (LMA)) and bag. In practice patients can have several airway techniques used



Figure 5.6 Two-person bag-mask ventilation during CPR

during a cardiac arrest as equipment arrives and according to the skills of the rescuer.

- If airway and ventilation equipment is unavailable, consider mouth-to-mouth ventilation. If there are clinical reasons to avoid mouth-to-mouth contact, or you are or unable to do this, do chest compressions until help or airway equipment arrives. There are often good clinical reasons to avoid mouth-to-mouth ventilation in clinical settings, and it is therefore not commonly used, but there will be situations where giving mouth-to-mouth breaths could be life-saving. A pocket mask with filter, or a barrier device with one-way valve will minimise infection risk during rescue breathing.
- Use an inspiratory time of about 1 s and give enough volume to produce a visible rise of the chest wall. Add supplemental oxygen as soon as possible.
- Avoid rapid or forceful breaths.
- Tracheal intubation should be attempted only by those who are trained, competent and experienced in this skill, and can insert the tracheal tube with minimal interruption (less than 5 s) to chest compressions. Waveform capnography should be routinely used for confirming that a tracheal tube is in the patient's airway and subsequent monitoring during CPR. Waveform capnography can also be used to monitor the quality of CPR, as an indicator of a return of spontaneous circulation (ROSC) and as a prognostic indicator. Once the patient's trachea has been intubated, continue chest compressions uninterrupted (except for defibrillation or pulse checks when indicated), at a rate of 100–120 min⁻¹, and ventilate the lungs at approximately 10 breaths min⁻¹ (i.e. do not stop chest compressions for ventilation). If a supraglottic airway (e.g. LMA) has been inserted it may also be possible to ventilate the patient without stopping chest compressions.
- When the defibrillator arrives, apply the self-adhesive defibrillation electrodes to the patient and analyse the rhythm. These should be applied whilst chest compressions are ongoing (Figure 5.7). The use of adhesive electrode pads will enable rapid assessment of heart rhythm compared with the use of ECG electrodes.
- For manual defibrillation, minimise the interruption to CPR to deliver a shock (Chapter 9). Using a manual defibrillator, it is possible to reduce the pause between stopping and restarting of chest compressions to less than 5 s.
- Once the pads are applied, pause briefly for a rapid rhythm check – aim for a pause in chest compression of less than 5 s for the rhythm check. If the rhythm is ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT), restart chest compressions. All other team members must now be informed to stand clear of the patient whilst the defibrillator is charged and a safety check performed. The defibrillator is charged and stop chest compressions, defibrillation is immediate.



Figure 5.7 Maintain chest compressions while adhesive pads are applied

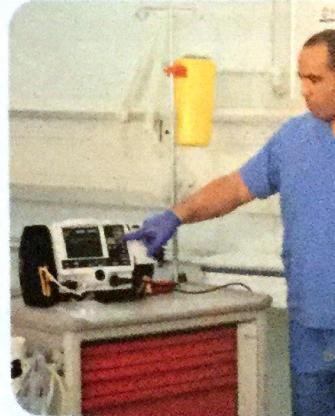


Figure 5.8 Continue chest compressions while defibrillator charged - all others stand clear

and a safety check performed. The defibrillator is charged and stop chest compressions, defibrillation is immediate.

- The length of the pre-shock pause between stopping chest compressions and giving the shock, is inversely proportional to the chance of successful defibrillation. Every second of the pre-shock pause reduces the chance of successful defibrillation by 7%.



Figure 5.7 Maintain chest compressions while self-adhesive pads are applied

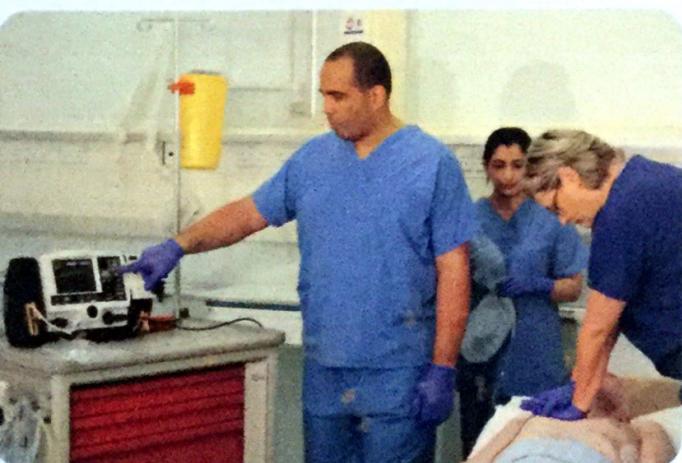


Figure 5.8 Continue chest compressions whilst defibrillator charged - all other team members must stand clear

and a safety check performed (Figure 5.8). Once the defibrillator is charged and the safety check completed, stop chest compressions, deliver the shock and restart chest compressions immediately.

The length of the pre-shock pause, the interval between stopping chest compressions and delivering a shock, is inversely proportional to the chance of successful defibrillation. Every 5 s increase in the duration of the pre-shock pause almost halves the chance of successful defibrillation, therefore it is critical to minimise this interval.

- If using an automated external defibrillator (AED) switch on the machine and follow the AED's audio-visual prompts.
- Rescuers must not compromise on safety. All actions should be planned by the team before pausing 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 a decision is made what to do when compressions are next paused.
- Continue resuscitation until the resuscitation team arrives or the patient shows signs of life. Follow the Advanced Life Support algorithm (Chapter 6).
- 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).
- Use a watch or clock for timing between rhythm checks. Any interruption to CPR should be planned. Assess the cardiac rhythm about every two minutes.
- 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.
- Identify one person to be responsible for handover to the resuscitation team leader. Use a structured communication tool for handover (e.g. SBAR, RSVP).
- Locate the patient's records.

4C If he is not breathing and has a pulse (respiratory arrest)

- Ventilate the patient's lungs (as described above) and check for a pulse every minute.
- This diagnosis can be made only if you are confident in assessing breathing and pulse or the patient has other signs of life (e.g. warm and well-perfused, normal capillary refill).
- If there are any doubts about the presence of a pulse, start chest compressions and continue ventilations until more experienced help arrives.
- All patients in respiratory arrest will develop cardiac arrest if the respiratory arrest is not treated rapidly and effectively.

5 If the patient has a monitored and witnessed cardiac arrest

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area, or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help.

- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and, if appropriate check for a pulse and other signs of ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful. These initial three stacked shocks are considered as giving the first shock in the ALS algorithm.
- This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is already connected to a manual defibrillator – these circumstances are rare.
- A precordial thump rarely works. Delivery of a precordial thump must not delay calling for help or accessing a defibrillator. Consider a precordial thump only when it can be used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest. 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.

Summary learning

- The exact sequence of actions after in-hospital cardiac arrest depends on the location, skills of the first responders, number of responders, equipment available, and the hospital response system to cardiac arrest and medical emergencies.
- Check for responsiveness, normal breathing, signs of life and if trained a carotid pulse to confirm cardiac arrest. This should take less than 10 s. Call for help and start CPR with chest compressions.
- Deliver high quality chest compressions with a depth of 5–6 cm, rate of 100–120 min⁻¹, and allow complete recoil between compressions.
- Minimise interruptions to chest compressions for other interventions – this means all interruptions must be planned before stopping compressions.

My key take-home messages from this chapter

Advanced algorithm

- The Advanced Life Support
- Treatment of shockable arrhythmias
- Monitoring during CPR including waveform analysis
- Identification and treatment of non-shockable rhythms

Further reading

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Leading the way

To enable you to:

- Manage a cardiac arrest using the ALS algorithm
- Understand the importance of high-quality chest compressions during CPR
- Appropriately and promptly manage non-shockable rhythms
- Know when and how to give effective defibrillation
- Treat the reversible causes of cardiac arrest
- Understand the role of waveform analysis in monitoring during CPR

Introduction

Heart rhythms associated with cardiac arrest are either shockable (ventricular fibrillation/pulseless ventricular tachycardia) or non-shockable (asystole and pulseless electrical activity). In the management of these two groups of rhythms, the focus is on defibrillation in patients with VF/pVT, airway management and ventilation, identification and correction of reversible causes of arrest, and resuscitation.

The ALS algorithm (Figure 6.1) is a standardised approach to the management of cardiac arrest. This has the advantage of enabling the team to work together without protracted discussion. It enables each member of the team to know what they need to do to prepare for the next stage in the patient's care. Although the ALS algorithm is designed for use in hospital, some interventions may be indicated for out-of-hospital cardiac arrest.

The interventions that unquestionably improve survival are prompt and effective bystander CPR, high-quality chest compressions, and early defibrillation. In the majority of cases, advanced airways are still included on the algorithm because their importance to high quality, uninterrupted CPR cannot be overemphasised.

Advanced Life Support algorithm

Chapter
6

Contents

- The Advanced Life Support (ALS) algorithm
- Treatment of shockable and non-shockable rhythms
- Monitoring during CPR including waveform capnography
- Identification and treatment of reversible causes of cardiac arrest

Learning outcomes

To enable you to:

- Manage a cardiac arrest using the ALS algorithm
- Understand the importance of minimising interruptions to chest compressions during CPR
- Appropriately and promptly treat shockable and non-shockable rhythms
- Know when and how to give drugs during cardiac arrest
- Treat the reversible causes of cardiac arrest
- Understand the role of waveform capnography during CPR

Introduction

Heart rhythms associated with cardiac arrest are divided into two groups: shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)) and non-shockable rhythms (asystole and pulseless electrical activity (PEA)). The principle difference in the management of these two groups of arrhythmias is the need for attempted defibrillation in patients with VF/pVT. Subsequent actions, including chest compressions, airway management and ventilation, venous access, injection of adrenaline and the identification and correction of reversible factors, are common to both groups.

The ALS algorithm (Figure 6.1) is a standardised approach to cardiac arrest management. This has the advantage of enabling treatment to be delivered expediently and without protracted discussion. It enables each member of the resuscitation team to predict and prepare for the next stage in the patient's treatment, further enhancing efficiency of the team. Although the ALS algorithm is applicable to most cardiac arrests, additional interventions may be indicated for cardiac arrest in special circumstances (Chapter 12).

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander cardiopulmonary resuscitation (CPR), uninterrupted, high quality chest compressions, and early defibrillation for VF/pVT. Although drugs and advanced airways are still included among ALS interventions, they are of secondary importance to high quality, uninterrupted chest compressions and when appropriate early defibrillation. In the most recent ALS guidelines there is an increase in emphasis on monitoring during CPR, especially the use of waveform capnography, and the identification and treatment of reversible causes of cardiac arrest, including the use of focused cardiac and lung ultrasound assessment.

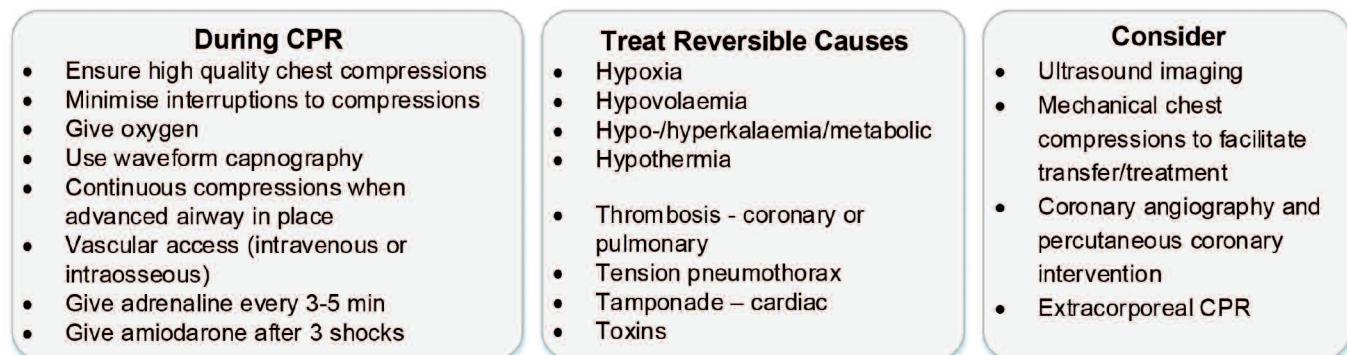
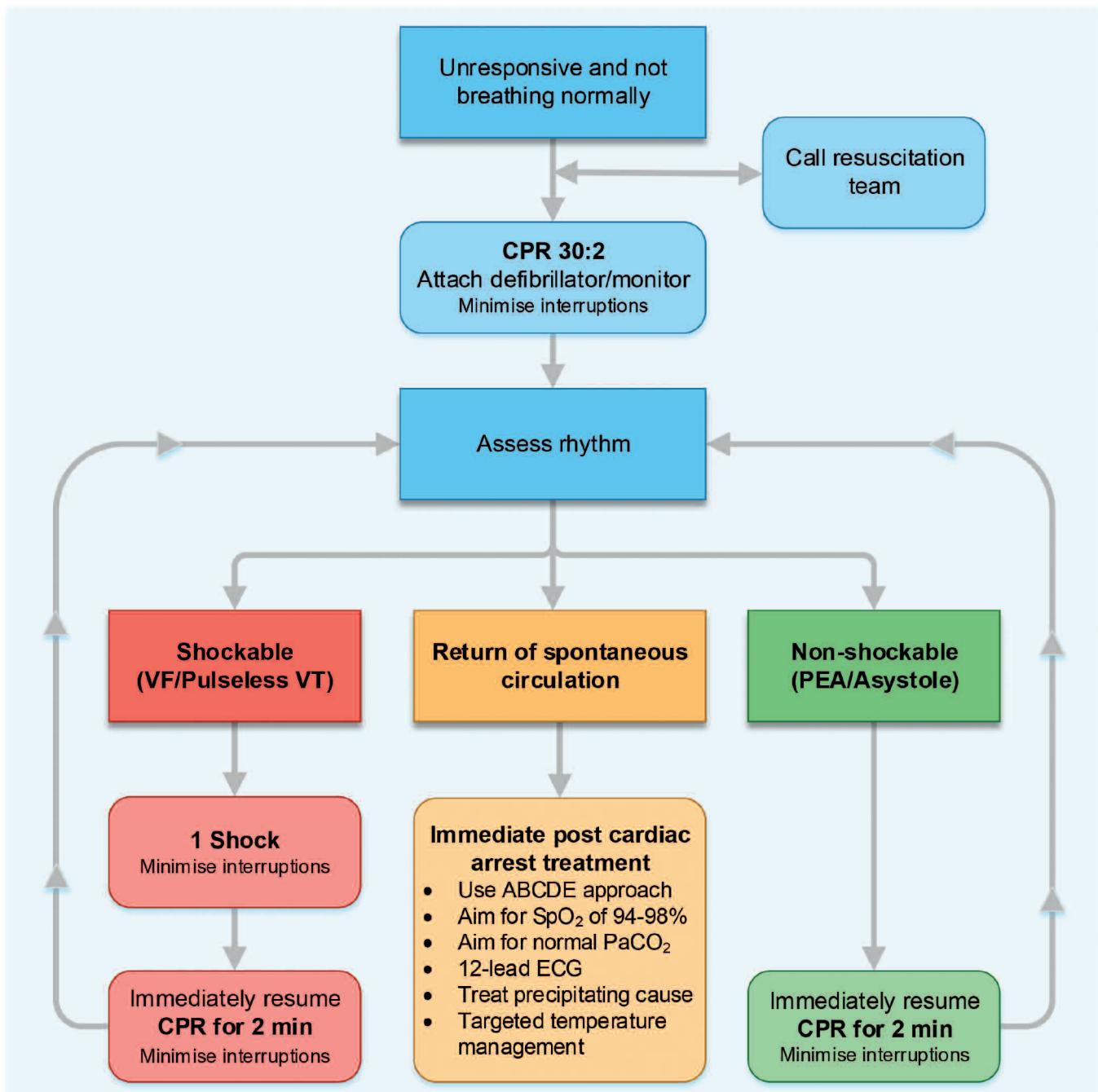


Figure 6.1 Adult Advanced Life Support algorithm

Shockable rhythms (VF/pVT)

The first monitored rhythm is VF/pVT in approximately 20% of cardiac arrests, both in-or out-of-hospital. VF/pVT will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA.

Treatment of shockable rhythms (VF/pVT)

A manual defibrillator is used in the sequence described below. Further information about defibrillation can be found in Chapter 9.

1. Confirm cardiac arrest – check for signs of life or if trained to do so, normal 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/pVT from the ECG. This pause in chest compressions should be brief and no longer than 5 s.
6. Resume chest compressions immediately; warn all rescuers **other than the individual performing the chest compressions** to “stand clear” and remove any oxygen delivery device as appropriate.
7. The designated person selects the appropriate energy on the defibrillator and presses the charge button (Figure 6.2). Choose an energy setting of at least 150 J for the first shock, the same or a higher energy for subsequent shocks, or follow the manufacturer’s guidance for the particular defibrillator.



Figure 6.2 Continuous chest compressions during charging with a manual defibrillator. Warn all rescuers other than the individual performing the chest compressions to “stand clear”. This patient has a tracheal tube and remains connected to the oxygen supply. A CPR quality and feedback device is used by the rescuer giving chest compressions.

8. Ensure that the rescuer giving the compressions is the only person touching the patient.
9. Once the defibrillator is charged and the safety check is complete, tell the rescuer doing the chest compressions to “stand clear”; when clear, give the shock (Figure 6.3).



Figure 6.3 Shock delivery. No one is touching the patient during shock delivery

10. After shock delivery immediately restart CPR using a ratio of 30:2, starting with chest compressions. Do not pause to reassess the rhythm or feel for a pulse. This pause in chest compressions should be brief and no longer than 5 s.
11. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.
12. Pause briefly to check the monitor.
13. If VF/pVT, repeat steps 6 – 12 above and deliver a second shock.
14. If VF/pVT persists, repeat steps 6 – 8 above and deliver a third shock. Resume chest compressions immediately. Give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR. Withhold adrenaline if there are signs of return of spontaneous circulation (ROSC) during CPR.
15. Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists.
16. Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).
17. If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of ROSC (check for signs of life, a central pulse and end-tidal CO₂ if available).
 - a. If there is ROSC, start post-resuscitation care.
 - b. If there are no signs of ROSC, continue CPR and switch to the non-shockable algorithm.
18. 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. Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation. Chest compressions are resumed immediately after delivering a shock (without checking the rhythm or a pulse) because:

- Even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation. The duration of asystole before ROSC can be longer than two minutes in as many as 25% of successful shocks.
- The delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.
- If a perfusing rhythm has been restored, giving chest compressions does not increase the chance of VF recurring.
- In the presence of post-shock asystole chest compressions may usefully induce VF.

Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs during CPR. The use of both adrenaline and amiodarone is currently recommended, based largely on an increased short-term survival in humans.

The first dose of adrenaline is given during the 2-min period of CPR after delivery of the third shock.

Give amiodarone 300 mg after three defibrillation attempts. Do not stop CPR to check the rhythm before giving drugs unless there are clear signs of ROSC.

Subsequent doses of adrenaline are given after alternate 2-min loops of CPR (which equates to every 3–5 min) for as long as cardiac arrest persists. If VF/pVT persists, or recurs, a further dose of 150 mg amiodarone may be given after a total of five defibrillation attempts. 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.

It is important in shock-refractory VF/pVT to check the position and contact of the defibrillation pads. The duration of any individual resuscitation attempt is a matter of clinical judgement, and should take into account the perceived prospect of a successful outcome. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing as long as the patient remains in identifiable VF/pVT.

When the rhythm is checked 2 min after giving a shock, if a non-shockable rhythm is present, and the rhythm is one that could be compatible with a pulse, try to palpate a central pulse and look for other evidence of ROSC (e.g. sudden increase in end-tidal CO₂ or evidence of cardiac output on any invasive monitoring equipment). Rhythm checks must be brief, and pulse checks undertaken only if a rhythm that could be compatible with a pulse is

observed. If a rhythm compatible with a pulse is seen during a 2-min period of CPR, do not interrupt chest compressions to palpate a pulse unless the patient shows signs of life suggesting ROSC. If there is any doubt about the presence of a palpable pulse, resume CPR. If the patient has ROSC, begin post-resuscitation care. If the patient's rhythm changes to asystole or PEA, see non-shockable rhythms, below.

If there is doubt about whether the rhythm is asystole or extremely fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation as continuing high quality CPR may improve the amplitude and frequency of the VF and improve the chance of subsequent successful defibrillation to a perfusing rhythm. If the rhythm is clearly VF, attempt defibrillation.

Precordial thump

A precordial thump has a very low success rate for cardioversion of a shockable rhythm. Its routine use is therefore not recommended. Consider a precordial thump only when it can be used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest. 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.

Witnessed and monitored VF/pVT cardiac arrest

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area, or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks
- Rapidly check for a rhythm change and, if appropriate check for a pulse and other signs of ROSC after each defibrillation attempt
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

This three-shock strategy may also be considered for an initial, witnessed VF/pVT 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/pVT.

If this initial three-shock strategy is unsuccessful for a monitored VF/pVT cardiac arrest, follow the ALS algorithm and treat these three-shocks as if only the first single shock has been given. These initial three stacked shocks are considered as giving the first shock in the ALS algorithm (i.e. the first dose of adrenaline should be given after another two shock attempts if VF/pVT persists).

Amiodarone is given after three shock attempts irrespective of when they are given during the cardiac arrest (i.e. give amiodarone during the 2 min of CPR after the three stacked-shock attempts).

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity (other than ventricular tachyarrhythmia) that would normally be associated with a palpable pulse. These patients often have some mechanical myocardial contractions but they are too weak to produce a detectable pulse or blood pressure. PEA may be caused by reversible conditions that can be treated (see below). Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated quickly and effectively.

Asystole is the absence of electrical activity on the ECG trace. During CPR, ensure the ECG pads are attached to the chest and the correct monitoring mode is selected. Ensure the gain setting is appropriate. Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because in this situation ventricular standstill may be treated effectively by cardiac pacing. Attempts to pace true asystole are unlikely to be successful.

Treatment for PEA and asystole

- Start CPR 30:2.
- Give adrenaline 1 mg IV/IO as soon as intravascular access is achieved.
- Continue CPR 30:2 until the airway is secured - then continue chest compressions without pausing during ventilation.
- Recheck the rhythm after 2 min:
 - If electrical activity compatible with a pulse is seen, check for a pulse and/or signs of life:
 - if a pulse and/or signs of life are present, start post resuscitation care
 - if no pulse and/or no signs of life are present (PEA OR asystole):
 - continue CPR
 - recheck the rhythm after 2 min and proceed accordingly
 - give further adrenaline 1 mg IV every 3–5 min (during alternate 2-min loops of CPR)
 - If VF/pVT at rhythm check, change to shockable side of algorithm.

During CPR

During the treatment of persistent VF/pVT or PEA/asystole, emphasis is placed on high quality chest compressions

between defibrillation attempts, recognising and treating reversible causes (4 Hs and 4 Ts), obtaining a secure airway and vascular access.

During CPR with a 30:2 ratio, the underlying rhythm may be seen clearly on the monitor as compressions are paused to enable ventilation. If VF is seen during this brief pause (whether on the shockable or non-shockable side of the algorithm) do not attempt defibrillation at this stage; instead, continue with CPR until the 2-min period is completed. Knowing that the rhythm is VF, the team should be fully prepared to deliver a shock with minimal delay at the end of the 2-min period of CPR.

Maintain high quality, uninterrupted chest compressions

The quality of chest compressions and ventilations are important determinants of outcome, yet are frequently performed poorly by healthcare professionals. Avoid interruptions in chest compressions because pauses cause coronary perfusion pressure to decrease substantially. Ensure compressions are of adequate depth (5–6 cm) and rate (100–120 min⁻¹), and ensure there is full recoil of the chest at the end of each compression.

As soon as the airway is secured, continue chest compressions without pausing during ventilation. To reduce fatigue, change the individual undertaking compressions every 2 min or earlier if necessary.

Airway and ventilation

A bag-mask, or preferably, a supraglottic airway (SGA) (e.g. laryngeal mask airway, i-gel) should be used in the absence of personnel skilled in tracheal intubation (Chapter 7). Once a SGA has been inserted, attempt to deliver continuous chest compressions, uninterrupted during ventilation. Ventilate the lungs at 10 breaths min⁻¹; do not hyperventilate the lungs. If excessive gas leakage causes inadequate ventilation of the patient's lungs, interrupt chest compressions to enable ventilation (using a compression-ventilation ratio of 30:2).

No studies have shown that tracheal intubation increases survival after cardiac arrest. Incorrect placement of the tracheal tube is common in cardiac arrest if intubation is attempted by unskilled personnel. Tracheal intubation should be attempted only if the healthcare provider is properly trained and has regular, ongoing experience with the technique. Avoid stopping chest compressions during laryngoscopy and intubation; if necessary, a brief pause in chest compressions may be required as the tube is passed between the vocal cords, but this pause should not exceed 5 s. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until after ROSC. After intubation, confirm correct tube position with waveform capnography, and secure the tube adequately. Once the patient's trachea has been intubated, continue chest compressions, at a rate of 100–120 min⁻¹ without pausing during ventilation.

Monitoring during CPR

Several methods can be used to monitor the patient during CPR and potentially help guide ALS interventions. These include:

- **Clinical signs** such as breathing efforts, movements and eye opening can occur during CPR. These can indicate ROSC and require verification by a rhythm and pulse check, but can also occur because high quality CPR can generate a sufficient circulation to restore signs of life including consciousness.
- **Pulse checks** can be used to identify ROSC when there is an ECG rhythm compatible with a pulse, but may not detect pulses in those with low cardiac output states and a low blood pressure. The value of attempting to feel arterial pulses during chest compressions to assess the effectiveness of chest compressions is unclear. A pulse that is felt in the femoral triangle can indicate venous rather than arterial blood flow. Carotid pulsation during CPR does not necessarily indicate adequate myocardial or cerebral perfusion.
- **Monitoring the heart rhythm** through pads, paddles or ECG electrodes is a standard part of ALS. Motion artefacts prevent reliable heart rhythm assessment during chest compressions.
- **End-tidal carbon dioxide measured with waveform capnography.** The use of waveform capnography during CPR is addressed in more detail below.
- **Feedback or prompt devices** can monitor CPR quality data such as compression rate and depth during CPR, and provide real-time feedback to rescuers (Figure 6.2). Be aware that some devices fail to compensate for compression of the underlying mattress during CPR on a bed when providing feedback. The use of these devices should be as part of a broader system of care that includes CPR quality improvement initiatives such as debriefing based on the data collected.
- **Blood sampling and analysis** during CPR can be used to identify potentially reversible causes of cardiac arrest. Avoid finger prick samples because they may not be reliable; instead, use samples from veins or arteries. Blood gas values are difficult to interpret during CPR. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state. Analysis of central venous blood may provide a better estimation of tissue pH.
- **Invasive cardiovascular monitoring** in critical care settings (e.g. continuous arterial blood pressure and central venous pressure monitoring). Invasive arterial pressure monitoring will enable the detection of even very low blood pressure values when ROSC is achieved.
- The use of **focused echocardiography/ultrasound** to identify and treat reversible causes of cardiac arrest, and identify low cardiac output states ('pseudo-PEA') is discussed below.

Waveform capnography during advanced life support

Carbon dioxide (CO_2) is a waste product of metabolism; approximately 400 L are produced each day. It is carried in the blood to the lungs where it is exhaled. The concentration in the blood is measured as the partial pressure of CO_2 (PCO_2) and in arterial blood (PaCO_2) is normally 5.3 kPa (range 4.7–6.0 kPa). The concentration of CO_2 can also be measured in expired air and is expressed as either a percentage by volume or as a partial pressure, both of which are very similar numerically. The concentration varies throughout expiration, being maximal at the end and it is this value, the end-tidal CO_2 that is most useful.

End-tidal carbon dioxide is the partial pressure of CO_2 at the end of an exhaled breath. It reflects cardiac output and pulmonary blood flow (CO_2 is transported by the venous system to the right side of the heart and then pumped to the lungs by the right ventricle), as well as the ventilation minute volume. During CPR, end-tidal CO_2 values are low, reflecting the low cardiac output generated by chest compression. Waveform capnography enables continuous real time end-tidal CO_2 to be monitored during CPR. It works most reliably in patients who have a tracheal tube, but can also be used with a supraglottic airway device or bag mask.

The role of waveform capnography during CPR

- **Ensuring tracheal tube placement in the trachea.** Correct tube placement also relies on observation and auscultation to ensure both lungs are ventilated.
- **Monitoring ventilation rate during CPR** and avoiding hyperventilation.
- **Monitoring the quality of chest compressions** during CPR. End-tidal CO_2 values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value.
- **Identifying ROSC during CPR.** An increase in end-tidal CO_2 during CPR may indicate ROSC, and prevent unnecessary and potentially harmful administration of adrenaline in a patient with ROSC. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.
- **Prognostication during CPR.** Precise values of end-tidal CO_2 depend on several factors including the cause of cardiac arrest, bystander CPR, chest compression quality, ventilation rate and volume, time from cardiac arrest and the use of adrenaline. Values are higher after an initial asphyxial arrest and with bystander CPR, and decline over time after cardiac arrest. Low end-tidal CO_2 values during CPR have been associated with lower ROSC rates and increased mortality, and high values with better ROSC and survival. End-tidal CO_2 values should be considered only as part of a multimodal approach to decision-making for prognostication during CPR.

Practical aspects of waveform capnography

A capnograph is a device that displays a waveform of the concentration of CO₂ as it varies during expiration and a numerical value. This is usually referred to as waveform capnography and is the most useful display for clinical use.

Equipment

- Portable monitors that measure the end-tidal CO₂ and display the waveform are now readily available (Figure 6.4).



Figure 6.4 Example of a monitor screen showing end-tidal CO₂ waveform (waveform capnography). This patient had a return of spontaneous circulation, and chest compressions have stopped. The ECG shows sinus rhythm with a rate of 72 min⁻¹. The end-tidal CO₂ waveform shows an end-tidal CO₂ value of 4.3 kPa, and a ventilation rate (RR) of 9 min⁻¹. On clinical assessment the patient had a palpable carotid pulse.

- Most capnographs use side-stream sampling. A connector (T-piece) is placed in the breathing system, usually on the end of the tracheal tube or supraglottic airway device. This has a small port on the side to which is attached a fine bore sampling tube. A continuous sample of gas is aspirated (about 50 mL min⁻¹) and analysed by using the property of absorption of infrared light.
- An alternative system is mainstream sampling in which the infrared source and detector are contained within a cell or cuvette which is placed directly in the breathing system, usually between the tracheal tube or supraglottic airway device and circuit. Gas is analysed as it passes through the sensor and none is removed from the system.

The capnography waveform

The shape of the waveform that is displayed will depend upon the time scale and the patient's condition. (Figure 6.5).

A-B: This is the baseline and indicates the end of inspiration. In effect the concentration of CO₂ is being measured in air (or whatever gas is being delivered) which of course is virtually zero.

B-C: This represents the start of expiration, with a rapid rise in

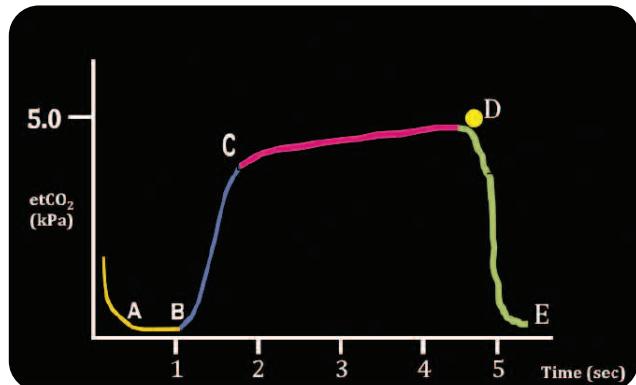


Figure 6.5 Typical capnography waveform from a single breath (note the time scale)

the concentration of expired CO₂. The expired gas initially contains no CO₂ as it has come from the anatomical dead space (i.e. the volume of the respiratory tract that does not take part in gas exchange (larynx, trachea, main bronchi)). As gas from alveoli that contains CO₂ starts to be expired, it mixes with this gas and the concentration rises.

C-D: Eventually the alveolar plateau is reached. This represents gas from alveoli taking part in gas exchange. The slight gradual increase during this phase is due to the fact that not all alveoli empty at the same rate.

D: At the end of expiration the concentration of CO₂ is maximal, this is the end-tidal CO₂. In healthy patients, this is normally 4.8 kPa (4.3–5.5 kPa), slightly lower than would be obtained if the arterial PaCO₂ was measured at the same point in time.

D-E: As inspiration starts, air containing no CO₂ is mixed with a small amount of residual expired gas in the breathing circuit. This is rapidly diluted until gas containing no CO₂ is being inspired.

End-tidal CO₂ during CPR

Reliable end-tidal CO₂ monitoring during CPR will usually require a tracheal tube or, when there is a good seal, a supraglottic airway device. Figure 6.6 shows how end-tidal CO₂ values can change during CPR.

- The presence of an end-tidal CO₂ waveform indicates the tracheal tube is positioned in the airway. Check that both lungs are being ventilated by looking and by listening with a stethoscope as the tube may have passed down into a bronchus and only be ventilating one lung.
- The rate of ventilation is 10 min⁻¹ (Figure 6.6).
- Soon after the second defibrillation attempt there is a significant increase in the end-tidal CO₂ value during CPR. This is often the first indicator of ROSC and often precedes other indicators such as the presence of a palpable pulse. It is a result of the improved circulation transporting accumulated CO₂ from the tissues to the lungs. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

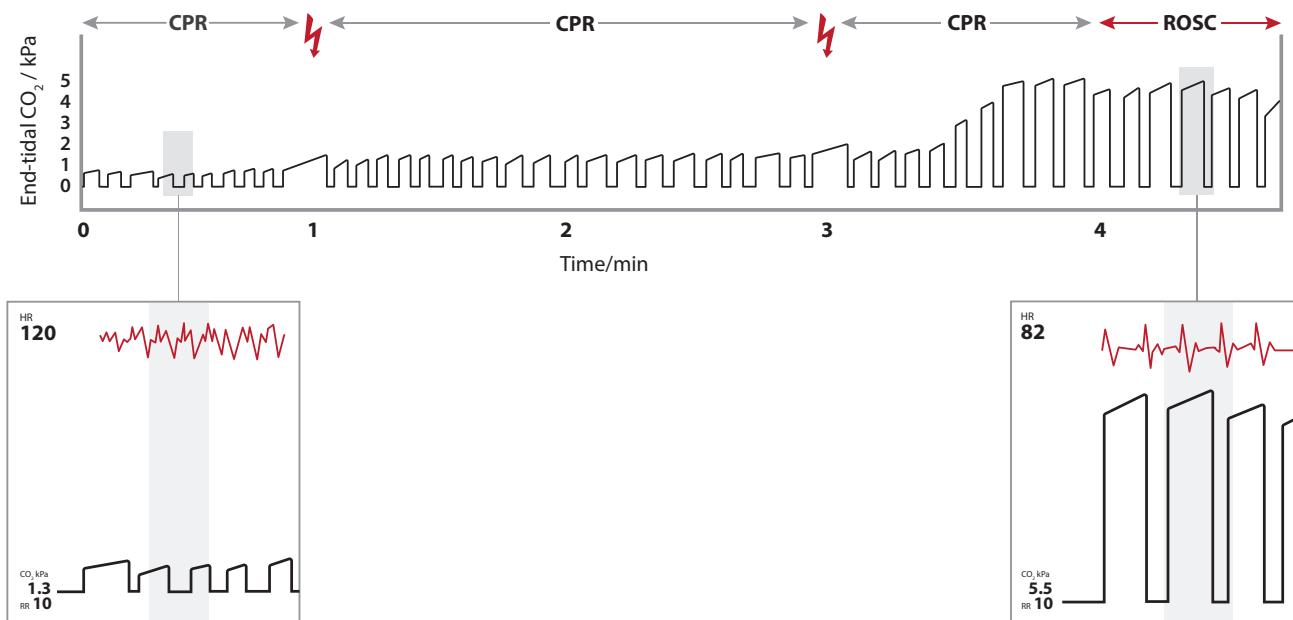


Figure 6.6 Waveform capnography showing changes in the end-tidal CO_2 during CPR and after ROSC. The boxes show the monitor displays at the times indicated. In this example the patient's trachea is intubated at zero minutes. The patient is ventilated at 10 min^{-1} and given chest compressions (indicated by CPR) at about two per second. A minute after tracheal intubation, there is pause in chest compressions and ventilation followed by a defibrillation attempt, and chest compressions and ventilation then continue. Higher-quality chest compressions lead to an increased end-tidal CO_2 value. There is a further defibrillation attempt after 2 min of chest compressions. There are then further chest compressions and ventilation. There is a significant increase in the end-tidal CO_2 value during chest compressions and the patient starts moving and eye opening. Chest compressions are stopped briefly, the monitor shows sinus rhythm and there is a pulse indicating ROSC. Ventilation continues at 10 min^{-1} .

CPR – cardiopulmonary resuscitation; ROSC – return of spontaneous circulation; HR – heart rate; RR – respiratory rate

- Failure to achieve an end-tidal CO_2 value $> 1.33 \text{ kPa}$ (10 mmHg) after 20 min of CPR is associated with a poor outcome in observational studies. A specific end-tidal CO_2 value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO_2 values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

Signs of life during CPR

If signs of life (such as regular respiratory effort, movement) or readings from patient monitors compatible with ROSC (e.g. increase in end-tidal CO_2 or arterial blood pressure waveform) appear during CPR, stop CPR briefly and check the monitor. If a rhythm compatible with a pulse is present, check for a pulse. If a pulse is palpable, continue post-resuscitation care and/or treatment of peri-arrest arrhythmias if appropriate. If no pulse is present, continue CPR.

Vascular access

The role of drugs during cardiac arrest is uncertain. Some patients will already have intravenous access before they have a cardiac arrest. If this is not the case ensure CPR had started and defibrillation, if appropriate, attempted before considering vascular access. Although peak drug concentrations are higher and circulation times shorter when drugs are injected into a central venous catheter compared with a peripheral cannula, insertion of a central

venous catheter requires interruption of CPR and is associated with several potential complications. Peripheral venous cannulation is quicker, easier, and safer. Drugs injected peripherally must be followed by a flush of at least 20 mL of fluid and elevation of the extremity for 10–20 s to facilitate drug delivery to the central circulation.

If IV access is difficult or impossible, consider the IO route (Figure 6.7). Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a vein.

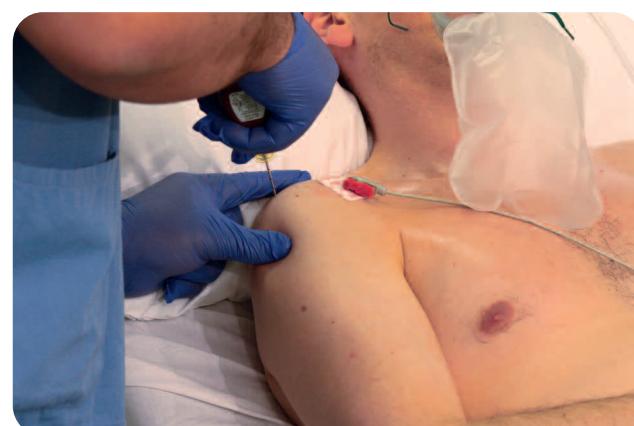


Figure 6.7 Intraosseous device insertion into proximal humerus

Use of intraosseous access during resuscitation

- The three main insertion sites for IO access recommended for use in adults are the proximal humerus, proximal tibia and distal tibia.
- Contraindications to IO access include trauma, infection or a prosthesis (e.g. joint replacement) at the target site, recent IO access (previous 48 h) in the same limb including a failed attempt, or a failure to identify the anatomical landmarks.
- Training in the specific device to be used in clinical practice is essential. Site of insertion, identification of landmarks and technique for insertion will differ depending on the device being used.
- Once inserted, confirm correct placement before delivery of drugs or infusion of fluids. Attempt to aspirate from the needle; presence of IO blood indicates correct placement, absence of aspirate does not necessarily imply a failed attempt.
- Flush the needle to ensure patency and observe for leakage or extravasation. This is best achieved using an extension set flushed with 0.9% saline attached to the hub of the needle before use.
- Once IO access has been confirmed, resuscitation drugs including adrenaline and amiodarone can be infused. Fluids and blood products can also be delivered but pressure will be needed to achieve reasonable flow rates using either a pressure bag or a syringe.
- Follow the manufacturer's guidance both for securing the needle and the maximum length of time it can be left in place.
- Complications associated with IO access include extravasation into the soft tissues surrounding the insertion site, dislodgement of the needle, compartment syndrome due to extravasation, fracture or chipping of the bone during insertion, pain related to the infusion of drugs/fluid, fat emboli and infection/osteomyelitis.

Identification and treatment of reversible causes

Potential causes or aggravating factors for which specific treatment exists must be considered during any cardiac arrest. For ease of memory, these are divided into two groups of four based upon their initial letter - either H or T (Figure 6.8). More details on many of these conditions are covered in Chapter 12.

- Hypoxia
- Hypovolaemia
- Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders
- Hypothermia
- Thrombosis (coronary or pulmonary)
- Tension pneumothorax

- Tamponade - cardiac
- Toxins

The four Hs

Minimise the risk of hypoxia by ensuring that the patient's lungs are ventilated adequately with 100% oxygen during CPR. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described in Chapter 7, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus. If ROSC is achieved adjust the inspired oxygen to target an oxygen saturation of 94–98% (Chapter 13).

Pulseless electrical activity caused by hypovolaemia is due usually to severe haemorrhage. Evidence of haemorrhage may be obvious, (e.g. trauma (Chapter 12)), or occult (e.g. gastrointestinal bleeding, or rupture of an aortic aneurysm). Intravascular volume should be restored rapidly with fluid and blood, coupled with urgent interventions to stop the haemorrhage.

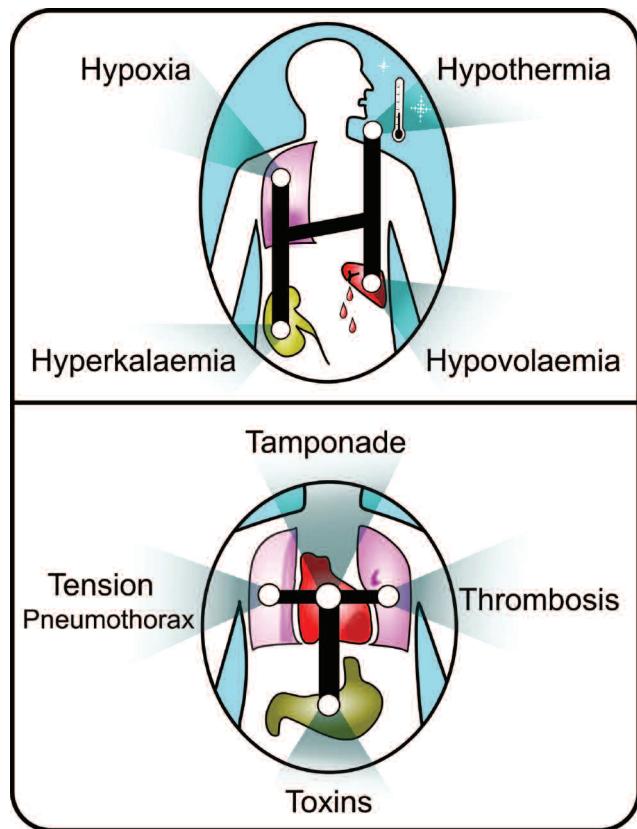
Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history (e.g. renal failure (Chapter 12)). Intravenous calcium chloride is indicated in the presence of hyperkalaemia, hypocalcaemia, and calcium channel-blocker overdose.

Suspect hypothermia in any drowning incident (Chapter 12); use a low reading thermometer.

The four Ts

Coronary thrombosis is a common cause of cardiac arrest. If an acute coronary syndrome is expected as the cause of a refractory cardiac arrest, it may be feasible to perform percutaneous coronary angiography and percutaneous coronary intervention during ongoing CPR. This would require an automated mechanical chest compression device and/or extracorporeal CPR to maintain a circulation during the procedure. A decision to transport a patient with ongoing CPR should take into consideration a realistic chance of survival (e.g. witnessed cardiac arrest with initial shockable rhythm (VF/pVT) and bystander CPR). Intermittent ROSC also strongly favours a decision to transport.

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause of cardiac arrest consider giving a fibrinolytic drug immediately. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported, even in cases requiring in excess of 60 min of CPR. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts. In some settings extracorporeal CPR, and/or surgical or mechanical thrombectomy can also be used to treat pulmonary embolism.

**Figure 6.8 The four Hs and four Ts**

A tension pneumothorax may be the primary cause of PEA. The diagnosis is made clinically. Decompress rapidly by thoracostomy or needle thoracocentesis and then insert a chest drain.

Cardiac tamponade is difficult to diagnose because the typical signs of distended neck veins and hypotension cannot be assessed during cardiac arrest. Focused cardiac ultrasound performed during CPR can be used to diagnose a pericardial effusion during CPR (see below). Cardiac arrest after penetrating chest trauma or after cardiac surgery should raise strong suspicion of tamponade and the need for resuscitative thoracotomy should be considered in this setting (Chapter 12).

In the absence of a specific history of accidental or deliberate ingestion, poisoning by therapeutic or toxic substances may be difficult to detect but in some cases may be revealed later by laboratory investigations (Chapter 12). Where available, the appropriate antidotes should be used but most often treatment is supportive.

Ultrasound during advanced life support

In skilled hands, focused echocardiography/ultrasound can be useful for the detection of potentially reversible causes of cardiac arrest (e.g. cardiac tamponade, pulmonary embolism, ischaemia (regional wall motion abnormality), aortic dissection, hypovolaemia,

**Figure 6.9 Use of focused cardiac ultrasound during advanced life support**

pneumothorax). The integration of ultrasound into advanced life support requires considerable training if interruptions to chest compressions are to be minimised. A sub-xiphoid probe position is recommended (Figure 6.9). 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. The Focused Echocardiography in Emergency Life Support (FEEL) course provides a valuable introduction to focused echocardiography/ultrasound in this setting.

The use of automated mechanical chest compression devices

Automated mechanical chest compression devices should not be used routinely to replace manual chest compressions. Automated mechanical chest compression devices may enable the delivery of high quality compressions in circumstances where this is not possible with manual compressions – CPR in a moving ambulance where safety is at risk, prolonged CPR (e.g. hypothermic arrest), and CPR during certain procedures (e.g. coronary angiography or preparation for extracorporeal CPR (ECPR)). Avoid interruptions to compressions during device deployment. Healthcare personnel who use mechanical CPR devices should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills.

Extracorporeal CPR

Extracorporeal resuscitation techniques require vascular access and a circuit with a pump and oxygenator and can provide a circulation of oxygenated blood to restore tissue perfusion. This has the potential to buy time for restoration of an adequate spontaneous circulation, and treatment of reversible underlying conditions. This is commonly called extracorporeal life support (ECLS), and more specifically ECPR when used during cardiac arrest. These techniques are becoming more commonplace and have been used for

both in-hospital and out-of-hospital despite limited observational data in selected patient groups. Observational studies suggest ECPR is associated with improved survival when there is a reversible cause for cardiac arrest (e.g. myocardial infarction, pulmonary embolism, severe hypothermia, poisoning), there is little comorbidity, the cardiac arrest is witnessed, the individual receives immediate high quality CPR, and ECPR is best implemented early and certainly within 1 h of collapse. The implementation of ECPR requires considerable resource and training, and availability is therefore limited.

The duration of a resuscitation attempt

If attempts at obtaining ROSC are unsuccessful the resuscitation team leader should discuss stopping CPR with the team. The decision to stop CPR requires clinical judgement and a careful assessment of the likelihood of achieving ROSC.

The duration of any resuscitation attempt should be based on the individual circumstances of the case and is a matter of clinical judgement, taking into consideration the circumstances and the perceived prospect of a successful outcome. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing, as long as the patient remains in VF/pVT, or there is a potentially reversible cause that can be treated. The use of mechanical compression devices and ECPR techniques make prolonged attempts at resuscitation feasible in selected patients. It is generally accepted that asystole for more than 20 min in the absence of a reversible cause and with ongoing ALS constitutes reasonable grounds for stopping further resuscitation attempts, although a shorter or longer time could be appropriate based on the circumstances of cardiac arrest.

Diagnosing death after unsuccessful resuscitation

If CPR is unsuccessful at achieving ROSC and a decision is made to discontinue CPR efforts, after stopping CPR observe the patient for a minimum of 5 min before confirming death. The absence of mechanical cardiac function is normally confirmed using a combination of the following:

- absence of a central pulse on palpation
- absence of heart sounds on auscultation.

One or more of the following can supplement these criteria:

- asystole on a continuous ECG display
- absence of pulsatile flow using direct intra-arterial pressure monitoring
- absence of contractile activity using echocardiography.

Any return of cardiac or respiratory activity during this period of observation should prompt a further 5 min observation from the next cardiorespiratory arrest. After 5 min of continued cardiorespiratory arrest, the absence of pupillary responses to light, corneal reflexes, and motor response to supra-orbital pressure should be confirmed. The time of death is recorded as the time at which these criteria are fulfilled.

Post-event tasks

At the end of the resuscitation further tasks include:

1. Ongoing care of the patient, and allocation of further team roles and responsibilities including handover to other teams.
2. Documentation of the resuscitation attempt. Use information from defibrillators and monitors to help document events and times.
3. Communication with relatives (Chapter 17).
4. An immediate post-event debriefing ('Hot' debriefing). This is normally led by the resuscitation team leader, focuses on immediate issues and concerns, and is usually of short duration. This can be difficult if the patient has a ROSC, as focus then inevitably shifts to post-resuscitation care. A delayed facilitated debriefing ('Cold' debriefing) is also useful (Chapter 2).
5. Ensuring equipment and drug trolleys are replenished.
6. Ensuring audit forms are completed.

Summary learning

- **The ALS algorithm provides a framework for the standardised resuscitation of all adult patients in cardiac arrest.**
- **The delivery of high quality chest compressions with minimal interruptions is an important determinant of outcome.**
- **Treatment depends on the underlying rhythm.**
- **Look for reversible causes and treat early.**
- **Secure the airway early to enable continuous chest compressions.**
- **Use waveform capnography to help assess and guide resuscitation interventions.**

My key take-home messages from this chapter

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Further reading

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Airway management and ventilation

Chapter 7

Contents

- Causes and recognition of airway obstruction
- Techniques for airway management when starting resuscitation
- The use of simple adjuncts to maintain airway patency
- Techniques for ventilating the lungs
- Supraglottic airway devices
- Tracheal intubation and cricothyroidotomy

Learning outcomes

To enable you to:

- Recognise the causes of airway obstruction
- Manage the airway effectively during resuscitation
- Understand the role of simple techniques and devices for maintaining the airway and ventilating the lungs
- Understand the role of supraglottic airways during CPR
- Consider the role of tracheal intubation during CPR
- Understand the role of needle and surgical cricothyroidotomy

Introduction

Patients requiring resuscitation often have an obstructed airway, usually caused by loss of consciousness, but occasionally it may be the primary cause of cardiorespiratory arrest. Prompt assessment, with control of airway patency and provision of ventilation if required are essential. This will help to prevent secondary hypoxic damage to the brain and other vital organs. Without adequate oxygenation it may be impossible to restore an organised, perfusing cardiac rhythm. These principles may **not apply to the witnessed primary cardiac arrest in the vicinity of a defibrillator**; in this case, the priority is immediate defibrillation followed by attention to the airway.

Causes of airway obstruction

Obstruction of the airway may be partial or complete. It may occur at any level from the nose and mouth down to the level of the carina and bronchi. In the unconscious patient, the commonest site of airway obstruction is the pharynx – more often at the **soft palate and epiglottis rather than the tongue**. Obstruction may also be caused by vomit or blood, following regurgitation of gastric contents or trauma, or by foreign bodies. Laryngeal obstruction may be caused by oedema from burns, inflammation or anaphylaxis. Upper airway stimulation or inhalation of foreign material may cause laryngeal spasm. Obstruction of the airway below the larynx is less common, but may be caused by excessive bronchial secretions, mucosal oedema, bronchospasm, pulmonary oedema, or aspiration of gastric contents. Extrinsic compression of the airway may also occur above or below the larynx (e.g. trauma, haematoma or tumour).

Recognition of airway obstruction

Airway obstruction can be subtle and is often missed by healthcare professionals. Recognition is best achieved by the look, listen and feel approach.

- **LOOK** for chest and abdominal movements.
- **LISTEN** and **FEEL** for airflow at the mouth and nose.

In partial airway obstruction, air entry is diminished and usually noisy.

- **Inspiratory stridor** - caused by obstruction at the laryngeal level or above.
- **Expiratory wheeze** - suggests obstruction of the lower airways, which tend to collapse and obstruct during expiration.
- **Gurgling** - suggests the presence of liquid or semisolid foreign material in the upper airways.
- **Snoring** - arises when the pharynx is partially occluded by the tongue or palate.

Complete airway obstruction in a patient who is making respiratory efforts causes paradoxical chest and abdominal movement, described as 'see-saw breathing'. As the patient attempts to breathe in, the chest is drawn in and the abdomen expands; the opposite occurs in expiration. This is in contrast to the normal breathing pattern of synchronous movement of the abdomen upwards and outwards (pushed down by the diaphragm) with lifting of the chest wall. During airway obstruction, accessory muscles of respiration are used - the neck and the shoulder muscles contract to assist movement of the thoracic cage. There may also be intercostal and subcostal recession and a tracheal tug. Full examination of the neck, chest and abdomen should enable differentiation of the movements associated with complete airway obstruction from those of normal breathing. Listen for airflow: normal breathing should be quiet, completely obstructed breathing will be silent, and noisy breathing indicates partial airway obstruction.

During apnoea, when spontaneous breathing movements are absent, complete airway obstruction is recognised by failure to inflate the lungs during attempted positive pressure ventilation. Unless airway obstruction can be relieved to enable adequate lung ventilation within a few minutes it will cause injury to the brain and other vital organs, and cardiac arrest if this has not already occurred. Whenever possible, give high-concentration oxygen during the attempt to relieve airway obstruction. Arterial blood oxygen saturation (SaO_2) measurements (normally using pulse oximetry (SpO_2)) will guide further use of oxygen as airway patency improves. If airway patency remains poor and SpO_2 remains low, continue to give high inspired oxygen concentration. As airway patency improves, blood oxygen saturation levels will be restored more rapidly if the inspired oxygen concentration is initially high. Inspired oxygen concentrations can then be adjusted to maintain SpO_2 at 94%–98%.

Patients with tracheostomies or permanent tracheal stoma

A patient with a tracheostomy tube or a permanent tracheal stoma (usually following a laryngectomy) may develop airway obstruction from blockage of the tracheostomy tube or stoma — airway obstruction can occur at the level of the pharynx in these patients. Remove any obvious foreign material from the stoma or tracheostomy tube. If necessary, remove the tracheostomy tube and exchange it if possible or, if present, exchange the tracheostomy tube liner alone. If a blocked tracheostomy tube is removed it may be possible to ventilate the patient's lungs by sealing the stoma and using a bag-mask applied to the face, or by intubating the trachea orally with a standard tracheal tube. This might not be possible if the tracheostomy was created because of significant upper airway obstruction (e.g. tumour).

In a patient with a permanent tracheal stoma, give oxygen and, if required, assist ventilation via the stoma, and not the mouth. The National Tracheostomy and Safety Project in collaboration with the Resuscitation Council (UK) has produced emergency guidelines and resources that are available at www.tracheostomy.org.uk

Choking

Recognition

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 Table 7.1.

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

Table 7.1 Signs of choking

Sequence for the treatment of adult choking

1. If the patient shows signs of **mild** airway obstruction (Figure 7.1):
 - Encourage him to continue coughing, but do nothing else.

Severe
Airway obstruc
(ineffective)

Unconscious

Start CPR

Figure 7.1 Adult choking algorithm

2. If the patient shows signs of **severe** airway obstruction and is conscious:
 - Give up to **5 back blows**.
 - Stand to the side and slightly behind the patient.
 - Support the chest with one hand.
 - Place the heel of the other hand on the upper part of the abdomen.
 - Give up to **5 sharp blows** with the heel of the other hand.
 - Check to see if each back blow relieves the airway obstruction.
 - If 5 back blows fail to relieve the airway obstruction, give up to **5 abdominal thrusts**.
 - Stand behind the patient and wrap your arms around the upper part of the abdomen.
 - Place a clenched fist just below the ribcage and grasp this hand with your other hand.
 - Sharply **inwards and upwards**.
 - Repeat up to 5 times.
 - If the obstruction is still present, continue giving **alternating 5 back blows and 5 abdominal thrusts**.
3. If the patient becomes unconscious, call the resuscitation team and start CPR.
4. As soon as an individual with training arrives, take over the resuscitation.

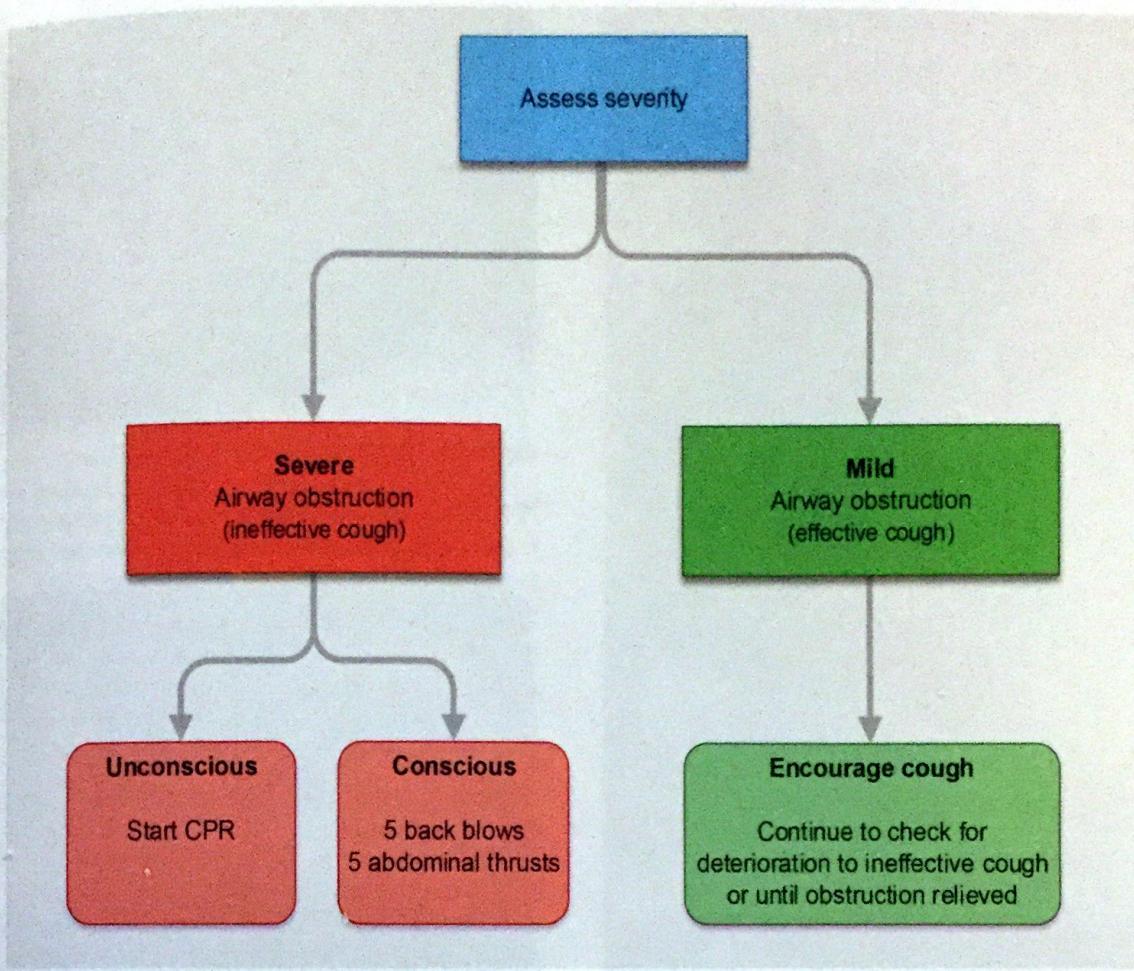


Figure 7.1 Adult choking algorithm

2. If the patient shows signs of **severe airway obstruction** and is conscious:
 - Give up to **5 back blows**.
 - Stand to the side and slightly behind the patient.
 - **Support the chest with one hand and lean the patient well forwards.**
 - Give up to **5 sharp blows between the scapulae** with the heel of the other hand.
 - Check to see if each back blow has relieved the airway obstruction.
 - If 5 back blows fail to relieve the airway obstruction give up to **5 abdominal thrusts**.
 - Stand behind the patient and put both arms round the **upper part of his abdomen**.
 - Place a **clenched fist** just under the **xiphisternum**; grasp this hand with your other hand and pull sharply **inwards and upwards**.
 - Repeat up to 5 times.
 - If the obstruction is still not relieved, continue alternating **5 back blows with abdominal thrusts**.
3. If the patient becomes unconscious, call the resuscitation team and start CPR.
4. As soon as an individual with appropriate skills is

present, undertake **laryngoscopy** and attempt to remove any foreign body with Magill's forceps.

Basic techniques for opening the airway

Once airway obstruction is recognised, take immediate action to relieve the obstruction and maintain a clear airway. Three manoeuvres that can be used to relieve upper airway obstruction are:

- **head tilt**
- **chin lift**
- **jaw thrust**.

Head tilt and chin lift

Place one hand on the patient's forehead and tilt the head back gently; place the fingertips of the other hand under the point of the patient's chin, and gently lift to stretch the anterior neck structures (Figure 7.2).

Jaw thrust

Jaw thrust is an alternative manoeuvre for bringing the mandible forward and relieving obstruction by the tongue, soft palate and epiglottis (Figure 7.3). It is most successful when applied with a head tilt.

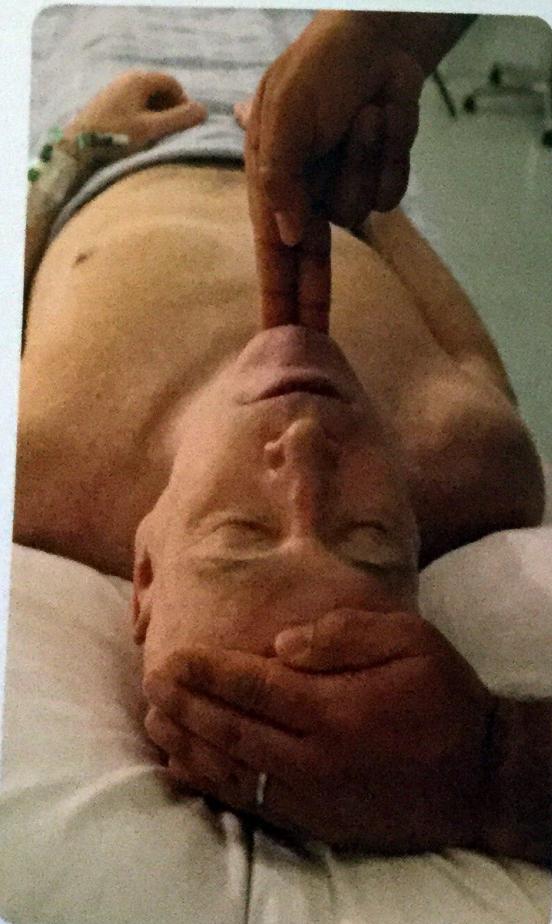


Figure 7.2 Head tilt and chin lift



Figure 7.3 Jaw thrust

Procedure for jaw thrust

- Identify the angle of the mandible.
- With the index and other fingers placed behind the angle of the mandible, apply steady **upwards** and **forward** pressure to lift the mandible.
- Using the thumbs, slightly open the mouth by downward displacement of the chin.

These simple positional methods are successful in most cases where airway obstruction is caused by loss of muscle tone in the pharynx. After each manoeuvre, check for success using the look, listen and feel sequence. If a clear airway cannot be achieved, look for other causes of airway obstruction. Carefully remove any visible foreign body with forceps or suction. Remove broken or displaced dentures but leave well-fitting dentures in place as they help to maintain the contours of the mouth, facilitating a good seal for ventilation by mouth-to-mask or bag-mask techniques.

Airway manoeuvres in a patient with suspected cervical spine injury

If spinal injury is suspected (e.g. if the victim has fallen, been struck on the head or neck, or has been rescued after diving into shallow water) maintain the head, neck, chest, and lumbar region in the neutral position during resuscitation. Excessive head tilt could aggravate the injury and damage the cervical spinal cord; however, this complication remains theoretical and the relative risk is unknown. When there is a risk of cervical spine injury, establish a clear upper airway by using **jaw thrust** or chin lift in combination with manual inline stabilisation (**MILS**) of the head and neck by an assistant. If life-threatening airway obstruction persists despite effective application of jaw thrust or chin lift, add head tilt a small amount at a time until the airway is open; establishing a patent airway takes priority over concerns about a potential cervical spine injury.

Adjuncts to basic airway techniques

Simple airway adjuncts are often helpful, and sometimes essential to maintain an open airway, particularly when resuscitation is prolonged. The position of the head and neck must be maintained to keep the airway aligned.

Oropharyngeal and nasopharyngeal airways may be used to overcome soft palate obstruction and bony displacement in an unconscious patient, but a jaw thrust may also be required.

Oropharyngeal airway

The oropharyngeal or **Guedel** airway is a soft, pliable tube, flanged and reinforced at the oral end in a flattened shape to ensure that it fits neatly around the tongue and hard palate (Figure 7.4). It is suitable for small and large adults. An easier fit may be obtained by selecting an airway length corresponding to the **vertical distance** between the patient's incisors and the **angle of the jaw**. The most common sizes are 2, 3 and 4 for small, medium and large adults respectively. An oropharyngeal airway that is slightly too big will be more beneficial than one that is slightly too small.



Figure 7.4 Oropharyngeal and nasopharyngeal airways



Figure 7.5 Sizing an oropharyngeal airway

During insertion of an oropharyngeal airway, the tube can occasionally be pushed backwards into the glottis instead of relieving it. This is because the airway may lodge in the vallecula, which is a depression that obstructs the lumen. Ensuring a correct fit of the airway should avoid this problem. Attempting to insert an oropharyngeal airway into an unconscious patient: vomiting or

Oropharyngeal and nasopharyngeal airways are designed to overcome soft palate obstruction and backward tongue displacement in an unconscious patient, but head tilt and jaw thrust may also be required.

Oropharyngeal airway

The oropharyngeal or **Guedel** airway is a curved plastic tube, flanged and reinforced at the oral end with a flattened shape to ensure that it fits neatly between the tongue and hard palate (Figure 7.4). It is available in sizes suitable for small and large adults. An estimate of the size required may be obtained by selecting an airway with a length corresponding to the **vertical distance** between the patient's incisors and the **angle of the jaw** (Figure 7.5). The most common sizes are **2, 3 and 4** for small, medium and large adults respectively. An oropharyngeal airway that is slightly too big will be more beneficial than one that is slightly too small.

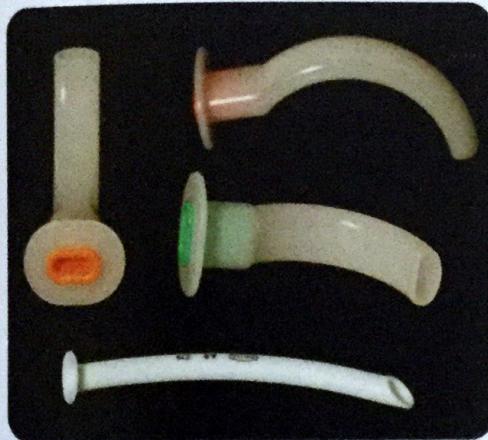


Figure 7.4 Oropharyngeal and nasopharyngeal airways



Figure 7.5 Sizing an oropharyngeal airway

During insertion of an oropharyngeal airway, the tongue can occasionally be pushed backwards, exacerbating obstruction instead of relieving it. The oropharyngeal airway may lodge in the vallecula, or the epiglottis may obstruct the lumen. Ensuring a correct insertion technique should avoid this problem. Attempt insertion only in unconscious patients: vomiting or laryngospasm may

occur if glossopharyngeal or laryngeal reflexes are present.

Technique for insertion of an oropharyngeal airway

- Open the patient's mouth and ensure that there is no foreign material that may be pushed into the larynx (if there is any, then use suction to remove it).
- Insert the airway into the oral cavity in the 'upside-down' position as far as the junction between the hard and soft palate and then rotate it through 180° (Figure 7.6). Advance the airway until it lies within the pharynx. This rotation technique minimises the chance of pushing the tongue backwards and downwards. Remove the airway if the patient gags or strains. Correct placement is indicated by an improvement in airway patency and by the seating of the flattened reinforced section between the patient's teeth or gums (if edentulous). A jaw thrust may further aid final placement of the airway as it is finally pushed into the correct position.



Figure 7.6 Oral airway insertion

After insertion, maintain head-tilt/chin-lift or jaw thrust, and check the patency of the airway and ventilation using the look, listen and feel technique. Where there is suspicion of an injury to the cervical spine, maintain alignment and immobilisation of the head and neck. Suction is usually possible through an oropharyngeal airway using a fine-bore flexible suction catheter.

Nasopharyngeal airway

This is made from soft malleable plastic, bevelled at one end and with a flange at the other (Figure 7.4). In patients who are not deeply unconscious, it is better tolerated than an oropharyngeal airway. It may be life-saving in patients with clenched jaws, trismus or maxillofacial injuries.

Inadvertent insertion of a nasopharyngeal airway through a fracture of the skull base and into the cranial vault is possible, but extremely rare. In the presence of a known or suspected basal skull fracture an oral airway is preferred, but if this is not possible, and the airway is obstructed, gentle insertion of a nasopharyngeal airway may be life-saving (i.e. the benefits may far outweigh the risks).

The tubes are sized in millimetres according to their internal diameter, and the length increases with diameter. The traditional methods of sizing a nasopharyngeal airway (measurement against the patient's little finger or anterior nares) do not correlate with the airway anatomy and are unreliable. Sizes 6–7 mm are suitable for adults. Insertion can cause damage to the mucosal lining of the nasal airway, resulting in bleeding in up to 30% of cases. If the tube is too long it may stimulate the laryngeal or glossopharyngeal reflexes to produce laryngospasm or vomiting.

Oxygen

In the absence of data indicating the optimal SaO_2 during CPR, ventilate the lungs with 100% until return of spontaneous circulation (ROSC) is achieved. After ROSC is achieved and in any acutely ill, or unconscious patient, give high-flow oxygen until the SaO_2 can be measured reliably. There are some registry data indicating an association between hyperoxaemia after ROSC and worse outcome. A standard oxygen mask will deliver up to 50%, providing the flow of oxygen is high enough. Initially, give the highest possible oxygen concentration - a mask with reservoir bag (non-rebreathing mask) can deliver an inspired oxygen concentration of 85% at flows of 10 L min⁻¹. Monitor the SpO_2 or arterial blood gases to enable titration of the inspired oxygen concentration. When blood oxygen saturation can be measured reliably,

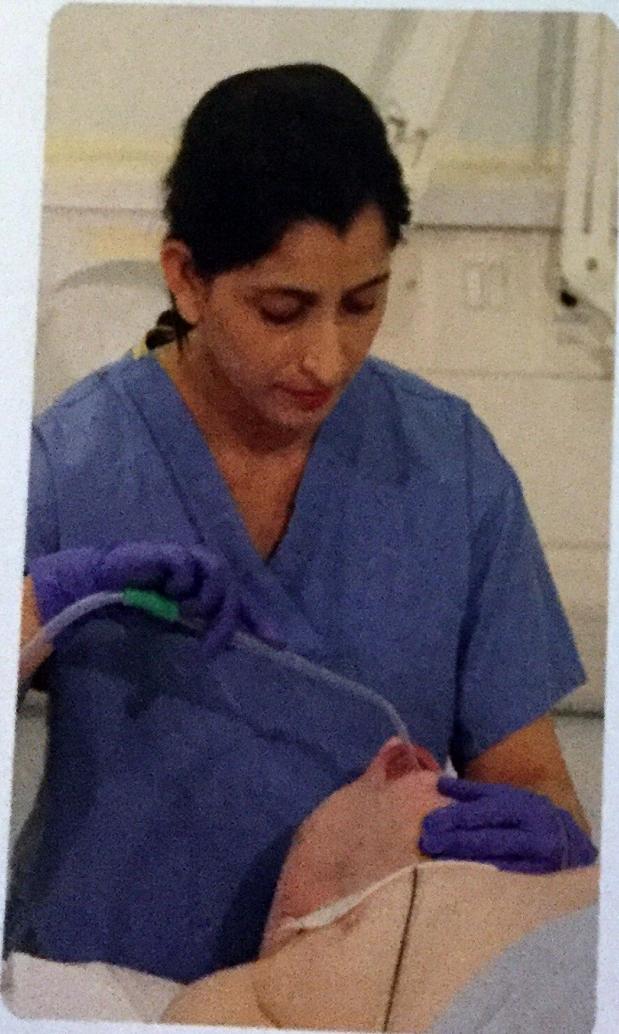


Figure 7.7 Suctioning

try to maintain the SpO_2 at 94%–98%; or 88%–92% if the patient has chronic obstructive pulmonary disease (COPD).

Suction

Use a wide-bore rigid sucker (Yankauer) to remove liquid (blood, saliva and gastric contents) from the upper airway (Figure 7.7). Use the sucker cautiously if the patient has an intact gag reflex – it can provoke vomiting. Fine-bore flexible suction catheters may be required in patients with limited mouth opening. These suction catheters can also be passed through oropharyngeal or nasopharyngeal airways.

Ventilation

Artificial ventilation is started as soon as possible in any patient in whom spontaneous ventilation is inadequate, absent. Expired air ventilation (rescue breathing) is effective but the rescuer's expired oxygen concentration, only 16–17%, so it must be replaced as soon as possible by ventilation with oxygen-enriched air. Although mouth-to-mouth ventilation has the benefit of not requiring any equipment, the technique is aesthetically unpleasant, particularly when vomit or blood is present, and the rescuer may be reluctant to place themselves in intimate contact with the victim who may be unknown to them.

There are only isolated reports of individuals acquiring infections after providing CPR (e.g. tuberculosis and seven acute respiratory distress syndrome (SARS)). Transmission of HIV during provision of CPR has never been reported. Simple adjuncts are available to enable direct person-to-person contact to be avoided; some of these devices may reduce the risk of cross infection between patient and rescuer.

The pocket resuscitation mask is used widely. It is similar to an anaesthetic face mask and enables mouth-to-mask ventilation. It has a unidirectional valve, which directs the patient's expired air away from the rescuer. The mask is transparent so that vomit or blood from the patient can be seen. Some masks have a port for the addition of oxygen. When using masks without an oxygen port, supplemental oxygen can be given by placing oxygen tubing underneath one side and ensuring an adequate seal. Use a two-hand technique to maximise the seal with the patient's face (Figure 7.8).

High airway pressures can be generated if the tidal volumes or inspiratory flows are too great, predisposing to gastric inflation and subsequent risk of regurgitation and pulmonary aspiration. As gastric inflation occurs, lung compliance is further reduced making ventilation more difficult. The possibility of gastric inflation is increased by:

- malalignment of the head and neck, and an obstructed airway
- an incompetent oesophageal sphincter (present in all patients with cardiac arrest)
- a high inflation pressure.

Tidal volumes in the resuscitated patient are adequate oxygenation and minimise the risk of gastric inflation. The inspiratory time will be prolonged to give chest compression over approximately 15% of the cycle to normal visible chest rise, minimising the risk of gastric inflation and hyperventilation as this will reduce coronary perfusion in an unprotected airway, giving a sequence of 30 chest compressions.

Technique for mouth-to-mouth ventilation

- Place the patient supine in a head extended position (i.e. neck slightly extended).
- Apply the mask to the patient's nose and mouth with both hands.
- Lift the jaw into the chin lift position (without exerting pressure beneath the chin or neck thrust). At the same time, support the patient's head with the thumbs to



Figure 7.8 Mouth-to-mouth ventilation

Tidal volumes in the region of 6–7 mL kg⁻¹ will provide adequate oxygenation and ventilation, and will reduce the risk of gastric inflation. If inspiratory flow is too slow, inspiratory time will be prolonged and the time available to give chest compressions is reduced. Deliver each breath over approximately 1 s and give a volume that corresponds to normal visible chest movement; this represents a compromise between giving an adequate volume, minimising the risk of gastric inflation, and allowing adequate time for chest compressions. Avoid hyperventilation as this will increase intrathoracic pressure and reduce coronary perfusion. During CPR with an unprotected airway, give 2 ventilations after each sequence of 30 chest compressions.

Technique for mouth-to-mask ventilation

- Place the patient supine with the head in a ‘sniffing’ position (i.e. neck slightly flexed on a pillow with the head extended (tilted backwards) on the neck).
- Apply the mask to the patient’s face using the thumbs of both hands.
- Lift the jaw into the mask with the remaining fingers by exerting pressure behind the angles of the jaw (i.e. jaw thrust). At the same time, press the mask onto the face with the thumbs to make a tight seal (Figure 7.8).

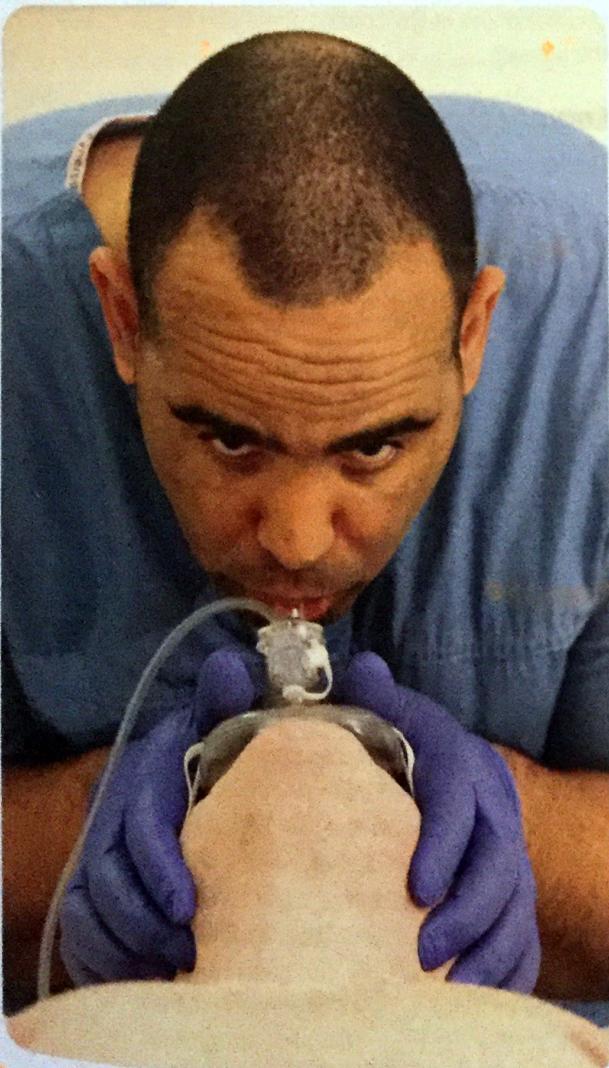


Figure 7.8 Mouth-to-mask ventilation

- Blow gently through the inspiratory valve and watch the chest rise normally.
- Stop inflation and observe the chest falling.
- Any leaks between the face and mask can be reduced by adjusting the contact pressure, altering the position of the fingers and thumbs, or increasing jaw thrust.
- If oxygen is available, add it via the port at a flow of 10 L min⁻¹.

Self-inflating bag

The self-inflating bag can be connected to a face mask, tracheal tube, or supraglottic airway (SGA). As the bag is squeezed, the contents are delivered to the patient’s lungs. On release, the expired gas is diverted to the atmosphere via a one-way valve; the bag then refills automatically via an inlet at the opposite end. When used without supplemental oxygen, the self-inflating bag ventilates the patient’s lungs with ambient air (oxygen concentration 21%) only. This is increased to around 45% by attaching high-flow oxygen directly to the bag adjacent to the air intake. An inspired oxygen concentration of approximately 85% is achieved if a reservoir system is attached and the oxygen flow is maximally increased. As the bag re-expands it fills with oxygen from both the reservoir and the continuous flow from the attached oxygen tubing.

Although the bag-mask apparatus enables ventilation with high concentrations of oxygen, its use by a single person requires considerable skill. When used with a face mask, it is often difficult to achieve a gas-tight seal between the mask and the patient’s face, and maintain a patent airway with one hand whilst squeezing the bag with the other. Any significant leak will cause hypoventilation and if the airway is not patent, gas may also be forced into the stomach. This will reduce ventilation further and greatly increase the risk of regurgitation and aspiration. There is a natural tendency to try to compensate for a leak by excessive compression of the bag, which causes high peak airway pressures and forces more gas into the stomach. Some self-inflating bags have flow restrictors that limit peak airway pressure with the aim of reducing gastric inflation.

Cricoid pressure can reduce the risk of gastric inflation but requires the presence of a trained assistant. Poorly applied cricoid pressure may make it more difficult to ventilate the patient’s lungs.

The two-person technique for bag-mask ventilation is preferable (Figure 7.9). One person holds the face mask in place using a jaw thrust with both hands and an assistant squeezes the bag. In this way, a better seal can be achieved and the patient’s lungs can be ventilated more effectively and safely.

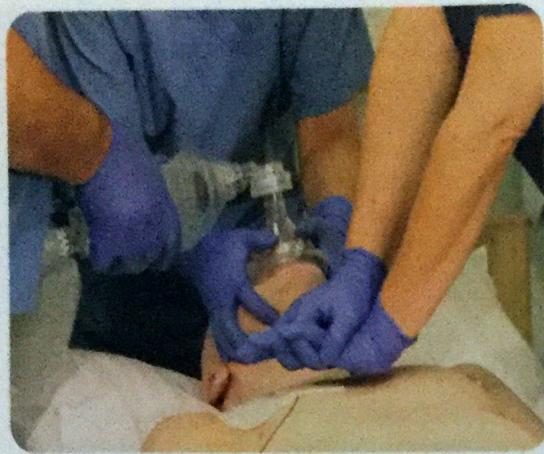


Figure 7.9 The two-person technique for bag-mask ventilation

Automatic resuscitators

Various small portable automatic ventilators may be used during resuscitation; they are more likely to be used prehospital rather than in-hospital. Most **automatic resuscitators** provide a **constant flow** of gas to the patient during inspiration; the **volume** delivered is **dependent** on the **inspiratory time** (a longer time provides a greater tidal volume). Because pressure in the airway rises during inspiration, these devices are often **pressure-limited** to protect the lungs against barotrauma.

Automatic resuscitators have some advantages over alternative methods of ventilation:

- In unintubated patients, the rescuer has both hands free for mask and airway alignment.
 - In intubated patients they free the rescuer for other tasks.
 - Once set, they provide a **constant** tidal volume, respiratory rate and minute ventilation; thus, they may help to avoid excessive ventilation.

Certain professional first responders (e.g. police, fire, and sports rescue personnel) may use simple automatic resuscitators provided that they have been trained adequately.

Passive oxygen delivery

In the presence of a patent airway, chest compressions alone may result in some ventilation of the lungs. Oxygen can be delivered passively, either via an adapted tracheal tube or with the combination of an oropharyngeal airway and standard oxygen mask with non-rebreath reservoir. There is insufficient evidence to support or refute the use of passive oxygen delivery during CPR to improve outcome when compared with oxygen delivery by positive pressure ventilation and until further data are available, passive oxygen delivery without ventilation is not currently recommended for routine use during CPR.

Supraglottic airways

Effective bag-mask ventilation requires a reasonable level of skill and experience: the inexperienced are likely to achieve ineffective tidal volumes and cause gastric inflation with risk of regurgitation and pulmonary aspiration. In comparison with bag-mask ventilation, use of SGAs may enable more effective ventilation and reduce the risk of gastric inflation. Furthermore, SGAs are easier to insert than a tracheal tube and, unlike tracheal intubation, they can generally be positioned without interrupting chest compressions.

Without adequate training and experience, the incidence of complications associated with attempted tracheal intubation is unacceptably high. Unrecognised oesophageal intubation is disastrous and prolonged attempts at tracheal intubation are harmful: the pause in chest compressions during this time will severely compromise coronary and cerebral perfusion. Alternative airway devices should be used if attempted tracheal intubation by those highly skilled to perform the technique has failed or by all other personnel not skilled in regular intubation of the trachea.

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.

Laryngeal mask airway

The laryngeal mask airway (LMA) consists of a wide-bore tube with an elliptical inflated cuff designed to seal around the laryngeal opening (Figure 7.10). It was introduced into anaesthetic practice in the middle of the 1980s and is a reliable and safe device, which can be introduced easily, with a high success rate after a short period of training. Ventilation using the LMA is more efficient and easier than with a bag-mask apparatus; provided high inflation pressures ($> 20 \text{ cmH}_2\text{O}$) are avoided, gastric inflation is minimised. When an LMA can be inserted without delay it is preferable to avoid bag-mask ventilation altogether: the risk of gastric inflation and regurgitation is reduced. Though not guaranteeing protection of the airway from gastric contents, pulmonary aspiration during use of the LMA is uncommon. The LMA does protect against sources of aspiration from above the larynx. Use of the LMA by nursing, paramedical and medical staff during resuscitation has been studied and reported to be effective. Like tracheal intubation, it requires the patient to be deeply unconscious. The LMA is particularly valuable if attempted intubation by skilled personnel has failed and bag-mask ventilation is impossible (the cannot ventilate, cannot intubate scenario). The original LMA (classic LMA (cLMA)), which is reusable, has been studied during CPR, but none of these studies has compared it directly with the tracheal tube. Although the cLMA remains in common use in elective anaesthetic practice, it has been superseded by



Figure 7.10 Laryngeal mask

Technique for inserting mask airway

- Try to maintain chest compressions during the insertion attempt; if it is difficult, pause in chest compressions.
 - Select a LMA of an appropriate size. Advance the cuff fully. A **size 4** for **men** and a **size 4** for **men**. Place the cuff area (the part with the larynx) with the patient's neck to maintain neutral alignment. If there is suspicion of cervical spine injury, hold the head still.
 - Holding the LMA like a pencil (Figure 7.11). Advance the LMA with the upper surface of the LMA reaching the posterior pharynx until a resistance is felt in the back of the pharynx. If possible, give a small jaw thrust after the LMA is inserted into the mouth - this increases the pressure on the posterior pharynx and makes the insertion easier. A slight 45 degree twist of the LMA may facilitate attempts at insertion if the LMA is difficult to insert.
 - Connect the inflation tube (up to **40 mL** for a **size 4** LMA). If insertion is difficult, withdraw the LMA out of the mouth as far as possible and then reinsert it.

several second generation SGAs that have more favourable characteristics, particularly when used for emergency airway management. Most of these SGAs are single-use devices and achieve higher oropharyngeal seal pressures than the cLMA, and some incorporate gastric drain tubes.



Figure 7.10 Laryngeal mask airway

Technique for insertion of a laryngeal mask airway

- Try to maintain chest compressions throughout the insertion attempt; if it is necessary to stop chest compressions during the insertion attempt, limit this pause in chest compressions to a maximum of 5 s.
- Select a LMA of an appropriate size for the patient and deflate the cuff fully. A size 5 will be correct for most men and a size 4 for most women. Lubricate the outer face of the cuff area (the part that will not be in contact with the larynx) with water-soluble gel.
- Flex the patient's neck slightly and extend the head (try to maintain neutral alignment of the head and neck if there is suspicion of cervical spine injury).
- Holding the LMA like a pen, insert it into the mouth (Figure 7.11). Advance the tip behind the upper incisors with the upper surface applied to the palate until it reaches the posterior pharyngeal wall. Press the mask backwards and downwards around the corner of the pharynx until a resistance is felt as it locates in the back of the pharynx. If possible, get an assistant to apply a jaw thrust after the LMA has been inserted into the mouth - this increases the space in the posterior pharynx and makes successful placement easier. A slight 45 degree twist will often aid placement if initial attempts at insertion beyond the pharynx are proving difficult.
- Connect the inflating syringe and inflate the cuff with air (up to 40 mL for a size 5 LMA and up to 30 mL for a size 4 LMA). If insertion is satisfactory, the tube will lift 1–2 cm out of the mouth as the cuff finds its correct position and

the larynx is pushed forward.

- If the LMA has not been inserted successfully after 30 s, oxygenate the patient using a pocket mask or bag-mask before reattempting LMA insertion.
- Confirm a clear airway by listening over the chest during inflation and observing bilateral chest movement. A large, audible leak suggests malposition of the LMA, but a small leak is acceptable provided chest rise is adequate.

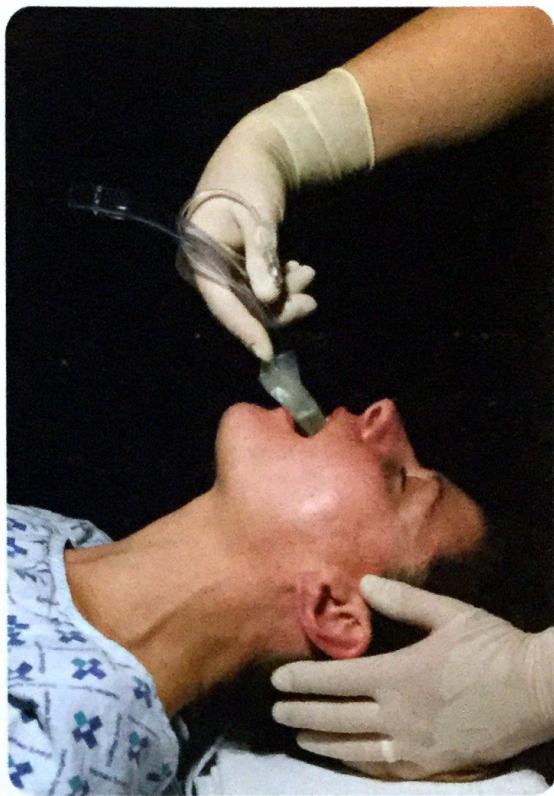


Figure 7.11 Insertion of a laryngeal mask airway

Limitations of the LMA

- In the presence of high airway resistance or poor lung compliance (pulmonary oedema, bronchospasm, chronic obstructive pulmonary disease) there is a risk of a significant leak around the cuff causing hypoventilation. Most of the gas leaking around the cuff normally escapes through the patient's mouth but some gastric inflation may occur.
- Uninterrupted chest compressions are likely to cause at least some gas leak from the LMA cuff when ventilation is attempted. Attempt continuous compressions initially but abandon this if persistent leaks and hypoventilation occur.
- There is a theoretical risk of aspiration of stomach contents because the LMA does not sit within the larynx like a tracheal tube; however, this complication is rarely documented in clinical practice.

- If the patient is not deeply unconscious, insertion of the LMA may cause coughing, straining or laryngeal spasm. This will not occur in patients in cardiorespiratory arrest.
- If an adequate airway is not achieved, withdraw the LMA, deflate the cuff and attempt reinsertion ensuring a good alignment of the head and neck.
- Uncommonly, airway obstruction may be caused by the epiglottis folding down to cover the laryngeal inlet. Withdraw the LMA, deflate the cuff and attempt reinsertion.

To become proficient in the insertion of an LMA requires practice on patients and this should be achieved under the supervision of an appropriately experienced person (e.g. anaesthetist) in a controlled environment.

i-gel airway

The i-gel incorporates a cuff 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 (Figure 7.12). It is easy to insert, requiring only **minimal training**, and a **laryngeal seal** pressure of 20–24 cmH₂O can be achieved. Insertion of the i-gel is **faster** than most other airway devices. The ease of insertion of the i-gel and its favourable leak pressure make it very attractive as a resuscitation airway device for those inexperienced in tracheal intubation. The i-gel is in widespread use in the UK for both in-hospital and out-of-hospital cardiac arrest.



Figure 7.12 i-gel

Technique for insertion of an i-gel

- Try to maintain chest compressions throughout the insertion attempt; if it is necessary to stop chest compressions during the insertion attempt, limit this pause in chest compressions to a maximum of 5 s.
- Select an appropriately sized i-gel: a **size 4** will function well in **most adults** although **small females** may require a **size 3** and **tall men** a **size 5**.

- Lubricate the back, sides and front of the i-gel cuff with a thin layer of lubricant.
- Grasp the lubricated i-gel firmly along the integral block. Position the device so that the i-gel cuff **faces** towards the chin of the patient.
- Ensure the patient is in the ‘sniffing the morning’ position with head extended and neck flexed. Gently press the chin down before inserting the i-gel.
- Introduce the leading soft tip into the mouth of the patient in a direction towards the hard palate (Figure 7.13).

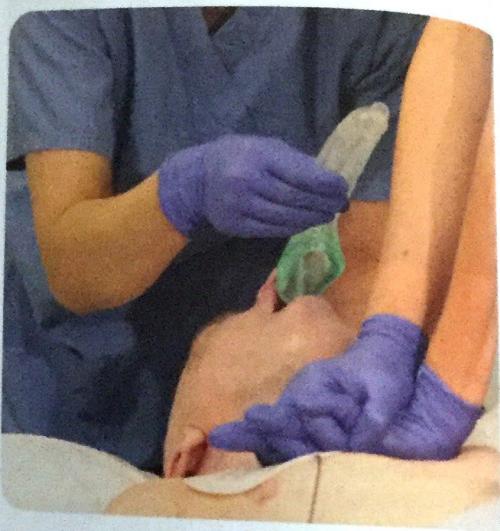


Figure 7.13 Insertion of an i-gel

- Do not apply excessive force to the device during insertion. It is not normally necessary to insert fingers or thumbs into the patient’s mouth during the process of inserting the device. If there is early resistance during insertion, get an assistant to apply a jaw thrust or rotate the device.
- Glide the device downwards and backwards along the hard palate with a continuous but gentle push until a definitive resistance is felt.
- At this point the tip of the airway should be located at the upper oesophageal opening and the cuff should be located against the larynx. The incisors should be resting on the integral bite-block.
- A horizontal line at the middle of the integral bite-block represents the approximate position of the teeth when the i-gel is positioned correctly. However, this line is only a guide – there is considerable variation in its location relative to the incisors. In short patients, this line may be at least 1 cm higher than the teeth, even when correctly positioned. In tall patients, the line may not be visible above the teeth.

The ProSeal LMA

The ProSeal LMA (PLMA) is a modified LMA. It has an additional posterior cuff. It has several attributes that make it more suitable than the original LMA for use in intubation with the larynx enabling ventilation at pressures (commonly up to 35–40 cmH₂O) and a gastric drain tube enabling venting of a gastric contents from the upper oesophagus. A gastric tube to drain liquid gastric inclusion of a bite block. The higher pressure with the PLMA may enable ventilation to be maintained during uninterrupted cardiopulmonary resuscitation (CPR).

Potential weaknesses of the PLMA compared with the original LMA are that it is slightly more difficult to intubate and to remove. Compared with the original LMA, it is relatively expensive and requires sterilisation between uses. A dispersive version of the LMA Supreme is more appropriate for use during CPR. It has a posterior cuff that allows ventilation to be maintained during CPR.

Tracheal intubation

There is **insufficient evidence** to support the use of any specific technique to manage the airway in adults with suspected cervical spine injury. In practice, airway management is usually undertaken using a stepwise approach, depending on available skills, until a satisfactory airway protection is achieved. Intubation should be attempted once the medical personnel are available to care for the patient. A high level of skill and competence are required. Advantages of tracheal intubation over bag-mask ventilation include maintenance of the airway, which is protected from aspiration of vomitus from the oropharynx, the ability to deliver tidal volume reliably even when the airway is unobstructed, the potential to perform other tasks, and the ability to provide oxygen. Use of a bag-mask is more likely to result in hypoxia, hypercapnia, and distension, which, theoretically, could lead to regurgitation and the risk of aspiration. There is no evidence that bag-mask ventilation is associated with a higher rate of aspiration than tracheal intubation.

The perceived disadvantages of tracheal intubation include the risk of a misplaced tracheal tube (which has been reported in older studies of out-of-hospital resuscitation), the time without chest compressions required to intubate (tracheal intubation is attempted in almost 25% of all CPR interventions in one study) and a comparative lack of intubation success rates compared with the experience attained by healthcare professionals who undertake prehospital resuscitation within a structured, monitored environment.

The ProSeal LMA

The ProSeal LMA (PLMA) is a modified version of the original LMA. It has an additional posterior cuff and a gastric drain tube. It has several attributes that, in theory, make it more suitable than the original LMA for use during CPR: improved seal with the larynx enabling ventilation at higher airway pressures (commonly up to 35–40 cmH₂O), the inclusion of a gastric drain tube enabling venting of liquid regurgitated gastric contents from the upper oesophagus and passage of a gastric tube to drain liquid gastric contents, and the inclusion of a bite block. The higher seal pressures achieved with the PLMA may enable ventilation volume to be maintained during uninterrupted chest compressions.

Potential weaknesses of the PLMA as an airway device for CPR are that it is slightly more difficult to insert than the original LMA, it is relatively expensive and it requires re-sterilisation between uses. A disposable form of the PLMA – the LMA Supreme is more appropriate than the reusable version for use during CPR. It has a more rigid shape and lacks a posterior inflatable cuff.

Tracheal intubation

There is insufficient evidence to support or refute the use of any specific technique to maintain an airway and provide ventilation in adults with cardiorespiratory arrest. In practice, airway management is most commonly undertaken using a stepwise approach, starting with basic techniques and moving on to more advanced techniques, depending on available skills, until effective ventilation and satisfactory airway protection is achieved. Tracheal intubation should be attempted only when trained personnel are available to carry out the procedure with a high level of skill and competence. The perceived advantages of tracheal intubation over bag-mask ventilation include maintenance of a patent airway which is protected from aspiration of gastric contents or blood from the oropharynx, the ability to provide an adequate tidal volume reliably even when chest compressions are uninterrupted, the potential to free the rescuers hands for other tasks, and the ability to suck-out airway secretions. Use of a bag-mask is more likely to cause gastric distension, which, theoretically, is more likely to cause regurgitation and the risk of aspiration. This theoretical risk has yet to be proven in randomised clinical trials.

The perceived disadvantages of tracheal intubation over bag-mask ventilation include the risk of an unrecognised misplaced tracheal tube (which is as high as 17% in some older studies of out-of-hospital cardiac arrest), a prolonged time without chest compressions while tracheal intubation is attempted (tracheal intubation attempts accounted for almost 25% of all CPR interruptions in one prehospital study) and a comparatively high failure rate. Tracheal intubation success rates depend on the intubation experience attained by the rescuer. Healthcare personnel who undertake prehospital intubation should do so only within a structured, monitored program, which should

include comprehensive competency-based training and regular opportunities to refresh skills.

Rescuers must therefore weigh the risks and benefits of tracheal intubation against the need to provide effective chest compressions. The intubation attempt will require some interruption of chest compressions but, once an advanced airway is in place, ventilation will not require further interruption of chest compressions. Personnel skilled in advanced airway management should 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 ROSC. The intubation attempt should interrupt chest compressions for less than 5 s; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After tracheal intubation, tube placement must be confirmed and the tube secured adequately. If there is any doubt about the correct position of the tube, remove it and re-oxygenate the patient before making another attempt.

In some cases, laryngoscopy and attempted intubation may prove impossible or cause life-threatening deterioration in the patient's condition. Such circumstances include acute epiglottitis, pharyngeal pathology, head injury (where coughing or straining may cause further increase in intracranial pressure), or in patients with cervical spine injury. In these circumstances, specialist skills such as the use of anaesthetic drugs, videolaryngoscopy or flexible fibroscopic laryngoscopy may be required. Such techniques require a high level of skill and training.

Essential equipment for tracheal intubation

- Laryngoscope - generally a curved Macintosh blade. Several sizes are available, but a size 4 will be adequate for most patients. Check the light source and battery regularly and just before use, and ensure that spares are immediately available. New videolaryngoscopes may be available in some settings (see below).
- Cuffed tracheal tubes - a selection should be available appropriate to the size of the patient. A 7.5 mm or 8.0 mm internal diameter tube is suitable for an adult male and a 7.0 mm internal diameter tube for a female.
- Sizes 6, 7 and 8 mm will generally cover the immediate needs of all adults. Availability of smaller tracheal tubes will be helpful for patients with conditions causing narrowing of the upper airway.
- Syringe for cuff inflation.
- Equipment for confirming correct placement of the tracheal tube.

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- Syringe for cuff inflation.
- Equipment for confirming correct placement of the tracheal tube.

- Extras:
 - water-soluble lubricating jelly
 - Magill's forceps
 - introducers: either an intubating bougie or a semi-rigid stylet
 - tape or bandage to secure tube in position
 - suction apparatus with a wide-bore rigid suction end (e.g. Yankauer) and a range of smaller flexible catheters.

Post-intubation procedures

- After successful intubation, connect the tracheal tube (via a catheter mount if necessary) to a ventilating device (e.g. self-inflating bag), and ventilate with the highest oxygen concentration available.
- Inflate the cuff of the tracheal tube just sufficiently to stop an air leak during inspiration.
- Confirm correct placement of the tracheal tube using clinical assessment AND waveform capnography. Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine use of waveform capnography to confirm correct placement of the tracheal tube will reduce this risk.
- Continue ventilation with a high-concentration of oxygen until ROSC and oxygen saturations are recordable.
- Secure the tube with a bandage or tie. Adhesive tape is not reliable if the face is moist.
- An oropharyngeal airway may be inserted alongside the tracheal tube to maintain the position of the tube, and prevent damage from biting when consciousness returns.

Clinical assessment

Primary assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and adequate) 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. The reported sensitivity (proportion of tracheal intubations correctly identified) and specificity (proportion of oesophageal intubations correctly identified) of clinical assessment varies.

Secondary confirmation of tracheal tube placement by waveform capnography will reduce the risk of unrecognised oesophageal intubation; however, this will differentiate between a tube placed in a main bronchus and one placed correctly in the trachea.

Carbon dioxide detectors

Carbon dioxide (CO_2) detector devices measure the concentration of exhaled carbon dioxide from the lungs. The persistence of exhaled CO_2 after six ventilations indicates placement of the tracheal tube in the trachea. Confirmation of correct placement of the tube in the main bronchus. Confirmation of correct placement of the tube in the trachea will require auscultation of the chest bilaterally in the mid-axillary lines. Broadly, there are three types of CO_2 detector device:

1. End-tidal CO_2 detectors that include a waveform graphical display (capnograph) are the most reliable for verification of tracheal tube position during cardiac arrest. Studies of waveform capnography to verify tracheal tube position in victims of cardiac arrest demonstrate 100% sensitivity and 100% specificity identifying correct tracheal tube placement.
2. Disposable colorimetric end-tidal CO_2 detectors use litmus paper to detect CO_2 , and these devices generally give readings of purple (end-tidal $\text{CO}_2 < 0.5\%$), tan (end-tidal $\text{CO}_2 0.5\%-2\%$) and yellow (end-tidal $\text{CO}_2 > 2\%$). In most studies, tracheal placement of the tube is considered verified if the tan colour persists after a few ventilations. Although colorimetric CO_2 detectors identify placement quite well in patients with good perfusion, these devices are less accurate than clinical assessment in cardiac arrest patients because pulmonary blood flow may be so low that there is insufficient exhaled CO_2 . Furthermore, if the tracheal tube is in the oesophagus, six ventilations may lead to gastric distension, vomiting and aspiration.
3. Non-waveform electronic digital end-tidal CO_2 devices generally measure end-tidal CO_2 using an infrared spectrometer and display the results with a number; they do not provide a waveform graphical display of the respiratory cycle on a capnograph.

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 tube through 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 all settings, including out-of-hospital, emergency department, and in-hospital locations where tracheal intubation is performed. Waveform capnography is also a sensitive indicator of ROSC and a monitor for the delivery of effective CPR.

The use of waveform capnography to confirm tracheal tube placement is now a standard of care. In the absence of waveform capnography, use a supraglottic airway device if advanced airway management is required.

Potential problems during tracheal intubation

Anatomical and pathological variations that make intubation difficult or impossible include a small jaw, short neck, poor movement at the temporomandibular joint, prominent incisors, narrow mouth, stiff tongue. If the vocal cords cannot be seen, do not insert the tube blindly. Often an intubating stylet can be inserted through the glottis more easily than the tube and, once in place, the tube may be passed over the bougie and guided (rail-roaded) into the trachea. An intubating stylet may also be used to correct the curvature of the tube or to guide it into the trachea. Problems during intubation may be caused by:

- Facial burns and trauma - it may be necessary to use BLS techniques or intubate patients with facial trauma or thermal injury to the upper airway. In some cases it may be necessary to establish a cricothyroidotomy (see below).
- Upper airway pathology (e.g. tumours, swelling from anaphylaxis, etc).
- Insecure/loose teeth or dental prostheses may be damaged or loosened if undue pressure is applied to them. Good intubation technique should be used.
- Gastric regurgitation - always have a nasogastric tube and wide-bore suction to hand. Intubation may prevent passive regurgitation and aspiration.
- Clenching of teeth - in the early stages of cardiac arrest, good CPR may prevent the profound unconsciousness required for tracheal intubation. In this case, use basic airway and ventilation.
- Oesophageal intubation - this should be recognised if tracheal tube placement is confirmed with waveform capnography. If in doubt, withdraw the tube and re-oxygenate the lungs using a bag-mask device.
- Possible cervical spine injury - suspect in patients who have a history of major blunt trauma. Perform inline stabilisation of the head and neck until an experienced operator undertakes the intubation.

Cricoid pressure

In non-arrest patients cricoid pressure is a measure of protection to the airway from regurgitation. It may also impede ventilation or interfere with intubation. The role of cricoid pressure in cardiac arrest has not been studied. Application of cricoid pressure during bag-mask ventilation reduces peak inspiratory pressures. Studies in anaesthetised patients, however, show that cricoid pressure impairs ventilation in the presence of a laryngeal mask airway, increases peak inspiratory pressures and causes airway obstruction in up to 50% of patients. The amount of cricoid pressure (in the range of 10-20 mmHg) is not well defined.

Potential problems during tracheal intubation

Anatomical and pathological variations that may make intubation difficult or impossible include receding lower jaw, short neck, poor movement at the atlanto-axial joint, prominent incisors, narrow mouth, stiff neck and trismus. If the vocal cords cannot be seen, do not attempt to insert the tube blindly. Often an intubating bougie can be inserted through the glottis more easily than a tracheal tube and, once in place, the tube may be placed over the bougie and guided (rail-roaded) into the trachea. The intubating stylet may also be used to stiffen and pre-form the curvature of the tube or to guide it into the larynx. Problems during intubation may be caused by:

- Facial burns and trauma - it may be impossible to use BLS techniques or intubate patients with severe facial trauma or thermal injury to the upper airway. In such cases it may be necessary to establish a surgical airway, (e.g. cricothyroidotomy (see below)).
- Upper airway pathology (e.g. tumours, infection, swelling from anaphylaxis, etc.).
- Insecure/loose teeth or dental prosthesis - these may be damaged or loosened if undue pressure is placed on them. Good intubation technique should reduce this risk.
- Gastric regurgitation - always have a functioning suction device and wide-bore suction to hand. Cricoid pressure may prevent passive regurgitation and pulmonary aspiration.
- Clenching of teeth - in the early stages of resuscitation good CPR may prevent the profound level of unconsciousness required for tracheal intubation. In this case, use basic airway and ventilation techniques.
- Oesophageal intubation - this should not go unrecognised if tracheal tube placement is confirmed with waveform capnography. If in doubt, take the tube out and re-oxygenate the lungs using a bag-mask.
- Possible cervical spine injury - suspect this in all patients who have a history of major blunt trauma. Use manual inline stabilisation of the head and neck and ensure an experienced operator undertakes the intubation.

Cricoid pressure

In non-arrest patients cricoid pressure may offer some measure of protection to the airway from aspiration but it may also impede ventilation or interfere with tracheal intubation. The role of cricoid pressure during cardiac arrest has not been studied. Application of cricoid pressure during bag-mask ventilation reduces gastric inflation. Studies in anaesthetised patients, however, show that cricoid pressure impairs ventilation in many patients, increases peak inspiratory pressures and causes complete obstruction in up to 50% of patients depending on the amount of cricoid pressure (in the range of recommended

effective pressure) that is applied. Do not use cricoid pressure routinely in cardiac arrest. If cricoid pressure is used, adjust, relax or release the pressure if it impedes ventilation or tracheal tube placement.

The cricoid cartilage is immediately below the thyroid cartilage, where it forms a complete ring at the upper end of the trachea. A pressure of **30 N (3 kg)** is applied anteroposteriorly, forcing the **cricoid ring** backwards, which compresses the oesophagus against the vertebral column. Do not apply cricoid pressure if there is active vomiting: it could cause oesophageal rupture.

Aids to intubation

Videolaryngoscopes

Videolaryngoscopes are being used increasingly in anaesthetic and critical care practice. In comparison with direct laryngoscopy, they enable a better view of the larynx and improve the success rate of intubation. Preliminary studies indicate that use of videolaryngoscopes improves laryngeal view and intubation success rates during CPR but further data are required before recommendations can be made for wider use during CPR.

Introducers

If visualisation is difficult, an intubating bougie may be helpful to guide the tracheal tube into the larynx. It is best inserted into the larynx separately - the tube is then passed over it into the trachea. When correctly placed, free passage of the bougie is stopped by the smaller airways of the bronchial tree; a bougie placed accidentally in the **oesophagus** can be inserted completely, without obvious resistance. Ultimately, when ventilation and intubation are impossible and alternatives (e.g. SGA), are not effective, it will be necessary to perform a cricothyroidotomy (see below).

Whilst descriptions of the advanced airway techniques above have been included, these descriptions are not intended as a substitute for practice on manikins or anaesthetised patients. Tracheal intubation during cardiac arrest should be attempted only by those undertaking this procedure regularly.

Suction

Use a wide-bore rigid suction end (Yankauer) to remove liquid (blood, saliva and gastric contents) from the upper airway. This is done best under direct vision during intubation but must not delay achieving a definitive airway. Apply suction to the trachea as briefly as possible and ventilate the lungs with 100% oxygen before and after the procedure. Use fine-bore suction catheters for tracheal suction and pass them directly down the tracheal tube.

Cricothyroidotomy

Occasionally it will be impossible to ventilate an apnoeic patient with a bag-mask, or to pass a tracheal tube or other airway device. This may occur in patients with extensive facial trauma or laryngeal obstruction caused by oedema (e.g. anaphylaxis, or foreign material). In these circumstances, it will be necessary to create a surgical airway below the level of the obstruction. A tracheostomy is contraindicated in an emergency because it is time consuming, hazardous and requires considerable surgical skill and equipment. Substantial bleeding can occur.

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. It requires a wide-bore, non-kinking cannula, a high-pressure oxygen source and may cause serious barotrauma. It is prone to failure because of kinking of the cannula, and is unsuitable for patient transfer. Unlike needle cricothyroidotomy, the surgical technique will result in an airway that is protected by a cuffed tube. Higher airway pressures can be generated and tracheal suction is possible. Surgical cricothyroidotomy enables ventilation of the lungs despite complete airway obstruction at, or above, the glottis.

Summary learning

- Airway patency and ventilating the lungs are important components of CPR.
- Use of simple airway manoeuvres, with or without basic adjuncts, will often achieve a patent airway.
- Give all patients high-concentration oxygen until the arterial oxygen saturation can be measured.
- Supraglottic airways are good alternatives to a bag-mask and are used instead of a bag-mask technique wherever possible.
- Supraglottic airways are used instead of tracheal intubation unless individuals highly skilled in intubation are immediately available. They are also used if attempted intubation is unsuccessful.
- In skilled hands, tracheal intubation is an effective airway management technique during cardiopulmonary resuscitation.
- In unskilled hands, prolonged interruptions of chest compressions, and high risk of failure and other complications (e.g. unrecognised oesophageal intubation) make attempted tracheal intubation potentially harmful.

My key take-home messages from this chapter

Rhythm

- Indications and
- The basis of the
- Reading an ECG
- Common arrhyth

To enable you to

- Understand the
- Know how to ma
- Consider the ori
- Understand the
- Identify the car
- Systematically

Introduction

During cardiac arrest, the correct choice of possible. In many pa substantial risk of fu people who have be risk of recurrence is

Some patients pres serious deterioration may prevent cardio others. Patients at structural heart dis patients who have monitoring and as will not always enc arrhythmia in 12 le

Some people exp arrhythmia that, if death. However, t people who prese lead ECG. People as cough syncop not require contin those who have

Rhythm recognition

Chapter 8

Contents

- Indications and techniques for ECG monitoring
- The basis of the ECG signal
- Reading an ECG rhythm strip
- Common arrhythmias

Learning outcomes

To enable you to:

- Understand the reasons for ECG monitoring
- Know how to monitor the ECG
- Consider the origin of the ECG
- Understand the importance of recording the ECG
- Identify the cardiac rhythms associated with cardiac arrest
- Systematically interpret rhythms on an ECG

Introduction

During cardiac arrest, assessment of the cardiac rhythm is crucial to enable you to make the correct choice of treatment (Chapter 6). Establish ECG monitoring as soon as possible. In many patients who have been resuscitated from cardiac arrest there is a substantial risk of further arrhythmia and cardiac arrest. Maintain ECG monitoring in people who have been resuscitated from cardiac arrest until you are confident that the risk of recurrence is very low.

Some patients present with an arrhythmia that may lead to cardiac arrest or other serious deterioration in their condition. Early detection and treatment of the arrhythmia may prevent cardiac arrest in some patients and prevent life-threatening deterioration in others. Patients at risk include those with persistent arrhythmia associated with structural heart disease, chest pain, heart failure, reduced conscious level or shock. In all patients who have persistent arrhythmia and are at risk of deterioration, establish ECG monitoring and as soon as possible record a good-quality 12-lead ECG. Monitoring alone will not always enable accurate rhythm recognition and it is important to document the arrhythmia in 12 leads for future reference if required.

Some people experience symptoms (usually syncope) caused by an intermittent cardiac arrhythmia that, if not documented and treated, could lead to cardiac arrest or sudden death. However, the arrhythmia may not be present at the time of initial assessment. In people who present with syncope undertake careful clinical assessment and record a 12-lead ECG. People who have experienced uncomplicated faints, situational syncope (such as cough syncope or micturition syncope) or syncope due to orthostatic hypotension do not require continuous ECG monitoring and do not usually require hospital admission. In those who have had unexplained syncope, especially during exercise, those who have

had syncope and have evidence of structural heart disease, and those who have had syncope and have an abnormal ECG (especially a prolonged QT interval) start ECG monitoring and arrange further expert cardiovascular assessment.

Monitoring the ECG by looking at only one of the 12 leads is not a reliable technique for detecting evidence of myocardial ischaemia (e.g. ST segment depression). Record serial 12-lead ECGs in people experiencing chest pain suggestive of an acute coronary syndrome.

During cardiac arrest, prompt recognition of ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT) as shockable rhythms will allow you to deliver effective treatment with minimum delay. Automated external defibrillators (AEDs) and shock-advisory defibrillators can identify these rhythms reliably by electronic analysis. If a shockable rhythm is present, the defibrillator will charge to the appropriate energy level and instruct the operator that a shock should be given. AEDs enable resuscitation from VF/pVT to be achieved by people who do not have skill in rhythm recognition and little or no training in AED use, both in hospitals and in the wider community.

Accurate analysis of some rhythm abnormalities requires experience and expertise; however, the non-expert can interpret most rhythms sufficiently to identify what immediate treatment is needed. The main priority is to recognise that the rhythm is abnormal and that the heart rate is inappropriately slow or fast. Use the structured approach to rhythm interpretation described in this chapter to help you to avoid errors. Any need for immediate treatment will be determined largely by the effect of the arrhythmia on the patient rather than by the nature of the arrhythmia. When an arrhythmia is present, first assess the patient (use the ABCDE approach), and then interpret the rhythm as accurately as possible. Always treat the patient and not their ECG.

Techniques for ECG monitoring

ECG monitors

ECG monitors display the ECG on a screen in real time. The signal is obtained from adhesive electrodes on the patient's skin and transmitted to the monitor either by wires or by telemetry. Many monitor systems have other features, such as the ability to print samples of the ECG rhythm display or to store samples of the ECG. Most monitors include a display of heart rate, and some have alarms that can be programmed to provide an alert when the heart rate goes below or exceeds preset limits.

Many systems enable monitoring of other variables such as blood pressure and oxygen saturation, which are important in the assessment of patients at risk. Digital processing of the ECG offers the potential for electronic analysis of the cardiac rhythm. If a patient requires monitoring, make sure that the monitor is being observed (and that alarms are programmed appropriately) so that immediate action can be taken if necessary, should the rhythm change.

How to attach a monitor

Attach ECG electrodes to the patient using the positions shown in Figure 8.1. These will enable monitoring using 'modified limb leads' I, II and III. Make sure that the skin is dry, not greasy (use an alcohol swab and/or abrasive pad to clean), and either place the electrodes on relatively hair-free skin or shave off dense hair. Place electrodes over bone rather than muscle, to minimise interference in the ECG signal from muscle artefact. Different electrode positions may be used when necessary (e.g. trauma, recent surgery, skin disease).



Figure 8.1 Electrode positions for ECG monitoring using modified limb leads

Most leads are colour-coded to help with correct connection. The usual colours (except in the United States) are:

- **Red** for the **Right** arm lead (usually placed over the right shoulder joint)
- **yellow** for the **Left** arm lead (usually placed over the left shoulder joint)
- **Green** for the **leg** lead (usually placed on the abdomen or lower left chest wall).

Begin by monitoring in modified lead II as this usually displays good amplitude sinus P waves and good amplitude QRS complexes, but switch to another lead if necessary to obtain the best ECG signal. Try to minimise muscle and movement artefact by explaining to patients what the monitoring is for and by keeping them warm and relaxed.

Emergency monitoring

In an emergency, such as a collapsed patient, assess the cardiac rhythm as soon as possible by applying adhesive defibrillator pads, which can be used for monitoring and for shock delivery (Figure 8.2). Apply the pads in the conventional positions, beneath the right clavicle and in the left mid-axillary line. Use anterior and posterior positions as an alternative if the conventional positions cannot be used (e.g. permanent pacemaker in right

pectoral position, chest wall trauma). Rapid application of manual defibrillator paddles also enables the cardiac rhythm to be determined rapidly. However in most UK healthcare environments paddles are no longer used.

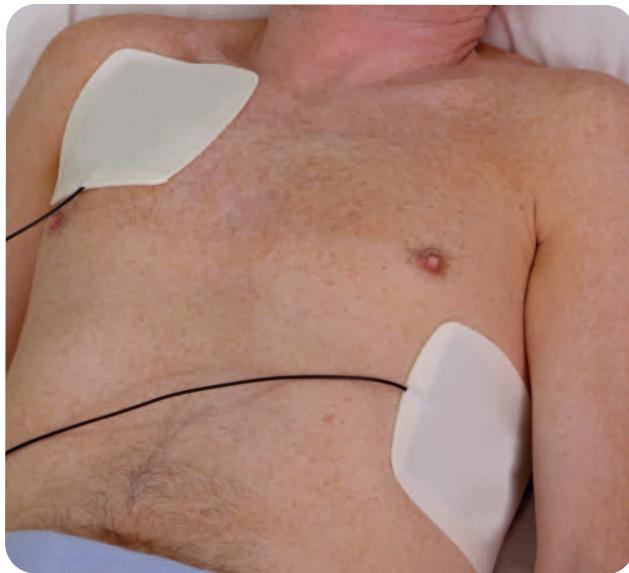


Figure 8.2 Pectoral and apical positions for defibrillator pads

Diagnosis from cardiac monitors

Use the ECG displays and printouts from monitors only for rhythm recognition. Do not attempt to interpret

ST-segment abnormalities or other more sophisticated elements of the ECG from monitors. When an arrhythmia is detected on a monitor, record a rhythm strip whenever possible.

If the arrhythmia persists for long enough, record a 12-lead ECG. It may not be possible to identify an arrhythmia by looking at only one of the 12 leads on a monitor or rhythm strip. The heart is a three-dimensional organ and the 12-lead ECG examines the electrical signals from the heart in three dimensions. Sometimes, features that enable precise identification of cardiac rhythm are visible in only one or two leads of the 12-lead ECG and would not be seen on a rhythm strip recorded in any other lead (Figure 8.3).

These recordings may assist with rhythm interpretation at the time but are also useful for later examination and planning of treatment in the longer term. Therefore effective management of any arrhythmia, including a cardiac arrest arrhythmia, includes recording and storing a good quality ECG, as well as interpretation and treatment of the rhythm at the time.

Valuable information about the nature and origin of a tachyarrhythmia can also be obtained by recording the response to treatment (e.g. carotid sinus massage, adenosine). Whenever possible, the effect of any such intervention should be documented on a continuous ECG recording, if possible using multiple leads (Figure 8.4).

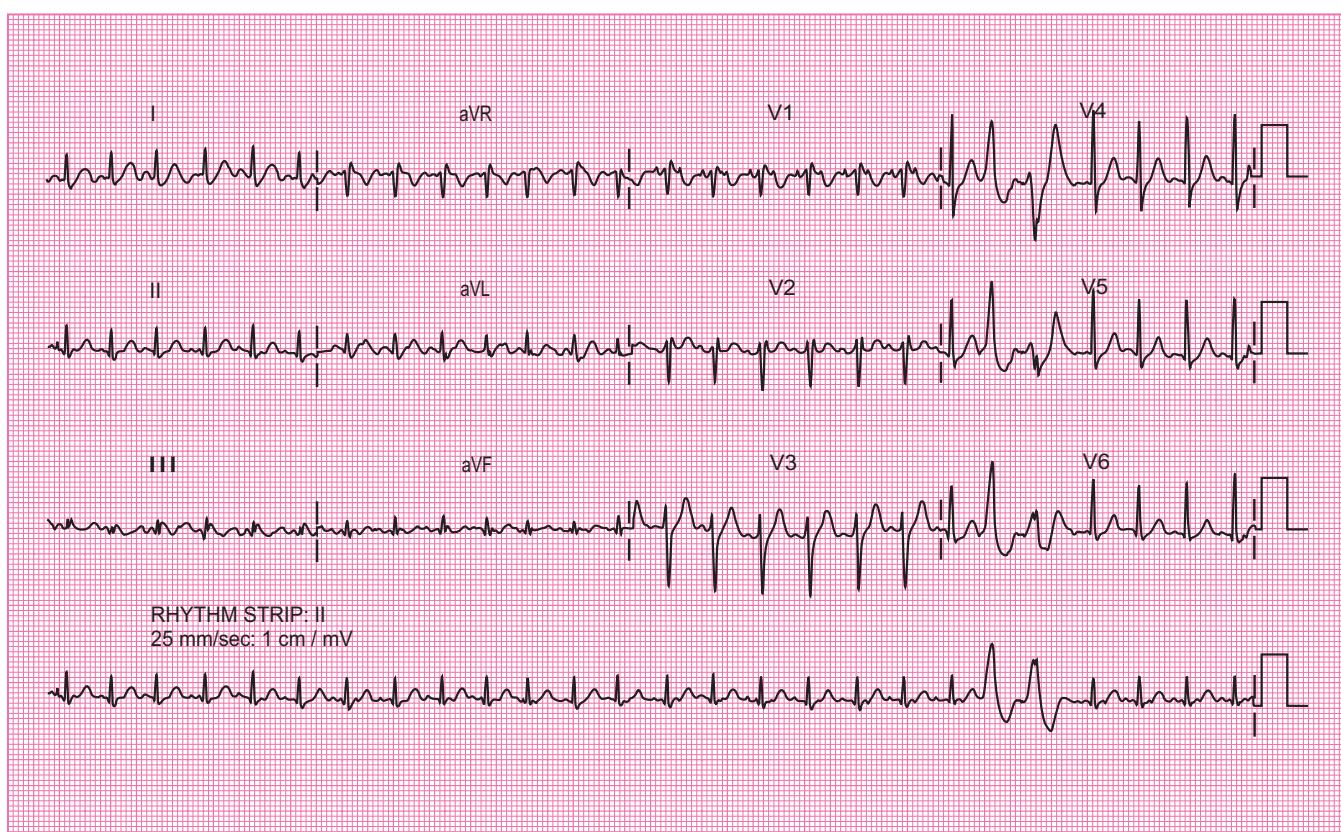


Figure 8.3 12-lead ECG showing atrial tachycardia with 2:1 AV conduction (and also 2 ventricular premature beats). Atrial activity is seen well only in lead V1, showing that the atrial rate is twice the ventricular rate.

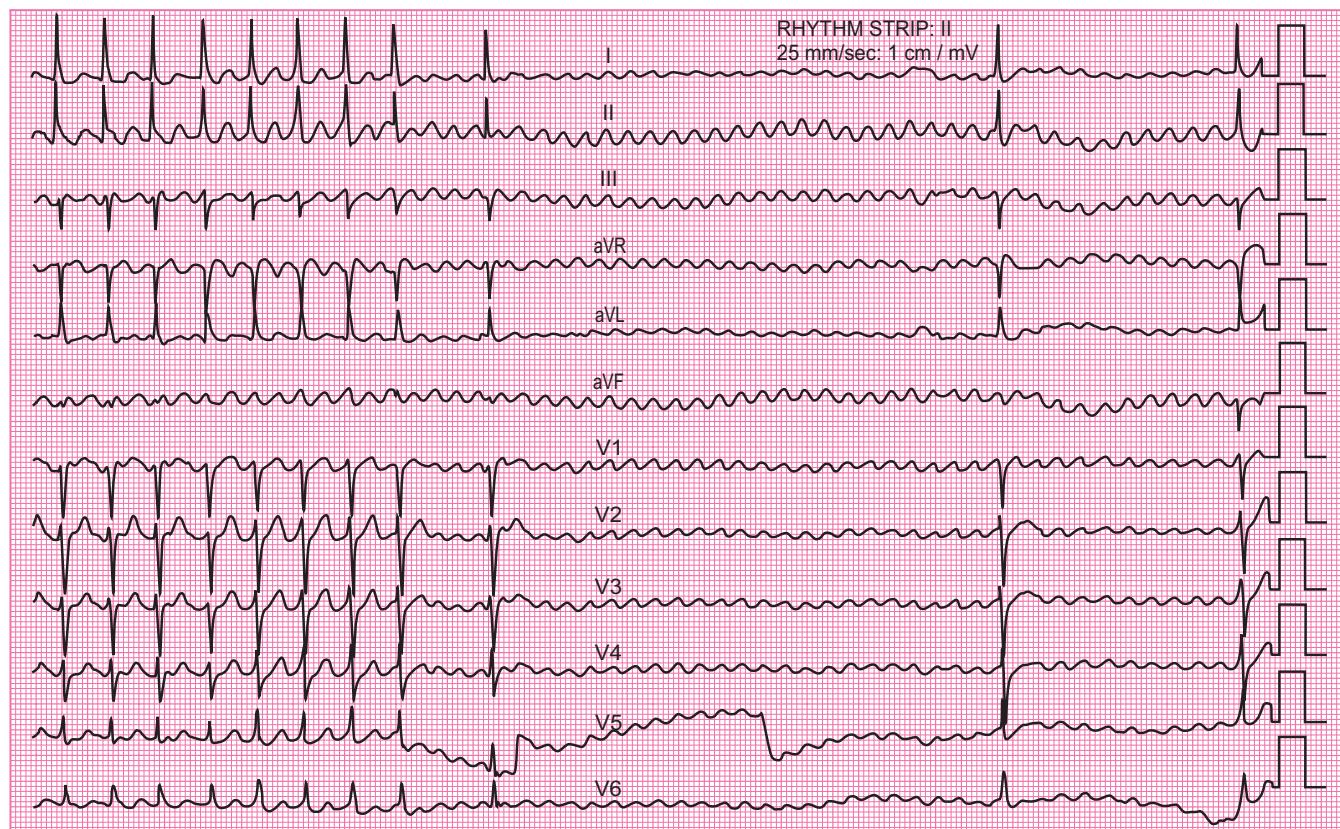


Figure 8.4 12-lead ECG showing initially a regular narrow-complex tachycardia. Adenosine did not terminate the arrhythmia but caused transient AV block, showing clearly that the initial rhythm was atrial flutter with 2:1 AV conduction.

Basic electrocardiography

At rest, the cells of the heart's conducting system and myocardium are 'polarised'. A potential difference of approximately 90 mV is present between the inside of the cell (which is negatively charged) and the extracellular space. A sudden shift of ions across the cell membrane triggers depolarisation, generating the electrical signal that travels through the conducting system and triggers contraction of myocardial cells.

In normal sinus rhythm, depolarisation begins in a group of specialised 'pacemaker' cells, called the sino-atrial (SA) node, located close to the entry of the superior vena cava into the right atrium. A wave of depolarisation then spreads from the SA node through the atrial myocardium. This is seen on the ECG as the P wave (Figure 8.5). Atrial contraction is the mechanical response to this electrical impulse.

The transmission of this electrical impulse to the ventricles occurs through specialised conducting tissue (Figure 8.6). Firstly, there is slow conduction through the atrioventricular (AV) node, followed by rapid conduction to the ventricular myocardium by specialised conducting tissue (Purkinje fibres). The bundle of His carries these fibres from the AV node and then divides into right and left bundle branches, spreading out through the right and left ventricles respectively. Rapid conduction down these fibres ensures that the ventricles contract in a co-ordinated fashion.

Depolarisation of ventricular myocardium is seen on the ECG as the QRS complex (Figure 8.5). Ventricular contraction is the mechanical response to this electrical impulse.

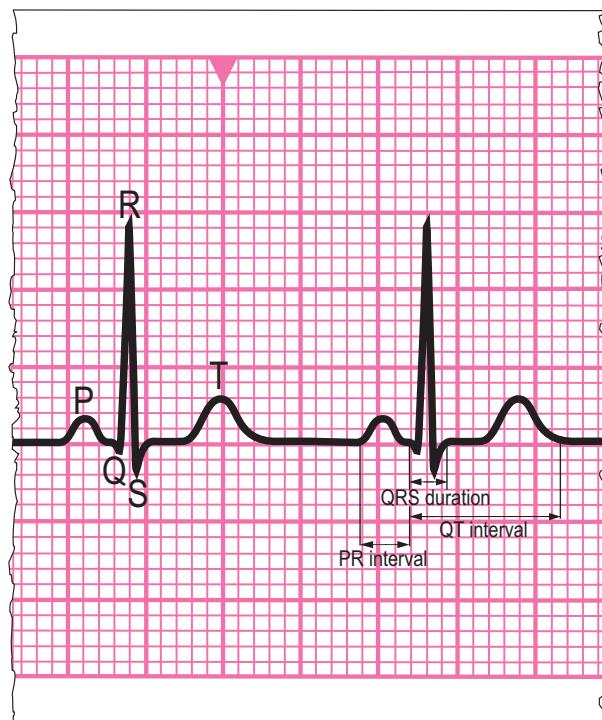


Figure 8.5 Components of the normal ECG signal

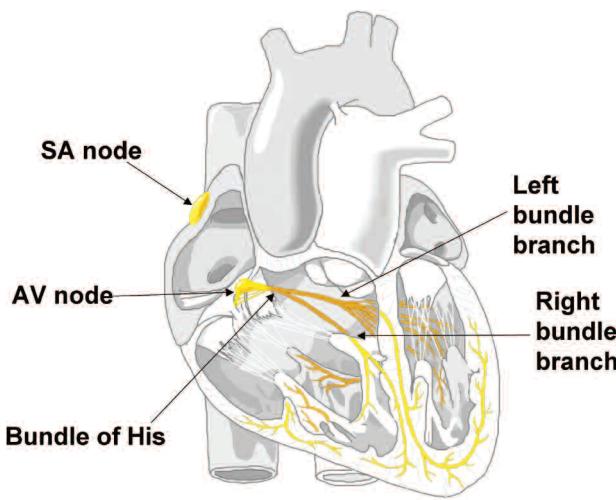


Figure 8.6 Electrical conduction in the heart

Between the P wave and QRS complex is a small isoelectric segment, which largely represents the delay in transmission through the AV node. The normal sequence of atrial depolarisation followed by ventricular depolarisation (P wave followed by QRS complex) is sinus rhythm (Rhythm strip 1).

The T wave, which follows the QRS complex, represents recovery of the resting potential in the cells of the conducting system and ventricular myocardium (ventricular repolarisation). Because the normal conducting system transmits the depolarising impulse rapidly to both ventricles, the normal QRS complex is of relatively short duration (normal < 0.12 s).

When one of the bundle branches is diseased or damaged, rapid conduction to the corresponding ventricle is prevented. The depolarising impulse travels more rapidly down the other bundle branch to its ventricle and then more slowly, through ordinary ventricular myocardium to the other ventricle. This situation is called bundle branch block. Because depolarisation of both ventricles takes longer than normal it is seen on the ECG as a broad QRS complex (0.12 s or longer).

How to read a rhythm strip

Experience and expertise may be needed to identify some rhythm abnormalities with complete accuracy and confidence. However, a simple, structured approach to interpreting the rhythm on any ECG recording will define any rhythm in sufficient detail to enable you to choose the most appropriate immediate treatment.

Use the 6-stage system to analyse rhythm on an ECG:

1. Is there any electrical activity?
2. What is the ventricular (QRS) rate?
3. Is the QRS rhythm regular or irregular?
4. Is the QRS complex width normal ('narrow') or broad?
5. Is atrial activity present?
6. Is atrial activity related to ventricular activity and, if so, how?

Any cardiac rhythm can be described accurately (e.g. irregular narrow complex tachycardia, regular broad-complex bradycardia, etc.) using the first four steps and its immediate management can then be decided safely and effectively. Do not go further to steps 5 and 6 unless you are confident that you can see atrial activity.

1. Is there any electrical activity?

If you cannot see any electrical activity, check that the gain control is not too low and that the electrodes and leads are connected securely to both the patient and the monitor.

Check the patient: is a pulse present? If the patient is pulseless and there is still no activity on the ECG this is asystole (Rhythm strip 2). Atrial and ventricular asystole are often both present, resulting in a line with no deflections. A completely straight line indicates usually that a lead has become disconnected. During asystole the ECG usually shows slight undulation of the baseline, and may show electrical interference due to respiratory movement, or chest compression.

Atrial activity (usually P waves but occasionally atrial fibrillation (AF) or atrial flutter) may continue for a short time after the onset of ventricular asystole. The ECG will show the atrial activity but no QRS complexes - ventricular standstill (Rhythm strip 3). Recognition of this is important because pacing is more likely to achieve a cardiac output in this situation than in most cases of complete asystole (Chapter 10).

If the patient is pulseless and electrical activity is present, decide whether recognisable QRS complexes are present. If not, and the ECG shows rapid, bizarre, irregular deflections of random frequency and amplitude, this is ventricular defibrillation (VF) (Rhythm strip 4). In VF all co-ordination of electrical activity is lost. There is no effective ventricular contraction and no detectable cardiac output. People in VF do not remain conscious for more than a few seconds after its onset, so a rhythm that appears to be sustained VF in a conscious patient is likely to have some other explanation (e.g. artefact – Rhythm strip 23).

Ventricular fibrillation is sometimes classified as coarse (Rhythm strip 4) or fine (Rhythm strip 5) depending on the amplitude of the complexes. If there is doubt about whether the rhythm is asystole or extremely fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation as continuing high quality CPR may improve the amplitude and frequency of the VF and improve the chance of subsequent successful defibrillation to a perfusing rhythm. If the rhythm is clearly VF, attempt defibrillation (Chapter 6). For Rhythm strips 4 and 5 defibrillation should be attempted as they both clearly show VF.

If electrical activity is present and contains recognisable QRS complexes, continue with the following steps in rhythm analysis. If the patient is pulseless and there are recognisable complexes on the ECG that would be expected to produce a pulse, this is pulseless electrical activity (PEA) and requires immediate CPR and consideration of a possible reversible cause. Do not delay CPR whilst the cardiac rhythm is analysed further.

2. What is the ventricular (QRS) rate?

The normal heart rate (ventricular rate) at rest is 60–100 beats min⁻¹. A bradycardia has a heart rate slower than 60 min⁻¹. A tachycardia has a rate faster than 100 min⁻¹.

ECG paper is calibrated in mm, with bolder lines every 5 mm. Standard paper speed in the UK is 25 mm s⁻¹. One second is represented by 5 large squares (25 small squares).

The best way of estimating the heart rate is to count the number of cardiac cycles (R wave to R wave, including fractions) that occur in 6 s (30 large squares) and multiply by 10. This provides an estimate of heart rate, even when the rhythm is irregular. For example, if 19.6 cardiac cycles occur in 30 large squares the rate is 196 min⁻¹ (Figure 8.7). For shorter rhythm strips count the number of cardiac cycles in 3 s (15 large squares) and multiply by 20.

3. Is the QRS rhythm regular or irregular?

This is not always as easy as it seems; at faster heart rates beat-to-beat variation during some irregular rhythms appears less obvious. Some rhythms may be regular in places but intermittent variation in R-R interval makes them irregular. Inspect an adequate length of rhythm strip carefully, measuring out each R-R interval and comparing it to others to detect any irregularity that is not obvious at first glance. Dividers are very useful for comparing the R-R intervals. Alternatively, the position of two adjacent identical points in the cardiac cycle (such as the tips of the R waves) can be marked on a strip of paper; this can then be moved to another section of the rhythm strip. If the rhythm is regular the marks will align precisely with each pair of R waves.

If the QRS rhythm is irregular, decide:

- Is this totally irregular, with no recognisable pattern of R-R interval?
- Is the basic rhythm regular, with intermittent irregularity?
- Is there a recurring cyclical variation in the R-R intervals?

If there is a cyclical pattern, the relationship between the QRS complexes and the P waves requires careful analysis, as described below. If the R-R intervals are totally irregular (irregularly irregular) and the QRS complex is of constant morphology, the rhythm is most likely to be AF (Rhythm strip 6).

A regular underlying rhythm may be made irregular by extrasystoles (ectopic beats). Extrasystoles can arise from

the atria or the ventricles, and the position in the heart from which they arise will determine their morphology on an ECG. If the QRS complex of ectopic beats is narrow (< 0.12 s), the beat is likely to have come from above the ventricular myocardium (i.e. from atrial muscle or the AV node). Broad-complex ectopic beats may be of ventricular origin or may be supraventricular ectopic beats with bundle branch block.

Broad-complex atrial premature beats can sometimes be identified by a preceding ectopic P wave. Some ventricular ectopic beats may be accompanied by a P wave occurring shortly after the QRS complex, caused by retrograde conduction from the ventricles to the atria. Ectopic beats that occur early (that is before the next regular sinus beat was due to occur) are referred to as premature beats (Rhythm strip 7).

A beat that arises from the AV node or from ventricular myocardium after a long pause, for example during sinus bradycardia or after sinus arrest, is referred to as an escape beat (Rhythm strip 8). This implies that the focus in the AV node or ventricle that generates this beat is acting as a back-up pacemaker, because the normal pacemaker function of the sinus node is too slow or absent. Complete AV block is an escape rhythm in which the cells generating the ventricular rhythm are acting as a pacemaker because no atrial impulses are transmitted to the ventricles.

Ectopic beats may occur singly, in pairs (couplets) or in threes (triplets). If more than three ectopic beats occur in rapid succession, this is regarded as a tachyarrhythmia. An arrhythmia that occurs intermittently, interspersed with periods of normal sinus rhythm, is described as paroxysmal. When ectopic beats occur alternately with sinus beats for a sustained period this is called bigeminy. It may be referred to as atrial bigeminy or ventricular bigeminy, depending on whether the ectopic beats are atrial or ventricular in origin.

4. Is the QRS complex duration normal or prolonged?

The upper limit of normal for the QRS duration is 0.12 s (3 small squares). If the QRS width is less than this, the rhythm originates from above the bifurcation of the bundle of His and may be from the SA node, atria or AV node, but not from the ventricular myocardium. If the QRS duration is 0.12 s or more the rhythm may be coming from ventricular myocardium or may be a supraventricular rhythm, transmitted with aberrant conduction (i.e. bundle branch block).



Figure 8.7 Calculation of heart rate from a rhythm strip. In the 30 large squares (between the arrows) there are 19.6 cardiac cycles (R wave to R wave). The heart rate is 196 min⁻¹.

5. Is atrial activity present?

Having defined the rhythm in terms of rate, regularity and QRS width, examine the ECG carefully for evidence of atrial activity. This may be difficult or impossible to identify, either because it is not visible or because atrial activity is partly or completely obscured by QRS complexes or T waves. Do not guess or try to convince yourself that you can identify atrial activity unless you are completely sure.

Depending on the nature of the arrhythmia and the ECG lead being examined, P waves may be present as positive deflections, negative deflections or biphasic deflections. When present, U waves may be mistaken for P waves. P waves may coincide with and cause distortion or variation of QRS complexes, ST segments, or T waves. Whenever possible, recording of a 12-lead ECG may enable P waves to be identified in one or more leads, even if they cannot be seen clearly in the initial monitoring lead. Lead V1 is often useful for clear demonstration of some types of atrial activity including sinus P waves and AF. Sinus P waves are usually seen clearly in lead II.

Other types of atrial activity may be present. During atrial flutter, atrial activity is seen as flutter waves - an absolutely regular repetitive deflection with a 'saw-tooth' appearance, often at a rate of about 300 min^{-1} . This is usually seen best in the inferior leads (II, III, aVF) (Figure 8.4).

During AF, circuits and waves of depolarisation travel randomly through both atria. There are no P waves. Atrial fibrillation waves may be seen as rapid deviations from the baseline of varying amplitude and duration, usually seen best in lead V1. In some patients this may be of such low amplitude that no definite atrial activity can be seen.

During a sustained tachycardia atrial activity may not be visible between the QRS complexes. If the rhythm is of atrial origin (e.g. atrial flutter or AF) it may be possible to reveal atrial activity by slowing the ventricular rate whilst recording an ECG, preferably in multiple leads. For example, if a regular tachycardia of 150 min^{-1} is due to atrial flutter with 2:1 conduction it may not be possible to identify flutter waves with confidence. A transient increase in AV block by vagal stimulation or by an intravenous bolus of adenosine will demonstrate the flutter waves and identify the rhythm accurately (Figure 8.4).

The shape and direction of P waves help to identify the atrial rhythm. For example, sinus P waves are upright in leads II and aVF. If retrograde activation of the atria is taking place from the region of the AV node (i.e. the rhythm is junctional or ventricular in origin), the P waves will be inverted in leads II and aVF because atrial depolarisation travels in the opposite direction to normal.

The rate and regularity of P waves and flutter waves are assessed in the same way as the rate and regularity of QRS

complexes. Suspected atrial flutter waves that are irregular are likely to be fibrillation waves.

6. Is atrial activity related to ventricular activity and, if so, how?

If there is a consistent interval between each P wave and the following QRS complex, it is likely that conduction between atrium and ventricle is intact and that ventricular depolarisation is triggered by atrial depolarisation. Examine a long rhythm strip to make sure that variation in the PR interval is not missed. Occasionally conduction between atria and ventricles is reversed (i.e. ventricular depolarisation is followed by retrograde conduction through the AV node and then by atrial depolarisation); the P wave occurs soon after the QRS complex. It may sometimes be difficult to distinguish between this situation and the presence of a very long PR interval.

In other circumstances careful inspection will detect no relationship between the timing of P waves and of QRS complexes. This will indicate that atrial and ventricular depolarisation is arising independently, sometimes referred to as atrioventricular dissociation. Examples of this include:

- Complete (third-degree) AV block, where a normal sinus rate (or AF) in the atria is accompanied by a regular bradycardia arising below the AV node.
- Some examples of ventricular tachycardia (VT) in which regular broad QRS complexes are present and regular P waves can be seen at a different, slower rate, out of phase with the QRS complexes.

Difficulty may arise when the relationship between the P waves and the QRS complexes varies in a recurring pattern. This may be misinterpreted as atrioventricular dissociation. This is seen most commonly in one form of second-degree AV block (called Wenckebach or Mobitz I AV block). Examine a long rhythm strip carefully for recurring patterns and plot and compare the timing of P waves and QRS complexes. In complete AV block, the QRS rhythm is usually completely regular.

In AF, the atrial activity is completely irregular, so there is no identifiable relationship between this atrial activity and the irregular ventricular rhythm that results from it. If AF is accompanied by a completely regular ventricular rhythm with a slow rate this is likely to be due to complete AV block in the presence of AF in the atria.

In atrial flutter there may be a consistent relationship between the flutter waves and the QRS complexes, giving rise to 1:1, 2:1, 3:1 conduction etc. In some instances, there is a varying relationship, producing an irregular QRS rhythm; this is atrial flutter with variable AV conduction.

Cardiac arrest rhythms

The rhythms seen during cardiac arrest can be classified into 3 groups:

1. Shockable rhythms: Ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT)
2. Asystole
3. Pulseless electrical activity (PEA)

Extreme bradycardia and, rarely, very fast supraventricular tachyarrhythmia may also cause such a severe fall in cardiac output to cause the clinical features of cardiac arrest.

Ventricular fibrillation

The characteristic appearance of VF (Rhythm strip 4) is usually easy to recognise, and this is the only rhythm that does not need the systematic rhythm analysis described earlier in this chapter. When a monitor appears to show VF check the patient immediately to establish whether this is VF requiring immediate defibrillation, or whether the appearance is due to artefact (Rhythm strip 23). If the patient is conscious or has a pulse, the rhythm is not VF.

Two rhythm abnormalities may resemble VF in some circumstances, since both produce an irregular, broad-complex, fast rhythm:

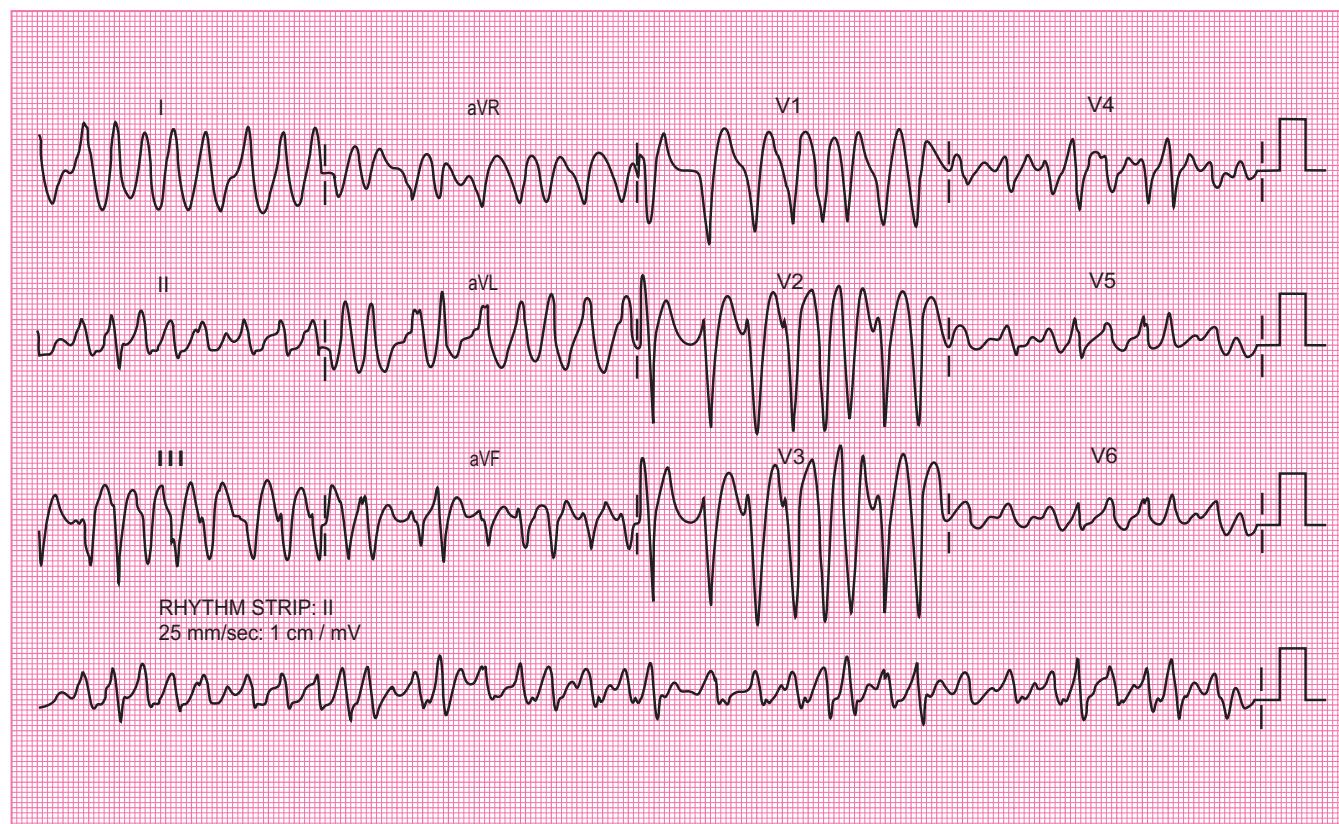


Figure 8.8 12-lead ECG showing pre-excited atrial fibrillation in a patient with Wolff-Parkinson-White syndrome

Ventricular tachycardia

Ventricular tachycardia may cause loss of cardiac output resulting in cardiac arrest, particularly at faster rates or in the presence of structural heart disease (e.g. impaired left ventricular function, severe left ventricular hypertrophy, aortic stenosis). VT may degenerate suddenly into VF. Pulseless VT is treated in the same way as VF – by immediate defibrillation.

In the presence of a cardiac output (i.e. palpable pulse), treatment of VT follows the broad-complex tachycardia algorithm described in Chapter 11. The QRS morphology may be monomorphic or polymorphic. In monomorphic VT (Rhythm strip 10), the rhythm is regular (or almost regular). The rate during VT may be anything from 100 to 300 min^{-1} , rarely faster. It is unusual to see more than slight variation in heart rate during any single episode of VT (other than in response to anti-arrhythmic drug therapy). Atrial activity may continue independently of ventricular activity; the identification of P waves, dissociated from QRS complexes during broad complex tachycardia, confirms the rhythm as VT. Occasionally these atrial beats may be conducted to the ventricles, causing capture beats or fusion beats (Rhythm strip 11). A capture beat produces a single normal-looking QRS complex during monomorphic VT, without otherwise interrupting the arrhythmia. In a fusion beat, a wave of depolarisation travelling down from the AV node occurs simultaneously with a wave of depolarisation travelling up from the ventricular focus producing the arrhythmia. This results in a hybrid QRS complex caused by fusion of the normal QRS complex with the complex of the monomorphic VT.

In the presence of bundle branch block, a supraventricular tachycardia (SVT) will produce a broad-complex tachycardia. After myocardial infarction, most broad-complex tachycardia will be ventricular in origin. The safest approach is to regard all broad-complex tachycardia as VT until, or unless, proved otherwise.

One important type of polymorphic VT is torsade de pointes (TdP) in which the axis of the electrical activity changes in a rotational way so that the overall appearance of the ECG on a rhythm strip produces a sinusoidal pattern (Rhythm strip 12). This arrhythmia usually arises in patients with a prolonged QT interval. This can occur as an inherited condition in some families (long QT syndromes). In some people it is caused by drugs, including some anti-arrhythmic drugs, and it may occur less commonly as a manifestation of myocardial ischaemia or other myocardial disease. Many patients with TdP are also hypokalaemic and/or hypomagnesaemic. It is important to recognise TdP because effective treatment (prevention of recurrent episodes) will require removal of any predisposing causes (i.e. drugs), treatment with intravenous magnesium and/or potassium, and may also require the use of overdrive pacing. Drugs that prolong QT interval (including amiodarone) should be avoided in patients with TdP. This arrhythmia can itself cause cardiac

arrest (in which case it is treated by defibrillation) and it can also degenerate into VF.

Asystole

The ECG features of asystole have been described already (Rhythm strip 2). Sometimes it may not be clear whether the observed rhythm is asystole or very fine VF. In this situation, immediate treatment is to provide high quality CPR. If fine VF was present, good CPR may increase the amplitude and frequency of the VF, making that diagnosis clear and increasing the probability of successful defibrillation.

Pulseless electrical activity

The term pulseless electrical activity (PEA) does not refer to a specific cardiac rhythm. It defines the clinical absence of cardiac output despite electrical activity that would normally be expected to produce a cardiac output. Potentially treatable causes include severe fluid depletion or blood loss, cardiac tamponade, massive pulmonary embolism and tension pneumothorax.

Peri-arrest arrhythmias

These are defined according to heart rate (bradycardia, tachyarrhythmia or arrhythmia with a normal rate), as this will dictate initial treatment (Chapter 11). In an unstable patient, concentrate on early treatment to prevent deterioration, rather than on prolonged attempts to identify the precise rhythm.

Bradycardia

A bradycardia is present when the ventricular (QRS) rate is $< 60\text{ min}^{-1}$ (Rhythm strip 13). Bradycardia may be a physiological state in very fit people or during sleep, or may be an expected result of treatment (e.g. with a beta blocker). Pathological bradycardia may be caused by malfunction of the SA node or from partial or complete failure of atrioventricular conduction. Some patients with these rhythm abnormalities may need treatment with an implanted pacemaker (Rhythm strip 14).

The emergency treatment of most bradycardia is with atropine and/or cardiac pacing (Chapter 10). Occasionally it may be necessary to use sympathomimetic drugs such as isoprenaline or adrenaline. The need for treatment depends on the haemodynamic effect of the arrhythmia and the risk of developing asystole, rather than the precise ECG classification of the bradycardia. Extreme bradycardia may sometimes precede cardiac arrest and this may be prevented by prompt and appropriate treatment of the bradycardia. In this context the most important bradycardia is acquired complete heart block (see below).

Heart block: first-degree atrioventricular block

The PR interval is the time between the onset of the P wave and the start of the QRS complex (whether this begins with a Q wave or R wave). The normal PR interval

is 0.12–0.20 s. First-degree atrioventricular (AV) block is present when the PR interval is > 0.20 s and is a common finding (Rhythm strip 15). It represents a delay in conduction through the AV junction (the AV node and/or bundle of His). In some instances this may be physiological (for example in trained athletes). There are many other causes of first-degree AV block, including primary disease (fibrosis) of the conducting system, various types of structural heart disease, ischaemic heart disease and use of drugs that delay conduction through the AV node. First-degree AV block rarely causes any symptoms and as an isolated finding rarely requires treatment.

Heart block: second-degree atrioventricular block

Second-degree AV block is present when some, but not all, P waves are conducted to the ventricles, resulting in absence of a QRS complex after some P waves. There are two types:

Mobitz Type I AV block (also called Wenckebach AV block)

The PR interval shows progressive prolongation after each successive P wave until a P wave occurs without a resulting QRS complex. Usually the cycle is then repeated (Rhythm strip 16).

Any condition that delays AV conduction can produce Wenckebach AV block. In some situations this may be physiological, for example in highly trained athletes with high vagal tone. Outside that setting Wenckebach AV block is usually pathological. Its many causes include acute myocardial infarction (especially inferior infarction). If asymptomatic, this rhythm does not usually require immediate treatment. The need for treatment is dictated by the effect of the bradycardia on the patient and the risk of developing more severe AV block or asystole.

Mobitz Type II AV block

There is a constant (often prolonged) PR interval in the conducted beats but some of the P waves are not followed by QRS complexes. This may occur randomly, without any consistent pattern. People with Mobitz II AV block have an increased risk of progression to complete AV block and asystole.

2:1 and 3:1 AV block

The term 2:1 AV block describes the situation in which alternate P waves are followed by a QRS complex (Rhythm strip 17). 2:1 AV block may be due to Mobitz I or Mobitz II AV block and it may be difficult to distinguish which it is from the ECG appearance. If bundle branch block is present as well as 2:1 block (broad QRS complexes) this is likely to be Mobitz II block. 3:1 AV block (Rhythm strip 18) is less common and is a form of Mobitz II AV block. Immediate decisions about treatment of these rhythms (see algorithm for treatment of bradycardia - Chapter 11) will be determined by the effect of the resulting bradycardia on the patient. After identifying and providing any necessary

immediate treatment continue ECG monitoring and arrange expert cardiology assessment.

Heart block: third-degree atrioventricular block

In third-degree (complete) AV block, there is no relationship between P waves and QRS complexes; atrial and ventricular depolarisation arises independently from separate ‘pacemakers’ (Rhythm strip 19). The site of the ‘pacemaker’ stimulating the ventricles will determine the ventricular rate and QRS width. A ‘pacemaker’ site in the AV node or proximal bundle of His may have an intrinsic rate of 40–50 min⁻¹ or sometimes higher and may produce a narrow QRS complex. A ‘pacemaker’ site in the distal His-Purkinje fibres or ventricular myocardium will produce broad QRS complexes, often have a rate of 30–40 min⁻¹ or less, and is more likely to stop abruptly, resulting in asystole.

Escape rhythms

If the normal cardiac ‘pacemaker’ (SA node) fails, or operates abnormally slowly, cardiac depolarisation may be initiated from a subsidiary ‘pacemaker’ in atrial myocardium, AV node, conducting fibres or ventricular myocardium. The resulting escape rhythm will be slower than the normal sinus rate. As indicated above, subsidiary ‘pacemakers’ situated distally in the conducting system tend to produce slower heart rates than those situated more proximally. Thus a ventricular escape rhythm will usually be slower than a junctional rhythm arising from the AV node or bundle of His.

The term idioventricular rhythm is used to describe a rhythm arising from ventricular myocardium. This includes ventricular escape rhythms seen in the presence of complete AV block. The term accelerated idioventricular rhythm is used to describe an idioventricular rhythm with a normal heart rate (usually faster than the sinus rate but not fast enough to be VT). This type of rhythm is observed quite frequently after successful thrombolysis or primary percutaneous coronary intervention for acute myocardial infarction (a ‘reperfusion arrhythmia’). Accelerated idioventricular rhythms do not usually present a serious risk unless they cause haemodynamic compromise or develop into VT or VF, which is uncommon. The QRS complex of an idioventricular rhythm will be broad (i.e. 0.12 s or greater), whereas a junctional rhythm may be narrow or broad, depending on whether conduction to the ventricles occurs normally, or with bundle branch block.

Agonal rhythm

Agonal rhythm occurs in dying patients. It is characterised by the presence of slow, irregular, wide ventricular complexes, often of varying morphology (Rhythm strip 20). An agonal rhythm does not usually generate a palpable pulse. This rhythm is seen commonly during the later stages of unsuccessful resuscitation attempts. The complexes slow inexorably and often become progressively broader before progressing to asystole.

Tachyarrhythmia

A pathological tachycardia may arise from atrial myocardium, the AV junction or ventricular myocardium. When a tachycardia arises from tissue situated above the bifurcation of the bundle of His, it is described as supraventricular (Rhythm strip 21). The QRS complexes will be narrow if ventricular depolarisation occurs normally, but will be broad if bundle branch block is present. Sinus tachycardia is not an arrhythmia and usually represents a response to some other physiological or pathological state (e.g. exercise, anxiety, blood loss, pain, fever etc).

Narrow-complex tachycardia

This results from a supraventricular tachycardia with normal conduction to the ventricles (Rhythm strip 21). QRS complexes may be regular in many rhythms or may be irregular in the presence of atrial fibrillation or variably conducted atrial flutter. Many tachycardias with narrow QRS complexes have a favourable prognosis, but the outlook will vary with individual clinical circumstances. These rhythms may be tolerated poorly by patients with structural heart disease and may provoke angina, especially in patients with coronary artery disease.

Atrial fibrillation

Atrial fibrillation is the most common sustained arrhythmia encountered in clinical practice. It is characterised by disorganised electrical activity in the atria. No recognisable P waves or co-ordinated atrial activity can be seen in any lead (Rhythm strip 6). The baseline is irregular and chaotic atrial activity is best seen in lead V1 where the atrial waveform is irregular in both amplitude and frequency. The QRS rhythm is irregularly irregular (i.e. the R-R interval varies in random fashion from beat to beat). The ventricular rate will depend on the refractory period of the AV junction. In the absence of drug treatment or pre-existing disease affecting the AV node, the resulting ventricular rate will be rapid, usually 120–180 min⁻¹ or faster.

Common causes of AF include hypertension, obesity, alcohol excess and structural heart disease. In coronary heart disease AF usually occurs as a result of left ventricular impairment (acute or chronic) and not as a direct result of ischaemia of the atrial myocardium.

Atrial flutter

In atrial flutter, atrial activity is seen on the ECG as flutter waves at a rate of about 300 min⁻¹ (Rhythm strip 22). These are usually best seen in the inferior leads II, III and aVF where they have a ‘saw-tooth’ appearance (Figure 8.4). The ventricular rate depends on AV conduction but there is often 2:1 (Rhythm strip 9) or 3:1 conduction (often referred to as atrial flutter with 2:1 or 3:1 block). If conduction is constant the ventricular rhythm will be regular, but variable conduction causes an irregular ventricular rhythm. Like AF, atrial flutter is often, but not always, associated with underlying disease. Atrial flutter usually arises in the right atrium, so is a recognised

complication of diseases that affect the right heart, including chronic obstructive pulmonary disease, major pulmonary embolism, complex congenital heart disease and chronic congestive heart failure of any cause.

Broad-complex tachycardia

Broad-complex tachycardia may be:

- a tachycardia arising in the ventricle below the bifurcation of the bundle of His (i.e. VT – Rhythm strip 10) or
- a supraventricular tachycardia conducted aberrantly (right or left bundle branch block) to the ventricles.

The clinical consequences depend on:

- the heart rate during the arrhythmia
- the presence or absence of structural heart disease or coronary disease
- the duration of the arrhythmia.

Ventricular tachycardia may degenerate into VF, especially if the VT is very fast (e.g. 200 min⁻¹ or faster) or if the myocardium is unstable as a consequence of acute ischaemia or infarction, or in the presence of electrolyte abnormality (hypokalaemia or hypomagnesaemia).

The safest approach is to treat all broad-complex tachycardia as ventricular tachycardia unless there is good evidence that it is supraventricular in origin. Patients with WPW syndrome have accessory pathways connecting atrial and ventricular myocardium. Some atrioventricular conduction occurs through these pathways as well as through the AV node. This results in widening of the QRS complexes by so-called delta waves (Figure 8.9). In the presence of such an accessory pathway that bypasses the AV node, AF may result in a ventricular rate that is so fast that cardiac output decreases dramatically. The ECG appearances are of a very rapid, irregular, broad complex tachycardia that usually shows variability in the width of QRS complexes (Figure 8.8). This rhythm may be misdiagnosed as ‘irregular VT’, polymorphic VT or possibly as VF. Overall the rhythm is more organised than ventricular fibrillation (it lacks the random chaotic activity of variable amplitude).

The QT interval

When identifying and treating rhythm abnormalities it is important to recognise likely underlying causes that may influence choice of effective treatment. These may be identified from clinical assessment (e.g. heart failure), laboratory tests (e.g. electrolyte abnormality) or from the ECG. Prolongation of the QT interval predisposes people to ventricular arrhythmia, in particular TdP and VF.

The QT interval is measured from the start of the QRS complex to the end of the T wave. It can be difficult to measure accurately, mainly because it may be difficult to identify the end of the T wave. This may be especially

difficult when prominent U waves are present, merging with the end of the T wave. U waves can be a feature of some abnormalities (e.g. hypokalaemia) and some types of inherited cardiac conditions but may be present in some healthy people with normal hearts.

The duration of the QT interval may also vary between different leads of the same 12-lead ECG. This may partly reflect variation in amplitude and direction of the T wave, making it more difficult to measure in some leads than others. Lead II is usually recommended for QT measurement, with leads I and V5 as other options, but your choice of lead may be dictated by the lead that best allows you to identify the end of the T wave.

The QT interval varies slightly with age and with gender and is usually slightly longer in the presence of bundle branch block. The QT interval is affected substantially by heart rate. The QT interval shortens as the heart rate increases. A correction can be made to allow for this, using the measured QT interval and heart rate to calculate the corrected QT interval (QTc). The normal range quoted for QTc varies slightly. It is reasonable to consider a QTc in men normal up to 0.43 s and in women normal up to 0.45 s. A QTc in a man > 0.45 s or in a woman > 0.47 s can be regarded as definitely prolonged. A QTc of 0.5 s or more indicates a high risk of cardiac arrest and sudden death.

Many modern ECG machines measure the QT interval and calculate the QTc automatically. These measurements are accurate only if the ECG recording is of good quality. Most

ECG machines cannot distinguish between T waves and U waves. Always look at the recording and make sure that the quoted measurements are not obviously inaccurate. If in doubt seek expert help with interpretation.

Abnormality of the QT interval can be seen in various situations. A shortened QT interval may be seen with hypercalcaemia and digoxin treatment. Hypokalaemia, hypomagnesaemia, hypocalcaemia, hypothermia, myocarditis and in some instances myocardial ischaemia can all cause QT prolongation. There is also a long list of drugs that may prolong the QT interval, including class I and class III anti-arrhythmic drugs.

There are several genetic abnormalities in which the QT interval is abnormal or there is abnormality of ventricular repolarisation (principally the long QT, short QT and Brugada syndromes). The repolarisation abnormality places them at risk of ventricular arrhythmia and sudden death. These people require expert assessment to identify whether treatment is needed to reduce this risk. For some effective treatment will include an implantable cardioverter-defibrillator to treat any episode of VF or VT immediately. It is especially important that patients with long QT syndromes are not given any drug that may cause further QT prolongation.

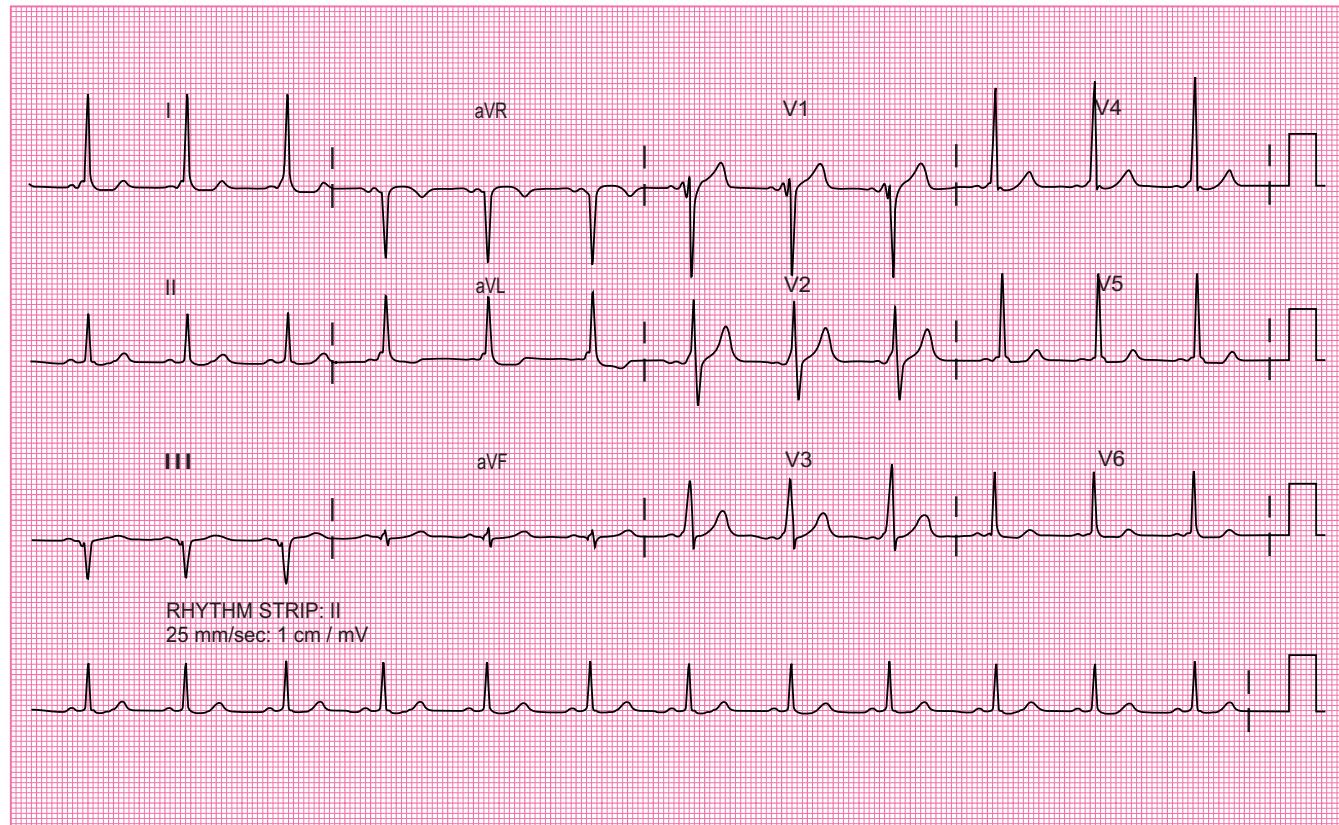


Figure 8.9 Delta waves in a patient with Wolff-Parkinson-White (WPW) syndrome

Summary learning

- A systematic approach to ECG rhythm analysis allows accurate assessment of any rhythm abnormality sufficiently to enable safe, effective immediate treatment.
- ECG recordings of any rhythm abnormality and of the ECG in sinus rhythm provide valuable diagnostic information and help the correct choice of longer-term treatment.
- Accurate monitoring of cardiac rhythm is essential for any patient at high risk of developing life-threatening arrhythmia.
- Accurate monitoring of the cardiac rhythm is essential in the management of cardiac arrest.

Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death) J Am Coll Cardiol 2006;48:e247-e346. www.escardio.org

My key take-home messages from this chapter

Further reading

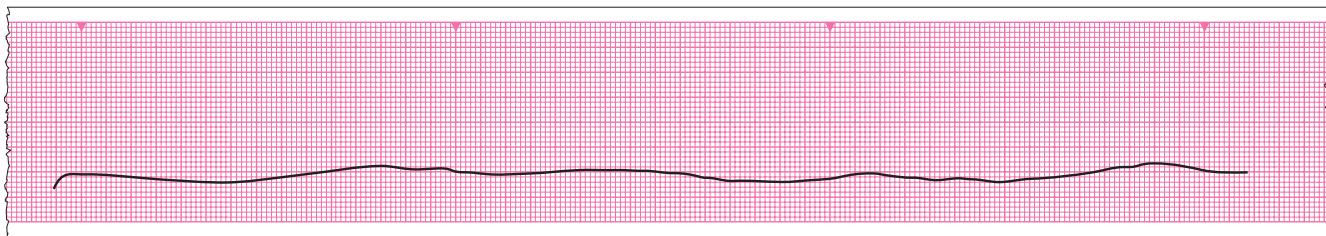
Blomstrom-Lundqvist C, Scheinmann M M (Co-Chairs). American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines. ACC/AHA/ESC Guidelines for the Management of Patients With Supraventricular Arrhythmias. European Heart Journal 2003;24:1857-1897. www.escardio.org

January CT, Wann LS, Alpert JS, et al. 2014 AHA/ACC/HRS guideline 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 Heart Rhythm Society. J Am Coll Cardiol 2014;64:e1-76.

Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of



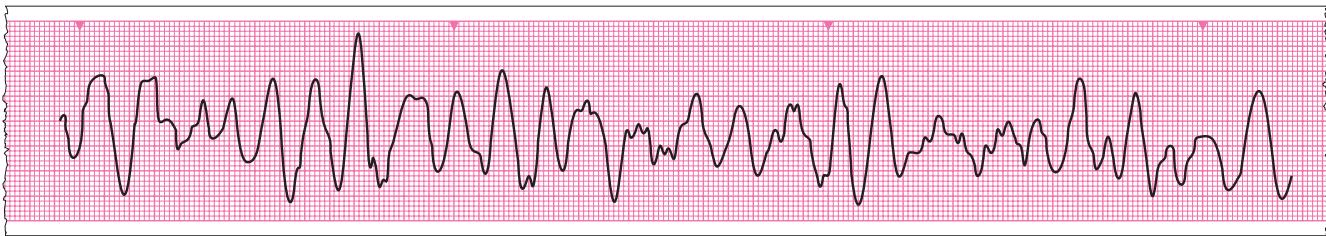
Rhythm strip 1 Sinus rhythm



Rhythm strip 2 Asystole



Rhythm strip 3 Ventricular standstill with continuing sinus P waves



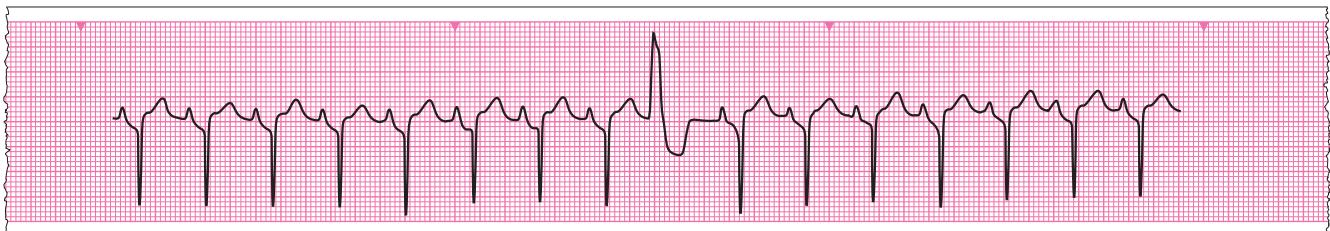
Rhythm strip 4 Coarse ventricular fibrillation



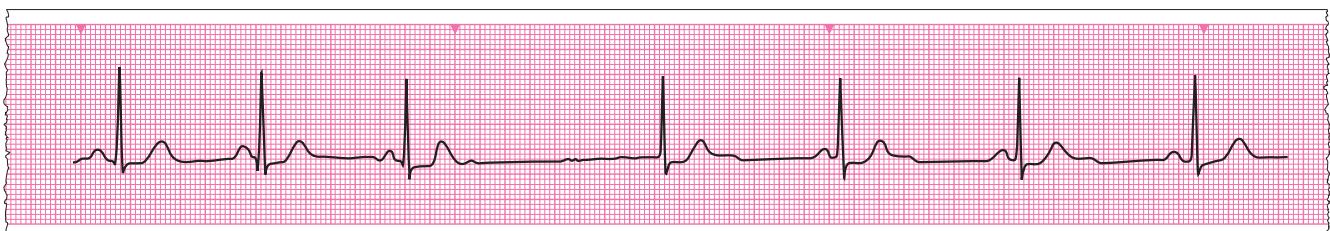
Rhythm strip 5 Fine ventricular fibrillation



Rhythm strip 6 Atrial fibrillation



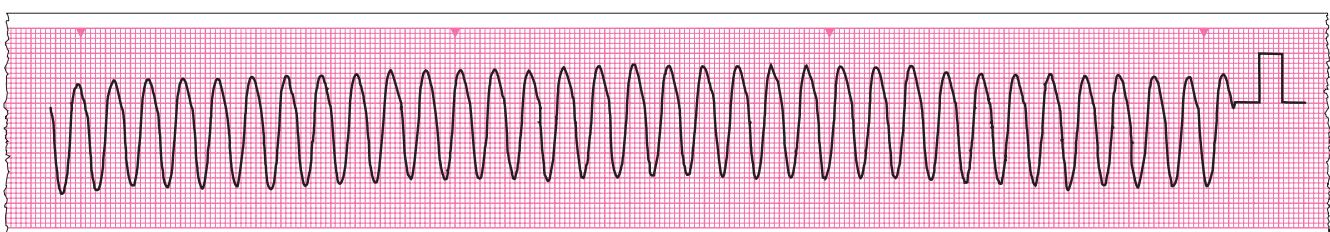
Rhythm strip 7 Ventricular premature beat



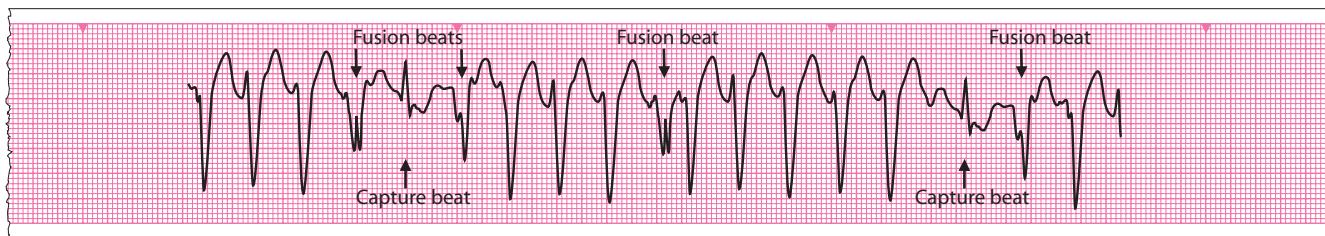
Rhythm strip 8 Junctional escape beat



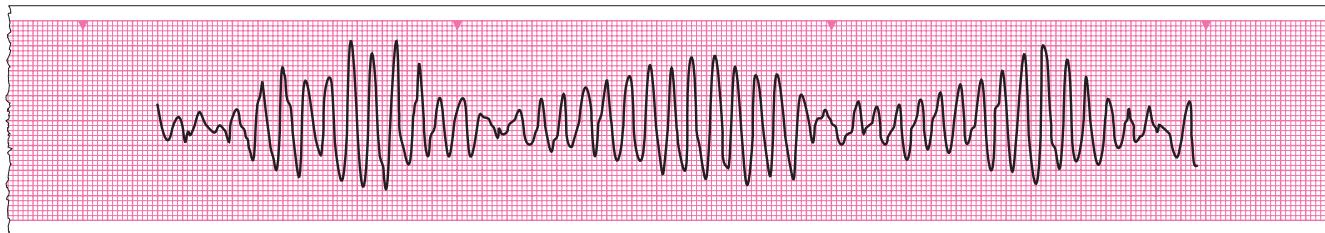
Rhythm strip 9 Atrial flutter with 2:1 AV conduction



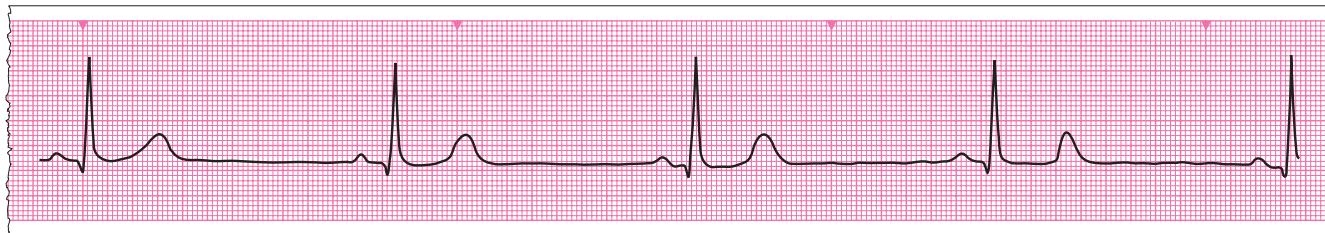
Rhythm strip 10 Monomorphic ventricular tachycardia



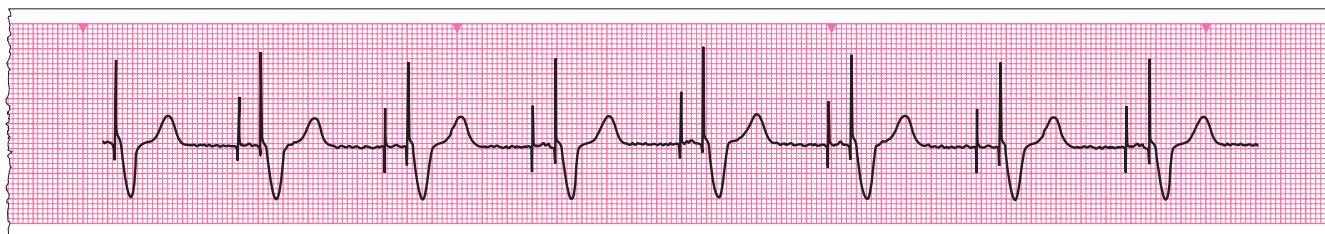
Rhythm strip 11 Ventricular tachycardia with capture beats and fusion beats



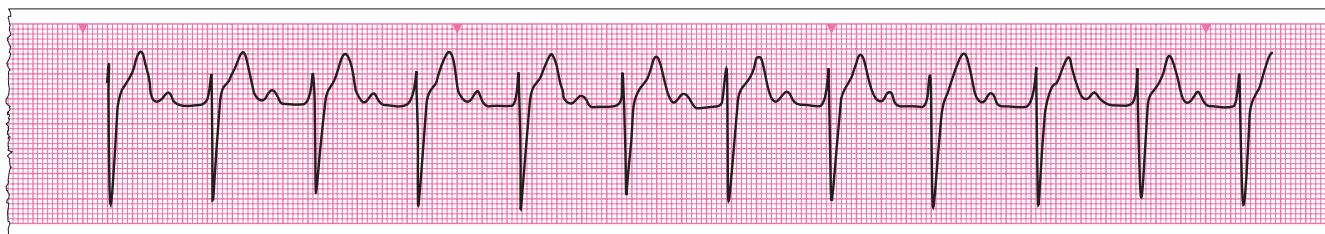
Rhythm strip 12 Torsade de pointes polymorphic ventricular tachycardia



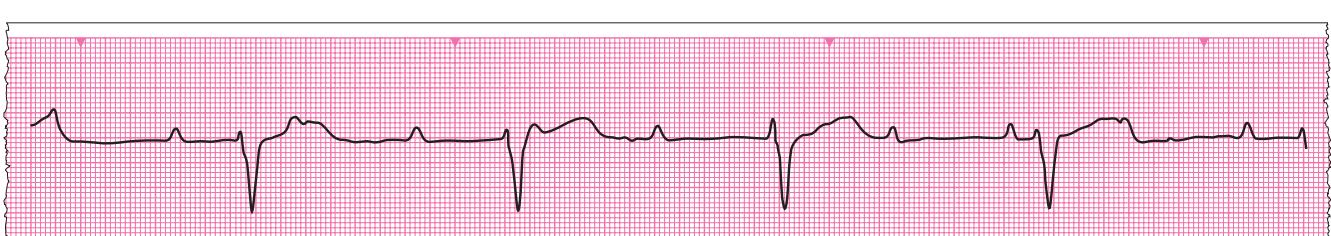
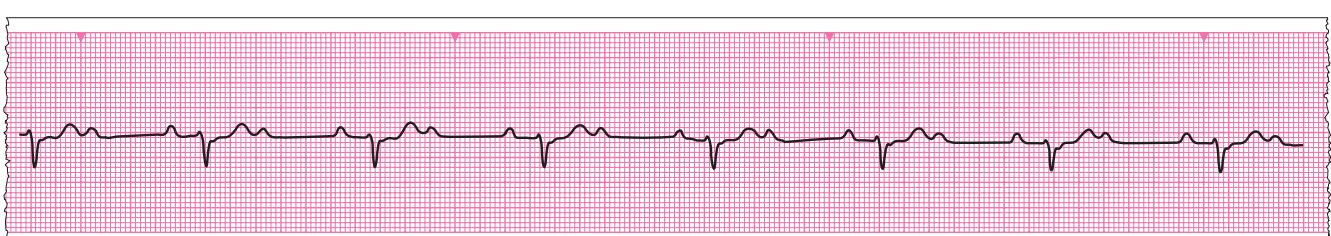
Rhythm strip 13 Sinus bradycardia

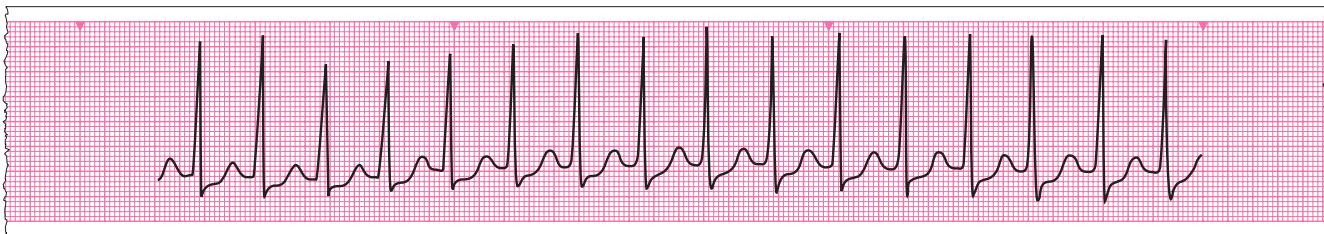


Rhythm strip 14 Sequential atrial and ventricular pacing



Rhythm strip 15 First-degree AV block

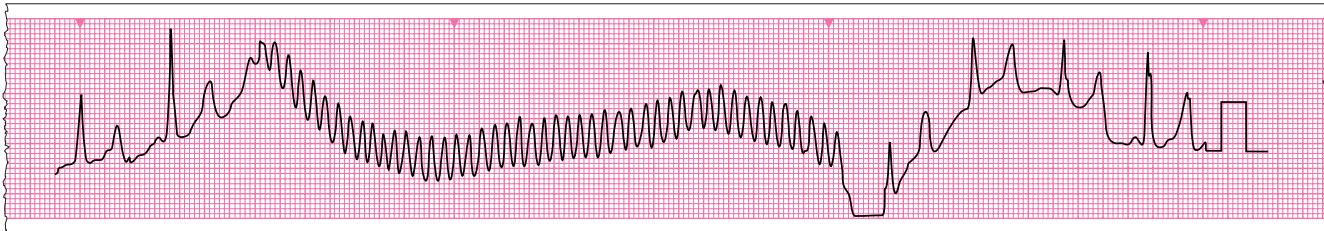




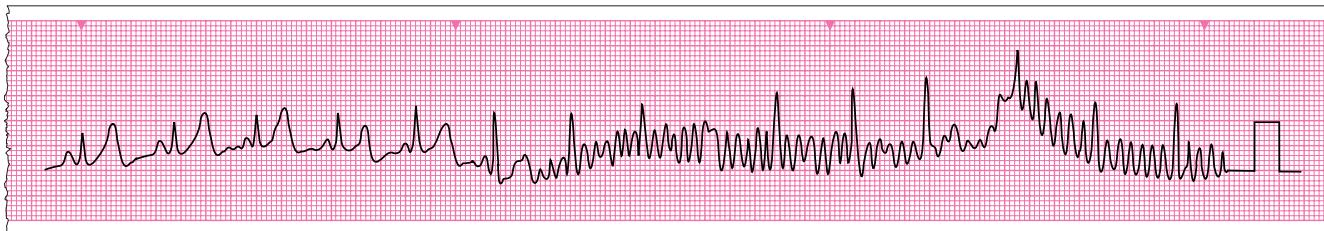
Rhythm strip 21 Regular narrow-complex tachycardia ('supraventricular tachycardia')



Rhythm strip 22 Atrial flutter with high-degree AV block causing extreme bradycardia (heart rate approximately 20 min⁻¹).



Rhythm strip 23a Artefact. A brief period of recurrent artefact that was misinterpreted as 'intermittent VF'.



Rhythm strip 23b Artefact from same patient. Artefact is seen superimposed on uninterrupted, regular QRS complexes, demonstrating that this could not be VF.

Contents

- Probability of successful defibrillation
- Mechanism of defibrillation
- Factors affecting defibrillation success
- Shock energies
- Safety during defibrillation
- Automated external defibrillators (AEDs) and sequence for use
- Manual defibrillation and sequence for use
- Defibrillation with an AED in children
- Synchronised cardioversion
- Implanted electronic devices (IEDs)

Learning outcomes

To enable you to:

- Understand how to safely deliver a shock with an AED or manual defibrillator
- Understand how to minimise pauses in chest compressions during defibrillation

Introduction

Following the onset of ventricular fibrillation or pulseless ventricular tachycardia (VF/pVT), cardiac output ceases and cerebral hypoxic injury starts within 3 min. If complete neurological recovery is to be achieved, early successful defibrillation with return of spontaneous circulation (ROSC) is essential. Defibrillation is a key link in the Chain of Survival and is one of the few interventions proven to improve outcome from VF/pVT cardiac arrest.

The probability of successful defibrillation

There are several factors that need to be taken into account:

1. Time from onset to delivery of shock. Early defibrillation is one of the most important factors in determining survival from cardiac arrest. In the absence of bystander CPR, for every minute that passes between collapse and attempted defibrillation, mortality increases by 7–10%. The shorter the interval between the onset of VF/pVT and delivery of the shock, the greater the chance of successful defibrillation and survival.
2. Continuous, uninterrupted chest compressions. Clinical studies have demonstrated that even short interruptions in chest compressions (e.g. to deliver rescue breaths or perform rhythm analysis) significantly reduce the chances of successful defibrillation. Animal studies show that even if defibrillation is successful, these short interruptions are associated with post-resuscitation myocardial dysfunction and reduced survival.

Analysis of CPR performance following both out-of-hospital and in-hospital cardiac arrest has shown that significant interruptions are common and every effort should be made to minimise interruptions. The aim should be to ensure that chest compressions are performed continuously throughout the resuscitation attempt, pausing only to enable specific interventions.

3. The duration of the interval between stopping chest compressions and delivering the shock: the pre-shock pause. The duration of the pre-shock pause is related inversely to the chance of successful defibrillation; one study has shown that every 5-second increase in the pre-shock pause almost halves the chance of successful defibrillation (defined by the absence of VF 5 s after shock delivery). Consequently, defibrillation must always be performed quickly and efficiently in order to maximise the chances of successful resuscitation.

If there is any delay in obtaining a defibrillator, and while the defibrillator pads are applied, start chest compressions and ventilation immediately. When bystander CPR is given the decrease in survival is more gradual and averages 3–4% per min from collapse to defibrillation. Bystander CPR doubles survival from witnessed cardiac arrest.

Mechanism of defibrillation

Defibrillation is the passage of an electrical current of sufficient magnitude across the myocardium to depolarise a critical mass of cardiac muscle simultaneously, enabling the natural pacemaker tissue to resume control. To achieve this, all defibrillators have three features in common:

- a power source capable of providing direct current
- a capacitor that can be charged to a pre-determined energy level
- two electrodes which are placed on the patient's chest, either side of the heart, across which the capacitor is discharged.

Successful defibrillation is defined as the absence of VF/pVT at 5 s after shock delivery, although the ultimate goal is ROSC.

Factors affecting defibrillation success

Defibrillation success depends on sufficient current being delivered to the myocardium. However, the delivered current is difficult to determine because it is influenced by transthoracic impedance (electrical resistance) and electrode position. Furthermore, much of the current is diverted along non-cardiac pathways in the thorax and, as a result, as little as 4% reaches the heart.

Transthoracic impedance

Current flow is inversely proportional to transthoracic impedance; however, biphasic defibrillators can measure

the transthoracic impedance and adjust the energy delivered to compensate. Their efficacy is therefore independent of transthoracic impedance (impedance compensation).

Ensure good contact between self-adhesive pads and the patient's skin. This may be compromised when:

- A transdermal drug patch is on the patient's chest. If it is in the area where self-adhesive pads would be applied, remove the patch and dry the skin. If this is likely to delay defibrillation, place the pads in an alternative position that avoids the patch (see below).
- The patient has a very hairy chest. If a razor is available immediately, use it to remove excessive hair from the area where the electrodes are placed. However, defibrillation should not be delayed if a razor is not to hand immediately. In very hairy patients, a bi-axillary electrode position may enable more rapid defibrillation.

Electrode position

No human studies have evaluated the electrode position as a determinant of ROSC or survival from cardiac arrest due to a shockable rhythm. Transmyocardial current during defibrillation is likely to be maximal when the electrodes are placed so that the area of the heart that is fibrillating lies directly between them (i.e. ventricles in VF/pVT, atria in atrial fibrillation (AF)). Therefore, the optimal electrode position may not be the same for ventricular and atrial arrhythmias.

When attempting to defibrillate a patient in VF/pVT, the standard procedure is to place one electrode to the right of the upper sternum below the clavicle. The apical pad is placed in the mid-axillary line, approximately level with the V6 ECG electrode or female breast. This position should be clear of any breast tissue. It is important that this electrode is placed sufficiently laterally (Figure 9.1). Although the electrodes are marked positive and negative, each can be placed in either position. Other acceptable pad positions include:

- One electrode anteriorly, over the left precordium, and the other electrode on the back behind the heart, just inferior to the left scapula (antero-posterior).
- One electrode placed in the mid-axillary line, approximately level with the V6 ECG electrode or female breast and the other electrode on the back, just inferior to the right scapula (postero-lateral).
- Each electrode on the lateral chest walls, one on the right and the other on the left side (bi-axillary).

More patients are presenting with implantable medical devices (e.g. permanent pacemaker, implantable cardioverter defibrillator (ICD)). Medic Alert bracelets are recommended for these patients. These devices may be damaged during defibrillation if current is discharged through electrodes placed directly over the device. Place



Figure 9.1 Standard electrode positions for defibrillation

the electrode away from the device (at least 10–15 cm) or use an alternative electrode position (anterior-lateral, anterior-posterior) as described above.

CPR or defibrillation first?

In any unwitnessed cardiac arrest, those responding should provide high quality, uninterrupted CPR while a defibrillator is retrieved, attached and charged. Attempt defibrillation as soon as possible; a period of CPR (e.g. 2–3 min) before rhythm analysis and shock delivery is not recommended.

Shock sequence

The optimal shock strategy for any survival end-point is unknown. There is no conclusive evidence that a single shock strategy increases rate of ROSC or reduces recurrence of VF compared with three stacked shocks, but given that outcome is improved by minimising interruptions to chest compressions, single shocks are recommended for most situations. The importance of early, uninterrupted chest compressions is emphasised throughout these guidelines, together with minimising the duration of pre-shock and post-shock pauses.

- Continue CPR while a defibrillator is retrieved and applied; as soon as the defibrillator is available, assess the rhythm and attempt defibrillation when indicated.
- Continue chest compressions during charging of the defibrillator. During compressions, ensure all rescuers stand clear other than the individual performing chest compressions.
- When defibrillating ensure that the total interruption to chest compressions is less than 5 s and after defibrillation immediately resume chest compressions.
- Immediately after a shock do not delay CPR for rhythm re-analysis or a pulse check.

- Continue CPR (30 compressions: 2 ventilations) for 2 min until rhythm re-analysis is undertaken and another shock is given (if indicated). Even if the defibrillation attempt is successful, it takes time until the post-shock circulation is established and a pulse is not usually palpable with a perfusing rhythm immediately after defibrillation.
- Patients can remain pulseless for over 2 min. The duration of asystole before ROSC can be longer than 2 min in as many as 25% of successful shocks.

Witnessed and monitored VF/pVT cardiac arrest

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area, or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and, if appropriate check for a pulse and other signs of ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is already connected to a manual defibrillator although these circumstances are rare. There are no data supporting a three-shock strategy in any of these circumstances, but 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/pVT.

Shock energies

Deliver the first shock with energy of at least 150 J. The shock energy for a particular defibrillator should be based on the manufacturer's guidance. Those using manual defibrillators should be aware of the appropriate energy settings for the type of device used, but in the absence of this and if appropriate energy levels are unknown, for adults use the highest available shock energy for all shocks.

For subsequent shocks, there remains no evidence to support either a fixed or escalating energy protocol, although an escalating protocol may be associated with a lower incidence of refibrillation. Both strategies are acceptable; however, if the first shock is not successful and the defibrillator is capable of delivering shocks of higher energy it is reasonable to increase the energy for subsequent shocks. With manual defibrillators it is also appropriate to consider escalating the shock energy in patients when refibrillation occurs.

Safety

Attempted defibrillation should be undertaken without risk to members of the resuscitation team. This is achieved best by using self-adhesive electrodes, which minimise the possibility of anyone touching the electrode during electrical discharge. Be wary of wet surroundings or clothing - wipe any water from the patient's chest before attempted defibrillation. No part of any person should make direct or indirect contact with the patient. Do not hold intravenous infusion equipment or the patient's trolley during shock delivery. The operator must ensure that everyone is clear of the patient before delivering a shock.

There is no evidence that continuous chest compressions during shock delivery increases the chance of achieving ROSC. Furthermore, the latex gloves routinely available and used by healthcare professionals do not provide sufficient protection from the electric current, therefore a shock is delivered only when everyone is clear of contact with the patient.

Safe use of oxygen during defibrillation

In an oxygen-enriched atmosphere sparking from poorly applied defibrillator paddles can cause a fire and significant burns to the patient. The use of self-adhesive electrodes is far less likely to cause sparks than manual paddles – no fires have been reported in association with the use of self-adhesive electrodes. The following are recommended as good practice:

- Take off any oxygen mask or nasal cannulae and place them at least 1 m away from the patient's chest.
- Leave the ventilation bag connected to the tracheal tube or supraglottic airway device; no increase in oxygen concentration occurs in the zone of defibrillation, even with an oxygen flow of 15 L min^{-1} .
- Alternatively, disconnect the ventilation bag from the tracheal tube or supraglottic airway device and remove it at least 1 m from the patient's chest during defibrillation.
- If the patient is connected to a ventilator, for example in the operating room or critical care unit, leave the ventilator tubing (breathing circuit) connected to the tracheal tube unless chest compressions prevent the ventilator from delivering adequate tidal volumes. In this case, the ventilator is usually substituted by a ventilation bag, which can be left connected or detached and removed to a distance of at least 1 m. Ensure that the disconnected ventilator tubing is kept at least 1 m from the patient or, better still, switch the ventilator to standby during the period of the resuscitation attempt; modern ventilators generate high oxygen flows when left disconnected. During normal use, when connected to a tracheal tube, oxygen from a ventilator in the critical care unit will be vented from the main ventilator housing well away from the defibrillation

zone. Patients in the critical care unit may be dependent on positive end expiratory pressure (PEEP) to maintain adequate oxygenation; during cardioversion, when the spontaneous circulation potentially enables blood to remain well oxygenated, it is particularly appropriate to leave the critically ill patient connected to the ventilator during shock delivery.

Automated external defibrillators

Automated external defibrillators (AEDs) are sophisticated, reliable, computerised devices that use voice and visual prompts to guide lay rescuers and healthcare professionals to attempt defibrillation safely in cardiac arrest victims (Figure 9.2). Advances in technology, particularly with respect to battery capacity, and software arrhythmia analysis have enabled the mass production of relatively cheap, reliable and easily operated portable defibrillators. Shock-advisory defibrillators have ECG-analysis capability but can usually be manually over-ridden by healthcare providers capable of rhythm recognition.



Figure 9.2 Automated external defibrillator

Automated rhythm analysis

Automated external defibrillators have microprocessors that analyse several features of the ECG, including frequency and amplitude. Some AEDs are programmed to detect spontaneous movement by the patient. Developing technology should soon enable AEDs to provide information about frequency and depth of chest compressions during CPR that may improve resuscitation performance by all rescuers.

Automated external defibrillators have been tested extensively against libraries of recorded cardiac rhythms and in many trials in adults and children. They are extremely accurate in rhythm analysis. Although AEDs are not designed to deliver synchronised shocks, all AEDs will also recommend shocks for VT if the rate and R-wave morphology exceed preset values.

In-hospital use of AEDs

When patients sustain cardiac arrest in unmonitored hospital beds and in outpatient departments, several minutes may elapse before a resuscitation team arrives with a defibrillator. There are no published randomised trials comparing in-hospital use of AEDs with manual defibrillators, and it has been shown that an AED can be used successfully before the arrival of the hospital resuscitation team.

AEDs should therefore be considered for areas of the hospital where there is a risk of delayed defibrillation because of the time taken for a resuscitation team to attend or where staff have no rhythm recognition skills or where they use defibrillators infrequently. In hospital areas where there is rapid access to manual defibrillation, either from trained staff or a resuscitation team, use manual defibrillation in preference to an AED. Ensure that an effective system for training and retraining is in place. Healthcare providers with a duty to perform CPR should be trained, equipped, and authorised to perform defibrillation, and sufficient numbers should be trained to enable the goal of providing the first shock within 3 min of collapse anywhere in the hospital. Hospitals should monitor collapse-to-first shock intervals.

Public access defibrillation (PAD) programmes

Public access defibrillation (PAD) and first responder AED programmes may increase the number of victims who receive bystander CPR and early defibrillation, thus improving survival from out-of-hospital cardiac arrest. Public access AED programmes are most effective when implemented in public places with a high density and movement of citizens such as airports, railway stations, bus terminals, sport facilities, shopping malls, and offices where cardiac arrests are usually witnessed and trained CPR providers can quickly be on scene. The Resuscitation Council (UK) and British Heart Foundation have published a guide to AEDs which provides information on how they can be deployed in the community. Registration of AEDs for public access, so that dispatchers can direct CPR providers to a nearby AED, may also help to optimise response.

Sequence for use of an AED (or shock-advisory defibrillator)

It is critically important that CPR providers pay attention to AED voice prompts and follow them without any delay.

1. Make sure the victim, any bystanders, and you are safe.
2. If the victim is unresponsive and not breathing normally:
 - ask someone to call for an ambulance or the resuscitation team and collect the AED. If you are on your own, do this yourself.
3. Start CPR according to the guidelines (Chapter 5).

4. As soon as the AED arrives:

- switch on the AED and attach the electrode pads.
- if more than one rescuer is present, continue CPR while this is done.
- follow the voice/visual directions.
- ensure that nobody touches the victim whilst the AED is analysing the rhythm.

- 5A. If a shock IS indicated:

- ensure that nobody touches the victim.
- push the shock button as directed.
- continue as directed by the voice/visual prompts.

- 5B. If NO shock is indicated:

- immediately resume CPR using a ratio of 30 compressions to 2 rescue breaths.
- continue as directed by the voice/visual prompts.

6. Continue to follow the AED prompts until:

- qualified help (e.g. ambulance or resuscitation team) arrives and takes over
- the victim starts to breathe normally, or
- you become exhausted.

Notes

- The carrying case with the AED must contain strong scissors for cutting through clothing and a disposable razor for shaving excessive chest hair in order to obtain good electrode contact.
- If ALS providers are using an AED, they should implement other ALS interventions (e.g. advanced airway techniques, ventilation, IV access, drug delivery, etc.) according to local protocols.

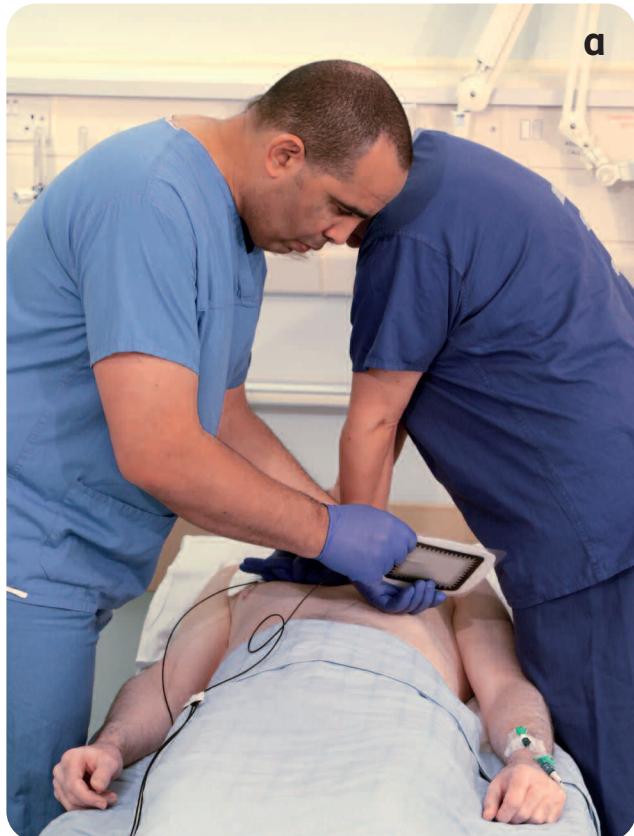
Manual defibrillation

Manual defibrillators have several advantages over AEDs. They enable the operator to diagnose the rhythm and deliver a shock rapidly without having to wait for rhythm analysis. This minimises the interruption in chest compressions. Manual defibrillators often have additional functions, such as the ability to deliver synchronised shocks, and external pacing facilities. The main disadvantage of these devices is that the operator has to be skilled in ECG rhythm recognition, therefore in comparison with AEDs, extra training is required.

Sequence for use of a manual defibrillator

This sequence is an integral part of the ALS algorithm in Chapter 6.

1. Confirm cardiac arrest – check for signs of life or if trained to do so, normal breathing and pulse simultaneously.
2. Call the resuscitation team.
3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads (Figure 9.3a) – 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/pVT from the ECG. This pause in chest compressions should be brief and no longer than 5 s.
6. Resume chest compressions immediately; warn all rescuers **other than the individual performing the chest compressions** to "stand clear" (Figure 9.3b) and remove any oxygen delivery device as appropriate.
7. The designated person selects the appropriate energy on the defibrillator and presses the charge button. Choose an energy setting of at least 150 J for the first shock, the same or a higher energy for subsequent shocks, or follow the manufacturer's guidance for the particular defibrillator.
8. Ensure that the rescuer giving the compressions is the only person touching the patient.
9. Once the defibrillator is charged and the safety check is complete, tell the rescuer doing the chest compressions to "stand clear" (Figure 9.3c); when clear, give the shock.
10. After shock delivery immediately restart CPR using a ratio of 30:2, starting with chest compressions (Figure 9.3d). Do not pause to reassess the rhythm or feel for a pulse. This pause in chest compressions should be brief and no longer than 5 s.
11. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.
12. Pause briefly to check the monitor.
13. If VF/pVT, repeat steps 6–12 above and deliver a second shock.
14. If VF/pVT persists repeat steps 6–8 above and deliver a third shock. Resume chest compressions immediately. Give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR. Withhold adrenaline if there are signs of ROSC during CPR.



Figures 9.3 a, b, c, d Operation of a manual defibrillator and efficient CPR

15. Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists.
16. Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).
17. If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of ROSC (check for signs of life, a central pulse and end-tidal CO₂ if available):
 - If there is ROSC, start post-resuscitation care.
 - If there are no signs of ROSC, continue CPR and switch to the non-shockable algorithm.
18. If asystole is seen, continue CPR and switch to the non-shockable algorithm.

Defibrillation with an AED in children

A standard AED using the energy settings already described is suitable for defibrillation of children above the age of 8 years. For defibrillation of children between 1 and 8 years, special paediatric electrodes with integral energy attenuators are recommended; these reduce the delivered energy to that recommended for manual defibrillation. If these electrodes are not available, use standard adult electrodes, ensuring that they do not overlap each other (antero-posterior electrode position may be necessary). For children below 1 year of age, based on some case reports documenting successful use in this group, it is acceptable to use an AED if no other option is available.

Synchronised cardioversion

If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronised with the R wave of the ECG (VF and pVT do not require synchronised shocks). By avoiding the relative refractory period in this way, the risk of inducing VF is minimised. Conscious patients must be anaesthetised or sedated before synchronised cardioversion is attempted. Most manual defibrillators incorporate a switch that enables the shock to be triggered by the R wave on the electrocardiogram. Electrodes are applied to the chest wall and cardioversion is achieved in the same way as attempted defibrillation, but the operator must anticipate the slight delay between pressing the buttons and the discharge of the shock when the next R wave occurs.

If synchronisation fails, choose another lead and/or adjust the amplitude. In patients with VT who are unstable, give unsynchronised shocks avoid prolonged delay in restoring sinus rhythm.

With some defibrillators, the synchronised mode has to be reset if a second or subsequent shocks are required. Other machines remain in the synchronised mode; be careful not to leave the synchronisation switch in the ‘on’ position following use as this will inhibit discharge of the defibrillator when it is next used for treating VF/pVT.

Energy doses for cardioversion are discussed in Chapter 11.

Implanted electronic devices

When a patient needs external defibrillation, effective measures to try to restore life take priority over concerns about any implanted device such as a pacemaker, implantable cardioverter-defibrillator, implantable event recorder or neurostimulator. Current resuscitation guidelines are followed, but awareness of the presence of an implanted device allows some additional measures to optimise outcome.

Cardiac pacemakers and implantable cardioverter-defibrillators

If the patient has an implanted electronic device in the chest wall, choose the position for defibrillator electrode placement carefully. Cardiac pacemakers and implantable cardioverter-defibrillators (ICDs) are usually implanted in the pectoral region, more commonly on the left than the right. Some people may have subcutaneous ICDs (S-ICDs), which are wholly subcutaneous, with a subcutaneous electrode running parallel to the sternum and the generator usually in a left lateral position.

Although modern electronic devices are designed to resist damage by external defibrillation currents, there is a remote possibility of damage when a shock is delivered through a defibrillation pad placed over, or close to, these implanted devices. There is also a theoretical risk of damage to the patient’s myocardium due to excess current flow. This may elevate pacing thresholds or damage the myocardium at the electrode-tissue interface.

To minimise this risk, place the defibrillator electrodes away from the pacemaker or ICD generator (at least 10–15 cm) without compromising effective defibrillation. If necessary place the pads in the antero-posterior, postero-lateral or bi-axillary position as described above. In people with S-ICDs (or with other devices in a left lateral position) place the external defibrillator electrodes in the antero-posterior position.

An implanted ICD gives no warning when it delivers a shock. On sensing a shockable rhythm, an ICD will discharge approximately 40 J (approximately 80 J for subcutaneous devices) through an internal pacing wire embedded in the right ventricle. The precise number of shocks that may be delivered in this situation will vary from one person/device to another, and is often up to eight, sometimes more. The ICD will re-start its discharge sequence if it detects even brief apparent cessation of the tachyarrhythmia (including transient slowing of heart rate below the rate programmed to trigger shocks). This could result in the patient receiving a large number of shocks, causing pain and distress. A ring magnet placed over the ICD will disable the defibrillation function in these circumstances (Chapter 10). Deactivation of an ICD in this way does not disable the ability of the device to act as a pacemaker if it has that capability.

During cardiorespiratory arrest in a shockable rhythm, external defibrillation should be attempted in the usual way if the ICD has not delivered a shock, or if its shocks have failed to terminate the arrhythmia.

Following successful resuscitation from cardiac arrest, interrogation of a pacemaker or ICD (by a cardiac physiologist from a pacemaker service) may provide valuable information about the rhythm behaviour that led to the cardiac arrest, as well as providing an opportunity to check lead thresholds and device function.

Implantable event recorders (also known as implantable loop recorders and implantable cardiac monitors)

These small devices (about the size of a computer memory stick) are used to record the heart's rhythm at the time of an event such as transient loss of consciousness. They are usually implanted under the skin on the anterior chest wall, overlying the heart, but occasionally may be placed in the axilla. They have no connected leads or other attachments and do not deliver any treatment. They present no risk to those giving CPR and no known risk to the patient during defibrillation. As with other devices there is a remote possibility of damage to the device itself by a high-energy shock if a defibrillator pad is placed directly over or close to the device, so careful pad placement (as far away from the device as possible without compromising effective defibrillation) is recommended.

If a person with an implantable event recorder suffers cardiorespiratory arrest, and CPR achieves ROSC, as soon as is clinically appropriate arrange to have the device interrogated (usually by a cardiac physiologist from a pacemaker service) because it may have recorded valuable information about the cardiac rhythm that initiated the arrest.

Implantable neurostimulators

Neurostimulators may be implanted subcutaneously in positions similar to those used for pacemakers and are similar in appearance. They are attached to the 'target' part of the nervous system by a lead, similar to a pacemaker lead. They present no known or likely risk to the patient or to those giving CPR. Similar principles to those used for ICDs are applied with these devices. Some manufacturers advise a minimum distance of 10–15 cm from the implanted device (between the edge of the implanted device and the edge of the defibrillator electrode).

Internal defibrillation

Internal defibrillation using paddles applied directly across the ventricles requires considerably less energy than that used for external defibrillation. For biphasic shocks, use 10–20 J, delivered directly to the myocardium through internal paddles. Monophasic shocks require approximately double these energy levels. Do not exceed 50 J when using internal defibrillation – failure to defibrillate at these energy levels requires myocardial optimisation before defibrillation is attempted again.

Summary learning

- **For the patient in VF/pVT, early defibrillation is the only effective means of restoring a spontaneous circulation.**
- **When using a defibrillator, minimise interruptions to chest compressions.**
- **Use at least 150 J for the first shock.**
- **For manual defibrillators, consider escalating energy levels for refractory and recurrent VF.**

My key take-home messages from this chapter

Further reading

A guide to automated external defibrillators (AEDs). Resuscitation Council (UK) and British Heart Foundation. December 2013.
<https://www.resus.org.uk/publications/a-guide-to-aeds/>

Cardiovascular implanted electronic devices in people towards the end of life, during cardiopulmonary resuscitation and after death. Guidance from the Resuscitation Council (UK), British Cardiovascular Society and National Council for Palliative Care. March 2015. <https://www.resus.org.uk/defibrillators/cardiovascular-implanted-electronic-devices/>

Soar J, Callaway CW, Aibiki M, et al. Part 4: Advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e71-e122.

Soar J, Nolan JP, Bottiger BW, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 3 Adult Advanced Life Support. Resuscitation 2015;95:99-146.

Contents

- Formation and transmission of the heart's spontaneous electrical signals
- Ways in which failure of these may result in a need for cardiac pacing
- Different methods of delivering cardiac pacing in various clinical settings
- How to recognise failure of cardiac pacing and reasons for it
- Safe and effective delivery of CPR for people who have implanted pacemakers or implanted cardioverter-defibrillators (ICDs)
- Consideration of the need for ICD implantation in people resuscitated from cardiac arrest in a shockable rhythm
- Actions needed after the death of a person with an implanted electronic device

Learning outcomes

To enable you to:

- Understand the indications for cardiac pacing in the peri-arrest setting
- Use percussion pacing
- Apply non-invasive, transcutaneous electrical pacing
- Describe the problems that may occur with temporary transvenous pacing and how to correct them
- Manage patients with implanted permanent pacemakers and cardioverter defibrillators in peri-arrest and cardiac arrest settings, and after death

Introduction

In some cardiac arrest or peri-arrest settings, use of cardiac pacing can be life-saving. Non-invasive pacing may be used to maintain cardiac output temporarily while expert help to deliver longer-term treatment is obtained. Non-invasive pacing can be established rapidly and as an ALS provider you should be able to achieve this.

The ALS provider does not need to have a detailed technical knowledge of permanent pacemakers and implanted cardioverter defibrillators (ICDs) but needs to be able to recognise when one of these devices is present, when they are failing, and how the presence of an implanted device may influence the management of a cardiac arrest.

Formation and failure of the heart's electrical signal

The electrical signal that stimulates each normal heartbeat arises in the sino-atrial (SA) node, which can be regarded as the heart's natural pacemaker. This depolarises

spontaneously and regularly, generating an electrical signal without any external stimulus. This behaviour is called ‘automaticity’, and any cardiac tissue that has this ability is capable of initiating a heartbeat and behaving as a ‘pacemaker’. Different parts of the conducting system generate spontaneous signals at different rates (Figure 10.1). The fastest ‘pacemaker’ will generate the cardiac rhythm and slower natural pacemakers will only take over if the faster ones slow down or stop working. For example, in sinus arrest or extreme sinus bradycardia the atrioventricular (AV) node may take over and provide a junctional escape rhythm and in complete atrioventricular block (complete heart block - CHB) the escape rhythm arises from ventricular myocardium or from conducting tissue below the atrioventricular node.

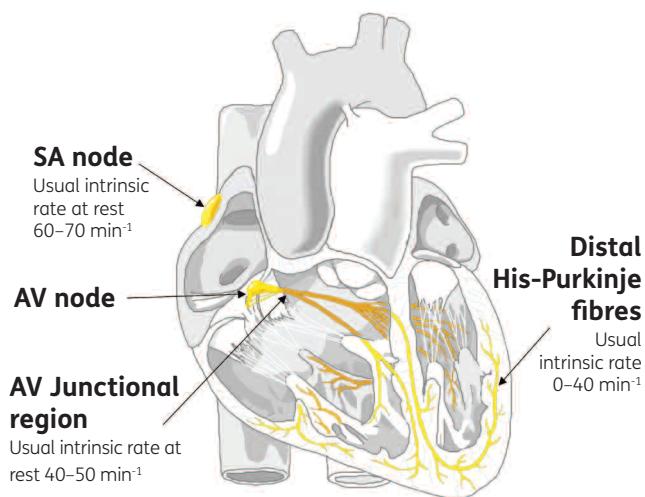


Figure 10.1 Cardiac conducting system

When CHB occurs at the level of the AV node, the most rapid automatic activity arises from cells immediately below the block and these become the new pacemaker. The heart rate produced by these cells is usually relatively fast (often about 50 min⁻¹). The resulting escape rhythm tends to be relatively stable and relatively unlikely to fail suddenly, and cause asystole.

The QRS complexes resulting from this type of block are narrow because the impulse is transmitted to the ventricles rapidly through an intact bundle of His and bundle branches. This situation may be seen complicating acute inferior myocardial infarction. In this setting, narrow-complex CHB often may not require pacing because the heart rate is not especially slow and the risk of asystole is usually low. Assess the patient (ABCDE) to identify the effect of the bradycardia and any need for treatment.

Complete heart block can occur lower in the conducting system. Examples of causes include:

- degenerative conducting tissue fibrosis
- extensive anteroseptal myocardial infarction affecting all the fibres of the bundle branches
- cardiomyopathies
- calcific valve disease.

Any automatic activity arising below this block in the distal Purkinje fibres or myocardium is likely to be slow and unreliable. In this situation, the resulting QRS complexes are broad, since the impulse passes slowly through ventricular muscle rather than rapidly through the conducting system. This escape rhythm is unreliable and may fail transiently, leading to syncope (Stokes-Adams attack), or fail completely, causing ventricular standstill and cardiac arrest. Broad-complex CHB requires cardiac pacing, and the occurrence of long ventricular pauses (> 3 s) makes this need urgent, as it implies a risk of asystole. The possible risk of more severe AV block and asystole should always be considered in a person who has presented with syncope and has any ECG evidence of conduction delay (e.g. long PR interval or bundle branch block). Start ECG monitoring and obtain expert assessment for all such patients.

In the peri-arrest setting, pacemakers are used when the heart rate is too slow or unreliable, and not responding to the treatment described in the peri-arrest algorithm for bradycardia (Chapter 11). Pacing will be effective only if the heart is able to respond to the pacing stimulus. In the setting of cardiac arrest the presence of P waves makes this more likely. Pacing is rarely successful in asystole in the absence of P waves and should not be attempted routinely in this situation.

The pacing stimulus may be mechanical, as in percussion pacing, or electrical, as in transcutaneous and transvenous pacing.

If a pacing stimulus induces an immediate QRS complex this is referred to as ‘capture’. Check that this electrical activity seen on the ECG is accompanied by mechanical activity that produces a palpable pulse.

Methods of pacing

Methods of pacing may be classified as:

Non-invasive

- Percussion pacing (“fist pacing”)
- Transcutaneous pacing

Invasive

- Temporary transvenous pacing
- Permanent pacing (using an implanted pacemaker)

Implanted devices that deliver pacing include pacemakers implanted for the treatment of bradycardia, biventricular pacemakers implanted for the treatment of heart failure (cardiac resynchronisation therapy) and implanted cardioverter defibrillators (ICDs), which also have a pacemaker function.

Non-invasive pacing

Percussion pacing

When bradycardia is so profound that it causes clinical cardiac arrest, percussion pacing (often called ‘fist pacing’) can be used in preference to CPR because it may produce an adequate cardiac output with less trauma to the patient. It is more likely to be successful when ventricular standstill is accompanied by continuing P wave activity (Chapter 8).

How to perform percussion pacing

1. With the side of a closed fist deliver repeated firm thumps to the praecordium, just lateral to the lower left sternal edge.
2. Raise the hand about 20 cm above the chest before each thump.
3. Monitor the ECG and assess whether a QRS complex is generated by each thump.
4. If possible a second person should check whether a pulse is generated by each QRS complex.
5. If initial thumps do not produce a QRS complex try using slightly harder thumps.
6. If this still fails to produce a QRS complex move the point of contact around the praecordium until a site is found that produces repeated ventricular stimulation.

Percussion pacing is not as reliable as electrical pacing in stimulating QRS complexes. If percussion does not produce a regular pulse promptly, regardless of whether or not it generates QRS complexes, start CPR immediately.

Like CPR, percussion pacing is an emergency measure that is used to try to maintain circulation to vital organs. In this way it may enable either recovery of a spontaneous cardiac rhythm or initiation of transcutaneous or transvenous pacing.

Transcutaneous pacing

Compared with transvenous pacing, non-invasive transcutaneous pacing has the following advantages:

- It can be established very quickly.
- It is easy to perform and requires a minimum of training.
- It can be initiated by healthcare providers including nurses, paramedics and doctors, while waiting for expert help to establish transvenous pacing.

The major disadvantage of transcutaneous pacing in the conscious patient is discomfort. The pacing impulse stimulates painful contraction of chest wall muscles as well as causing some direct discomfort. Many defibrillators also have the ability to deliver transcutaneous pacing. Stand-alone, non-invasive pacing devices may also be available in some hospital departments.

Most transcutaneous pacing systems are capable of demand pacing in which the device detects spontaneous QRS complexes and delivers a pacing stimulus only when it is needed.

How to perform transcutaneous pacing

1. Avoid delay, but pay careful attention to technique in order to increase the chance of success.
2. If necessary, use scissors or a razor to quickly remove excess chest hair from the skin where the electrode (pad) is to be applied.
3. Make sure that the skin is dry.
4. If necessary attach ECG monitoring electrodes and leads - these are needed with some transcutaneous pacing devices.
5. Position the pads in the ‘conventional’ right pectoral and apical positions if possible (Figure 10.2 a). For right pectoral and apical positions place one pad over the right pectoral muscle, just below the clavicle. Place the apical pad in the left mid-axillary line, overlying the V6 ECG electrode position. Apply this pad to the chest wall, not over breast tissue. If this is prevented (e.g. by chest trauma or an implanted device in this position) anterior-posterior (A-P) pad positions can be used.
6. For A-P positions (Figure 10.2b-d) place the anterior pad on the left anterior chest wall, beside the sternum, overlying the V2 and V3 ECG electrode positions. Place the posterior pad between the lower part of the left scapula and the spine, at the same horizontal level on the trunk as the anterior pad. These precise positions may not be optimal for cardioversion of atrial tachyarrhythmia, for which the anterior pad is best placed overlying the right sternal border.
7. If you are using a pacing device that is not capable of defibrillation, use A-P positions for the pacing electrode pads so that defibrillator pads can still be applied in the right pectoral and apical positions.
8. Make sure that you are familiar with the device that you are using and that you know how to operate it. Different transcutaneous pacing devices have different operational features. For example, some require the operator to increase the current delivered with each pacing stimulus until electrical capture is achieved, whilst others use a constant current that cannot be adjusted and a longer pulse duration (duration of the pacing stimulus) than other devices.

9. Most transcutaneous pacing devices pace the heart in demand mode. The pacemaker will be inhibited if it detects a spontaneous QRS complex. However, if there is movement artefact on the ECG this may inhibit the pacemaker. Avoid causing movement artefact as far as possible. If artefact still appears to be inhibiting the pacemaker, switch the device to deliver fixed-rate pacing.
10. Select an appropriate pacing rate. This will usually be in the range $60\text{--}90 \text{ min}^{-1}$ for adults, but in some circumstances (e.g. complete AV block with an idioventricular rhythm at 50 min^{-1}) a slower pacing rate of 40 or even 30 min^{-1} may be appropriate in order to deliver pacing only during sudden ventricular standstill or more extreme bradycardia.
11. If the pacing device has an adjustable energy output set this at its lowest value and turn on the pacemaker. Gradually increase the output while observing the patient and the ECG. As the current is increased the muscles of the chest wall will contract with each impulse and a pacing ‘spike’ will appear on the ECG (Figure 10.3a). Increase the current until each pacing spike is followed immediately by a QRS complex, indicating electrical capture (typically with a current of $50\text{--}100 \text{ mA}$). This means that the pacing stimuli are causing depolarisation of the ventricles (Figure 10.3b).
12. Check that the apparent QRS complex is followed by a T wave. Occasionally, artefact generated by the pacing current travelling through the chest may look like a QRS complex, but such artefact will not be followed by a T wave (Figure 10.3).

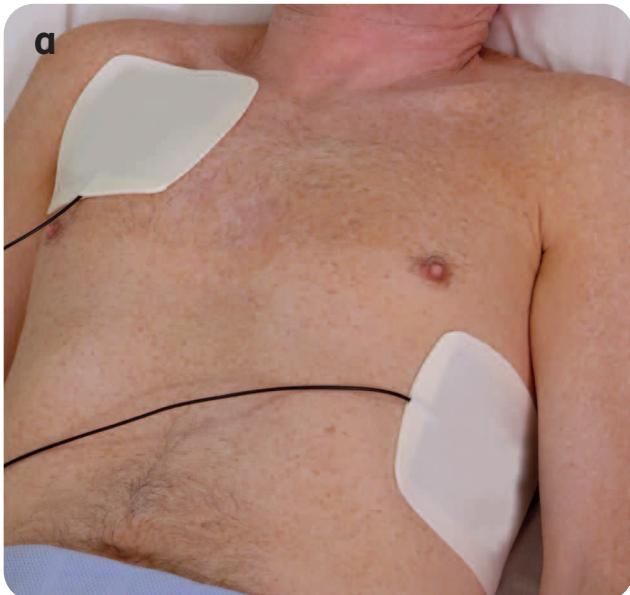


Figure 10.2a Conventional pectoral and apical pad positions can be used for pacing and for defibrillation

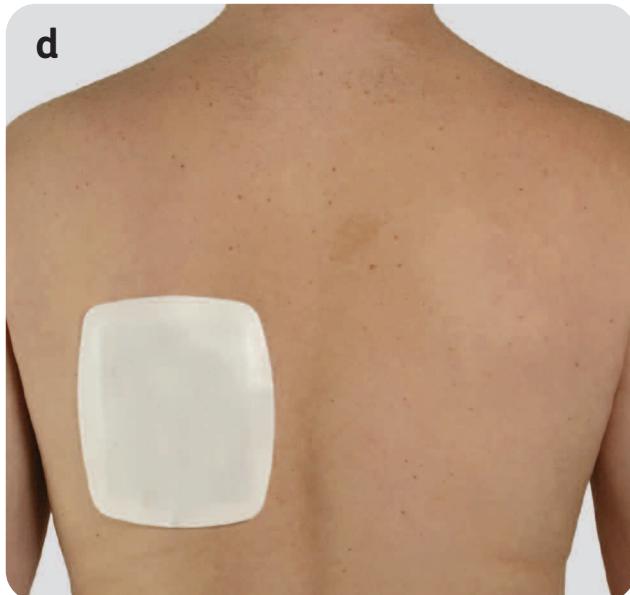
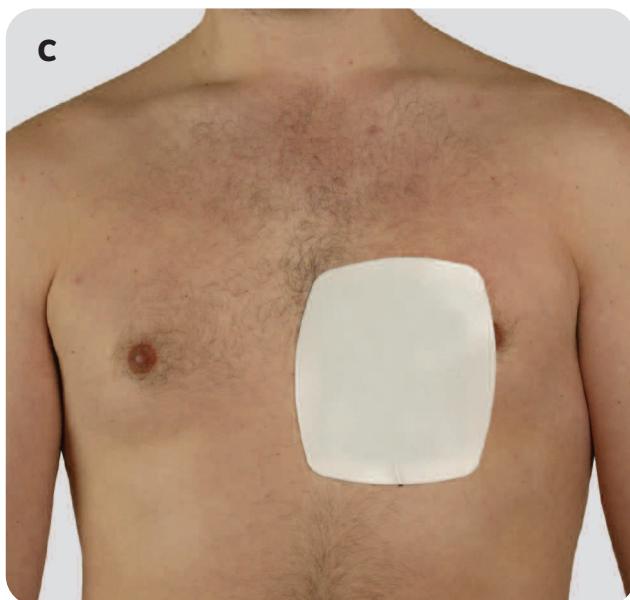
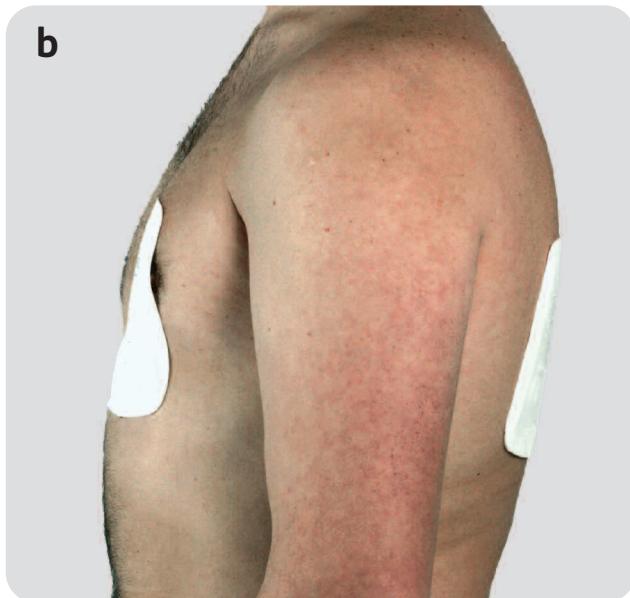


Figure 10.2b - d. Anterior-posterior (AP) pad positions for external pacing.

13. If the highest current setting is reached and electrical capture has not occurred, try changing the electrode positions. Continued failure to achieve electrical capture may indicate non-viable myocardium, but other conditions (e.g. severe hyperkalaemia) may prevent successful pacing.
14. Having achieved electrical capture with the pacemaker, check that each paced QRS complex is followed by a pulse. A palpable pulse confirms a mechanical response of the heart (i.e. contraction of the myocardium) to the paced QRS complex. Good electrical capture that fails to generate a pulse constitutes pulseless electrical activity (PEA). This may be due to severe myocardial failure but consider other possible causes of PEA in these circumstances.
15. Warn conscious patients that they are likely to experience considerable discomfort during transcutaneous pacing. Be ready to give them intravenous analgesia and/or sedation as required, especially if prolonged transcutaneous pacing is needed. If sedation is used, reassess the patient frequently (ABCDE) because sedative drugs may suppress respiratory effort.
16. If necessary, provide good-quality chest compressions. These can be given and other manual contact with the patient maintained as necessary with transcutaneous electrodes in place. There is no hazard from transcutaneous pacing to people who are in contact with the patient. However, there is no benefit in trying to deliver transcutaneous pacing during chest compressions, so it is best to turn off the pacemaker whilst CPR is in progress.
17. When transcutaneous pacing produces an adequate cardiac output seek expert help immediately to arrange emergency transvenous pacing.

Invasive pacing

Temporary transvenous pacing

It is rarely appropriate to try to attempt to insert a transvenous pacing wire during a cardiac arrest. In this setting, use non-invasive pacing to attempt to achieve a cardiac output, and then seek expert help with transvenous pacing. Failure of an existing temporary transvenous pacing system may cause cardiac arrest, particularly when the patient is pacing-dependent. Temporary transvenous pacing systems can fail in three ways:

1. High threshold

When a temporary pacing lead is inserted the usual aim is to position its tip in the apex of the right ventricle, where it is least likely to be displaced. After positioning the lead, it is used to pace the heart. The pacing 'threshold' is measured by gradually reducing the voltage delivered by the pacemaker to determine the minimum voltage needed to

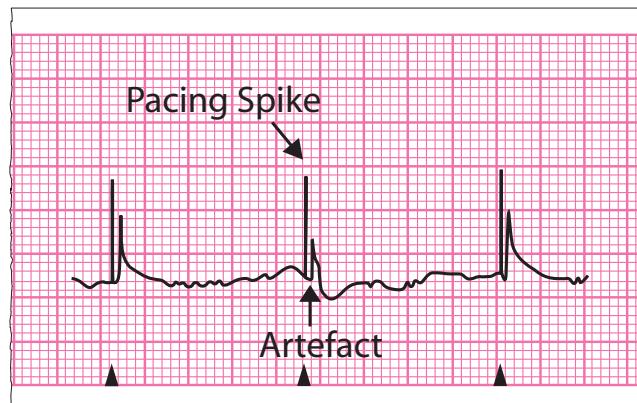


Figure 10.3a Transcutaneous pacing. ECG appearance of pacing spikes without ventricular capture

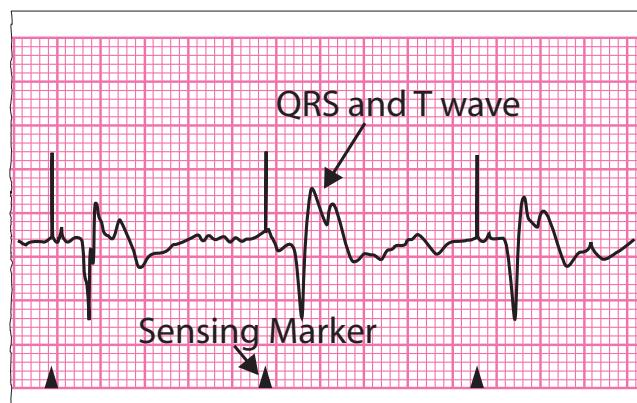


Figure 10.3b. Transcutaneous pacing. ECG shows ventricular capture after each pacing spike

stimulate the ventricle. The usual aim is to achieve a threshold of < 1.0 V at the time of lead insertion. Higher thresholds suggest that the electrode is not making satisfactory contact with the myocardium, so there may be a need to reposition the lead.

It is usual to pace the heart with a 3–4 V stimulus, well above the initial pacing threshold. Over the first days and weeks after insertion of a pacing lead (temporary or permanent) a transient rise in the threshold can be expected.

Check the threshold on temporary pacing leads at least daily to make sure that the output of the pacemaker is well above the threshold. If not, loss of capture may occur. This is seen on the ECG as a pacing spike without a subsequent QRS complex. Loss of capture may be intermittent, so any apparent 'missed beat' of this nature should prompt a repeat check of the pacing threshold.

If loss of capture occurs because of a high threshold, increase the output of the pacemaker immediately to well above the threshold. A sudden increase in pacing threshold may be caused by lead displacement, so obtain prompt expert help, as repositioning of the lead may be needed.

2. Connection failure

Most temporary transvenous pacing leads are bipolar. One electrode is at the tip of the lead and the second is about

1 cm proximal to the tip. Each electrode is connected by the lead to separate connectors at the other end, outside the patient. These are usually inserted into sockets at one end of a connecting cable that in turn is connected to the terminals of the pacemaker. Make sure that all connections between the lead and the pacemaker are making good, secure contact that is unlikely to be lost easily, for example by minor movement of the lead or cable.

Failure of any of these connections will prevent delivery of the pacing stimulus to the heart, seen on the ECG as absence of a pacing spike. This may be intermittent and symptomless, or may be sudden and total and may result in syncope or cardiac arrest in asystole. When pacing failure is accompanied by loss of the pacing spike on the ECG, check all connections immediately. Check that the pacemaker has not been turned off inadvertently and check that its batteries are not depleted. If no such cause is present another possible explanation is a fracture of a wire within its insulation. This usually causes intermittent pacing failure and the fracture is more likely to be in the connecting cable than in the pacing lead. If this is suspected change the connecting cable immediately.

3. Lead displacement

The tip of an endocardial transvenous pacing lead is usually positioned in the apex of the right ventricle. There should be enough slack in the lead in the right atrium to allow for changes in posture and deep inspiration, but not so much as to encourage displacement of the lead tip.

The tip of a pacing lead may also perforate the wall of the right ventricle and enter the pericardium with little or no apparent change in position on chest radiography. Very rarely, this may cause cardiac tamponade, so consider this possibility if a patient with a recently implanted pacing lead suffers cardiac arrest with pulseless electrical activity.

When displacement or perforation occurs, the ECG will still show a pacing spike, but there is likely to be intermittent or complete loss of capture of the pacing stimulus (when the pacing spike is not followed by a QRS complex). When a pacing lead displaces but remains in the right ventricle it may trigger ventricular extrasystoles or more serious ventricular arrhythmia, including VT and VF. When transvenous pacing fails, there is a risk of ventricular standstill. This may be relatively short-lived and cause syncope, or prolonged and cause cardiac arrest in asystole. In this situation use non-invasive pacing until effective transvenous pacing can be re-established.

Cardiovascular implanted electronic devices

This term refers mainly to implanted pacemakers and implantable cardioverter-defibrillators (ICDs). Detailed guidance and quality standards for the management of these devices towards the end of life, during cardiac arrest and after death have been published by the Resuscitation Council (UK), the British Cardiovascular Society and the National Council for Palliative Care (see ‘Further reading’ below).

Implanted permanent pacemakers

Problems with permanent pacing systems are rare, because the connections between pacing electrodes and the pacemaker are much more secure. Lead displacement may occur as an early complication in the first few days after implantation, but becomes progressively less likely thereafter and rarely occurs more than 4–6 weeks after implantation. Occasional fracture of a permanent pacing lead may occur, usually following trauma such as a fall on to an outstretched arm on the side of the pacemaker. This may cause permanent or intermittent loss of the pacing spike.

When assessing a patient using the ABCDE approach check (during ‘E’) for the presence of an implanted device. These devices are usually implanted below the clavicle, often but not always on the left side. If a device is identified consider whether it is a pacemaker or an ICD and in the case of a pacemaker try to establish whether it was implanted as treatment for bradycardia or as treatment for heart failure. Be aware also that leadless pacemakers now exist. These are implanted transvenously, entirely within the right ventricle and will not be detectable on clinical examination.

If a patient with an implanted subcutaneous pacemaker or ICD has a cardiac arrest or requires cardioversion, place defibrillation pads at least 10–15 cm from the device. Devices that are implanted below the left clavicle usually present no problem with the use of standard defibrillator pad positions. If a device has been implanted below the right clavicle or just below the left axilla, use A-P positions for defibrillation or cardioversion if possible.

Biventricular pacing systems

In the past, the usual reason for implantation of a permanent pacemaker was treatment of bradycardia, caused mostly by malfunction of atrioventricular conduction or the sino-atrial node. Over several years there has been increasing use of biventricular pacemakers as ‘cardiac resynchronisation therapy’ in patients with heart failure. Many of these patients do not need pacing for bradycardia. The aim of these devices is to pace the apex of the right ventricle and the lateral wall of the left ventricle simultaneously in order to improve the co-ordination of left ventricular contraction. These pacemakers require the same precautions during defibrillation and cardioversion as any other pacemaker, but failure of a pacemaker that has been inserted for this purpose will not usually cause any major change in heart rate or any dangerous rhythm abnormality. Some biventricular pacing systems also incorporate an ICD function (see below).

Implantable cardioverter-defibrillators

These devices resemble large implanted pacemakers. Unlike a simple pacemaker, the primary function of an ICD is to terminate a life-threatening tachyarrhythmia. A ‘simple’ ICD can deliver a shock when it detects VF or very fast VT. Many of these devices are programmed also to deliver critically timed pacing stimuli to attempt to terminate VT that is not especially fast and is unlikely in

itself to cause cardiac arrest, resorting to defibrillation only if the VT accelerates or degenerates into VF. Most ICDs can function as demand pacemakers in the event of bradycardia and some devices will also deliver biventricular pacing for heart failure, as well as delivering defibrillation if required.

National and international guidelines define indications for ICD implantation. These include indications for their use in ‘primary prevention’ in people who have not experienced cardiac arrest but are at high risk of VF or VT causing sudden cardiac death. ICDs may improve survival in selected patients after major myocardial infarction, selected patients with heart failure and in some people with certain types of inherited cardiac condition.

ICDs are implanted usually in the pectoral region in a similar subcutaneous position to pacemakers. Though these devices may seem complex, the means by which they sense changes in cardiac rhythm is relatively simple, depending mainly on automated detection of very rapid heart rates (from an ECG signal). Consequently, ICDs may occasionally misdiagnose an arrhythmia, or misinterpret other electrical signals, and deliver inappropriate shocks, which are very unpleasant for a conscious patient. If necessary, to prevent inappropriate shocks, ICDs can be disabled temporarily by holding or taping a ring magnet on the skin overlying the device (Figure 10.4). This will also prevent the ICD from recognising and shocking VF and VT. Seek urgent expert help if ICD malfunction is suspected.

If a patient with an ICD has a cardiac arrest that is not terminated by the ICD, deliver CPR in the usual way. It is believed that chest compressions can be delivered without major risk to the rescuer, even if the ICD delivers an internal shock to the patient during chest compressions. However, there have been rare reports of shocks from an ICD causing transient myalgia and paraesthesia in the arms of a person delivering chest compressions. If a shockable cardiac arrest rhythm is present and is not terminated by the ICD, use external defibrillation in a standard way, taking the same precautions with choice of defibrillator pad positions as in a patient with an implanted pacemaker. During CPR, if an ICD delivers repeated inappropriate shocks that are impeding delivery of high-quality CPR consider deactivating the ICD using a ring magnet as above. Do not delay or interrupt CPR while locating or positioning a magnet for this purpose.

Consider the possible requirement for ICD implantation in any patient who has been resuscitated from cardiac arrest in a shockable rhythm outside the context of proven acute ST segment elevation myocardial infarction. All such patients should be referred before discharge from hospital for assessment by a cardiologist with expertise in heart rhythm disorders.

Implanted electronic devices – management after death

When a person dies with an active ICD in place arrange for its deactivation (usually done by a cardiac physiologist) as soon as is reasonably practicable. An ICD must be deactivated prior to its removal from the body or performance of an autopsy. Any implanted electronic devices (including pacemakers, ICDs, event ('loop') recorders and neurostimulators) must be removed prior to cremation.

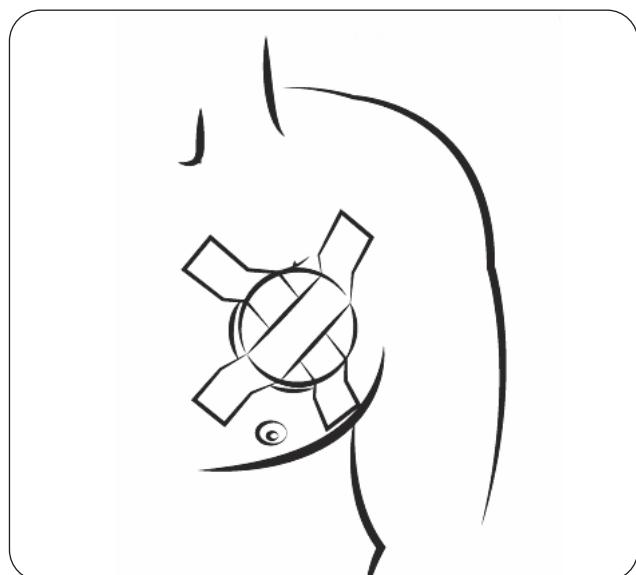
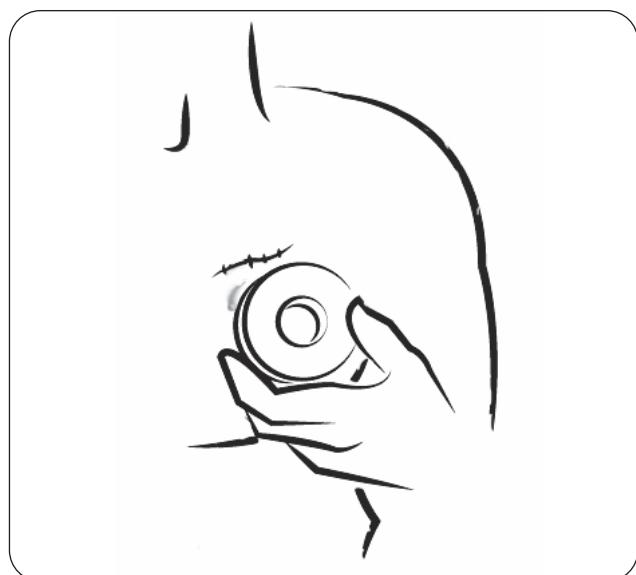
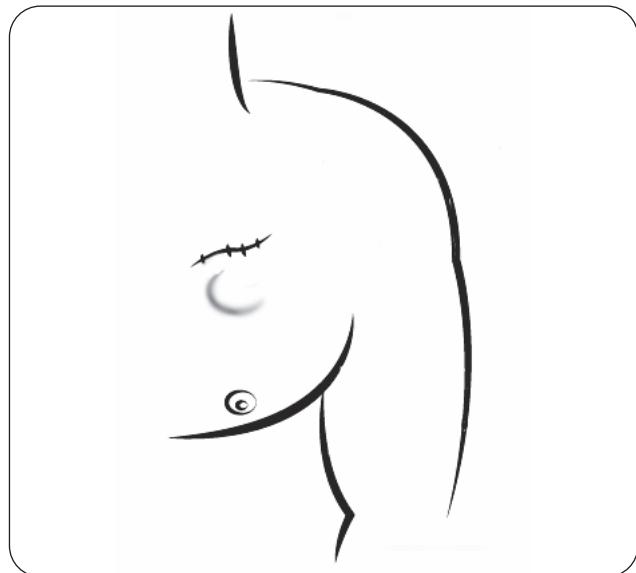


Figure 10.4 An implantable cardioverter-defibrillator can be disabled by taping a ring magnet on the skin overlying the device.

Summary learning

- Non-invasive pacing can be delivered by an ALS provider and is the immediate treatment for severe bradycardia that is a potential risk to a patient who does not respond to initial drug treatment
- Non-invasive pacing is a temporary, emergency measure to be used briefly until either a stable and effective spontaneous rhythm returns, or a competent person establishes transvenous pacing
- During resuscitation attempts in patients with implanted pacemakers and ICDs deliver CPR in the usual way, taking care not to place external defibrillator pads over or close to an implanted device
- Consider the possible need for an ICD in patients resuscitated from cardiac arrest in VT or VF, in whom there is a possible risk of recurrence
- Active ICDs should be deactivated as soon as practicable after a person's death. Remove all implanted electronic devices before cremation

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My key take-home messages from this chapter

Further reading

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Contents

- Arrhythmias that may lead to cardiac arrest or recurrent cardiac arrest
- Assessment of a patient with an arrhythmia or suspected arrhythmia
- Adverse features indicating a need for urgent treatment
- Immediate treatment of cardiac arrhythmia by non-specialists
- Tachyarrhythmia and bradyarrhythmia algorithms
- When to seek specialist/expert help

Learning outcomes

To enable you to:

- Understand the importance of arrhythmias that may precede or follow a cardiac arrest
- Assess peri-arrest arrhythmias
- Understand the principles of treating peri-arrest arrhythmias

Introduction

Rhythm abnormalities that occur in the ‘peri-arrest’ period may be considered in two main categories:

- **Arrhythmias that may lead to cardiac arrest**

Many rhythm abnormalities occur without causing cardiac arrest. Although arrhythmias are a relatively common complication of acute myocardial infarction (AMI) they are common also in patients with other cardiac abnormalities and in people who do not have coronary disease or structural heart disease. Untreated, some of these arrhythmias may lead to cardiac arrest or to avoidable deterioration in the patient’s condition. Others require no immediate treatment.

- **Arrhythmias that occur after initial resuscitation from cardiac arrest**

These often indicate that the patient’s condition is still unstable and that there is a risk of deterioration or further cardiac arrest.

You should be able to recognise common arrhythmias, know how to assess whether or not they require immediate treatment and know when to call for expert help. Arrhythmias may be categorised by heart rate as tachyarrhythmias, bradyarrhythmias and arrhythmias with a normal heart rate. The majority of peri-arrest arrhythmias will fall into the first two categories, so the remainder of this chapter will focus on these. However, some arrhythmias with a normal rate may indicate a potentially treatable risk of cardiac arrest (e.g. hypokalaemia), so any arrhythmia or suspected arrhythmia should be

assessed carefully in the context of the individual circumstances at the time. The treatment algorithms described in this section have been designed to enable the non-specialist advanced life support (ALS) provider to treat a 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 treatment options, including the use of drugs (oral or parenteral), that will be less familiar to the non-expert. In this situation you should, whenever possible, seek advice from cardiologists or other senior specialists with the appropriate expertise.

Actions to take in all arrhythmias

Sequence of actions

When an arrhythmia is present or suspected, start by assessing the patient using the ABCDE approach, including early ECG monitoring (Chapter 8). Assess the patient specifically for adverse features (see below). Clinical assessment is of limited value in identifying the precise rhythm abnormality. Whenever possible, record a 12-lead ECG at the earliest opportunity. This will help to identify the precise rhythm, either before treatment or retrospectively, if necessary with the help of an expert. Insert an intravenous cannula. If hypoxia is present give oxygen, aiming for an SpO_2 of 94–98% (88–92% in COPD).

For any patient with an arrhythmia or suspected arrhythmia assess and document:

1. the condition of the patient (presence or absence of adverse features)
2. the heart rate (bradycardia, tachycardia or normal heart rate)
3. the nature of the arrhythmia.

Adverse features

The presence or absence of adverse symptoms and/or signs will dictate the urgency and choice of treatment for most arrhythmias. The following adverse features indicate that a patient is unstable and at risk of deterioration, whether entirely or partly because of the arrhythmia:

- **Shock** - hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold extremities, confusion or impaired consciousness.
- **Syncope** - transient loss of consciousness because of global reduction in blood flow to the brain.
- **Heart failure** - pulmonary oedema and/or raised jugular venous pressure (with or without peripheral oedema and liver enlargement).
- **Myocardial ischaemia** - typical ischaemic chest pain and/or evidence of myocardial ischaemia on a 12-lead ECG.
- **Extremes of heart rate** - in addition to the above adverse features it may be appropriate to consider extremes of heart rate as adverse features in

themselves, requiring more urgent assessment and treatment than less extreme tachycardia or bradycardia with no adverse features.

(a) **Extreme tachycardia:** when heart rate increases, diastole is shortened to a greater degree than systole. Rhythm abnormalities that cause very fast heart rates (e.g. $> 150 \text{ min}^{-1}$) reduce cardiac output dramatically (because diastole is very short and the heart does not have time to fill properly) and reduce coronary blood flow (because this mostly occurs during diastole), potentially causing myocardial ischaemia. The faster the heart rate, the less well it will be tolerated.

(b) **Extreme bradycardia:** in general, the slower the bradycardia the less well it will be tolerated and heart rates below 40 min^{-1} are often tolerated poorly. This is especially so when people have severe heart disease and cannot compensate for the bradycardia by increasing stroke volume. Some people with very severe heart disease require faster than normal heart rates to maintain cardiac output, and even a ‘normal’ heart rate may be inappropriately slow for them.

Treatment options

Depending on the presence or absence of adverse features and the nature of the arrhythmia, choose one of four options for immediate treatment:

1. no treatment needed
2. simple clinical intervention (e.g. vagal manoeuvres, percussion pacing)
3. pharmacological (drug treatment)
4. electrical (cardioversion for tachyarrhythmia or pacing for bradyarrhythmia).

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 as a complication of some other condition (e.g. infection, AMI, heart failure), make sure that the underlying condition is assessed and treated appropriately, involving relevant experts if necessary.

Subsequent monitoring and treatment

After successful treatment of an arrhythmia continue to monitor the patient until you are confident that the risk of further arrhythmia is low. Remember always to record a 12-lead ECG after successful treatment of an arrhythmia because this may show abnormalities (or absence of abnormalities) that will be important in planning future management. Correct all reversible factors that may predispose to further arrhythmia. Ensure that appropriate further expert help and advice is obtained at the most appropriate time for the patient.

Tachyarrhythmia

If the patient has adverse features

These imply that the patient's condition is unstable and that they are at risk of deterioration. If this appears to be because of the tachyarrhythmia, attempt to correct this using synchronised cardioversion (Figure 11.1). In people with otherwise normal hearts, adverse signs and symptoms are uncommon if the heart rate is $< 150 \text{ min}^{-1}$ (so correction of the rhythm by cardioversion may be of limited benefit). However, people 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 $100\text{--}150 \text{ min}^{-1}$ (so may be more likely to benefit from rhythm correction by cardioversion).

If cardioversion fails to terminate the arrhythmia, and adverse features persist, give amiodarone 300 mg IV over 10–20 min and attempt further synchronised cardioversion. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 h, given into a large vein (preferably via central venous cannula).

Synchronised cardioversion

Carry out cardioversion under conscious sedation or general anaesthesia, administered by a healthcare professional competent in the technique being used. Ensure that the defibrillator is set to deliver a synchronised shock. This delivers the shock to coincide with the R wave. An unsynchronised shock could coincide with a T wave and cause ventricular fibrillation (VF).

For a broad-complex tachycardia or atrial fibrillation (AF), start with 120–150 J and increase in increments if this fails. Atrial flutter and regular narrow-complex tachycardia will often be terminated by lower-energy shocks: start with 70–120 J. For atrial fibrillation and flutter use anteroposterior defibrillator pad positions when it is practicable to do so.

When delivering the shock, press the shock button and keep it pressed until after the shock has occurred – there may be a slight delay before the shock is delivered. If a second shock is needed, remember to reactivate the synchronisation switch if necessary.

If the patient has no adverse features

If there are no adverse features consider firstly whether or not any treatment is necessary. If treatment is needed consider using drug treatment in the first instance. Assess the ECG and measure the QRS duration. If the QRS duration is 0.12 s (3 small squares at ECG paper speed 25 mm s^{-1}) or more this is a broad-complex tachycardia. If the QRS duration is $< 0.12 \text{ s}$ it is a narrow-complex tachycardia. Following any drug therapy, continue to reassess the patient (ABCDE) and monitor heart rate and rhythm to assess the response to treatment. Some anti-arrhythmic drugs cause myocardial depression, which may cause or worsen heart failure or hypotension, and in some cases an

anti-arrhythmic drug may cause other tachyarrhythmia or provoke severe bradycardia.

Broad-complex tachycardia

Broad-complex tachycardia ($\text{QRS} \geq 0.12 \text{ s}$) may be ventricular in origin or may be a supraventricular rhythm with aberrant conduction (i.e. bundle branch block).

In a person with adverse features due to the arrhythmia the distinction will not affect the immediate treatment needed. Attempt synchronised cardioversion as described above.

If a patient has a broad-complex tachycardia but no adverse features, next determine whether the rhythm is regular or irregular.

Regular broad-complex tachycardia

A regular broad-complex tachycardia may be ventricular tachycardia (VT) or a supraventricular rhythm with bundle branch block.

If the broad-complex tachycardia is considered to be VT, treat with amiodarone 300 mg IV 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 treatment strategy recommended for narrow-complex tachycardia (below).

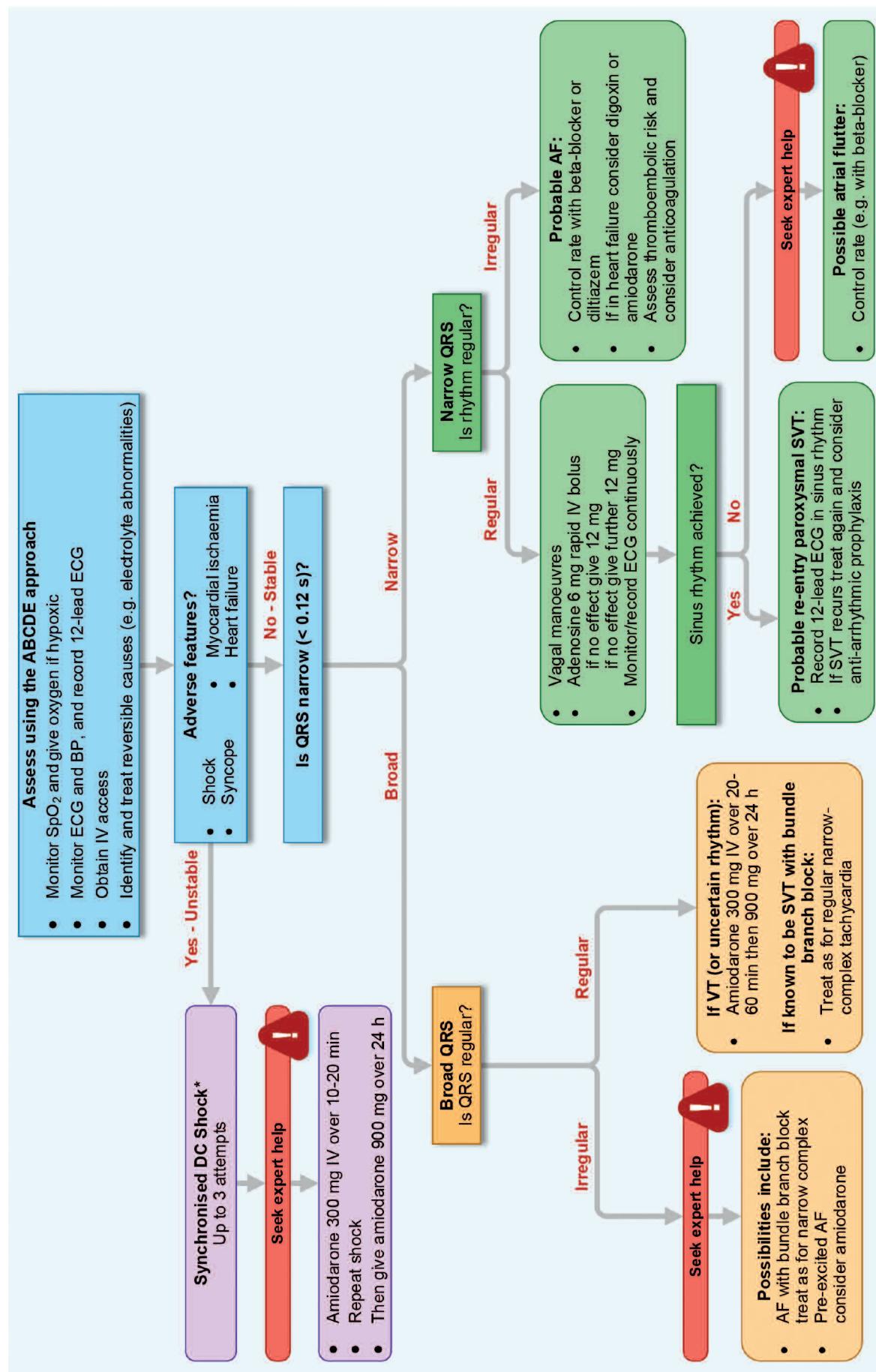
Irregular broad-complex tachycardia

This is most likely to be AF with bundle branch block, but careful examination of a 12-lead ECG (if necessary by an expert) may provide 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 by stopping immediately all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulfate 2 g IV over 10 min. 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 sinus tachycardia, atrioventricular nodal re-entry tachycardia (AVNRT) – the commonest type of regular narrow-complex tachyarrhythmia, atrioventricular re-entry tachycardia (AVRT) – due to WPW syndrome, and atrial flutter with regular AV conduction (usually 2:1).



*Conscious patients require sedation or general anaesthesia for cardioversion

Figure 11.1 Tachycardia algorithm

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 situations will usually make the situation worse. Do not attempt to treat sinus tachycardia with cardioversion or anti-arrhythmic drugs.

AVNRT and AVRT (paroxysmal supraventricular tachycardia)

Atrioventricular 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 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 usually well above the upper limit of sinus rate at rest (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.

Atrioventricular 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 with no visible atrial activity on the ECG. Like AVNRT, it may cause frightening symptoms.

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 on 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 ($125\text{--}150\text{ min}^{-1}$) may be caused by 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 tachyarrhythmia

If the patient has adverse features and is at risk of deterioration because of the tachyarrhythmia, perform synchronised cardioversion. In this situation it is

reasonable to attempt vagal manoeuvres (see below) or to give intravenous adenosine (see below) to a patient with a regular narrow-complex tachyarrhythmia while preparations are being made for synchronised cardioversion. However, do not delay electrical cardioversion if these treatments fail to terminate the arrhythmia.

In the absence of adverse features:

1. 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 12-lead) during each manoeuvre. If the rhythm is atrial flutter with 2:1 conduction, transient slowing of the ventricular response will often occur and reveal flutter waves.
2. If the arrhythmia persists and is not atrial flutter, give adenosine 6 mg as a very rapid intravenous bolus followed by a flush. 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 12-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 (i.e. no transient slowing or termination of the tachyarrhythmia) 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 be likely if the bolus is given too slowly or into a peripheral vein.
3. 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 peripheral vein).
4. If adenosine is contraindicated, or fails to terminate a regular narrow-complex tachycardia without demonstrating that it is atrial flutter, consider giving verapamil 2.5–5 mg intravenously over 2 min.

Rapid narrow-complex tachycardia with no pulse

Rarely, a very rapid (usually $> 250\text{ min}^{-1}$) narrow-complex tachycardia can impair cardiac output to such an extent that the pulse may be impalpable and consciousness impaired or lost. If the patient is pulseless and unconscious this situation is pulseless electrical activity (PEA) and you should start CPR. As the arrhythmia is potentially treatable by DC shock the most appropriate treatment then is immediate synchronised cardioversion, so this is an exception to the non-shockable limb of the ALS algorithm (Chapter 6).

Irregular narrow-complex tachycardia

An irregular narrow-complex tachycardia is most likely to be AF with a rapid ventricular response or, less commonly, atrial flutter with variable AV conduction. Record a 12-lead ECG to identify the rhythm.

If the patient has adverse features and is at risk of deterioration because of the tachyarrhythmia, perform synchronised cardioversion. In the absence of contraindications, start anticoagulation prior to cardioversion, initially with low-molecular-weight heparin or unfractionated heparin (see below), at the earliest opportunity. Do not allow this treatment to delay cardioversion.

If there are no adverse features, immediate treatment options include:

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

Obtain expert help to determine the most appropriate treatment for the individual patient. The longer a person remains in AF the greater is the likelihood of atrial thrombus developing. In general, people who have been in AF for > 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least 3 weeks, or unless transoesophageal echocardiography has detected no evidence of atrial thrombus. If the clinical situation dictates that cardioversion is needed more urgently, give either 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 1.5–2 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 may be used in patients in whom beta-blockade is contraindicated or not tolerated. 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 < 48 h and rhythm control is considered the appropriate strategy, chemical cardioversion may be appropriate. Seek expert help with

the use of drugs such as propafenone or flecainide. Do not use propafenone or flecainide in the presence of heart failure, 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 be used to attempt chemical cardioversion but is less often effective and takes longer to act. Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

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

Bradycardia

Bradycardia is defined as a resting heart rate of < 60 min⁻¹. It may be:

- physiological (e.g. in athletes or during sleep)
- 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 a patient with bradycardia using the ABCDE approach. Consider the possible cause of the bradycardia and look for adverse signs (Figure 11.2). Treat any reversible causes of bradycardia identified in the initial assessment.

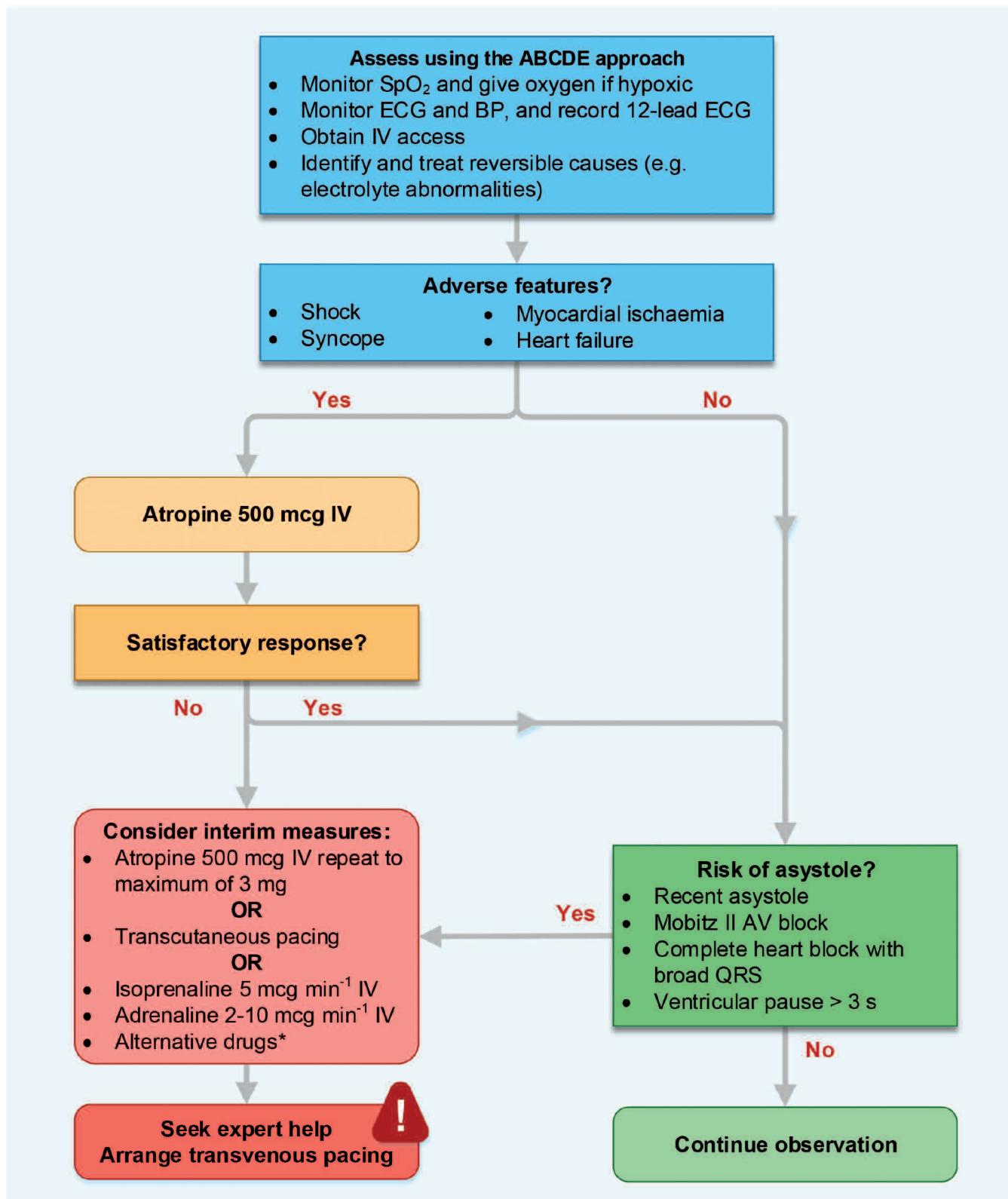
If the patient has adverse features

If adverse features are present start to treat the bradycardia. Initial treatment is usually pharmacological. Pacing is used for people in whom initial pharmacological treatment is ineffective or inadequate and those at risk of asystole.

Pharmacological treatment for bradycardia

If adverse features are present give atropine 500 mcg IV and, if necessary, repeat every 3–5 min to a total of 3 mg. Doses of atropine of < 500 mcg can cause paradoxical slowing of the heart rate. In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart rate. Use atropine cautiously in the presence of acute myocardial ischaemia or myocardial infarction; the resulting increase in heart rate may worsen ischaemia or increase the size of the infarct.

If bradycardia with adverse signs persists 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.



* Alternatives include:

- Aminophylline
- Dopamine
- Glucagon (if bradycardia is caused by beta-blocker or calcium channel blocker)
- Glycopyrrrolate (may be used instead of atropine)

Figure 11.2 Bradycardia algorithm

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 caused by digoxin toxicity. Consider using aminophylline (100–200 mg by slow IV injection) for bradycardia complicating acute inferior wall myocardial infarction, spinal cord injury or cardiac transplantation. 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. Other options for second-line drug therapy include infusion of isoprenaline (starting dose 5 mcg min⁻¹), adrenaline (2–10 mcg min⁻¹), or dopamine (2.5–10 mcg kg⁻¹ min⁻¹).

Cardiac pacing for bradycardia

For a patient with bradycardia and adverse features, if there is no response to atropine or if atropine is contraindicated initiate transcutaneous pacing immediately (Chapter 10). In the presence of life-threatening, extreme bradycardia use percussion pacing as an interim measure until transcutaneous pacing is achieved. Give repeated rhythmic thumps with the side of a closed fist over the left lower edge of the sternum at a rate of 50–70 min⁻¹ to try to stimulate the heart with each thump.

Transcutaneous pacing can be painful and may fail to achieve effective electrical ‘capture’ (i.e. a QRS complex after each pacing stimulus) or fail to achieve a mechanical response (i.e. palpable pulse). Check for electrical capture on the monitor or ECG and, when achieved, check whether it is producing a pulse. Reassess the patient’s condition (ABCDE). Use analgesia and sedation as necessary to control pain and distress. Remember that sedation may compromise respiratory effort, so continue to reassess the patient at frequent intervals. Attempt to identify the cause of the bradyarrhythmia.

Seek expert help to assess the need for temporary transvenous pacing and to initiate this when appropriate. Consider temporary transvenous pacing (or early permanent pacemaker implantation as appropriate) if there is documented recent asystole (ventricular standstill of > 3 s), Mobitz type II AV block, or complete (third-degree) AV block (especially with broad QRS or initial heart rate < 40 min⁻¹).

If the patient has no adverse features

For a patient with bradycardia who has no adverse features and no high risk of progression to asystole, do not initiate immediate treatment. Continue to monitor the patient and assess them to identify the cause of the bradycardia. If the cause is physiological or easily reversible (e.g. by stopping suppressant drug therapy) no further treatment may be needed. Seek expert help with further assessment and treatment of people with other causes of bradycardia.

Summary learning

- **Arrhythmias occurring after resuscitation from cardiac arrest and ROSC may need treatment to stabilise the patient and prevent recurrence of cardiac arrest.**
- **In other settings some arrhythmias require prompt treatment to prevent deterioration (including progression to cardiac arrest) and others do not require immediate treatment.**
- **The urgency for treatment and the best choice of immediate treatment is determined by the condition of the patient (presence or absence of adverse features) and by the nature and cause of the arrhythmia.**
- **Assessment of a patient with an arrhythmia should follow the ABCDE approach.**
- **Whenever possible the arrhythmia should be documented on a 12-lead ECG.**

My key take-home messages from this chapter

Further reading

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Resuscitation in special circumstances

Chapter
12

Contents

- Life-threatening electrolyte disorders
- Sepsis
- Toxins (poisoning)
- Asthma
- Anaphylaxis
- Pregnancy
- Traumatic cardiorespiratory arrest
- Perioperative cardiac arrest (including after cardiac surgery)
- Drowning
- Accidental hypothermia
- Hyperthermia
- Lightning strike and electrical injuries

Learning outcomes

To enable you to:

- Use the ABCDE approach for early recognition and treatment to prevent cardiac arrest
- Consider the modifications required before and after cardiac arrest in special circumstances

Introduction

Resuscitation needs to be modified in specific circumstances. Early recognition of signs and symptoms and effective treatment will often prevent cardiac arrest. These conditions account for a large proportion of cardiac arrests in younger patients with no co-existing disease. It is essential to ask for expert help early for most of these conditions because they can require specialist interventions.

Survival in all these conditions still relies on using the ABCDE approach to help prevent cardiac arrest. If cardiac arrest does occur, high quality CPR with minimal interruption and treatment of reversible causes are still the most important interventions.

Life-threatening electrolyte disorders

Electrolyte abnormalities can cause cardiac arrhythmias or cardiorespiratory arrest. Life-threatening arrhythmias are most commonly associated with potassium disorders, particularly hyperkalaemia, and less commonly with disorders of serum calcium and magnesium.

Consider starting treatment in life-threatening electrolyte disorders before laboratory results are available. Electrolyte values for definitions are quoted as a guide to clinical decision-making. The precise values that trigger treatment decisions will depend on the patient's clinical condition and rate of change of electrolyte values.

Prevention of electrolyte disorders

- Treat life-threatening electrolyte abnormalities before cardiac arrest occurs.
- Remove precipitating factors (e.g. drugs, diet) and monitor electrolyte concentrations to prevent recurrence of the abnormality.
- Monitor renal function in patients at risk of electrolyte disorders (e.g. patients with acute and chronic kidney disease, heart failure) and avoid combinations of drugs that may exacerbate electrolyte disorders.
- Review renal replacement therapy (e.g. haemodialysis) regularly to avoid inappropriate electrolyte shifts during treatment.

Potassium disorders

Potassium homeostasis

Extracellular potassium concentration is tightly regulated between 3.5–5.0 mmol L⁻¹. A large concentration gradient normally exists between intracellular and extracellular fluid compartments. This potassium gradient across cell membranes contributes to the excitability of nerve and muscle cells, including the myocardium. Evaluation of serum potassium must take into consideration the effects of changes in serum pH. When serum pH decreases (acidosis), serum potassium increases because potassium shifts from the cellular to the vascular space; a process that is reversed when serum pH increases (alkalosis). Anticipate the effects of pH changes on serum potassium during therapy for hyperkalaemia or hypokalaemia.

Hyperkalaemia

Hyperkalaemia is the most common electrolyte disorder associated with cardiac arrest and occurs in up to 10% of hospitalised patients. Acute hyperkalaemia is more likely than chronic hyperkalaemia to cause life-threatening cardiac arrhythmias or cardiac arrest. Hyperkalaemia is usually caused by increased potassium release from cells or impaired excretion by the kidneys.

Definition of hyperkalaemia

There is no universal definition. We have defined hyperkalaemia as a serum potassium concentration > 5.5 mmol L⁻¹; in practice, hyperkalaemia is a continuum. As the potassium concentration increases above 5.5 mmol L⁻¹, the risk of adverse events increases and the need for urgent treatment increases. Severe hyperkalaemia has been defined as a serum potassium concentration > 6.5 mmol L⁻¹.

Causes of hyperkalaemia

The main causes of hyperkalaemia are:

- Renal failure (i.e. acute kidney injury or chronic kidney disease).
- Drugs (e.g. angiotensin converting enzyme inhibitors (ACE-I), angiotensin II receptor antagonists (ARB), potassium sparing diuretics, non-steroidal anti-inflammatory drugs, beta-blockers, trimethoprim).
- Tissue breakdown (e.g. rhabdomyolysis, tumour lysis, haemolysis).
- Metabolic acidosis (e.g. renal failure, diabetic ketoacidosis).
- Endocrine disorders (e.g. Addison's disease).
- Diet (may be sole cause in patients with advanced chronic kidney disease).
- Spurious – pseudo-hyperkalaemia describes the finding of a raised serum (clotted blood) K⁺ value, when the actual value in plasma (non-clotted blood) is normal. The clotting process releases K⁺ from cells and platelets. The most common cause is a prolonged transit time to the laboratory or poor storage conditions.

The risk of hyperkalaemia is even greater when there is a combination of factors.

Recognition of hyperkalaemia

Exclude hyperkalaemia in all patients with an arrhythmia or cardiac arrest. Patients can present with weakness progressing to flaccid paralysis, paraesthesia, or depressed deep tendon reflexes. Most patients will have ECG abnormalities at a serum potassium concentration > 6.7 mmol L⁻¹. The use of a blood gas analyser that measures potassium helps reduce delays in recognition. The effect of hyperkalaemia on the ECG depends on the absolute serum potassium concentration as well as the rate of increase (Figure 12.1).

ECG changes with hyperkalaemia are usually progressive and include:

- first degree heart block (prolonged PR interval > 0.2 s)
- flattened or absent P waves
- tall, peaked (tentied) T waves (i.e. T wave larger than R wave in more than one lead)
- ST-segment depression
- S and T wave merging (sine wave pattern)
- widened QRS (> 0.12 s)
- ventricular tachycardia
- bradycardia
- cardiac arrest (PEA, ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT), asystole).

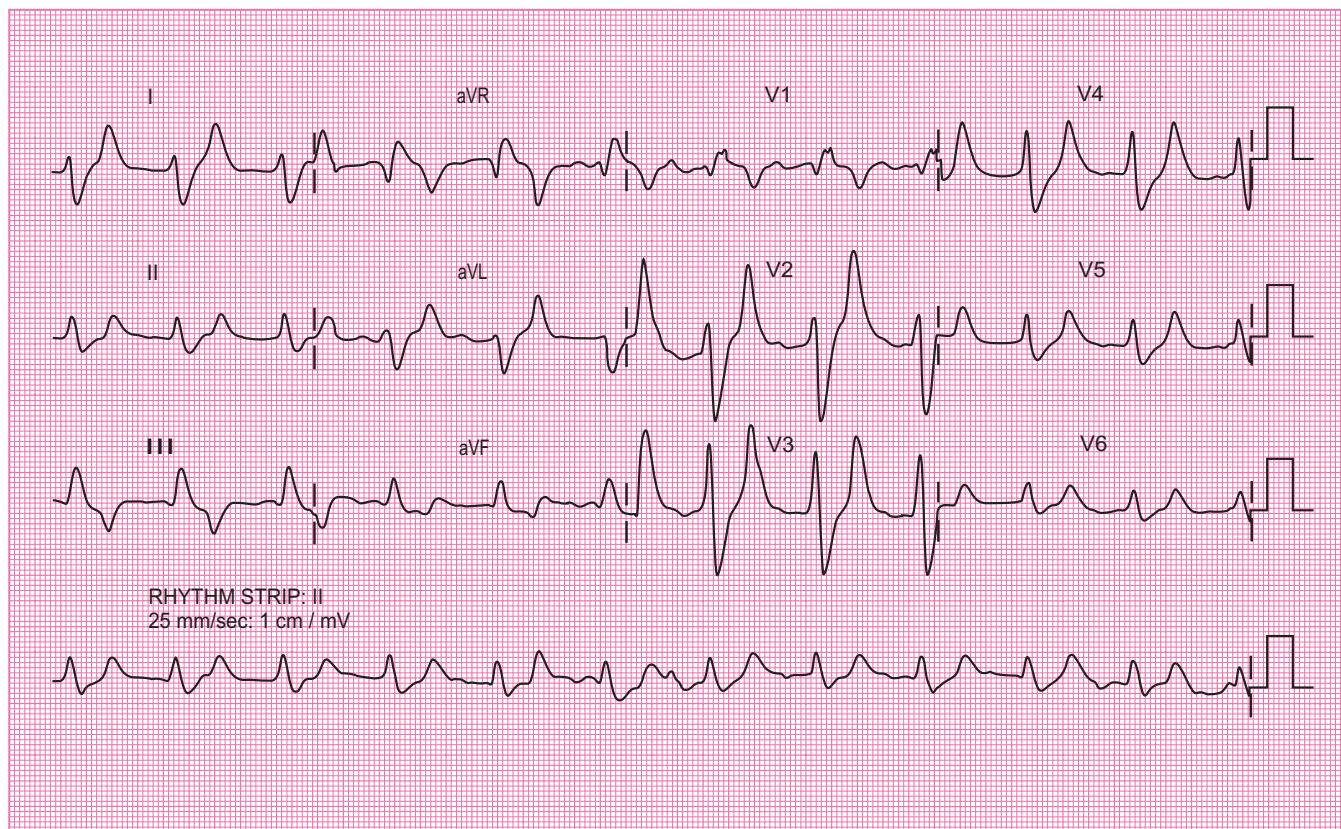


Figure 12.1 12-lead ECG showing features of hyperkalaemia

Treatment of hyperkalaemia

The five key steps in treating hyperkalaemia are:

1. Cardiac protection.
2. Shifting potassium into cells.
3. Removing potassium from the body.
4. Monitoring serum potassium concentration.
5. Prevention of recurrence.

When hyperkalaemia is strongly suspected (e.g. in the presence of ECG changes), start life-saving treatment even before laboratory results are available. Involve expert help from renal or intensive care teams at an early stage, especially for those patients who might require renal replacement therapy (e.g. haemodialysis).

The main risks associated with the treatment of hyperkalaemia are:

- Hypoglycaemia following insulin-glucose administration (usually occurs within three hours of treatment, but may occur up to six hours after the infusion ends). Monitor blood glucose and treat hypoglycaemia promptly.
- Tissue necrosis secondary to extravasation of intravenous calcium salts. Ensure secure vascular access prior to administration.
- Intestinal necrosis or obstruction following use of potassium exchange resins. Avoid prolonged use of resins and give laxative.
- Rebound hyperkalaemia after the effect of drug treatment has worn off (i.e. within 4–6 h). Continue to

monitor serum potassium for a minimum of 24 h after an episode.

Patient not in cardiac arrest

Assess patient:

- Use the ABCDE approach and correct any abnormalities, obtain IV access
- Check serum potassium
- Record a 12-lead ECG.

Treatment is determined according to severity of hyperkalaemia. Approximate values are provided to guide treatment.

Mild elevation ($5.5\text{--}5.9 \text{ mmol L}^{-1}$):

- Address cause of hyperkalaemia to correct and avoid further rise in serum potassium (e.g. drugs, diet).
- If treatment is indicated, remove potassium from the body: potassium exchange resins – calcium resonium 15–30 g, or sodium polystyrene sulfonate (Kayexalate) 15–30 g given either orally or by retention enema (onset in > 4 h).

Moderate elevation ($6.0\text{--}6.4 \text{ mmol L}^{-1}$) without ECG changes:

Shift potassium intracellularly with glucose/insulin: 10 units short-acting insulin and 25 g glucose IV over 15–30 min (onset in 15–30 min; maximal effect at 30–60 min;

duration of action 4–6 h; monitor blood glucose). Remove potassium from the body (see above; consider dialysis guided by clinical setting).

Severe elevation ($\geq 6.5 \text{ mmol L}^{-1}$) without ECG changes:

- Seek expert help.
- Give glucose/insulin (see above).
- Give salbutamol 10–20 mg nebulised (onset in 15–30 min; duration of action 4–6 h).
- Remove potassium from the body (consider dialysis).

Severe elevation ($\geq 6.5 \text{ mmol L}^{-1}$) with toxic ECG changes:

- Seek expert help.
- Protect the heart with calcium chloride: 10 mL 10% calcium chloride IV over 2–5 min to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane. This protects the heart by reducing the risk of VF/pVT but does not lower serum potassium (onset in 1–3 min).
- Use shifting agents (glucose/insulin and salbutamol).
- Remove potassium from the body (consider dialysis at outset or if refractory to medical treatment).

Modifications to cardiopulmonary resuscitation associated with hyperkalaemia

The following modifications to standard ALS guidelines are recommended in the presence of severe hyperkalaemia during CPR:

- Confirm hyperkalaemia using a blood gas analyser if available.
- Protect the heart: give 10 mL calcium chloride 10% IV by rapid bolus injection.
- Shift potassium into cells: give glucose/insulin: 10 units short-acting insulin and 25 g glucose IV by rapid injection. Monitor blood glucose.
- Give sodium bicarbonate: 50 mmol IV by rapid injection (if severe acidosis or renal failure).
- Remove potassium from body: consider dialysis for hyperkalaemic cardiac arrest resistant to medical treatment. Several dialysis modalities have been used safely and effectively in cardiac arrest, but this may only be available in specialist centres. Consider the use of a mechanical chest compression device if prolonged CPR is needed.

Cardiac arrest during haemodialysis

Sudden cardiac death is the most common cause of death in haemodialysis patients and is usually preceded by ventricular arrhythmias.

- Call the resuscitation team and seek expert help immediately.

- Start resuscitation according to standard ALS protocols ensuring high quality CPR and minimising interruptions.
- Assign a trained dialysis nurse to the dialysis machine. Stop ultrafiltration (i.e. fluid removal) and give a fluid bolus. Return the patient's blood volume and disconnect from the dialysis machine.
- Leave dialysis access open and use for drug administration.
- Minimise delay in delivering defibrillation. VF/pVT is more common in patients undergoing haemodialysis than in the general population. Most haemodialysis machine manufacturers recommend disconnection from the dialysis equipment prior to defibrillation. Ensure familiarity with local dialysis equipment and check if equipment has a defibrillator-proof label.
- All of the standard reversible causes (4 Hs and 4 Ts) apply to dialysis patients. Electrolyte disorders, particularly hyperkalaemia, and fluid overload (e.g. pulmonary oedema) are most common causes.

Hypokalaemia

Hypokalaemia is the most common electrolyte disorder in clinical practice. It is seen in up to 20% of hospitalised patients. Hypokalaemia increases the incidence of arrhythmias and sudden cardiac death (SCD). The risk is increased further in patients with pre-existing heart disease and in those treated with digoxin.

Definition of hypokalaemia

Hypokalaemia is defined as a serum potassium level $< 3.5 \text{ mmol L}^{-1}$. Severe hypokalaemia is defined as a serum potassium level $< 2.5 \text{ mmol L}^{-1}$ and may be associated with symptoms.

Causes of hypokalaemia

The main causes of hypokalaemia include:

- Gastrointestinal loss (e.g. diarrhoea).
- Drugs (e.g. diuretics, laxatives, steroids).
- Renal losses (e.g. renal tubular disorders, diabetes insipidus, dialysis).
- Endocrine disorders (e.g. Cushing's syndrome, hyperaldosteronism).
- Metabolic alkalosis.
- Magnesium depletion.
- Poor dietary intake.

Treatment strategies used for hyperkalaemia may also induce hypokalaemia.

Recognition of hypokalaemia

Exclude hypokalaemia in every patient with an arrhythmia or cardiac arrest. In dialysis patients, hypokalaemia may occur at the end of a haemodialysis session or during treatment with peritoneal dialysis.

As serum potassium concentration decreases, the nerves and muscles are predominantly affected, causing fatigue, weakness, leg cramps, constipation. In severe cases (serum potassium $< 2.5 \text{ mmol L}^{-1}$), rhabdomyolysis, ascending paralysis and respiratory difficulties may occur.

ECG features of hypokalaemia are:

- U waves
- T wave flattening
- ST segment changes
- arrhythmias, especially if patient is taking digoxin
- cardiac arrest (VF/pVT, PEA, asystole).

Treatment of hypokalaemia

This depends on the severity of hypokalaemia and the presence of symptoms and ECG abnormalities. Gradual replacement of potassium is preferable, but in an emergency, IV potassium is required. The maximum recommended IV dose of potassium is 20 mmol h^{-1} , but more rapid infusion (e.g. 2 mmol min^{-1} for 10 min, followed by 10 mmol over 5–10 min) is indicated for unstable arrhythmias when cardiac arrest is imminent.

Continuous ECG monitoring is essential during IV infusion and the dose should be titrated after repeated sampling of serum potassium levels.

Disorder	Causes	Presentation	ECG	Treatment
Hypercalcaemia Total Calcium* $> 2.6 \text{ mmol L}^{-1}$	Primary or tertiary hyperparathyroidism Malignancy Sarcoidosis Drugs	Confusion Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Short QT interval Prolonged QRS Interval Flat T waves AV block Cardiac arrest	Fluid replacement IV Furosemide 1 mg kg^{-1} IV Hydrocortisone 200–300 mg IV Pamidronate 30–90 mg IV Treat underlying cause
Hypocalcaemia Total Calcium* $< 2.1 \text{ mmol L}^{-1}$	Chronic renal failure Acute pancreatitis Calcium channel blocker overdose Toxic shock syndrome Rhabdomyolysis Tumour lysis syndrome	Paraesthesia Tetany Seizures AV-block Cardiac arrest	Prolonged QT interval T wave inversion Heart block Cardiac arrest	Calcium chloride 10% 10–40 mL IV Magnesium sulfate (2–4 mL: 4–8 mmol) IV if necessary
Hypermagnesaemia [Magnesium] $> 1.1 \text{ mmol L}^{-1}$	Renal failure Iatrogenic	Confusion Weakness Respiratory depression AV-block Cardiac arrest	Prolonged PR and QT intervals T wave peaking AV block Cardiac arrest	Consider treatment when magnesium $> 1.75 \text{ mmol L}^{-1}$ Calcium chloride 10% 5–10 mL IV repeated if necessary Ventilatory support if necessary Saline diuresis—0.9% saline with furosemide 1 mg kg^{-1} IV Haemodialysis
Hypomagnesaemia [Magnesium] $< 0.6 \text{ mmol L}^{-1}$	GI loss Polyuria Starvation Alcoholism Malabsorption	Tremor Ataxia Nystagmus Seizures Arrhythmias – torsade de pointes Cardiac arrest	Prolonged PR and QT Intervals ST-segment depression T-wave inversion Flattened P waves Increased QRS duration Torsade de pointes	Severe or symptomatic: 2 g 50% magnesium sulfate (4 mL; 8 mmol) IV over 15 min Torsade de pointes: 2 g 50% magnesium sulfate (4 mL; 8 mmol) IV over 1–2 min Seizure: 2 g 50% magnesium sulfate (4 mL; 8 mmol) IV over 10 min

Table 12.1 Calcium and magnesium disorders

* A normal total calcium is about $2.2\text{--}2.6 \text{ mmol L}^{-1}$. A normal ionised calcium is about $1.1\text{--}1.3 \text{ mmol L}^{-1}$. Interpret calcium values cautiously. Seek expert help if not sure. Total calcium depends on serum albumin values and will need to be corrected for low albumin values (corrected total calcium). Ionised calcium values are often measured by blood gas machines. Do not confuse ionised calcium, total calcium, and corrected calcium values.

Many patients who are potassium deficient are also deficient in magnesium. Magnesium is important for potassium uptake and for the maintenance of intracellular potassium values, particularly in the myocardium. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases of hypokalaemia.

Calcium and magnesium disorders

The recognition and management of calcium and magnesium disorders is summarised in Table 12.1.

Sepsis

Sepsis is a common cause of acute deterioration and accounts for 37,000 deaths annually in the United Kingdom. Consider sepsis in any deteriorating patient who has clinical suspicion of infection. Cardiorespiratory arrest associated with sepsis has a poor survival. Early recognition and treatment of sepsis is key to improving outcomes.

Definition of sepsis

Sepsis is the body's response to an infection: sources include the chest, abdomen, urinary tract, skin and soft tissues. The clinical course is variable: some patients will not deteriorate beyond a systemic inflammatory response syndrome (SIRS), whereas others will deteriorate rapidly to organ failure ('severe sepsis'). Septic shock (sepsis with a serum lactate $> 4 \text{ mmol L}^{-1}$ or hypotension unresponsive to fluid resuscitation) has a 50% mortality.

Treatment of sepsis

Supportive care based on the ABCDE approach while controlling the underlying source of infection is critical in the prevention of shock, multi-organ failure and cardiorespiratory arrest. Once immediately life-threatening problems have been addressed, initial resuscitation in patients with severe sepsis is covered by the **Sepsis Six** (www.survivesepsis.org), underpinned by the Surviving Sepsis Campaign guidelines (www.survivingsepsis.org).

Escalation of care

Early application of the Sepsis Six will frequently be sufficient to prevent organ dysfunction. Request senior assistance urgently at any time life is clearly immediately threatened, or if any of the following persist after appropriate fluid resuscitation:

- hypotension
- oliguria
- acute confusion
- lactate $> 4 \text{ mmol L}^{-1}$.

Cardiac arrest and sepsis

Treat cardiac arrest in a patient with sepsis or suspected sepsis according to standard ALS guidelines. Correct hypoxia and hypovolaemia, and look for other potentially reversible causes using the 4 Hs and 4Ts approach.

Sepsis Six

Step 1: Give high-flow oxygen

Hypotension, disordered capillary beds, and microthrombi compromise oxygen delivery to tissues. Give high-flow oxygen during initial resuscitation and consider intubation in patients showing signs of fatigue or cardiorespiratory compromise.

Step 2: Take blood cultures

Send at least one set of blood cultures and samples of any other fluid specific to the presumed source of infection (e.g. sputum) before starting antibiotic therapy. If a presumed source of infection is amenable to control (e.g. a line removed or an abscess drained) complete this as soon as possible and always within 12 h.

Step 3: Give broad-spectrum antibiotics

Give appropriate antibiotics as soon as possible, and always within the first hour, according to local prescribing guidelines. Delays in the administration of antibiotics are directly linked to poor outcome.

Step 4: Initiate fluid resuscitation

Circulatory shock, resulting from a combination of vasodilatation and hypovolaemia (due to leaky capillaries), is the most common form of organ dysfunction in sepsis. Give fluid challenges in divided boluses of 250–500 mL of crystalloid to a maximum volume of 30 mL kg^{-1} body weight in patients with hypotension, serum lactate $> 2 \text{ mmol L}^{-1}$, or other signs of circulatory dysfunction such as low urine output.

Step 5: Measure lactate

Lactic acid is a product of anaerobic metabolism. While elevated lactate is not specific to sepsis, it does indicate circulatory compromise and can be used to predict outcome. An initially high lactate which decreases with fluid challenges can be used to guide on-going resuscitation.

Step 6: Measure urine output

Accurate hourly measurement of urine output can therefore guide further fluid challenges and the need for critical care input.

Toxins (poisoning)

Poisoning is an infrequent cause of cardiac arrest, but remains a leading cause in victims younger than 40 years. It is also a common cause of non-traumatic coma in this age group. Self-poisoning with therapeutic or recreational drugs is the main reason for hospital admission. Drug toxicity can also be caused by inappropriate dosing and drug interactions. Accidental poisoning is commonest in

children. Homicidal poisoning is uncommon. Industrial accidents, warfare or terrorism may cause chemical, biological, radiological or nuclear (CBRN) exposure.

Prevention of cardiac arrest

Assess and treat the patient using the ABCDE approach. Airway obstruction and respiratory arrest secondary to a decreased conscious level is a common cause of death after self-poisoning (benzodiazepines, alcohol, opioids, tricyclics, barbiturates). Early tracheal intubation of unconscious patients by trained personnel can decrease the risk of aspiration. Drug-induced hypotension usually responds to IV fluids, but occasionally vasopressor support (e.g. noradrenaline infusion) is required. Measure electrolytes (particularly potassium), blood glucose and arterial blood gases. Retain samples of blood and urine for analysis. Patients with severe poisoning should be cared for in a critical care setting.

Modifications to resuscitation

- Ensure your personal safety and wear appropriate personal protective equipment.
- Avoid mouth-to-mouth breathing in the presence of chemicals such as cyanide, hydrogen sulphide, corrosives and organophosphates.
- Treat life-threatening tachyarrhythmias with cardioversion, according to the peri-arrest arrhythmia guidelines (Chapter 11). This includes correction of electrolyte, glucose and acid-base abnormalities.
- Once resuscitation has started, try to identify the poison(s). Relatives, friends and ambulance crews can provide useful information. Examination of the patient may reveal diagnostic clues such as odours, needle marks, pupil size, and signs of corrosion in the mouth.
- Measure the patient's temperature because hypo- or hyperthermia can occur after drug overdose.
- Provide standard basic and advanced life support if cardiac arrest occurs.
- Be prepared to continue resuscitation for a prolonged period, particularly in young patients, as the poison may be metabolised or excreted during extended resuscitation measures. Consider extracorporeal life support (ECLS).
- There is an increase in the use of new 'designer drugs' that can have actions similar to stimulants or depressants. In addition to supportive and symptomatic treatment expert advice will be required.
- Consult a regional or national poisons centre for information on treatment of the poisoned patient. In the UK, specialist advice about specific poisons can be obtained by accessing TOXBASE® (www.toxbase.org).

Specific treatments

There are few specific therapies for poisons that are useful immediately. The emphasis is on intensive supportive therapy using the ABCDE approach, with correction of

hypoxia, hypotension, acid/base, and electrolyte disorders.

Therapies include decontamination, limiting absorption of ingested poisons, enhancing elimination, or the use of specific antidotes. Seek advice from a poisons centre for up-to-date guidance for severe or uncommon poisonings.

- For skin exposures initial management consists of removing clothes.
- Routine use of gastric lavage for gastrointestinal decontamination is no longer recommended.
- Activated charcoal adsorbs certain drugs. Its efficacy decreases over time following ingestion. There is little evidence that treatment with activated charcoal improves clinical outcome. Consider giving a single dose of activated charcoal to patients who have ingested a potentially toxic amount of a poison known to be adsorbed by activated charcoal up to one hour previously. Give only to patients with an intact or protected airway. Multiple doses may be beneficial in life-threatening poisoning with carbamazepine, dapsone, phenobarbital, quinine and theophylline.
- Whole-bowel irrigation can reduce drug absorption by cleansing the gastro-intestinal tract using enteral administration of a polyethylene glycol solution. Consider in potentially toxic ingestion of sustained release or enteric-coated drugs, oral iron poisoning, and the removal of ingested packets of illicit drugs.
- Laxatives (cathartics) or emetics (e.g. ipecacuanha) have no role in the management of the acutely poisoned patient and are not recommended.
- Urine alkalinisation (urine pH > 7.5) by giving IV sodium bicarbonate can be useful in moderate to severe salicylate poisoning in patients who do not need haemodialysis.
- Haemodialysis removes drugs or metabolites with low molecular weight, low protein binding, small volumes of distribution and high water solubility.
- Consider the use of lipid emulsion (Intralipid) for cardiac arrest caused by local anaesthetic toxicity (see below).
- Specific antidotes include: acetylcysteine for paracetamol; high-dose atropine for organophosphate insecticides; sodium nitrite, sodium thiosulfate, hydroxocobalamin, and amyl nitrite for cyanides; digoxin-specific Fab antibodies for digoxin; flumazenil for benzodiazepines; naloxone for opioids.

Specific antidotes

This section addresses only some causes of cardiac arrest from poisoning.

Opioid poisoning

Opioid poisoning causes respiratory depression, pinpoint pupils and coma followed by respiratory arrest. The opioid antagonist naloxone rapidly reverses these effects.

The route for giving naloxone depends on the skills of the rescuer: intravenous (IV), intramuscular (IM), subcutaneous (SC), and intranasal (IN) routes can be used. The non-IV routes may be quicker because time is saved in not having to establish IV access, which can be extremely difficult in an IV drug user. The initial doses of naloxone are 400 mcg IV, 800 mcg IM, 800 mcg SC or 2 mg IN. Large opioid overdoses require titration to a total naloxone dose of 10 mg. The duration of action of naloxone is 45–70 min, but respiratory depression may persist for 4–5 h after opioid overdose. Thus, the clinical effects of naloxone may not last as long as those of a significant opioid overdose. Give increments of naloxone until the victim is breathing adequately and has protective airway reflexes.

Acute withdrawal from opioids produces a state of sympathetic excess and can cause complications such as pulmonary oedema, ventricular arrhythmia, and severe agitation. Use naloxone reversal of opioid intoxication with caution in patients suspected of opioid dependence.

Cardiac arrest is usually secondary to a respiratory arrest and associated with severe brain hypoxia. Prognosis is poor. Once cardiac arrest has occurred, follow standard resuscitation guidelines.

Benzodiazepines

Overdose of benzodiazepines can cause loss of consciousness, respiratory depression and hypotension. Flumazenil, a competitive antagonist of benzodiazepines can be used to reverse sedation caused by benzodiazepines when there is no history or risk of

seizures. Reversal of benzodiazepine intoxication with flumazenil can cause significant toxicity (seizure, arrhythmia, hypotension, and withdrawal syndrome) in patients with benzodiazepine dependence or co-ingestion of proconvulsant medications such as tricyclic antidepressants. Do not use flumazenil routinely in the comatose patient. There are no specific modifications required for cardiac arrest caused by benzodiazepines.

Tricyclic antidepressants

This includes tricyclic and related cyclic drugs (e.g. amitriptyline, desipramine, imipramine, nortriptyline, doxepin, and clomipramine). Self-poisoning with tricyclic antidepressants is common and can cause hypotension, seizures, coma and life-threatening arrhythmias. Cardiac toxicity mediated by anticholinergic and sodium channel-blocking effects can produce a broad-complex tachycardia (VT). Consider tricyclic overdose as a possible diagnosis in any child presenting with a shockable rhythm. Hypotension is exacerbated by alpha-1 receptor blockade. Anticholinergic effects include mydriasis, fever, dry skin, delirium, tachycardia, ileus, and urinary retention. Most life-threatening problems occur within the first 6 h after ingestion.

A widening QRS complex and right axis deviation indicates a greater risk of arrhythmias (Figure 12.2). Consider sodium bicarbonate for the treatment of tricyclic-induced ventricular conduction abnormalities. While no study has investigated the optimal target arterial pH with bicarbonate therapy, a pH of 7.45–7.55 is commonly accepted.

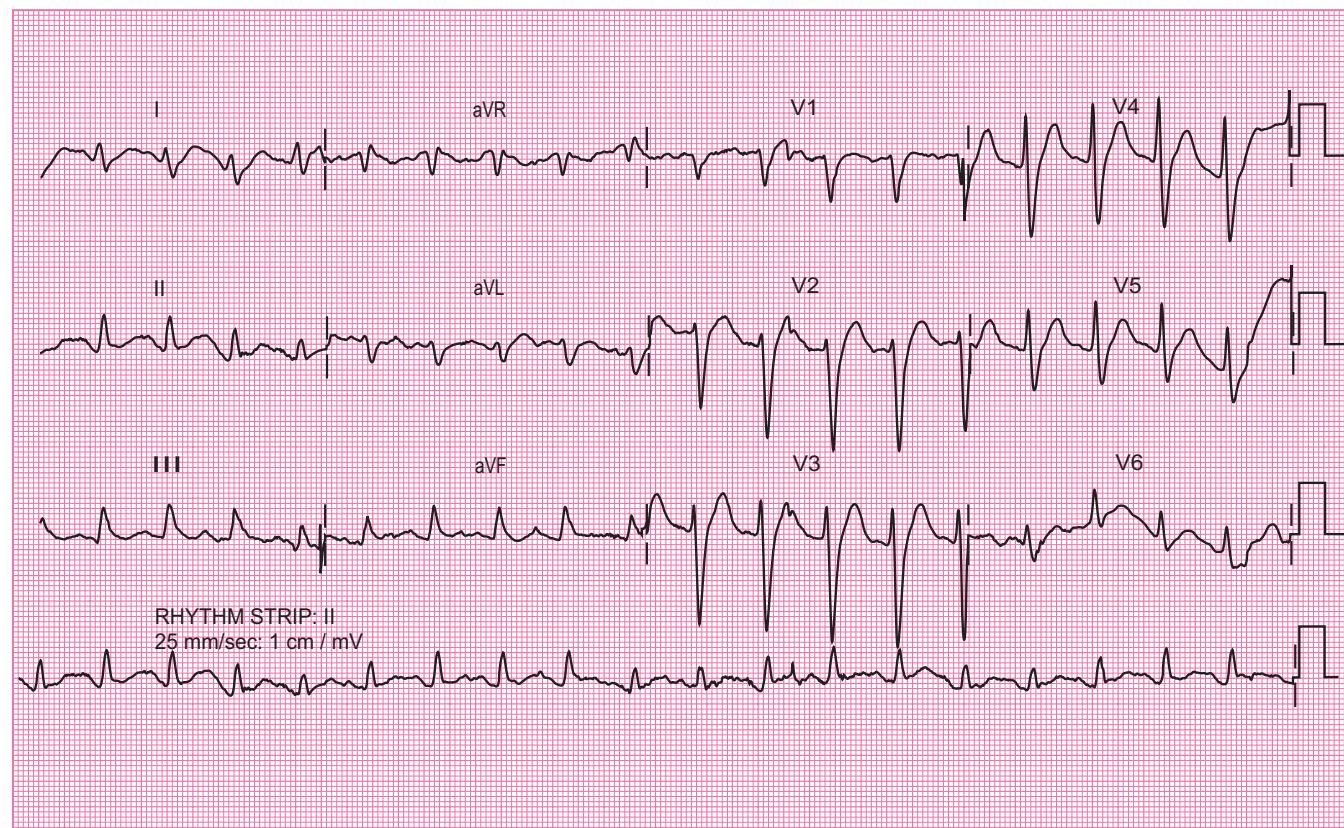


Figure 12.2 12-lead ECG showing features of severe tricyclic antidepressant toxicity

Local anaesthetic toxicity

Local anaesthetic toxicity occurs typically in the setting of regional anaesthesia, when a bolus of local anaesthetic inadvertently enters an artery or vein. Systemic toxicity of local anaesthetics involves the central nervous system, and the cardiovascular system. Severe agitation, loss of consciousness, with or without tonic-clonic convulsions, sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmia can all occur. Toxicity can be potentiated in pregnancy, extremes of age, or hypoxaemia.

Follow standard resuscitation measures. In addition, patients with both cardiovascular collapse and cardiac arrest attributable to local anaesthetic toxicity may benefit from treatment with intravenous 20% lipid emulsion in addition to standard advanced life support. Give an initial intravenous bolus of 1.5 mL kg^{-1} 20% lipid emulsion followed by an infusion at $15 \text{ mL kg}^{-1} \text{ h}^{-1}$. Give up to three bolus doses of lipid at 5-min intervals and continue the infusion until the patient is stable or has received up to a maximum of 12 mL kg^{-1} of lipid emulsion (Figure 12.3).

Cocaine toxicity

Sympathetic overstimulation associated with cocaine toxicity may cause agitation, symptomatic tachycardia, hypertensive crisis, hyperthermia and myocardial ischaemia with angina. Small doses of intravenous benzodiazepines (midazolam, diazepam, lorazepam) are effective first-line drugs. Glyceryl trinitrate and phentolamine can reverse cocaine-induced coronary vasoconstriction. Use nitrates only as second-line therapy for myocardial ischaemia. The evidence for or against the use of beta-blocker drugs, including those beta-blockers with alpha blocking properties (carvedilol and labetolol) is limited. The optimal choice of anti-arrhythmic drug for the treatment of cocaine-induced tachyarrhythmias is not known. Seek expert advice if chest pain does not settle, recurs or there is evidence suggesting an acute coronary syndrome. If cardiac arrest occurs, follow standard resuscitation guidelines. There is no evidence to guide the optimal dose of adrenaline during cardiac arrest associated with cocaine toxicity and standard adrenaline doses are recommended.

Drug-induced severe bradycardia

Severe bradycardia from poisoning or drug overdose may be refractory to standard ALS protocols because of prolonged receptor binding or direct cellular toxicity. Atropine can be life-saving in organophosphate, carbamate or nerve agent poisoning. Give atropine for bradycardia caused by acetylcholinesterase-inhibiting substances. Large (2–4 mg IV) and repeated doses may be required to achieve a clinical effect. Isoprenaline may be useful at high doses in refractory bradycardia induced by beta-receptor blockade. Heart block and ventricular arrhythmias associated with digoxin or digitalis glycoside poisoning may be treated effectively with digoxin-specific antibody fragments.

Vasopressors, inotropes, calcium, glucagon, phosphodiesterase inhibitors and high-dose insulin-glucose-

potassium infusions may all be useful in beta-blocker and calcium channel blocker overdose. Transcutaneous pacing may be effective for severe bradycardia caused by poisoning and overdose (Chapters 10 and 11).

Asthma

Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma with a high prevalence in some European countries (United Kingdom, Ireland, Netherlands and Scandinavia). Annual worldwide deaths from asthma have been estimated at 250,000. A national confidential enquiry in the UK in 2014 showed that most asthma-related deaths occurred before admission to hospital. Good asthma control and prevention of acute asthma is therefore important. The British Thoracic Society (BTS) and Scottish Intercollegiate Guidelines Network (SIGN) have published guidelines for the management of asthma available at www.brit-thoracic.org.uk

This guidance focuses on the treatment of patients with near-fatal asthma and cardiac arrest.

Patients at risk of asthma-related cardiac arrest

The risk of near-fatal asthma attacks is not necessarily related to asthma severity. Patients most at risk include those with:

- a history of near-fatal asthma requiring intubation and mechanical ventilation
- hospitalisation or emergency care for asthma in the past year
- low or no use of inhaled corticosteroids
- increasing use and dependence of beta-2 agonists
- anxiety, depressive disorders and/or poor compliance with therapy
- food allergy in a patient with asthma.

Causes of cardiorespiratory arrest associated with asthma

Cardiac arrest in a patient with asthma is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden. Cardiac arrest in patients with asthma has been linked to:

- Severe bronchospasm and mucous plugging leading to asphyxia (this condition causes the vast majority of asthma-related deaths).
- Cardiac arrhythmias caused by hypoxia, which is the commonest cause of asthma-related arrhythmia. Arrhythmias can also be caused by stimulant drugs (e.g. beta-adrenergic agonists, aminophylline) or electrolyte abnormalities.
- Dynamic hyperinflation (auto positive end-expiratory pressure (auto-PEEP)) can occur in mechanically ventilated patients with asthma. Auto-PEEP is caused by

AAGBI Safety Guideline

Management of Severe Local Anaesthetic Toxicity



1 Recognition

Signs of severe toxicity:

- Sudden alteration in mental status, severe agitation or loss of consciousness, with or without tonic-clonic convulsions
- Cardiovascular collapse: sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias may all occur
- Local anaesthetic (LA) toxicity may occur some time after an initial injection

2 Immediate management

- Stop injecting the LA
- Call for help
- Maintain the airway and, if necessary, secure it with a tracheal tube
- Give 100% oxygen and ensure adequate lung ventilation (hyperventilation may help by increasing plasma pH in the presence of metabolic acidosis)
- Confirm or establish intravenous access
- Control seizures: give a benzodiazepine, thiopental or propofol in small incremental doses
- Assess cardiovascular status throughout
- Consider drawing blood for analysis, but do not delay definitive treatment to do this

3 Treatment

IN CIRCULATORY ARREST

- Start cardiopulmonary resuscitation (CPR) using standard protocols
- Manage arrhythmias using the same protocols, recognising that arrhythmias may be very refractory to treatment
- Consider the use of cardiopulmonary bypass if available

GIVE INTRAVENOUS LIPID EMULSION

(following the regimen overleaf)

- Continue CPR throughout treatment with lipid emulsion
- Recovery from LA-induced cardiac arrest may take >1 h
- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

WITHOUT CIRCULATORY ARREST

Use conventional therapies to treat:

- hypotension,
- bradycardia,
- tachyarrhythmia

CONSIDER INTRAVENOUS LIPID EMULSION

(following the regimen overleaf)

- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

4 Follow-up

- Arrange safe transfer to a clinical area with appropriate equipment and suitable staff until sustained recovery is achieved
- Exclude pancreatitis by regular clinical review, including daily amylase or lipase assays for two days
- Report cases as follows:
 - in the United Kingdom to the National Patient Safety Agency (via www.npsa.nhs.uk)
 - in the Republic of Ireland to the Irish Medicines Board (via www.imb.ie)

If Lipid has been given, please also report its use to the international registry at www.lipidregistry.org. Details may also be posted at www.lipidrescue.org

Your nearest bag of Lipid Emulsion is kept.....

This guideline is not a standard of medical care. The ultimate judgement with regard to a particular clinical procedure or treatment plan must be made by the clinician in the light of the clinical data presented and the diagnostic and treatment options available.

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IMMEDIATELY

Give an initial intravenous bolus injection of 20% lipid emulsion $1.5 \text{ ml}.\text{kg}^{-1}$ over 1 min

AND

Start an intravenous infusion of 20% lipid emulsion at $15 \text{ ml}.\text{kg}^{-1}.\text{h}^{-1}$

AFTER 5 MIN

Give a **maximum of two** repeat boluses (same dose) if:

- cardiovascular stability has not been restored **or**
- an adequate circulation deteriorates

Leave 5 min between boluses

A maximum of **three** boluses can be given (including the initial bolus)

AND

Continue infusion at same rate, but:

- Double the rate to $30 \text{ ml}.\text{kg}^{-1}.\text{h}^{-1}$ at any time after 5 min, if:
- cardiovascular stability has not been restored
 - an adequate circulation deteriorates
- Continue infusion until stable and adequate circulation restored or maximum dose of lipid emulsion given

Do not exceed a maximum cumulative dose of $12 \text{ ml}.\text{kg}^{-1}$

An approximate dose regimen for a 70-kg patient would be as follows:

IMMEDIATELY

Give an initial intravenous bolus injection of 20% lipid emulsion 100 ml over 1 min

AND

Start an intravenous infusion of 20% lipid emulsion at $1000 \text{ ml}.\text{h}^{-1}$

AFTER 5 MIN

Give a **maximum of two** repeat boluses of 100 ml

AND

Continue infusion at same rate but **double** rate to $2000 \text{ ml}.\text{h}^{-1}$ if indicated at any time

Do not exceed a maximum cumulative dose of 840 ml



This AAGBI Safety Guideline was produced by a Working Party that comprised:
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This Safety Guideline is endorsed by the Australian and New Zealand College of Anaesthetists (ANZCA).

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Figure 12.3 Management of severe local anaesthetic toxicity

air trapping and ‘breath stacking’ (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure.

- Tension pneumothorax (occasionally bilateral).

The 4 Hs and 4 Ts approach to reversible causes will help identify these causes in cardiac arrest.

Initial assessment and treatment of asthma to prevent cardiorespiratory arrest

Use the ABCDE approach to assess severity and guide treatment. The severity of acute asthma is summarised in Table 12.2.

Asthma severity	
Near-fatal asthma	Raised PaCO ₂ and/or mechanical ventilation with raised inflation pressures
Life-threatening asthma	Any one of the following in a patient with severe asthma:
	Clinical signs
	Measurements
	Altered conscious level PEF < 33% best or predicted
	Exhaustion SpO ₂ < 92%
	Arrhythmia PaO ₂ < 8 kPa
	Hypotension ‘normal’ PaCO ₂ (4.6–6.0 kPa)
	Cyanosis
	Silent chest
	Poor expiratory effort
Acute severe asthma	Any one of: <ul style="list-style-type: none"> - PEF 33–50% best or predicted - respiratory rate ≥ 25 min⁻¹ - heart rate ≥ 110 min⁻¹ - inability to complete sentences in one breath

Table 12.2 Severity of asthma exacerbations

From British Thoracic Society/Scottish Intercollegiate Guidelines Network Guideline on the Management of Asthma - www.brit-thoracic.org.uk

- Wheezing is a common physical finding, but severity does not correlate with the degree of airway obstruction. The absence of wheezing may indicate critical airway obstruction, whereas increased wheezing may indicate a positive response to bronchodilator therapy.
- Oxygen saturation of arterial blood (SpO₂) may not reflect progressive alveolar hypoventilation, particularly

if oxygen is being given. SpO₂ may initially decrease during therapy because beta-agonists cause both bronchodilation and vasodilation, initially increasing intrapulmonary shunting.

- Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis, foreign bodies, pulmonary embolism, and subglottic mass.
- The patient with acute severe asthma requires aggressive medical management to prevent deterioration. Experienced clinicians should treat these patients in a critical care area.
- Use a concentration of inspired oxygen that will achieve an SpO₂ of 94–98%. High-flow oxygen by mask is sometimes necessary.
- Salbutamol (5 mg delivered by oxygen driven nebuliser) is the main therapy for acute asthma. Repeated doses every 15–30 min, or continuous doses (5–10 mg h⁻¹ – requires a special nebuliser), may be needed. Remember that nebulised drugs will not be delivered to the lungs effectively if the patient is tired and hypoventilating. If a nebuliser is not immediately available beta-2 agonists can be temporarily administered by repeating activations of a metered dose inhaler via a large volume spacer device.
- Add nebulised ipratropium bromide (500 mcg 4–6 hourly) to beta-2 agonist treatment for patients with acute severe or life-threatening asthma or those with a poor initial response to beta-2 agonist therapy.
- Give steroids (prednisolone 40–50 mg orally or hydrocortisone 100 mg IV 6-hourly) early. Oral formulations have a longer half-life but the IV route is easier to give in near fatal asthma.
- Give a single dose of IV magnesium sulfate (2 g (8 mmol) IV over 20 min) to patients with acute severe asthma (PEF < 50% best or predicted) who have not had a good initial response to inhaled bronchodilator therapy. The most commonly reported adverse effects of IV magnesium sulfate are flushing, fatigue, nausea, headache and hypotension.
- Consider intravenous salbutamol (250 mcg IV slowly) only when inhaled therapy is not possible (e.g. a patient receiving bag-mask ventilation).
- Following senior advice, consider aminophylline in severe or near-fatal asthma only. A loading dose of 5 mg kg⁻¹ IV is given over 20 min (unless on maintenance therapy), followed by an infusion of 500–700 mcg kg⁻¹ h⁻¹. Maintain serum theophylline concentrations below 20 mcg mL⁻¹ to avoid toxicity.
- These patients are often dehydrated or hypovolaemic and will benefit from fluid replacement. Beta-2 agonists and steroids may induce hypokalaemia, which should be corrected with electrolyte supplements.

- An intensive care specialist should assess patients who fail to respond to initial treatment, or develop signs of life-threatening asthma.
- Consider tracheal intubation and controlled ventilation if, despite efforts to optimise drug therapy, the patient has:
 - deteriorating peak flow
 - a decreasing conscious level, or coma
 - persisting or worsening hypoxaemia
 - deteriorating respiratory acidosis
 - severe agitation, confusion and fighting against the oxygen mask (clinical signs of hypoxaemia)
 - progressive exhaustion
 - respiratory or cardiac arrest.
- Elevation of the PaCO₂ alone does not indicate the need for tracheal intubation.
- The role of non-invasive ventilation (NIV) in patients with severe acute asthma is uncertain. NIV should be considered only in an ICU or equivalent clinical setting.

Cardiorespiratory arrest associated with asthma

- Follow standard BLS and ALS protocols. Ventilation will be difficult because of increased airway resistance; try to avoid gastric inflation.
- Intubate the trachea early. There is a significant risk of gastric inflation and hypoventilation of the lungs when attempting to ventilate a severe asthmatic without a tracheal tube.
- The recommended respiratory rate (10 breaths min⁻¹) and tidal volume required for a normal chest rise during CPR should not cause dynamic hyperinflation of the lungs (gas trapping).
- If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest wall and/or a period of apnoea (disconnection of tracheal tube) may relieve gas-trapping. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.
- Dynamic hyperinflation increases transthoracic impedance, but modern impedance-compensated biphasic defibrillation waveforms are no less effective in patients with a higher impedance. As with standard ALS defibrillation protocols, consider increasing defibrillation energy if the first shock is unsuccessful and a manual defibrillator is available.
- Look for reversible causes using the 4 Hs and 4 Ts approach.
- Tension pneumothorax can be difficult to diagnose in cardiac arrest; it may be indicated by unilateral expansion of the chest wall, shifting of the trachea, and

subcutaneous emphysema. Pleural ultrasound in skilled hands is faster and more sensitive than chest X-ray for the detection of pneumothorax. Early needle decompression (thoracocentesis) followed by chest drain insertion is needed. Needle decompression may fail due to inadequate needle length. In the ventilated patient, thoracostomy (a surgical hole in the chest wall and pleura) may be quicker to perform and more effective for decompressing the chest (see trauma section).

- Always consider bilateral pneumothoraces in asthma-related cardiac arrest.
- Extracorporeal life support (ECLS) can provide both organ perfusion and gas exchange in cases of otherwise refractory respiratory and circulatory failure. Cases of successful treatment of asthma-related cardiac arrest in adults using ECLS have been reported

Anaphylaxis

This guidance is based on Emergency Treatment of Anaphylactic Reactions, Resuscitation Council (UK). For more details see www.resus.org.uk.

Definition

Anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction. This is characterised by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.

Epidemiology

Anaphylaxis is common and affects about 1 in 300 of the European population at some stage in their lives. Anaphylaxis can be triggered by any of a very broad range of triggers with food, drugs, stinging insects, and latex the most commonly identified triggers. Food is the commonest trigger in children and drugs the commonest in adults. Virtually any food or drug can be implicated, but certain foods (nuts) and drugs (muscle relaxants, antibiotics, nonsteroidal anti-inflammatory drugs and aspirin) cause most reactions. A significant number of cases of anaphylaxis are idiopathic.

The overall prognosis of anaphylaxis is good, with a case fatality ratio of less than 1% reported in most population-based studies. The European Anaphylaxis Registry reported that only 2% of 3333 cases progressed to cardiac arrest.

Anaphylaxis and risk of death is increased in those with pre-existing asthma, particularly if the asthma is poorly controlled, severe, or in asthmatics who delay treatment. When anaphylaxis is fatal, death usually occurs very soon after contact with the trigger. From a case series, fatal food reactions cause respiratory arrest typically within 30–35 min; insect stings cause collapse from shock within 10–15 min; and deaths caused by intravenous medication occur most commonly within 5 min. Death was not reported more than six hours after contact with the trigger.

Recognition of anaphylaxis

- Anaphylaxis is likely if a patient who is exposed to a trigger (allergen) develops a sudden illness (usually within minutes of exposure) with rapidly progressing skin changes and life-threatening airway and/or breathing and/or circulation problems. The reaction is usually unexpected.
- The lack of any consistent clinical manifestation and a range of possible presentations cause diagnostic difficulty. Patients have been given injections of adrenaline inappropriately for allergic reactions just involving the skin, or for vasovagal reactions or panic attacks. Guidelines for the treatment of an anaphylactic reaction must therefore take into account some inevitable diagnostic errors, with an emphasis on the need for safety.

Anaphylaxis is likely when all of the following three criteria are met:

1. Sudden onset and rapid progression of symptoms.
2. Life-threatening Airway and/or Breathing and/or Circulation problems.
3. Skin and/or mucosal changes (flushing, urticaria, angioedema).

Exposure to a known allergen for the patient supports the diagnosis.

Remember:

- Skin or mucosal changes alone are not a sign of anaphylaxis.
- Skin and mucosal changes can be subtle or absent in up to 20% of reactions (some patients can have only a decrease in blood pressure (i.e. a Circulation problem)).
- There can also be gastrointestinal symptoms (e.g. vomiting, abdominal pain, incontinence).

Sudden onset and rapid progression of symptoms

- The patient will feel and look unwell.
- Most reactions occur over several minutes. Rarely, reactions may be slower in onset.
- An intravenous trigger will cause a more rapid onset of reaction than stings which, in turn, tend to cause a more rapid onset than orally ingested triggers.
- The patient is usually anxious and can experience a ‘sense of impending doom’.

Life-threatening Airway, Breathing and Circulation problems

Use the ABCDE approach to recognise life-threatening Airway, Breathing and Circulation problems.

Airway problems

- Airway swelling, (e.g. throat and tongue swelling (pharyngeal/laryngeal oedema)). The patient has difficulty in breathing and swallowing and feels that the throat is closing up.
- Hoarse voice.
- Stridor - this is a high-pitched inspiratory noise caused by upper airway obstruction.

Breathing problems

- Shortness of breath - increased respiratory rate
- Wheeze
- Patient becoming tired
- Confusion caused by hypoxia
- Cyanosis - this is usually a late sign
- Respiratory arrest

Circulation problems

- Signs of shock - pale, clammy
- Tachycardia
- Hypotension - feeling faint, collapse
- Decreased conscious level or loss of consciousness
- Anaphylaxis can cause myocardial ischaemia and ECG changes even in individuals with normal coronary arteries
- Cardiac arrest

Circulation problems (often referred to as anaphylactic shock) can be caused by direct myocardial depression, vasodilation and capillary leak, and loss of fluid from the circulation.

The above Airway, Breathing and Circulation problems can all alter the patient’s neurological status (**Disability problems**) because of decreased brain perfusion. There may be confusion, agitation and loss of consciousness.

Skin and mucosal changes

These should be assessed as part of the **Exposure** when using the ABCDE approach.

- They are often the first feature and present in over 80% of anaphylactic reactions.
- They can be subtle or dramatic.
- There may be changes just to the skin, just to the mucosal, or both.

- There may be erythema - a patchy, or generalised, red rash.
- There may be urticaria (also called hives, nettle rash, weals or welts), which can appear anywhere on the body. The weals may be pale, pink or red, and may look like nettle stings. They can be different shapes and sizes, and are often surrounded by a red flare. They are usually itchy.
- Angioedema is similar to urticaria but involves swelling of deeper tissues, most commonly in the eyelids and lips, and sometimes in the mouth and throat.
- Although skin changes can be worrying or distressing for patients and those treating them, skin changes without airway, breathing or circulation problems do not signify anaphylaxis.

Differential diagnosis for anaphylaxis

Life-threatening conditions

- Sometimes an anaphylactic reaction can present with symptoms and signs that are very similar to life-threatening asthma.
- A low blood pressure (or normal in children) with a petechial or purpuric rash can be a sign of septic shock. Seek help early if there are any doubts about the diagnosis and treatment.
- Following the ABCDE approach will help with treating the differential diagnoses.

Non life-threatening conditions (these usually respond to simple measures)

- Faint (vasovagal episode)
- Panic attack
- Breath-holding episode in child
- Idiopathic (non-allergic) urticaria or angioedema

There can be confusion between an anaphylactic reaction and a panic attack. Victims of previous anaphylaxis may be particularly prone to panic attacks if they think they have been re-exposed to the allergen that caused a previous problem. The sense of impending doom and breathlessness leading to hyperventilation are symptoms that can resemble anaphylaxis. While there is no hypotension, pallor, wheeze, or urticarial rash or swelling, there may sometimes be flushing or blotchy skin associated with anxiety, adding to the diagnostic difficulty. Diagnostic difficulty may also occur with vasovagal attacks after immunisation procedures, but the absence of rash, breathing difficulties, and swelling are useful distinguishing features, as is the slow pulse of a vasovagal attack compared with the rapid pulse of a severe anaphylactic episode. Fainting will usually respond to lying the patient down and raising the legs.

Treatment of anaphylaxis to prevent cardiorespiratory arrest

As the diagnosis of anaphylaxis is not always obvious, all those who treat anaphylaxis must use the ABCDE approach to the sick patient. Treat life-threatening problems as you find them. The key steps are described in the anaphylaxis algorithm (Figure 12.4).

- All patients should be placed in a comfortable position. Patients with airway and breathing problems may prefer to sit up as this will make breathing easier. Lying flat with or without leg elevation is helpful for patients with a low blood pressure (Circulation problem). If the patient feels faint, do not sit or stand them up - this can cause cardiac arrest. Patients who are breathing and unconscious should be placed on their side (recovery position).
- Removing the trigger for an anaphylactic reaction is not always possible. Stop any drug suspected of causing an anaphylactic reaction (e.g. stop IV infusion of a gelatin solution or antibiotic). Do not delay definitive treatment if removing the trigger is not feasible.
- Monitor all patients who have suspected anaphylaxis as soon as possible (e.g. by ambulance crew, in the emergency department etc.). Minimum monitoring includes pulse oximetry, non-invasive blood pressure and a 3-lead ECG.
- Initially, give the highest concentration of oxygen possible using a mask with an oxygen reservoir. Once pulse oximetry is feasible target an SpO₂ of 94–98%.

Adrenaline is the most important drug for the treatment of an anaphylactic reaction. As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. Its beta-receptor activity dilates the bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release. Adrenaline works best when given early after the onset of the reaction. Adverse effects are extremely rare with correct doses injected intramuscularly (IM). Sometimes there has been uncertainty about whether complications (e.g. myocardial ischaemia) have been caused by the allergen itself or by the adrenaline given to treat it.

- The intramuscular (IM) route is the best for most individuals who have to give adrenaline to treat an anaphylactic reaction and should be administered as early as possible.
- **For adults give an initial IM adrenaline dose of 0.5 mg (0.5 mL of 1:1000 adrenaline = 0.5 mg = 500 mcg). Further doses can be given at about 5-min intervals, according to the patient's response.**
- The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The needle used for injection needs to be sufficiently long to ensure that the adrenaline is injected into muscle.

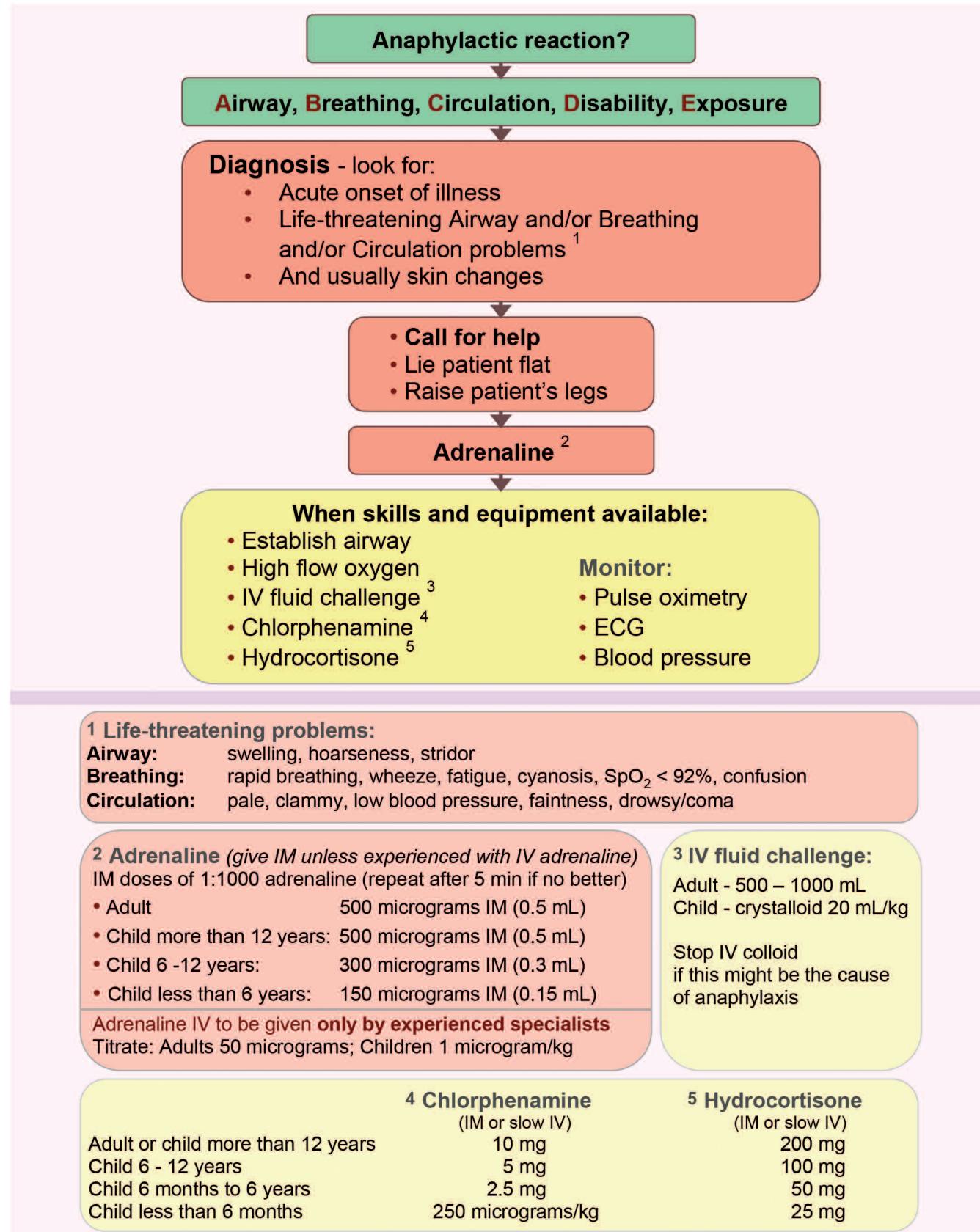


Figure 12.4 Anaphylaxis algorithm

- The use of IV adrenaline applies only to those experienced in the use and titration of vasopressors in their normal clinical practice (e.g. anaesthetists, emergency physicians, intensive care doctors). In patients with a spontaneous circulation, intravenous adrenaline can cause life-threatening hypertension, tachycardia, arrhythmias, and myocardial ischaemia. Patients who are given IV adrenaline must be monitored closely - continuous ECG and pulse oximetry and frequent non-invasive blood pressure measurements as a minimum. Patients who require repeated IM doses of adrenaline may benefit from IV adrenaline. It is essential that these patients receive expert help early.
- Titrate IV adrenaline using 50 mcg boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion. The pre-filled 10 mL syringe of 1:10,000 adrenaline contains 100 mcg mL⁻¹. A dose of 50 mcg is 0.5 mL, which is the smallest dose that can be given accurately. Do not give the undiluted 1:1000 adrenaline concentration IV.
- Auto-injectors are often given to patients at risk of anaphylaxis for their own use. Healthcare professionals should be familiar with the use of the most commonly available auto-injector devices. If an adrenaline auto-injector is the only available adrenaline preparation when treating anaphylaxis, healthcare providers should use it.
- Give a rapid IV fluid challenge (500–1000 mL in an adult) and monitor the response; give further doses as necessary. Consider colloid infusion as a cause in a patient receiving a colloid at the time of onset of an anaphylactic reaction and stop the infusion. Hartmann's solution or 0.9% saline are suitable fluids for initial resuscitation. A large volume of fluid may be needed.
- Antihistamines are a second line treatment for an anaphylactic reaction. Antihistamines (H1-antihistamine) may help counter histamine-mediated vasodilation and bronchoconstriction. They may not help in reactions depending in part on other mediators, but they have the virtue of safety. Used alone, they are unlikely to be life-saving in a true anaphylactic reaction. Give chlorphenamine 10 mg IM or IV slowly.
- Steroids may help prevent or shorten protracted reactions. Inject hydrocortisone 200 mg IM or IV slowly.
- If the patient has asthma-like features alone, treat as for asthma. As well as the drugs listed above, consider further bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), aminophylline (IV) or magnesium (IV). Remember that intravenous magnesium is a vasodilator and can cause hot flushes and make hypotension worse.
- Adrenaline remains the first line vasopressor for the treatment of anaphylactic reactions. Consider other vasopressors and inotropes (noradrenaline, vasopressin, metaraminol and glucagon) when initial resuscitation with adrenaline and fluids has not been successful. Only use these drugs in specialist settings (e.g. intensive care units) where there is experience in their use. Glucagon can be useful to treat an anaphylactic reaction in a patient taking a beta-blocker.
- Airway obstruction may occur rapidly in severe anaphylaxis, particularly in patients with angioedema. Warning signs are swelling of the tongue and lips, hoarseness and oropharyngeal swelling.
- Consider early tracheal intubation; delay may make intubation extremely difficult. As airway obstruction progresses, supraglottic airway devices (e.g. LMA) are likely to be difficult to insert. Attempts at tracheal intubation may exacerbate laryngeal oedema. Early involvement of a senior anaesthetist is mandatory when managing these patients. A surgical airway may be required if tracheal intubation is not possible.

Cardiac arrest associated with anaphylaxis

Start CPR immediately and follow current guidelines. Prolonged CPR may be necessary. Rescuers should ensure that help is on its way as early ALS is essential. Cardiac arrest with suspected anaphylaxis should be treated with the standard 1 mg dose of IV or intraosseous (IO) adrenaline for cardiac arrest. If this is not feasible, consider 0.5 mg IM adrenaline if cardiac arrest is imminent or has just occurred. The benefit of IM adrenaline in these circumstances is uncertain.

Investigation of anaphylaxis

Undertake the usual investigations appropriate for a medical emergency (e.g. 12-lead ECG, chest X-ray, urea and electrolytes, arterial blood gases etc).

The specific test to help confirm a diagnosis of an anaphylactic reaction is measurement of mast cell tryptase. In anaphylaxis, mast cell degranulation leads to markedly increased blood tryptase concentrations.

Mast cell tryptase sample timing

The time of onset of the anaphylactic reaction is the time when symptoms were first noticed.

- Minimum: one sample at 1–2 h after the start of symptoms.
- Ideally: Three timed samples:
 - Initial sample as soon as feasible after resuscitation has started - do not delay resuscitation to take sample.
 - Second sample at 1–2 h after the start of symptoms
 - Third sample either at 24 h or in convalescence. This provides baseline tryptase levels - some individuals have an elevated baseline level.
- Use a serum or clotted blood ('liver function test' bottle) sample.
- Record the timing of each sample accurately on the sample bottle and request form.
- Consult your local laboratory if you have any queries.

Discharge and follow-up after anaphylaxis

Patients who have had suspected anaphylaxis (i.e. an airway, breathing or circulation problem) should be treated and then observed in a clinical area with facilities for treating life-threatening ABC problems. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation. The exact incidence of biphasic reactions is unknown. Although studies quote an incidence of 1–20%, it is not clear whether all the patients in these studies actually had anaphylaxis or whether the initial treatment was appropriate. There is no reliable way of predicting who will have a biphasic reaction. It is therefore important that decisions about discharge are made for each patient by an experienced clinician.

Before discharge from hospital, all patients must:

- Be reviewed and have a treatment plan based on their individual risk.
- Be given clear instructions to return to hospital if symptoms return.
- Be considered for an adrenaline auto-injector, or given replacements and ensured that appropriate training has been given.
- Have a plan for follow-up, including contact with the patient's general practitioner.

All patients presenting with anaphylaxis should be referred to an allergy clinic to identify the cause, and thereby reduce the risk of future reactions and prepare the patient to manage future episodes themselves. Patients need to know the allergen responsible (if identified) and how to avoid it. Patients need to be able to recognise the early symptoms of anaphylaxis, so that they can summon help quickly and prepare to use their emergency medication.

Pregnancy

Mortality related to pregnancy in developed countries is rare, occurring in an estimated 16 per 100,000 live births. Both the mother and fetus must be considered in emergencies during pregnancy. Effective resuscitation of the mother is often the best way to optimise fetal outcome. Significant physiological changes occur during pregnancy; for example, cardiac output, circulatory volume, minute ventilation, and oxygen consumption all increase. The gravid uterus can cause compression of the abdominal vessels when the mother is in the supine position, resulting in reduced cardiac output, hypotension and reduced uterine perfusion. Resuscitation guidelines for pregnancy are based largely on case series, extrapolation from non-pregnant cardiac arrests, manikin studies and expert opinion based on the physiology of pregnancy and changes that occur in labouring women.

Causes of cardiac arrest in pregnancy

Cardiac arrest in pregnancy is most commonly caused by:

- cardiac disease (congenital and acquired)
- pulmonary embolism
- psychiatric disorders (suicide)
- hypertensive disorders of pregnancy
- sepsis
- haemorrhage
- amniotic fluid embolism
- ectopic pregnancy.

Pregnant women can also have the same causes of cardiac arrest as females of the same age group (e.g. anaphylaxis, drug overdose, trauma).

Prevention of cardiac arrest in pregnancy

In an emergency, use the ABCDE approach. Many cardiovascular problems associated with pregnancy are caused by compression of the inferior vena cava. Treat a distressed or compromised pregnant patient as follows:

- Place the patient in the left lateral position or manually displace the uterus to the left.
- Give high-flow oxygen, guided by pulse oximetry.
- Give a fluid bolus if there is hypotension or evidence of hypovolaemia.
- Immediately re-evaluate the need for any drugs currently being given.
- Seek expert help and involve obstetric and neonatal specialists early in the resuscitation.
- Identify and treat the underlying cause.

Modifications for cardiac arrest in pregnancy

- In cardiac arrest, all the principles of basic and advanced life support apply.
- Summon help immediately. Obtain expert help, including an obstetrician, anaesthetist, and neonatologist, to facilitate effective resuscitation of both mother and fetus.
- Start CPR according to standard ALS guidelines. Ensure high quality chest compressions with minimal interruptions. The hand position may have to be slightly higher on the sternum in advanced pregnancy.

- After approximately 20 weeks gestation, the pregnant woman's uterus can press down against the inferior vena cava (IVC) and the aorta, impeding venous return, cardiac output and uterine perfusion. IVC compression limits the effectiveness of chest compressions.
- The potential for IVC compression suggests that IV or IO access should ideally be established above the diaphragm.
- Manually displace the uterus to the left to minimise IVC compression.
- Add left lateral tilt only if this is feasible. The patient's body will need to be supported on a firm surface to allow effective chest compressions (e.g. a full length tilting operating table). The optimal angle of tilt is unknown. Aim for between 15–30°. Even a small amount of tilt may be better than no tilt. The angle of tilt used needs to permit high quality chest compressions and if needed caesarean delivery of the fetus (see below).
- If tilting on a firm surface is not possible then maintain left uterine displacement and continue effective chest compressions with the patient supine.
- The use of soft pillows and wedges for the pregnant patient requiring chest compressions is ineffective.
- Start preparing for emergency caesarean section (see below) - the fetus will need to be delivered if initial resuscitation efforts fail.
- There is an increased risk of pulmonary aspiration of gastric contents in pregnancy. Early tracheal intubation decreases this risk. Tracheal intubation can be more difficult in the pregnant patient however. Expert help, a failed intubation drill, and the use of alternative airway techniques may be needed.
- Attempt defibrillation using standard energy doses. Left lateral tilt and large breasts can make it difficult to place an apical defibrillator pad.

Reversible causes of collapse and cardiac arrest in pregnancy

Look for reversible causes using the 4 Hs and 4 Ts approach. Abdominal ultrasound by a skilled operator to detect possible causes during cardiac arrest can be useful. It can also permit an evaluation of fetal viability, multiple pregnancy and placental localisation. It should not however delay treatments. Specific reversible causes of collapse or cardiac arrest in pregnancy include:

Haemorrhage

This can occur both antenatally and postnatally. Causes include ectopic pregnancy, placental abruption, placenta praevia and uterine rupture. Maternity units should have a

massive haemorrhage protocol. Treatment is based on the ABCDE approach. The key step is to stop the bleeding. Consider the following:

- fluid resuscitation including use of a rapid transfusion system and cell salvage
- tranexamic acid and correction of coagulopathy
- oxytocin, ergometrine, prostaglandins and uterine massage to correct uterine atony
- uterine compression sutures, uterine packs, and intrauterine balloon devices
- interventional radiology to identify and control bleeding
- surgical control including aortic crossclamping/compression and hysterectomy. Placenta percreta may require extensive intra-pelvic surgery.

Drugs

Overdose can occur in women with eclampsia receiving magnesium sulfate, particularly if the patient becomes oliguric. Give calcium to treat magnesium toxicity (see electrolyte abnormalities section above). Central neural blockade for analgesia or anaesthesia can cause problems due to sympathetic blockade (hypotension, bradycardia) or local anaesthetic toxicity (see Toxins section above).

Cardiovascular disease

Myocardial infarction and aneurysm or dissection of the aorta or its branches, and peripartum cardiomyopathy cause most deaths from acquired cardiac disease. Patients with known cardiac disease need to be managed in a specialist unit. Pregnant women may develop an acute coronary syndrome, typically in association with risk factors such as obesity, older age, higher parity, smoking, diabetes, pre-existing hypertension and a family history of ischaemic heart disease. Pregnant patients can have atypical features such as epigastric pain and vomiting. Percutaneous coronary intervention (PCI) is the reperfusion strategy of choice for ST-elevation myocardial infarction in pregnancy. Consider fibrinolysis if urgent PCI is unavailable. More women with congenital heart disease are becoming pregnant – they should be managed in specialist centres.

Pre-eclampsia and eclampsia

Eclampsia is defined as the development of convulsions and/or unexplained coma during pregnancy or postpartum in patients with signs and symptoms of pre-eclampsia. Magnesium sulfate treatment may prevent eclampsia developing in labour or immediately postpartum in women with pre-eclampsia.

Amniotic fluid embolism

Amniotic fluid embolism usually presents around the time of delivery, often in the labouring mother, with sudden cardiovascular collapse, breathlessness, cyanosis, arrhythmias, hypotension and haemorrhage associated

with disseminated intravascular coagulopathy. Treatment is supportive based on the ABCDE approach and correction of coagulopathy.

Pulmonary embolus

Pulmonary embolism causing cardiopulmonary collapse can present throughout pregnancy. CPR is started with modifications as necessary. The use of fibrinolysis (thrombolysis) needs considerable thought, particularly if a peri-mortem caesarean section is being considered. If the diagnosis is suspected and maternal cardiac output cannot be restored it should be given.

Peri-mortem caesarean section

When initial resuscitation attempts fail, delivery of the fetus may improve the chances of successful resuscitation of both the mother and fetus. The best survival rate for infants over 24–25 weeks gestation occurs when delivery of the infant is achieved within 5 min after the mother's cardiac arrest. This is a difficult time to achieve in reality, but consideration of peri-mortem caesarean section should be made at an early stage after cardiac arrest has occurred, and resuscitation started.

Delivery relieves IVC compression and may improve the likelihood of resuscitating the mother by permitting an increase in venous return during the CPR attempt. Delivery also enables access to the abdominal cavity so that aortic clamping or compression is possible. Internal cardiac massage may also be possible. Once the fetus has been delivered, resuscitation of the newborn child can also begin.

In the supine position, the gravid uterus begins to compromise blood flow in the IVC and abdominal aorta at approximately 20 weeks' gestation; however, fetal viability currently begins at approximately 24 weeks.

- **Gestational age < 20 weeks.** Urgent caesarean delivery need not be considered, because a gravid uterus of this size is unlikely to compromise maternal cardiac output and fetal viability is not an issue.
- **Gestational age approximately 20–23 weeks.** Initiate emergency delivery of the fetus to permit successful resuscitation of the mother, not survival of the delivered infant, which is unlikely at this gestational age.
- **Gestational age approximately > 24 weeks.** Initiate emergency delivery to help save the life of both the mother and the infant.

Post-resuscitation care

Post-resuscitation care should follow standard guidelines. Targeted temperature management (TTM) has been used safely and effectively in early pregnancy with fetal heart monitoring and resulted in favourable maternal and fetal outcome after a term delivery. Implantable cardioverter defibrillators (ICDs) have been used in patients during pregnancy.

Planning for resuscitation in pregnancy

Advanced life support in pregnancy requires co-ordination of maternal resuscitation, early caesarean delivery of the fetus if maternal cardiac output cannot be restored rapidly, and newborn resuscitation. To achieve this, units likely to deal with cardiac arrest in pregnancy should:

- have in place plans and equipment for resuscitation of both the pregnant patient and the newborn child
- ensure early involvement of obstetric, anaesthetic and neonatal teams
- ensure regular training of staff in obstetric emergencies.

Traumatic cardiorespiratory arrest

Traumatic cardiac arrest has a very high mortality, but when ROSC can be achieved, neurological outcome in survivors appears to be much better than in other causes of cardiac arrest. The response to traumatic cardiac arrest is time-critical and success depends on rapidly identifying and treating the reversible causes.

The history and a scene assessment may provide clues to the cause of cardiac arrest. Cardiac arrest from a primary medical problem (e.g. cardiac arrhythmia, hypoglycaemia, seizure) can also cause a secondary traumatic event (e.g. fall, road traffic accident). In these cases, traumatic injuries may not be the primary cause of a cardiorespiratory arrest and standard advanced life support, including chest compressions and defibrillation are appropriate.

Causes of cardiac arrest in trauma patients include: severe traumatic brain injury, hypovolaemia from massive blood loss, hypoxia from respiratory arrest, direct injury to vital organs and major vessels, tension pneumothorax, and cardiac tamponade.

Commotio cordis is actual or near cardiac arrest caused by a blunt impact to the chest wall over the heart. A blow to the chest can cause VF. Commotio cordis occurs mostly during sports (most commonly baseball) and recreational activities and victims are usually teenage males. Follow standard ALS guidelines. Early defibrillation is important for survival.

Treatment of traumatic cardiac arrest

- Survival from traumatic cardiac arrests is correlated with duration of CPR and pre-hospital time. Factors that are associated with survival include the presence of reactive pupils, an organised ECG rhythm and respiratory activity.
- Prolonged CPR is associated with a poor outcome. If there is no response to 20 min of advanced life support, stop further resuscitation efforts.
- Chest compressions are still the standard of care in patients with cardiac arrest, irrespective of cause. In cardiac arrest caused by hypovolaemia, cardiac

tamponade or tension pneumothorax, chest compressions are unlikely to be as effective as in normovolaemic cardiac arrest. Thus, chest compressions have a lower priority than the immediate treatment of reversible causes (e.g. thoracotomy, controlling haemorrhage etc). In an out-of-hospital setting, only essential life-saving interventions are undertaken on scene before rapid transfer to the nearest appropriate hospital. Do not delay for spinal immobilisation.

- Focused ultrasound can help diagnose haemoperitoneum, haemopneumothorax, tension pneumothorax and cardiac tamponade. This requires a trained operator and should not delay treatment. In more stable patients who are not in cardiac arrest, early rapid whole-body computed tomography scanning as part of the primary survey will help identify injuries and guide treatment.

Hypovolaemia – haemorrhage control

Uncontrolled haemorrhage is the cause of traumatic cardiac arrest in 48% of cases, so early haemorrhage control is essential.

- Treat compressible external haemorrhage with elevation and direct pressure (with or without a dressing), use tourniquets if needed and/or apply topical haemostatic agents.
- Non-compressible haemorrhage is more difficult. Use splints (pelvic splint), blood products, IV fluids and tranexamic acid while moving the patient to surgical/radiological haemorrhage control.
- In the presence of uncontrolled bleeding, excessive fluid will increase the bleeding. In the UK, the National Institute for Health and Clinical Excellence (NICE) has published guidelines on pre-hospital fluid replacement in trauma. The recommendations include giving 250 mL boluses of crystalloid solution until a radial pulse is achieved and not delaying rapid transport of trauma victims for fluid infusion.
- Use IO access when IV access is not initially feasible.
- The early use of blood products as a primary resuscitation fluid may improve outcomes and prevent trauma-induced coagulopathy.
- Aiming for a systolic blood pressure of 80–90 mmHg until definitive bleeding control may help prevent further blood loss but may be harmful in patients with a traumatic brain injury, where a higher blood pressure can be beneficial to the injured brain.

Hypoxia

- Effective airway management is essential to maintain oxygenation of the severely compromised trauma patient. Early tracheal intubation by experienced rescuers can be beneficial. Use basic airway

management manoeuvres and alternative airways to maintain oxygenation if tracheal intubation cannot be accomplished immediately. If these measures fail, a surgical airway is indicated.

- In low cardiac output conditions, positive pressure ventilation causes further circulatory depression, or even cardiac arrest, by impeding venous return to the heart. Monitor ventilation with continuous waveform capnography. This may enable slow respiratory rates and low tidal volumes and the corresponding decrease in transpulmonary pressure may increase venous return and cardiac output.
- During CPR, use 100% oxygen. In peri-arrest or post-ROSC patients, titrate oxygen levels to achieve an SpO₂ of 94–98%.

Tension pneumothorax

- To decompress the chest in traumatic cardiac arrest, perform bilateral thoracostomies in the 5th intercostal space, extending to a clamshell thoracotomy if required. In the presence of positive pressure ventilation, thoracostomies are likely to be more effective than needle thoracocentesis and quicker than inserting a chest tube.
- A significant proportion of patients have a chest wall that is too thick to be decompressed with a standard length 14-gauge cannula. Cannulae are also prone to kinking and blockage so this is a very temporary measure. Any attempt at needle decompression should be followed by insertion of a chest tube (see asthma). The needle can be placed in the 2nd intercostal space in the midclavicular line or in the 5th intercostal space in the midaxillary line.

Cardiac tamponade

- Cardiac tamponade occurs when the pericardial sac is filled with fluid under pressure, which leads to compromise of cardiac function and ultimately cardiac arrest.
- It most commonly occurs after penetrating trauma and cardiac surgery. Mortality is high and immediate decompression of the pericardium is required to give any chance of survival.
- In traumatic cardiac arrest with penetrating trauma to the chest or epigastrium, immediate resuscitative thoracotomy with a clamshell incision can be life-saving.
- Needle aspiration of tamponade, with or without ultrasound guidance, is unreliable because the pericardium is commonly full of clotted blood.

Resuscitative thoracotomy

- Consider resuscitative thoracotomy for patients with penetrating torso trauma and less than 15 min of CPR and patients with blunt trauma with less than 10 min of pre-hospital CPR. Thoracotomy will enable evacuation of pericardial blood to relieve cardiac tamponade and repair of simple cardiac wounds.
- Patients with no pulse after penetrating chest or cardiac injuries, who arrive at hospital after a short on-scene and transport time with witnessed signs of life or ECG activity, are candidates for emergency department resuscitative thoracotomy.
- The chances of success are much lower when cardiac arrest results from blunt trauma.

Perioperative cardiac arrest

Perioperative cardiac arrest can be caused by the underlying condition being treated, physiological effects of the surgery, anaesthetic drugs and fluids, complications relating to existing co-morbidities, or adverse events.

- Overall survival from perioperative cardiac arrest is high, compared to cardiac arrest in other settings.
- The incidence of perioperative cardiac arrest during general anaesthesia (GA) is higher than that of regional anaesthesia (RA).
- Causes of perioperative cardiac arrest include hypovolaemia (e.g. bleeding), cardiac problems, and anaesthesia related problems.
- The commonest cause of anaesthesia-related cardiac arrest involves airway management. Failure of ventilation, medication-related events, complications associated with central venous access, and perioperative myocardial infarction are also common.
- The primary arrest rhythms during perioperative cardiac arrest recorded in a large single centre (Mayo Clinic) series were asystole in 41.7%, VF in 35.4%, PEA in 14.4% and unknown in 8.5%. In contrast to cardiac arrest in other circumstances, the rhythm associated with the best chance of survival to hospital discharge was asystole (43% survival).

Management of perioperative cardiac arrest

Patients are normally fully monitored and, as such, there should be little or no delay in diagnosing cardiac arrest. High-risk patients will often also have invasive blood pressure monitoring, which is invaluable in the event of cardiac arrest. If cardiac arrest is a strong possibility, apply self-adhesive defibrillation electrodes before induction of anaesthesia, ensure adequate venous access and prepare

resuscitation drugs and fluids. Use fluid warmers and forced air warmers to limit perioperative hypothermia and monitor the patient's temperature.

- Asystole and VF will be detected immediately, but the onset of PEA might not be so obvious – loss of the pulse oximeter signal and very low end-tidal CO₂ values will be good clues and should provoke a pulse check. Do not waste time attempting to measure non-invasive blood pressure
- In the event of cardiac arrest, follow the ALS algorithm, but with appropriate modifications. Adjust the position and height of the operating table or trolley to optimise delivery of chest compressions.
- CPR is optimal in the supine position, but is possible in patients who are prone and where immediate turning to a supine position is not possible. Risk factors for cardiac arrest in prone patients include cardiac abnormalities in patients undergoing major spinal surgery, hypovolaemia, air embolism, wound irrigation with hydrogen peroxide, and occluded venous return. The Resuscitation Council (UK) has produced specific guidance on the Management of cardiac arrest during neurosurgery in adults
<https://www.resus.org.uk/publications/management-of-cardiac-arrest-during-neurosurgery-in-adults/>

Identification of causes

In many cases of perioperative cardiac arrest, physiological deterioration is gradual and the cause of the cardiac arrest is known and hence the arrest anticipated. In those where this is not the case, follow the standard ABCDE approach to identify and treat reversible causes.

- c** - Catastrophic haemorrhage is usually obvious, but may be occult if it involves bleeding into body compartments (abdomen, chest) or into soft tissues in patients with multiple limb fractures. Pelvic and retroperitoneal haemorrhage can also cause rapid hypovolaemia and should be excluded (e.g. by ultrasound if pre-operative haemodynamic instability). In cases where direct surgical intervention is unable to control haemorrhage, consider early interventional radiography.
- A** Loss of the airway is a common cause of perioperative cardiac arrest. Always use waveform capnography to ensure the lungs are ventilated.
- B** Undiagnosed tension pneumothorax is a readily treatable cause of cardiac arrest (see the management of tension pneumothorax above).
- C** Cardiovascular collapse has several causes, but in the context of perioperative cardiac arrest, common causes include hypovolaemia, anaphylaxis (most commonly with neuromuscular blockers), and vagal

stimulation. Use of focused transthoracic echocardiography is a useful tool to exclude cardiac tamponade (if suspected) and to assess myocardial contractility and filling.

Specific modifications for perioperative cardiac arrest

- Chest compression in the prone position can be achieved with or without sternal counter-pressure. Consider open cardiac compressions in patients where the thorax is open or the heart can be easily accessed.
- In the case of VF/pVT, call for a defibrillator. If one is not immediately available, give a precordial thump. If that is unsuccessful, give chest compressions and ventilation until the defibrillator arrives. Look for reversible causes immediately – hypoxaemia and hypovolaemia will be the most common in this setting.
- If there is asystole or extreme bradycardia stop any surgical activity likely to be causing excessive vagal activity – if this is the likely cause, give 0.5 mg atropine IV and further doses as required. Start CPR and immediately look for other reversible causes.
- If adrenaline is required according to the ALS algorithm, give the initial dose in increments (e.g. 50–100 micrograms IV), rather than a 1 mg bolus. If 1 mg in total has been given with no response, consider further adrenaline doses of 1 mg IV.
- Waveform capnography and invasive arterial monitoring (when available) will help guide resuscitation (Chapter 6).
- See section on Anaphylaxis (above) for the treatment of cardiac arrest associated with anaphylaxis. Titrate IV adrenaline using 50 mcg boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion. The pre-filled 10 mL syringe of 1:10,000 adrenaline contains 100 mcg mL⁻¹.
- See section on Toxins (above) for the treatment of systemic toxicity of local anaesthetics with IV lipid.
- Stop operative surgery unless it is addressing a reversible cause of the cardiac arrest.

Cardiac arrest following cardiac surgery

Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase, with a reported incidence of 0.7–8%.

There are usually specific causes of cardiac arrest, such as tamponade, hypovolaemia, myocardial ischaemia, tension pneumothorax, or pacing failure. These are all potentially reversible and if treated promptly cardiac arrest after cardiac surgery, have a relatively high survival rate.

- The key to successful resuscitation is recognition of the need to perform emergency resternotomy early, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective.
- If VF or asystole, attempt external defibrillation or emergency temporary pacing at maximum amplitude.
- Start external chest compressions immediately in patients who arrest with monitoring indicating no output. Verify the effectiveness of compressions by looking at the arterial trace, aiming to achieve a systolic blood pressure > 60 mmHg and a diastolic blood pressure > 25 mmHg at a rate of 100–120 min⁻¹. Inability to obtain these targets with external chest compressions indicates the need for emergency resternotomy because ineffective chest compressions may indicate cardiac tamponade and/or hypovolaemia.
- There is concern that external chest compressions can cause sternal disruption or cardiac damage.
- Consider other reversible causes:
 - Hypoxia – check tracheal tube position, use waveform capnography, and ventilate with 100% oxygen during resuscitation.
 - Tension pneumothorax.
 - Pacing failure – check pacing box output and pacing wire integrity. In asystole, secondary to a loss of cardiac pacing, chest compressions may be delayed momentarily as long as the surgically inserted temporary pacing wires can be connected rapidly and pacing re-established (DDD mode at 100 min⁻¹ at maximum amplitude).
- Treat a witnessed and monitored VF/pVT cardiac arrest immediately with up to three quick successive (stacked) defibrillation attempts. Three failed shocks in the post-cardiac surgery setting should trigger the need for emergency resternotomy. Further defibrillation is attempted as indicated in the ALS algorithm and is performed with internal paddles at 20 J if resternotomy has been performed.
- Use adrenaline very cautiously and titrate to effect (IV doses of up to 100 mcg in adults). Consider amiodarone 300 mg in patients with refractory shockable rhythms (VF/pVT), but do not delay resternotomy. Atropine is not recommended for asystole and temporary or external pacing should be employed.
- Emergency resternotomy is an integral part of resuscitation after cardiac surgery, once all other reversible causes have been excluded. Once adequate airway and ventilation has been established, and if three attempts at defibrillation have failed in VF/pVT, undertake resternotomy without delay. Emergency resternotomy is also indicated in asystole or PEA, when other treatments have failed, and should be performed within 5 min of the cardiac arrest by anyone with appropriate training.

Drowning

Drowning is a common cause of accidental death. The World Health Organization (WHO) reports that every hour of every day, more than 40 die from drowning. In general, males are much more likely to drown than females. In the UK, there are approximately 350 accidental deaths from drowning each year. Drowning is commonest in males aged 20–30, and occurs mostly in inland waters (e.g. lakes, rivers) and during summer months.

Definition of drowning

Drowning is defined as a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium. Implicit in this definition is that a liquid/air interface is present at the entrance of the victim's airway, preventing the victim from breathing air. The victim may live or die after this process, but whatever the outcome, he or she has been involved in a drowning incident.

Submersion occurs when the face is underwater or covered in water. Asphyxia and cardiac arrest occurs within a matter of minutes of submersion. Immersion, by contrast, is when the head remains above water, in most cases by means of the support of a lifejacket. In most situations of immersion, the victim remains immersed with an open airway and becomes hypothermic, although aspiration of water may occur if water splashes over the face or if the victim becomes unconscious with their face in the water. The difference between submersion and immersion is important in understanding the difference in epidemiology, pathophysiology, clinical course and prognosis.

Pathophysiology of drowning

Following submersion, the victim initially breath holds by reflex, and frequently swallows water. As breath holding continues, hypoxia and hypercapnia develop. A reflex laryngospasm may temporarily prevent the entrance of water into the lungs. Eventually these reflexes abate and the victim aspirates water. The key is that bradycardia as a consequence of hypoxia occurs before sustaining a cardiac arrest. Correction of hypoxaemia by ventilation-only resuscitation is critical and in itself may lead to ROSC in some cases, probably because the presence of a circulation had not been detected.

Water rescue

- Whenever possible, bystanders should attempt to save the drowning victim without entry into the water. Talking to the victim, reaching with a rescue aid (e.g. stick or clothing), or throwing a rope or buoyant rescue aid may be effective if the victim is close to dry land.
- Rescue can present significant risk to the rescuer, but a sensible risk assessment is necessary to ensure that potentially survivable victims are rescued promptly.
- If entry into the water is essential, take a buoyant

rescue aid, flotation device or boat. It is safer to enter the water with two rescuers than alone.

- Trained rescuers are often professionals with specialist equipment to assist with search and rescue and will base rescue efforts on the likelihood of survival. Submersion durations of less than 10 min are associated with a very high chance of a good outcome, and submersion durations of more than 25 min are associated with a low chance of good outcome.
- In the UK combined emergency services guidance recommends review of search and rescue efforts at 30 and 60 min for submersion. Extended rescue efforts up to 90 min may be appropriate for children or those submerged in icy cold water, although the protective effects of extreme hypothermia are unlikely to be sufficient in the UK where water is insufficiently cold to cool rapidly and provide neuroprotection.
- Trained individuals should consider in-water ventilation (with the support of a buoyant rescue aid) only if there is likely to be a delay in reaching land or a rescue craft.
- Remove the victim from the water promptly. The chances of a drowning victim sustaining a spinal injury are very low. Spinal precautions are unnecessary unless there is a history of diving in shallow water, or signs of severe injury after water-slide use, water-skiing, kite-surfing, or watercraft racing. If the victim is pulseless and apnoeic, remove them from the water as quickly as possible while attempting to limit neck flexion and extension.
- Hypovolaemia after prolonged immersion can cause cardiovascular collapse/arrest on removal from water, especially if the victim is upright. Keep the victim in a horizontal position during and after retrieval from the water.

Initial resuscitation once retrieved from water

- Check for response by opening the airway and check for signs of life. The drowning victim rescued from the water within a few minutes of submersion is likely to exhibit abnormal (agonal) breathing. Do not confuse this with normal breathing.
- Give 5 initial rescue breaths supplemented with oxygen if available.
- If the victim has not responded to initial ventilations, place them on a firm surface before starting chest compressions. Provide CPR in a ratio of 30 compressions to 2 ventilations. Most drowning victims will have sustained cardiac arrest secondary to hypoxia. In these patients, compression-only CPR is likely to be ineffective and should be avoided.

- Massive amounts of foam caused by mixing moving air with water and surfactant can sometimes come out of the mouth of victims. If this occurs, continue rescue breaths/ventilation until an ALS provider arrives and is able to intubate the victim's trachea.
- Regurgitation of stomach contents and swallowed water is common. If this prevents ventilation, turn the victim on their side and remove the regurgitated material using directed suction if possible.

Modifications to advanced life support after drowning

Airway and breathing

- Give high-flow oxygen ($10\text{--}15 \text{ L min}^{-1}$), ideally through an oxygen mask with reservoir bag to the spontaneously breathing victim.
- For victims who fail to respond to initial basic airway measures, who have a reduced level of consciousness or are in cardiac arrest, consider early tracheal intubation and controlled ventilation by skilled personnel. Reduced pulmonary compliance requiring high inflation pressures may limit the use of a supraglottic airway device.
- In the drowning patient who has not arrested or who has achieved ROSC, titrate the inspired oxygen concentration to achieve an SpO_2 of 94–98%. Confirm adequate oxygenation and ventilation with arterial blood gases once available. Set positive end expiratory pressure (PEEP) to at least 5–10 cm H_2O . However, PEEP values of 15–20 cm H_2O may be required if the patient is severely hypoxaemic. Decompress the stomach with a gastric tube.

Circulation and defibrillation

- Palpation of the pulse as the sole indicator of the presence or absence of cardiac output is not always reliable, particularly in the wet and cold drowning patient. As soon as possible, check the ECG and end-tidal CO_2 , and consider echocardiography to confirm the presence or not of a cardiac output.
- If the victim is in cardiac arrest, follow standard ALS protocols. If the victim is hypothermic, modify the approach in accordance with the guidance for treatment of hypothermia (see below).
- Assess the rhythm and attempt defibrillation if indicated according to standard guidelines. Dry the victim's chest before applying defibrillator pads.
- After prolonged immersion, most victims will have become hypovolaemic due to the cessation of the hydrostatic pressure of water on the body. Give rapid IV fluid to correct hypovolaemia. This should commence out-of-hospital, if transfer time is prolonged.

Discontinuing resuscitation efforts

- Making a decision to discontinue resuscitation efforts on a victim of drowning is difficult. No single factor accurately predicts good or poor survival. Frequently, decisions may later prove to have been incorrect.
- Continue resuscitation unless there is clear evidence that such attempts are futile (e.g. massive traumatic injuries, rigor mortis, putrefaction etc.), or timely evacuation to a medical facility is not possible.
- Neurologically intact survival has been reported in several victims submerged for longer than 25 min, however these rare case reports almost invariably occur in children submerged in ice-cold water, when immersion hypothermia has preceded hypoxia or in submersion of car occupants. A retrospective study of 160 children who drowned in the Netherlands found that outcomes were extremely poor if ALS took longer than 30 min to achieve ROSC even if hypothermia was present.

Post-resuscitation care after drowning

- Follow standard post-resuscitation guidelines (Chapter 13).
- Many victims of drowning are at risk of developing acute respiratory distress syndrome (ARDS) and standard protective ventilation strategies for ARDS should be followed.
- Extracorporeal membrane oxygenation (ECMO) has been used for those in refractory cardiac arrest, those with refractory hypoxaemia and in selected cases of submersion in ice cold water, although success rates remain low.
- Pneumonia is common after drowning. Prophylactic antibiotics have not been shown to be of benefit but they may be considered after submersion in grossly contaminated water such as sewage.
- Neurological outcome, notably severe permanent neurological damage, is primarily determined by the duration of hypoxia.

Accidental hypothermia

Definition of hypothermia

Hypothermia exists when the body core temperature is below 35°C and is classified arbitrarily as mild (32–35°C), moderate (28–32°C), or severe (< 28°C). The Swiss staging system based on clinical signs can be used by rescuers at the scene to describe victims:

- Stage I: mild hypothermia (conscious, shivering, core temperature 35–32°C).
- Stage II: moderate hypothermia (impaired consciousness without shivering, core temperature 32–28°C).

- Stage III: severe hypothermia (unconscious, vital signs present, core temperature 28–24°C).
- Stage IV: cardiac arrest or low flow state (no or minimal vital signs, core temperature < 24°C).
- Stage V: death due to irreversible hypothermia (core temperature < 13.7°C).

Diagnosis of hypothermia

- Accidental hypothermia may be under-diagnosed in countries with a temperate climate. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia may follow a mild insult. The risk of hypothermia is increased by alcohol or drug ingestion, exhaustion, illness, injury or neglect especially when there is a decrease in the level of consciousness.
- Hypothermia may be suspected from the clinical history or a brief external examination of a collapsed patient.
- A low-reading thermometer is needed to measure core temperature and confirm the diagnosis. The core temperature in the lower third of the oesophagus correlates well with heart temperature.
- Tympanic measurement using a thermocouple is a reliable alternative, but may be considerably lower than core temperature if the environment is very cold, the probe is not well insulated, or the external auditory canal is filled with snow or water. Commonly used tympanic thermometers based on infrared technique do not seal the ear canal and are not designed for low core temperature readings.
- Once in hospital, use a consistent core temperature measurement site throughout resuscitation and rewarming. Bladder and rectal temperatures lag behind core temperature and are not recommended in patients with severe hypothermia.

Decision to resuscitate the hypothermic patient

- Cooling of the human body decreases cellular oxygen consumption by about 6% per 1°C decrease in core temperature. In some cases, hypothermia can exert a protective effect on the brain and vital organs and intact neurological recovery is possible, even after prolonged cardiac arrest if deep hypothermia develops before asphyxia.
- Beware of diagnosing death in a hypothermic patient, because hypothermia can produce a very slow, small-volume, irregular pulse and unrecordable blood pressure. In a hypothermic patient, no signs of life (Swiss hypothermia stage IV) alone are unreliable for declaring death. At 18°C the brain can tolerate periods of circulatory arrest for ten times longer than at 37°C. Dilated pupils can be caused by a variety of insults and

must not be regarded as a sign of death. Good quality survival has been reported after cardiac arrest and a core temperature of 13.7°C after immersion in cold water and with CPR for as long as six and a half hours in an adult.

- Intermittent CPR during rescue can be of benefit if continuous CPR cannot be delivered. In these circumstances, in a patient with hypothermic cardiac arrest and a core temperature < 28°C (or unknown), give 5 min of CPR, alternating with periods ≤ 5 min without CPR. Patients with a core temperature < 20°C, should receive 5 min of CPR, alternating with periods of up to 10 min without CPR.
- In the prehospital setting, resuscitation should be withheld only if the cause of a cardiac arrest is clearly attributable to a lethal injury, fatal illness, prolonged asphyxia, or if the chest is incompressible. In all other patients the traditional guiding principle that ‘no one is dead until warm and dead’ should be considered. In remote wilderness areas, the impracticalities of achieving rewarming have to be considered. In the hospital setting, involve senior doctors and use clinical judgment to determine when to stop resuscitating a hypothermic arrest victim.
- Avalanche victims are not likely to survive when they are:
 - buried > 60 min (or if the initial core temperature is < 30°C) and in cardiac arrest with an obstructed airway on extrication
 - buried and in cardiac arrest on extrication with an initial serum potassium > 8 mmol L⁻¹.

Modifications to advanced life support in the hypothermic patient

- Check for signs of life for up to 1 min. Palpate a central artery and assess the cardiac rhythm (if ECG monitor available). Echocardiography, near-infrared spectroscopy or ultrasound with Doppler can be used to establish whether there is an adequate cardiac output or peripheral blood flow. If there is any doubt, start CPR immediately.
- Hypothermia can cause stiffness of the chest wall, making ventilations and chest compressions difficult. Consider the use of mechanical chest compression devices.
- Do not delay careful tracheal intubation when it is indicated. The advantages of adequate oxygenation and protection from aspiration outweigh the minimal risk of triggering VF by performing tracheal intubation.
- Once CPR is under way, confirm hypothermia with a low-reading thermometer.

- The hypothermic heart may be unresponsive to cardioactive drugs, attempted electrical pacing and defibrillation. Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drug given. The evidence for the efficacy of drugs in severe hypothermia is limited. It is reasonable to withhold adrenaline, and other CPR drugs until the patient has been warmed to a core temperature $\geq 30^{\circ}\text{C}$. Once 30°C has been reached, the intervals between drug doses should be doubled when compared to normothermia (i.e. adrenaline every 6–10 min). As normothermia is approached ($\geq 35^{\circ}\text{C}$), use standard drug protocols.

Treatment of arrhythmias associated with hypothermia

- As core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation followed by VF and finally asystole.
- Arrhythmias other than VF tend to revert spontaneously as core temperature increases, and usually do not require immediate treatment. Bradycardia is physiological in severe hypothermia. Cardiac pacing is not indicated unless bradycardia associated with haemodynamic compromise persists after rewarming.
- The temperature at which defibrillation should first be attempted, and how often it should be attempted in the severely hypothermic patient, has not been established. If VF is detected, defibrillate according to standard protocols. If VF persists after three shocks, delay further attempts until core temperature is $\geq 28\text{--}30^{\circ}\text{C}$. CPR and rewarming may have to be continued for several hours to facilitate successful defibrillation.

Rewarming after accidental hypothermia

Insulation

- General measures for all victims include removal from the cold environment, prevention of further heat loss and rapid transfer to hospital.
- In the field, a patient with moderate or severe hypothermia (hypothermia stage $\geq \text{II}$) should be immobilised and handled carefully, oxygenated adequately, monitored (including ECG and core temperature), and the whole body dried and insulated.
- Remove wet clothes, while minimising excessive movement of the victim.
- Conscious victims (hypothermia stage I) can mobilise as exercise warms a person more rapidly than shivering.
- Patients will continue cooling after removal from a cold environment (i.e. afterdrop). This can result in a life-threatening decrease in core temperature triggering a cardiac arrest during transport (i.e. ‘rescue death’).
- Patients who stop shivering (e.g. hypothermia stage II–

IV, and sedated or anaesthetised patients) will cool faster.

Prehospital rewarming and transfer

- Rewarming may be passive, active external, or active internal.
- In mild hypothermia, passive rewarming is appropriate as patients are still able to shiver. This is best achieved by full body insulation with wool blankets, aluminium foil, cap and a warm environment.
- In hypothermia stages II–IV the application of chemical heat packs to the trunk has been recommended. In conscious patients who are able to shiver, this improves comfort but does not speed rewarming. If the patient is unconscious and the airway is not secured, arrange the insulation around the patient lying in a recovery (lateral decubitus) position.
- Rewarming in the field with heated intravenous fluids and warm humidified gases is not feasible.
- Intensive active rewarming must not delay transport to a hospital where advanced rewarming techniques, continuous monitoring and observation are available.
- The choice of hospital for transfer depends on the patients condition:
 - Stage I: those with mild hypothermia (conscious, shivering, core temperature $35\text{--}32^{\circ}\text{C}$) should go to the nearest hospital.
 - In hypothermia stages II–IV, consider direct transfer to a hospital with facilities for ECLS, especially when there is cardiac instability (i.e. systolic blood pressure $< 90 \text{ mmHg}$, ventricular arrhythmia, core temperature $< 28^{\circ}\text{C}$).
 - In hypothermia stage V, consider reasons for withholding or terminating CPR (e.g. obvious signs of irreversible death, a valid DNACPR decision, conditions unsafe for rescuer, avalanche burial $\geq 60 \text{ min}$ and airway packed with snow and asystole). In the absence of any of these signs, start CPR and transfer the patient to an ECLS centre.

In-hospital rewarming and post-resuscitation care

- Rewarm using active external methods (i.e. with forced warm air) and minimally invasively methods (i.e. with warm IV infusions).
- With a core temperature $< 32^{\circ}\text{C}$ and $\text{K}^{+} < 8 \text{ mmol L}^{-1}$, consider ECLS rewarming. ECLS was commonly undertaken using cardiopulmonary bypass, but more recently, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) has become the preferred method because it can be made available more rapidly, needs less anticoagulation and can provide prolonged cardiorespiratory support after rewarming.

- If an ECLS centre is not available, rewarming can be attempted using a combination of external and internal rewarming techniques (e.g. forced warm air, warm infusions, forced peritoneal lavage).
- Continuous haemodynamic monitoring and warm IV fluids are essential. Patients will require large volumes of IV fluids during rewarming, as vasodilation causes expansion of the intravascular space. Avoid hyperthermia during and after rewarming.
- Once ROSC has been achieved, use standard post-resuscitation care.

Hyperthermia

Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds that normally maintained by homeostatic mechanisms. Hyperthermia may be exogenous, caused by environmental conditions or secondary to endogenous heat production.

Environment-related hyperthermia occurs where heat, usually in the form of radiant energy, is absorbed by the body at a rate faster than can be lost by thermoregulatory mechanisms. Hyperthermia occurs along a continuum of heat-related conditions starting with heat stress, progressing to heat exhaustion, heat stroke and culminating in multi-organ dysfunction and cardiac arrest in some instances.

Malignant hyperthermia (MH) is a rare disorder of skeletal muscle calcium homeostasis characterised by muscle contracture and life-threatening hypermetabolic crisis following exposure of genetically predisposed individuals to halogenated anaesthetics and depolarising muscle relaxants.

Heat stroke

Heat stroke is a systemic inflammatory response with a core temperature $> 40.6^{\circ}\text{C}$ accompanied by a change in mental state and varying levels of organ dysfunction. Mortality from heat stroke ranges between 10–50%. There are two forms of heat stroke:

- Classic non-exertional heat stroke occurs during high environmental temperatures and often affects the elderly during heat waves.
- Exertional heat stroke occurs during strenuous physical exercise in high environmental temperatures and/or high humidity and usually effects healthy young adults.

Predisposing factors for heat stroke

The elderly are at increased risk for heat-related illness because of underlying illness, medication use, declining thermoregulatory mechanisms, and limited social support. There are several risk factors: lack of acclimatisation, dehydration, obesity, alcohol, cardiovascular disease, skin conditions (psoriasis, eczema, scleroderma, burn, cystic fibrosis) hyperthyroidism, phaeochromocytoma, and drugs (anticholinergics, diamorphine, cocaine, amphetamine,

phenothiazines, sympathomimetics, calcium channel blockers, beta-blockers).

Clinical presentation of heat stroke

Heat stroke can resemble septic shock and may be caused by similar mechanisms. Features include:

- core temperature $\geq 40^{\circ}\text{C}$
- hot, dry skin (sweating present in about 50% of cases of exertional heat stroke)
- early signs and symptoms (e.g. extreme fatigue, headache, fainting, facial flushing, vomiting and diarrhoea)
- cardiovascular dysfunction including arrhythmias and hypotension
- respiratory dysfunction including acute respiratory distress syndrome (ARDS)
- central nervous system dysfunction including seizures and coma
- liver and renal failure
- coagulopathy
- rhabdomyolysis.

Other clinical conditions presenting with increased core temperature need to be considered, including drug toxicity, drug withdrawal syndrome, serotonin syndrome, neuroleptic malignant syndrome, sepsis, central nervous system infection, endocrine disorders (e.g. thyroid storm, phaeochromocytoma).

Treatment of heat stroke

The mainstay of treatment is supportive therapy based on optimising the ABCDEs and rapidly cooling the patient.

- Start cooling before the patient reaches hospital. Aim to rapidly reduce the core temperature to approximately 39°C . Patients with severe heat stroke need to be managed in a critical care setting.
- Use haemodynamic monitoring to guide fluid therapy. Large volumes of fluid may be required. Correct electrolyte abnormalities.
- If cardiac arrest occurs, follow standard procedures for basic and advanced life support and cool the patient. Attempt defibrillation, if appropriate, according to current guidelines, while continuing to cool the patient. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest. The risk of unfavourable neurological outcome increases for each degree of body temperature $> 37^{\circ}\text{C}$.
- Provide post-resuscitation care according to normal guidelines (Chapter 13).

Cooling techniques for heat stroke

Several cooling methods have been described but there are few formal trials on which method is best.

- Simple techniques include cool drinks, fanning the undressed patient and spraying tepid water on the patient. Ice packs over areas where there are large superficial blood vessels (axillae, groins, neck) are also useful. Surface cooling may cause shivering.
- In cooperative stable patients, immersion in cold water is effective; however, this can cause peripheral vasoconstriction and reduce heat dissipation. Immersion is not practical in very sick patients.
- Use the same advanced cooling techniques as for targeted temperature management after cardiac arrest (Chapter 13). Consider the use of cold IV fluids, intravascular cooling catheters, surface cooling devices and extracorporeal circuits.
- No specific drugs lower core temperature in heat stroke. There is no good evidence that antipyretics (e.g. non-steroidal anti-inflammatory drugs or paracetamol) are effective in heat stroke. Diazepam may be useful to treat seizures and facilitate cooling. Dantrolene (see below) has not been shown to be beneficial.

Malignant hyperthermia

Malignant hyperthermia is a life-threatening genetic sensitivity of skeletal muscles to volatile anaesthetics and depolarising neuromuscular blocking drugs occurring during or after anaesthesia. Stop triggering agents immediately; give oxygen, correct acidosis and electrolyte abnormalities. Start active cooling and give dantrolene. Other drugs such as 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines also cause a condition similar to malignant hyperthermia and the use of dantrolene may be beneficial.

Lightning strike and electrical injuries

Electrical injury is a relatively infrequent but potentially devastating multi-system injury with high morbidity and mortality. Most electrical injuries in adults occur in the workplace and are associated generally with high voltage, whereas children are at risk primarily at home, where the voltage is lower (220 V in Europe, Australia, Asia; 110 V in the USA and Canada). Electrocution from lightning strikes is rare, but causes about 1000 deaths worldwide each year.

Factors influencing the severity of electrical injury include whether the current is alternating (AC) or direct (DC), voltage, magnitude of energy delivered, resistance to current flow, pathway of current through the patient, and the area and duration of contact. Skin resistance is decreased by moisture, which increases the likelihood of injury. Electric current follows the path of least resistance; conductive neurovascular bundles within limbs are particularly prone to damage.

Contact with AC may cause tetanic contraction of skeletal muscle, which may prevent physical release from the source of electricity. Myocardial or respiratory failure may cause immediate death:

- Respiratory arrest may be caused by central respiratory depression or paralysis of the respiratory muscles.
- Current may precipitate VF if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon). Electrical current may also cause myocardial ischaemia because of coronary artery spasm.
- Asystole may be primary, or secondary to asphyxia following respiratory arrest.

Current that traverses the myocardium is more likely to be fatal. A transthoracic (hand-to-hand) pathway is more likely to be fatal than a vertical (hand-to-foot) or straddle (foot-to-foot) pathway. There may be extensive tissue destruction along the current pathway.

Lightning strikes deliver as much as 300 kV over a few milliseconds. Most of the current from a lightning strike passes over the surface of the body in a process called external flashover. Both industrial shocks and lightning strikes cause deep burns at the point of contact - in industry the points of contact are usually on the upper limbs, hands and wrists, whilst with lightning they are mostly on the head, neck and shoulders. Injury may also occur indirectly through ground current or current 'splashing' from a tree or other object that is hit by lightning. Explosive force generated by a lightning strike may cause blunt trauma.

The pattern and severity of injury from a lightning strike varies considerably. As with industrial and domestic electric shock, death is caused by cardiac or respiratory arrest. In those who survive the initial shock, extensive catecholamine release or autonomic stimulation may occur, causing hypertension, tachycardia, nonspecific ECG changes (including prolongation of the QT interval and transient T wave inversion), and myocardial necrosis. Creatine kinase may be released from myocardial and skeletal muscle. Lightning also causes various central and peripheral neurological problems.

Treatment of lightning strike and electrical injuries

Ensure that any power source is switched off and do not approach the victim until it is safe. High voltage (above domestic mains) electricity can arc and conduct through the ground for up to a few metres around the victim. It is safe to approach and handle casualties after lightning strike, although it would be wise to move to a safer environment. Follow standard resuscitation guidelines.

- Airway management can be difficult if there are electrical burns around the face and neck. Intubate the trachea early in these cases as soft tissue oedema can cause subsequent airway obstruction. Consider cervical spine immobilisation. This should not delay airway management.

- Muscular paralysis, especially after high voltage, may persist for several hours; ventilatory support is required during this period.
- VF is the commonest initial arrhythmia after high voltage AC shock; treat with prompt attempted defibrillation. Asystole is more common after DC shock; use standard guidelines for treatment of this and other arrhythmias.
- Remove smouldering clothing and shoes to prevent further thermal injury.
- Give IV fluids if there is significant tissue destruction. Maintain a good urine output to increase excretion of myoglobin, potassium and other products of tissue damage.
- Consider early surgical intervention in patients with severe thermal injuries.
- Conduct a thorough secondary survey to exclude injuries caused by tetanic muscular contraction or from the person being thrown by the force of the shock.
- Electrocution can cause severe, deep soft tissue injury with relatively minor skin wounds because current tends to follow neurovascular bundles; look carefully for features of compartment syndrome, which will necessitate fasciotomy.

Further treatment and prognosis

Immediate resuscitation in young victims of cardiac arrest due to electrocution can result in survival. Successful resuscitation has been reported after prolonged life support. All those who survive electrical injury should be monitored in hospital if they have a history of cardiorespiratory problems or have had:

- loss of consciousness
- cardiac arrest
- electrocardiographic abnormalities
- soft tissue damage and burns.

Severe burns (thermal or electrical), myocardial necrosis, the extent of central nervous system injury, and secondary multiple system organ failure, determine the morbidity and long-term prognosis. There is no specific therapy for electrical injury, and the management is symptomatic. Prevention remains the best way to minimise the prevalence and severity of electrical injury.

Summary learning

- The conditions described in this chapter account for a large proportion of cardiac arrests in younger patients.
- Use the ABCDE approach for early recognition and treatment to prevent cardiac arrest
- High quality CPR and treatment of reversible causes is the mainstay of treatment of cardiac arrest from any cause.
- Call for expert help early when specialist procedures are needed (e.g. delivery of the fetus for cardiac arrest in pregnancy).

My key take-home messages from this chapter

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post-resuscitation care

Chapter
13

Contents

- The post-cardiac arrest syndrome
- Optimising organ function
- The post-resuscitation care algorithm
- Prognostication

Learning outcomes

To enable you to:

- Understand the need for continued resuscitation after return of spontaneous circulation
- Understand the post-cardiac arrest syndrome
- Facilitate safe transfer of the patient
- Consider the role and limitations of assessing prognosis after cardiac arrest

Introduction

Return of a spontaneous circulation (ROSC) is an important step in the continuum of resuscitation. However, the next goal is to return the patient to a state of normal cerebral function, and to establish and maintain a stable cardiac rhythm and normal haemodynamic function. This requires further treatment, tailored to each patient's individual needs. The quality of treatment provided in this post-resuscitation phase – the final ring in the Chain of Survival – significantly influences the patient's ultimate outcome. The post-resuscitation phase starts at the location where ROSC is achieved but, once stabilised, the patient needs transfer to the most appropriate high-care area (e.g. intensive care unit (ICU), coronary care unit (CCU)) for continued monitoring and treatment.

The post-cardiac arrest syndrome

The post-cardiac arrest syndrome comprises:

- post-cardiac arrest brain injury
- post-cardiac arrest myocardial dysfunction
- systemic ischaemia/reperfusion response
- persistent precipitating pathology.

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, hypotension, hypercarbia, hypoxaemia, hyperoxaemia, pyrexia, hypoglycaemia, hyperglycaemia and seizures. Significant myocardial dysfunction is

common after cardiac arrest but typically starts to recover by 2–3 days, although full recovery may take significantly longer. The whole body ischaemia/reperfusion of cardiac arrest activates immune and coagulation pathways contributing to multiple organ failure and increasing the risk of infection. Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion, vasodilation, endothelial injury and abnormalities of the microcirculation.

Continued resuscitation

In the immediate post-resuscitation phase, pending transfer to an appropriate high-care area, treat the patient by following the ABCDE approach described in the post-resuscitation care algorithm (Figure 13.1).

Airway and breathing

Patients who have had a brief period of cardiac arrest and have responded immediately to appropriate treatment (e.g. witnessed ventricular fibrillation (VF) reverting to sinus rhythm after early defibrillation) may achieve an immediate return of normal cerebral function. These patients do not require tracheal intubation and ventilation, but should be given oxygen by face mask to maintain a normal arterial oxygen saturation.

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 (excessively high arterial blood oxygen values) causes oxidative stress and harms post-ischaemic neurones. Observational studies, using data from intensive care registries, have produced conflicting results: some have shown an association between hyperoxaemia and a poor outcome while others have not. A study of air versus supplemental oxygen in ST-elevation myocardial infarction showed that supplemental oxygen therapy increased myocardial injury, recurrent myocardial infarction and major cardiac arrhythmia, and was associated with larger infarct size. Based on this evidence, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry (SpO_2)), 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. Observational studies using cardiac arrest registries document an association between hypoxaemia and poor neurological outcome; therefore, adjust ventilation to achieve normocarbia and monitor this using the end-tidal CO_2 and arterial blood gas values.

Examine the patient's chest and look for symmetrical chest movement. Listen to ensure that the breath sounds are equal on both sides. A tracheal tube that has been inserted too far will tend to go down the right main bronchus and fail to ventilate the left lung. If ribs have been fractured during chest compression (documented in up to 70% of out-of-hospital cardiac arrests receiving CPR) there may be a pneumothorax (reduced or absent breath

sounds) or a flail segment. Listen for evidence of pulmonary oedema or pulmonary aspiration of gastric contents. Insert a gastric tube – this will decompress the stomach following mouth-to-mouth or bag-mask ventilation, prevent splinting of the diaphragm, and enable drainage of gastric contents.

If the intubated patient regains consciousness soon after ROSC, and is cooperative and breathing normally, consider immediate extubation: coughing on the tracheal tube will increase the patient's plasma catecholamine concentrations significantly, which may provoke arrhythmias and/or hypertension. Ensure that a rigid sucker is available. If immediate or early extubation is not possible, sedate the patient to ensure the tracheal tube is tolerated, and provide ventilatory support.

Circulation

Cardiac rhythm and haemodynamic function are likely to be unstable following a cardiac arrest. Continuous monitoring of the ECG is essential. Seek evidence of poor cardiac function. Record the pulse and blood pressure and assess peripheral perfusion: warm, pink digits with a rapid capillary refill usually imply adequate perfusion. Grossly distended neck veins when the patient is semi-upright may indicate right ventricular failure, but in rare cases could indicate pericardial tamponade. Left ventricular failure may be indicated by fine inspiratory crackles heard on auscultation of the lungs, and the production of pink frothy sputum. Try to optimise right and left heart filling pressures: measurement of central venous pressure will guide this. If the facility for direct continuous arterial blood pressure monitoring is available (e.g. in the emergency department) insert an arterial cannula to enable reliable monitoring during transfer. Once in a high-care area, the use of non-invasive cardiac output monitoring devices may be valuable. Infusion of fluids may be required to increase right heart filling pressures or conversely, diuretics and vasodilators may be needed to treat left ventricular failure.

Record a 12-lead ECG as soon as possible. Acute ST-segment elevation or new left bundle branch block in a patient with a typical history of acute myocardial infarction is an indication for treatment to try to re-open an occluded coronary artery (reperfusion therapy) – this is usually achieved by emergency percutaneous coronary intervention (PCI) (Chapter 4). Services should aim to achieve a 'call-to-balloon' time (i.e. time from call for help to attempted re-opening of the culprit artery) of < 120 min whenever possible. If this is not feasible consider fibrinolytic therapy. Cardiopulmonary resuscitation, even if prolonged, is not a contraindication to fibrinolytic therapy.

In post-cardiac arrest patients, chest pain and/or ST elevation are relatively poor predictors of acute coronary occlusion; for this reason primary PCI should be considered and discussed with an interventional cardiologist in all post-cardiac-arrest patients who are suspected of having coronary artery disease as the cause of their arrest, even if

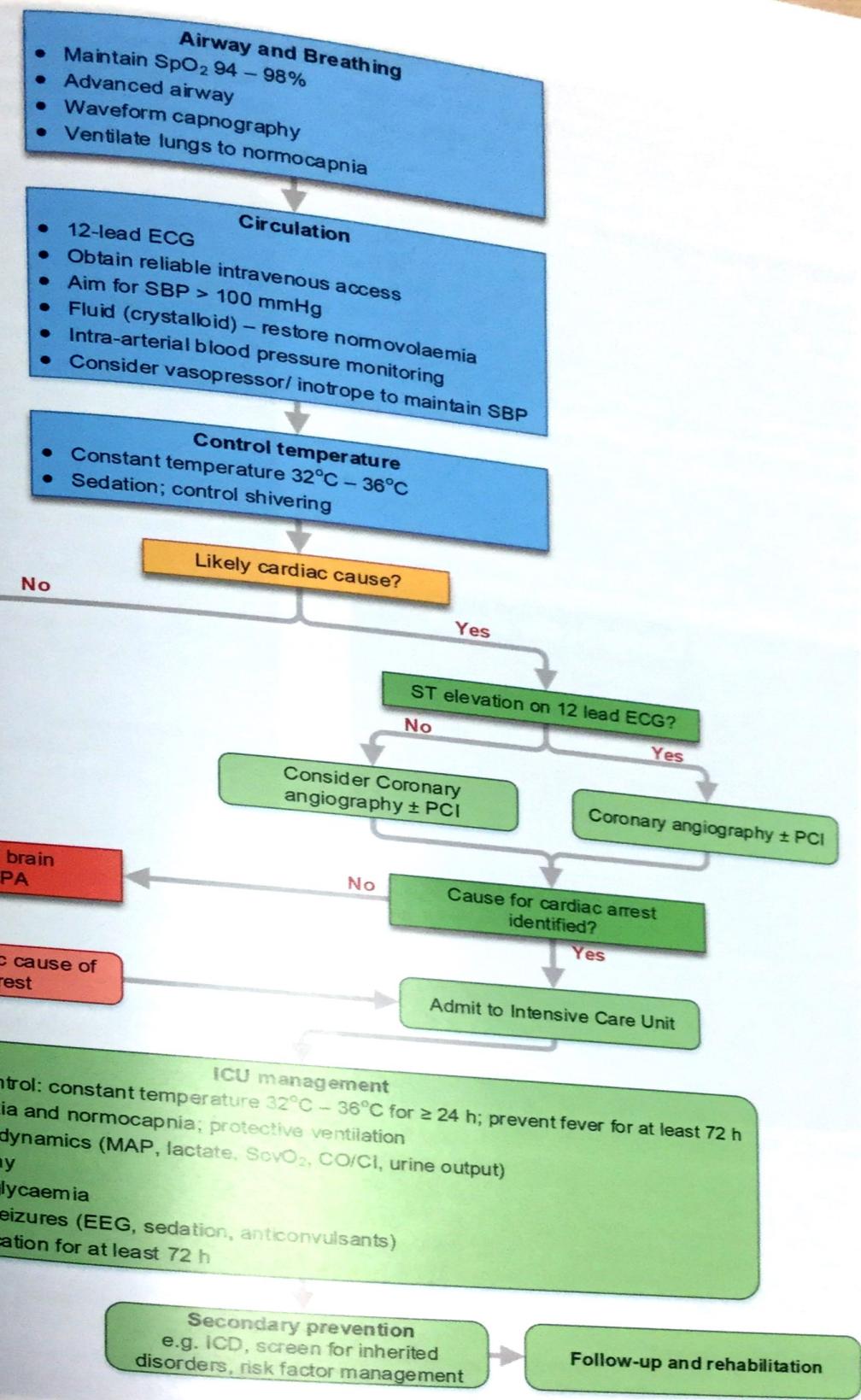


Figure 13.1 Post-resuscitation care algorithm

systolic blood pressure; **PCI** percutaneous coronary intervention; **CTPA** computed tomography pulmonary angiogram; **MAP** mean arterial pressure; **ScvO₂** central venous oxygenation; **CO/CI** cardiac output/cardiac index; **EEG** electroencephalography; **ICD** implanted cardioverter defibrillator.

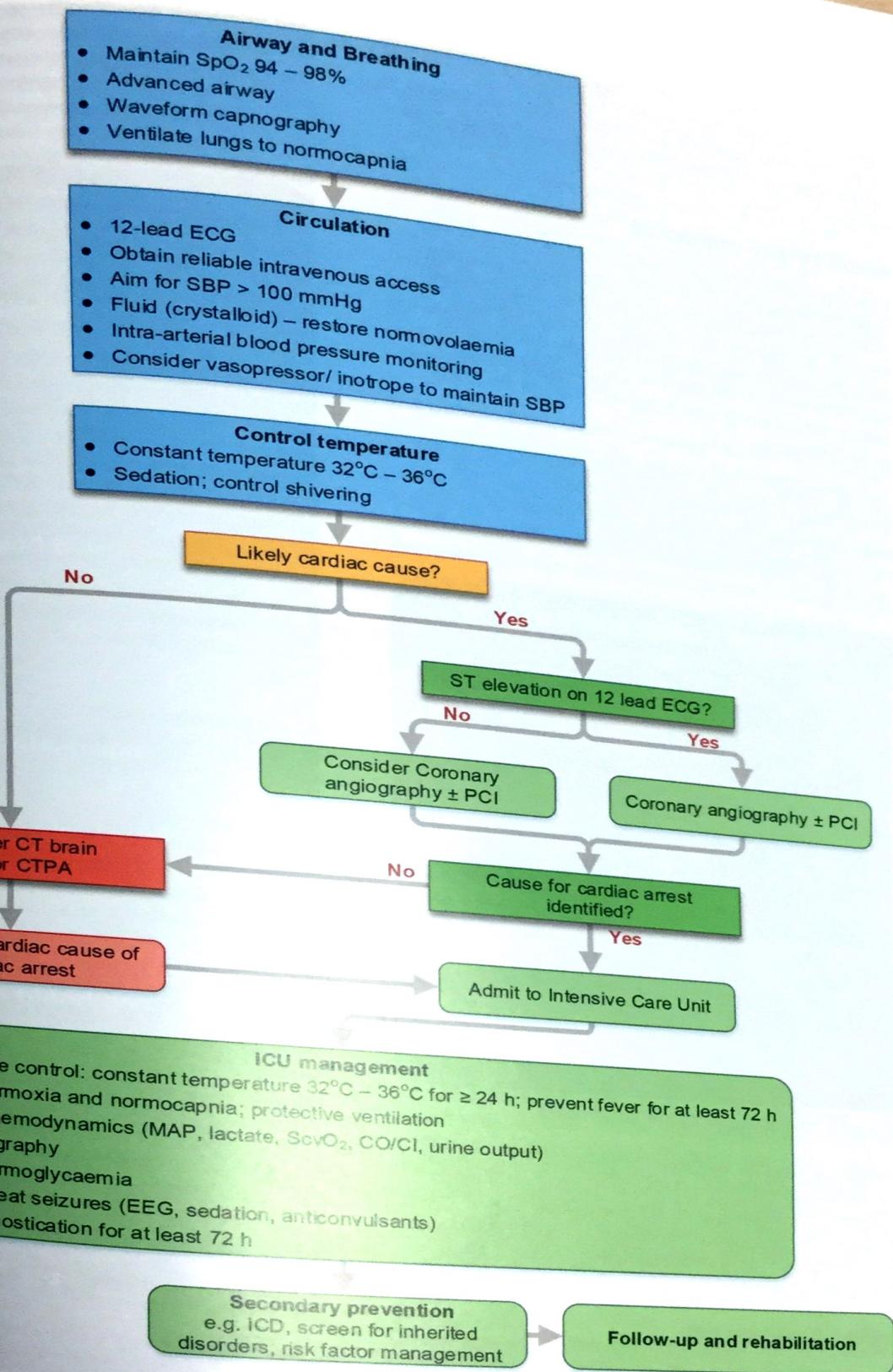


Figure 13.1 Post-resuscitation care algorithm

SBP systolic blood pressure; PCI percutaneous coronary intervention; CTPA computed tomography pulmonary angiogram; MAP mean arterial pressure; ScvO_2 central venous oxygenation; CO/CI cardiac output/cardiac index; EEG electroencephalography; ICD implanted cardioverter defibrillator.

they are sedated and mechanically ventilated. Several studies indicate that the combination of targeted temperature management (see below) and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction.

Disability and exposure

Although cardiac arrest is frequently caused by primary cardiac disease, other precipitating conditions must be excluded, particularly in hospital patients (e.g. massive blood loss, respiratory failure, pulmonary embolism). Assess the other body systems rapidly so that further resuscitation can be targeted at the patient's needs. To examine the patient properly full exposure of the body may be necessary.

Although it may not be of immediate significance to the patient's management, assess neurological function rapidly and record the Glasgow Coma Scale score (Table 13.1). The maximum score possible is 15; the minimum score possible is 3.

Glasgow Coma Scale score

Eye Opening	Spontaneously	4
	To speech	3
	To pain	2
	Nil	1
Verbal	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	Nil	1
Best Motor Response	Obeys commands	6
	Localises	5
	Normal flexion	4
	Abnormal flexion	3
	Extension	2
	Nil	1

Table 13.1 The Glasgow Coma Scale score

Consider the need for targeted temperature management (TTM) in any patient that remains comatose after initial resuscitation from cardiac arrest (see below). When TTM is considered an appropriate treatment, it should start as soon as possible – do not wait until the patient is in the ICU before starting to cool.

Further Assessment

History

Obtain a comprehensive history as quickly as possible. Those involved in caring for the patient immediately before the cardiac arrest may be able to help (e.g. emergency medical personnel, primary/community care physician,

and relatives). Specifically, symptoms of cardiac disease should be sought. Consider other causes of cardiac arrest if there is little to suggest primary cardiac disease (e.g. drug overdose, subarachnoid haemorrhage). Make a note of any delay before the start of resuscitation, and the duration of the resuscitation; this may have prognostic significance, although is generally unreliable and certainly should not be used alone to predict outcome. The patient's baseline physiological reserve (before the cardiac arrest) is one of the most important factors taken into consideration by the ICU team when determining whether prolonged multiple organ support is appropriate.

Monitoring

Continuous monitoring of ECG, arterial and possibly central venous blood pressures, respiratory rate, pulse oximetry, capnography, core temperature and urinary output is essential to detect changes during the period of instability that follows resuscitation from cardiac arrest. Monitor continuously the effects of medical interventions (e.g. assisted ventilation, diuretic therapy).

Investigations

Several physiological variables may be abnormal immediately after a cardiac arrest and urgent biochemical and cardiological investigations should be undertaken (Table 13.2).

Arterial blood gases

Guidance on the interpretation of arterial blood gas values is given in Chapter 15.

Hypoperfusion during the period of cardiac arrest will usually cause a metabolic acidosis. This will cause a low pH (acidaemia), low standard bicarbonate and a base deficit. The rate at which the acidaemia resolves in the post-resuscitation period is an important guide to the adequacy of tissue perfusion. The most effective way of correcting any acidaemia is by addressing the underlying cause. For example, poor peripheral perfusion is treated best by giving fluid and inotropic drugs and not by giving sodium bicarbonate.

Giving bicarbonate may, paradoxically, increase intracellular acidosis, as it is converted to carbon dioxide with the release of hydrogen ions within the cell. Indications for bicarbonate include cardiac arrest associated with hyperkalaemia or tricyclic overdose. Do not give bicarbonate routinely to correct acidaemia after cardiac arrest.

Indications and timing of computed tomography (CT) scanning

Early identification of a respiratory or neurological cause for out-of-hospital cardiac arrest (OHCA) can be achieved by performing a brain and chest CT-scan at hospital admission, before or after coronary angiography. In the absence of signs or symptoms suggesting a neurological or respiratory cause (e.g. headache, seizures or neurological

for neurological causes, shortness of breath or augmented hypoxia in patients suffering from a known worsening respiratory disease) or if there is clinical or evidence of myocardial ischaemia, coronary angiography is undertaken first, followed by CT scan in the absence of causative lesions.

Full blood count

- To exclude anaemia as contributor to myocardial ischaemia and provide baseline values.

Biochemistry

- To assess renal function.
- To assess electrolyte concentrations (K^+ , Mg^{2+} , and Ca^{2+}).
- To ensure normoglycaemia.
- To commence serial cardiac troponin measurements.
- To provide baseline values.

12-lead ECG

- To record cardiac rhythm.
- To look for evidence of acute coronary syndrome.
- To look for evidence of old myocardial infarction.
- To provide a baseline record.

Chest radiograph

- To establish the position of a tracheal tube, a gastric tube, and/or a central venous catheter.
- To check for evidence of pulmonary oedema.
- To check for evidence of pulmonary aspiration.
- To exclude pneumothorax.
- To assess cardiac contour.

Arterial blood gas

- To ensure adequacy of ventilation and oxygenation.
- To ensure correction of acid/base imbalance.

Echocardiography

- To identify contributing causes to cardiac arrest.
- To assess LV and RV structure and function.

CT scan

- To rule out intracranial bleed or stroke as a primary cause of cardiac arrest particularly if there were neurological symptoms before the cardiac arrest.
- To rule out pulmonary embolus or other respiratory cause of cardiac arrest.

Patient transfer

Following the period of initial post-resuscitation care and stabilisation, the patient will need to be transferred to an appropriate critical care environment (e.g. ICU or CCU). The decision to transfer a patient from the place where stabilisation has been achieved should be made only after discussion with senior members of the admitting team. Continue all established monitoring during the transfer and secure all cannulae, catheters, tubes and drains. Make a full re-assessment immediately before the patient is transferred. Ensure that portable suction apparatus, an oxygen supply and a defibrillator/monitor accompany the patient and transfer team.

The transfer team should comprise individuals capable of monitoring the patient and responding appropriately to any change in patient condition, including a further cardiac arrest. The Intensive Care Society (UK) has published guidelines for the transport of the critically ill adult (<http://www.ics.ac.uk/ics-homepage/guidelines-and-standards/>). These outline the requirements for equipment and personnel when transferring critically ill patients.

Optimising organ function

The extent of secondary organ injury after ROSC depends on the ability to minimise the harmful consequences of post-cardiac-arrest syndrome. There are opportunities to limit the insult to organs following cardiac arrest.

Heart and cardiovascular system

Post-resuscitation myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias. Perform early echocardiography in all patients in order to detect and quantify the degree of myocardial dysfunction. Post-resuscitation myocardial dysfunction often requires inotropic support, at least transiently. Although dobutamine is the most established treatment in the setting of myocardial dysfunction, the systematic inflammatory response that occurs frequently in post-cardiac arrest patients causes vasoplegia and severe vasodilation. Thus, noradrenaline, with or without dobutamine, and fluid is usually the most effective treatment. Infusion of relatively large volumes of fluid is tolerated remarkably well by patients with post-cardiac arrest syndrome. If treatment with fluid resuscitation, inotropes and vasoactive drugs is insufficient to support the circulation, consider insertion of an intra aortic balloon pump (IABP). Treatment may be guided by blood pressure, heart rate, urine output, rate of plasma lactate clearance, and central venous oxygen saturation.

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 normal blood pressure, the cause of the arrest and the severity of any myocardial dysfunction. These targets may vary depending on individual physiology and co-morbid status but a

systolic blood pressure of at least 100 mmHg is usually required.

Immediately after a cardiac arrest there is typically a period of hyperkalaemia. Subsequent endogenous catecholamine release and correction of metabolic and respiratory acidosis promotes intracellular transportation of potassium, causing hypokalaemia. Hypokalaemia may predispose to ventricular arrhythmias. Give potassium to maintain the serum potassium concentration between 4.0–4.5 mmol L⁻¹.

Referral for implantable cardioverter defibrillator

Consider the possible requirement for an implantable cardioverter defibrillator (ICD) in any patient who has been resuscitated from cardiac arrest in a shockable rhythm outside the context of proven acute ST segment elevation myocardial infarction. All such patients should be referred before discharge from hospital for assessment by a cardiologist with expertise in heart rhythm disorders (Chapter 4).

Brain: optimising neurological recovery

Cerebral perfusion

Immediately after ROSC there is a period of cerebral hyperaemia. After asphyxial cardiac arrest, brain oedema may occur transiently after ROSC but it is associated only rarely with clinically relevant increases in intracranial pressure. Autoregulation of cerebral blood flow is impaired for some time after cardiac arrest, which means that cerebral perfusion varies with cerebral perfusion pressure instead of being linked to neuronal activity. As discussed above, following ROSC, maintain mean arterial pressure near the patient's normal level.

Sedation

Although it has been common practice to sedate and ventilate patients for at least 24 h after ROSC, there are no data to support a defined period of ventilation, sedation and neuromuscular blockade after cardiac arrest. Patients need to be well sedated during treatment with targeted temperature management, and the duration of sedation and ventilation is therefore influenced by this treatment. There are no data to indicate whether or not the choice of sedation influences outcome, but a combination of opioids and hypnotics is usually used. Short-acting drugs (e.g. propofol, alfentanil, remifentanil) will enable earlier neurological assessment. Adequate sedation will reduce oxygen consumption. During hypothermia, optimal sedation can reduce or prevent shivering, which enables the target temperature to be achieved more rapidly.

Control of seizures

Seizures are common after cardiac arrest and occur in approximately one-third of patients who remain comatose after ROSC. Myoclonus is most common and occurs in 18–25%, the remainder having focal or generalized tonic-

clonic seizures or a combination of seizure types. Use intermittent electroencephalography (EEG) to detect epileptic activity in patients with clinical seizure manifestations. Patients with electrographic status epilepticus may or may not have clinically detectable seizure manifestations that may be masked by sedation. Whether systematic detection and treatment of electrographic epileptic activity improves patient outcome is not known.

Seizures may increase the cerebral metabolic rate and have the potential to exacerbate brain injury caused by cardiac arrest: treat with sodium valproate, levetiracetam, phenytoin, benzodiazepines, propofol, or a barbiturate. Myoclonus can be particularly difficult to treat; phenytoin is often ineffective. Propofol is effective to suppress post-anoxic myoclonus. Clonazepam, sodium valproate and levetiracetam are antmyoclonic drugs that may be effective in post-anoxic myoclonus. After the first event, start maintenance therapy once potential precipitating causes (e.g. intracranial haemorrhage, electrolyte imbalance) are excluded. No studies directly address the use of prophylactic anticonvulsant drugs after cardiac arrest in adults. Routine seizure prophylaxis in post-cardiac arrest patients is not recommended because of the risk of adverse effects and the poor response to anti-epileptic agents among patients with clinical and electrographic seizures.

Myoclonus and electrographic seizure activity, including status epilepticus, are related to a poor prognosis but individual patients may survive with good outcome. Prolonged observation may be necessary after treatment of seizures with sedatives, which will decrease the reliability of a clinical examination.

Glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome. However, severe hypoglycaemia is associated with increased mortality in critically ill patients, and comatose patients are at particular risk from unrecognised hypoglycaemia. Based on the available data and expert consensus, following ROSC, maintain blood glucose at ≤ 10 mmol L⁻¹ and avoid hypoglycaemia (< 4.0 mmol L⁻¹).

Temperature control

Treatment of hyperpyrexia

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

Targeted temperature management

Animal and human data indicate that mild induced hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia. Hypothermia suppresses many of the pathways leading to increased cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate of oxygen (CMRO_2) by about 6% for each 1°C reduction in temperature and this may reduce the release of excitatory amino acids and free radicals. Hypothermia reduces the intracellular consequences of excitotoxin exposure (high calcium and glutamate concentrations) and reduces the inflammatory response associated with post-cardiac arrest syndrome.

Studies of post-cardiac arrest mild induced hypothermia have included only patients in coma. One randomised trial and a pseudo-randomised trial demonstrated improved neurological outcome at hospital discharge or at six 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 $32\text{--}34^\circ\text{C}$ was maintained for 12–24 h. In the Targeted Temperature Management (TTM) trial, 950 all-rhythm OHCA patients were randomised to 36 h of temperature control comprising 28 h at the target temperature followed by rewarming at either 33°C or 36°C . There was no difference in mortality and detailed neurological outcome at six months was also similar. Importantly, patients in both arms of this trial had their temperature well controlled so that fever was prevented in both groups.

The term targeted temperature management or temperature control is now preferred over the previous term therapeutic hypothermia. The Advanced Life Support Task Force of the International Liaison Committee on Resuscitation (ILCOR) made several treatment recommendations on TTM:

• Maintain a constant, target temperature between $32\text{--}36^\circ\text{C}$ for those patients in whom temperature control is used.

• TTM is recommended for adults after out-of-hospital cardiac arrest with an initial shockable rhythm who remain unresponsive after ROSC.

• TTM is suggested for adults after out-of-hospital cardiac arrest with an initial nonshockable rhythm who remain unresponsive after ROSC.

• TTM is suggested for adults after in-hospital cardiac arrest with any initial rhythm who remain unresponsive after ROSC.

• If TTM is used, it is suggested that the duration is at least 24 h.

Following the TTM trial, 36°C is becoming the preferred target temperature for post-cardiac arrest temperature control. This has several advantages compared with a

target temperature of 33°C :

- There is a reduced need for vasopressor support.
- Lactate values are lower (the clinical significance of this is unclear).
- The rewarming phase is shorter.
- There is reduced risk of rebound hyperthermia after rewarming.

How to control temperature

A target temperature of 36°C is not the same as normothermia and in most cases will still require cooling and active temperature management to achieve and maintain this target.

The practical application of TTM is divided into three phases: induction, maintenance and rewarming. External and/or internal cooling techniques can be used to initiate and maintain TTM.

Animal data indicate that earlier cooling after ROSC produces better outcome but this has yet to be demonstrated in humans. External and/or internal cooling techniques can be used to initiate cooling. If a lower target temperature (e.g. 33°C) is chosen, an infusion of 30 mL kg^{-1} of 4°C saline or Hartmann's solution will decrease core temperature by approximately $1.0\text{--}1.5^\circ\text{C}$ and is probably safe in a well monitored environment. Prehospital cooling using this technique is not recommended because there is some evidence of increased risk of pulmonary oedema and re-arrest during transport to hospital.

Methods of inducing and/or maintaining TTM include:

- Simple ice packs and/or wet towels are inexpensive; however, these methods may be more time consuming for nursing staff, may result in greater temperature fluctuations, and do not enable controlled rewarming. Ice cold fluids alone cannot be used to maintain hypothermia, but even the addition of simple ice packs may control the temperature adequately.
- Cooling blankets or pads.
- Water or air circulating blankets.
- Water circulating gel-coated pads.
- Transnasal evaporative cooling – this technique enables cooling before ROSC and is undergoing further investigation in a large multicentre randomised controlled trial.
- Intravascular heat exchanger, placed usually in the femoral or subclavian veins.
- Extracorporeal circulation (e.g. cardiopulmonary bypass, extracorporeal membrane oxygenation (ECMO)).

In most cases, it is easy to cool patients initially after ROSC because the temperature normally decreases within this first hour. Admission temperature after OHCA is usually between 35°C–36°C. If a target temperature of 36°C is chosen allow a slow passive rewarm to 36°C. If a target temperature of 33°C is chosen, initial cooling is facilitated by neuromuscular blockade and sedation, which will prevent shivering. Magnesium sulfate, a naturally occurring NMDA receptor antagonist, that reduces the shivering threshold slightly, can also be given to reduce the shivering threshold.

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. The temperature is typically monitored from a thermistor placed in the bladder and/or oesophagus. As yet, there are no data indicating that any specific cooling technique increases survival when compared with any other cooling technique; however, internal devices enable more precise temperature control compared with external techniques.

Plasma electrolyte concentrations, effective intravascular volume and metabolic rate can change rapidly during rewarming, as they do during cooling. Rebound hyperthermia is associated with worse neurological outcome. Thus, rewarming should be achieved slowly: the optimal rate is not known, but the consensus is currently about 0.25–0.5°C of rewarming per hour.

Physiological effects and complications of hypothermia

The well-recognised physiological effects of hypothermia need to be managed carefully.

- Shivering will increase metabolic and heat production, thus reducing cooling rates.
- Mild hypothermia increases systemic vascular resistance, and can cause arrhythmias (usually bradycardia).
- Hypothermia causes a diuresis and electrolyte abnormalities such as hypophosphataemia, hypokalaemia, hypomagnesaemia and hypocalcaemia.
- Hypothermia decreases insulin sensitivity and insulin secretion, causing hyperglycaemia, which will need treatment with insulin.
- Mild hypothermia impairs coagulation and increases bleeding although this has not been confirmed in many clinical studies.
- Hypothermia can impair the immune system and increase infection rates.
- The clearance of sedative drugs and neuromuscular blockers is reduced by up to 30% at a core temperature of 34°C.

Contraindications to hypothermia

Generally recognised contraindications to therapeutic hypothermia, but which are not applied universally, include: severe systemic infection, established multiple organ failure, and pre-existing medical coagulopathy (fibrinolytic therapy is not a contraindication to therapeutic hypothermia). These contraindications may not apply if a temperature target of 36°C is selected.

Prognostication

The prognostication of comatose post-cardiac arrest patients is very complex, should be undertaken only by experienced clinicians and is outside the scope of the Advanced Life Support course. This brief summary covers only the broad concepts of prognostication; detailed guidance is provided in the Advisory Statement on Neurological Prognostication in Comatose Survivors of Cardiac Arrest, written by members of the European Resuscitation Council ALS Working Group and of the Trauma and Emergency Medicine (TEM) Section of the European Society of Intensive Care Medicine (ESICM).

Hypoxic-ischaemic brain injury is common after resuscitation from cardiac arrest. Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury. Most of these deaths are due to active withdrawal of life sustaining treatment (WLST) based on prognostication of a poor neurological outcome. For this reason, when dealing with patients who are comatose after resuscitation from cardiac arrest minimising the risk of a falsely pessimistic prediction is essential. Ideally, when predicting a poor outcome these tests should have 100% specificity or zero false positive rate (FPR) (i.e. no individuals should have a 'good' long-term outcome if predicted to have a poor outcome). However, most prognostication studies include so few patients that it is very difficult to be completely confident in the results. Moreover, many studies are confounded by self-fulfilling prophecy, which is a bias occurring when the treating physicians are not blinded to the results of the outcome predictor and use it to make a decision on WLST. Finally, both TTM itself and sedatives or neuromuscular blocking drugs used to maintain it may potentially interfere with prognostication tests, especially those based on clinical examination.

Prognostication of the comatose post-cardiac arrest patient should be multimodal, in other words involve multiple types of tests of brain injury, and should be delayed sufficiently to enable full clearance of sedatives and any neurological recovery to occur – in most cases, prognostication is not reliable until after 72 h from cardiac arrest. The tests are categorised:

- clinical examination – GCS score, pupillary response to light, corneal reflex, presence of seizures
- neurophysiological studies – somatosensory evoked potentials (SSEPs) and electroencephalography (EEG)
- biochemical markers – neuron-specific enolase (NSE) is the most commonly used
- imaging studies – brain CT and magnetic resonance imaging (MRI).

Rehabilitation

Although neurological outcome is considered to be good for the majority of cardiac arrest survivors, cognitive and emotional problems and fatigue are common. Long-term cognitive impairments are present in half of survivors. Memory is most frequently affected, followed by problems in attention and executive functioning (planning and organisation). The cognitive impairments can be severe, but are mostly mild. These patients may benefit from a formal program of rehabilitation but such an approach is currently rare in the UK.

Organ donation

Post-cardiac arrest patients who do not survive represent an opportunity to increase the number of potential organ donors, either after brain death or as non-heart-beating donors. Non-randomised studies have shown that graft survival at one year is similar from donors who have had CPR compared with donors who have not had CPR.

Cardiac arrest centres

There is wide variation in patient survival rates among hospitals caring for patients after resuscitation from cardiac arrest. There is indirect evidence that regional cardiac systems of care improve outcome after ST elevation myocardial infarction (STEMI). Many studies have reported an association between survival to hospital discharge and transport to a cardiac arrest centre but there is inconsistency in the hospital factors that are most related to patient outcome. There is also inconsistency in the services that together define a cardiac arrest centre. Most experts agree that such a centre must have a cardiac catheterisation laboratory that is immediately accessible 24/7 and the facilities to provide intensive care with targeted temperature management. The availability of a neurology service that can provide neuroelectrophysiological monitoring (electroencephalography (EEG)) and investigations (e.g. EEG and somatosensory evoked potentials (SSEPs)) is also essential. Despite the lack of high quality data to support implementation of cardiac arrest centres, it seems likely that post-cardiac arrest care will be increasingly regionalised.

Summary learning

- After cardiac arrest, return of spontaneous circulation is just the first stage in a continuum of resuscitation.
- The quality of post-resuscitation care will influence significantly the patient's final outcome.
- These patients require appropriate monitoring, safe transfer to a critical care environment, and continued organ support.
- The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and persistence of precipitating pathology.
- Prognosticating the final neurological outcome for those patients remaining comatose after cardiopulmonary resuscitation is complex.

My key take-home messages from this chapter

Prehospital cardiac arrest

Contents

- Resuscitation at the scene versus transfer to hospital
- Team approach to prehospital cardiac arrest
- Prehospital airway management
- Circulatory support
- Recognition of life extinct
- Prehospital post-resuscitation care
- Team handover and crew debriefing
- Audit

Learning outcomes

To enable you to:

- Apply the team approach to prehospital resuscitation
- Understand the optimal approach to managing the airway, breathing and circulation in the prehospital setting
- Consider the principles of recognition of life extinct (ROLE)
- Consider the main features of a team handover and crew debriefing

Introduction

The principles of basic and advanced life support are the same for the prehospital setting as they are for in-hospital cardiac arrest, but out-of-hospital can be constrained by a combination of factors including a lack of trained staff, the environment (both on scene or during transport), equipment and drugs and physical access to the patient. The actions described in the Advanced Life Support (ALS) algorithm can also be applied to the prehospital setting (Chapter 6). Resuscitation in special circumstances (e.g. drowning, trauma) is addressed in Chapter 12. Most prehospital services will have standard operating procedures (SOPs) that cover many of the topics discussed below.

Survival after out-of-hospital cardiac arrest (OHCA) relies on early recognition and CPR from bystanders. When an ambulance is called, ambulance service telephone dispatchers will provide assistance to bystanders to help identify cardiac arrest and start CPR before the ambulance arrives. Automated external defibrillators (AEDs) are available in many public places (e.g. train stations, shopping centres). An AED should be used as soon as possible. In some areas the ambulance dispatcher will be able to tell bystanders the location of the nearest AED. Resuscitation by bystanders with CPR and an AED before the ambulance arrives offers the best chance of survival after OHCA.

Resuscitation at scene versus transfer to hospital

Treatment on scene aims to identify and treat immediate life-threatening problems and achieve return of spontaneous circulation (ROSC) in those who have had a cardiac arrest. Remain on scene to achieve ROSC unless reversible causes that cannot be dealt with on

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Resuscitation at scene versus transfer to hospital

Treatment on scene aims to identify and treat immediate life-threatening problems and achieve return of spontaneous circulation (ROSC) in those who have had a cardiac arrest. To remain on scene to achieve ROSC unless reversible causes that cannot be dealt with on

scene are identified and can potentially be dealt with in hospital (e.g. emergency department, cardiac catheterisation laboratory, operating theatre). Ideally the decision to transport a patient to hospital should be reached as soon as the need for skills/interventions not available on scene becomes apparent. The deteriorating patient that cannot be stabilised on scene requires early recognition and transport to a suitable hospital (e.g. patient with penetrating trauma and haemodynamic compromise).

Team approach

Prehospital cardiac arrest requires a system to be in place to achieve the best possible chance of survival. The system not only requires technical skills, but also requires non-technical skills:

- teamwork
- situational awareness
- leadership
- decision making.

Appoint an experienced team leader who should assign each member of the team a specific role. Ensure that there is 360° access to the patient (Figure 14.1).

• Position 1

Airway (at head of patient) - the person must be trained and equipped to provide the full range of airway skills.

• Position 2

High quality chest compressions and defibrillation if needed - at patient's left side. Be prepared to alternate with operator at position 3 to avoid fatigue.

• Position 3

High quality chest compressions and access to the circulation (intravenous (IV), intraosseous (IO)) - at patient's right side.

• Position 4

Team leader - stand back and oversee the resuscitation attempt, only becoming involved if required. The team leader should have an awareness of the whole incident and ensure high quality resuscitation is maintained and appropriate decisions made.

Prehospital airway management

The aim is to maintain a patent airway enabling adequate oxygenation and ventilation of the lungs. Although tracheal intubation has been considered the gold standard in airway management, lack of supportive outcome data, the practical challenges of implementing its use in the prehospital setting and the risks of very serious harm and death (e.g. prolonged desaturation, unrecognised oesophageal intubation or bronchial intubation) have questioned the role of intubation. In particular, do not attempt tracheal intubation in children unless you are expert in the procedure in this age group, as simpler and safer techniques (e.g. bag-mask ventilation) may be just as effective at maintaining adequate oxygenation and ventilation.

Stepwise approach to airway
In practice, several airway interventions (e.g. interventions, mouth-to-mouth, bag-mask, stepwise manner during CPR and after ROSC) should use the skills in which they are progressive and maintaining an open airway. During airway care provides a very important to maintain high quality c with minimal interruption for airway inter at risk of cervical spine injury. Simple airway assist with opening the airway at the unresponsive patients, particularly those where ventilation is required, a supraglottic insertion may be most appropriate.

Tracheal intubation

The tracheal tube is the most challenging devices to insert successfully and require training and ongoing practice. There is outcome from out-of-hospital cardiac tracheal intubation and several studies that simpler airway techniques during SGA result in at least as good, if not better outcomes.

Prehospital tracheal intubation considerations

Views of the larynx are often suboptimal, restricted neck movement/jaw opening and other airway secretions, poor patient position, difficult access to the patient. The following achieve safe and successful prehospi

- Ensure 360° access around the patient
- Ensure all the necessary equipment is available patient as a 'kit dump' (Figure 14.2)



Figure 14.2 An example of a prehospital kit dump.

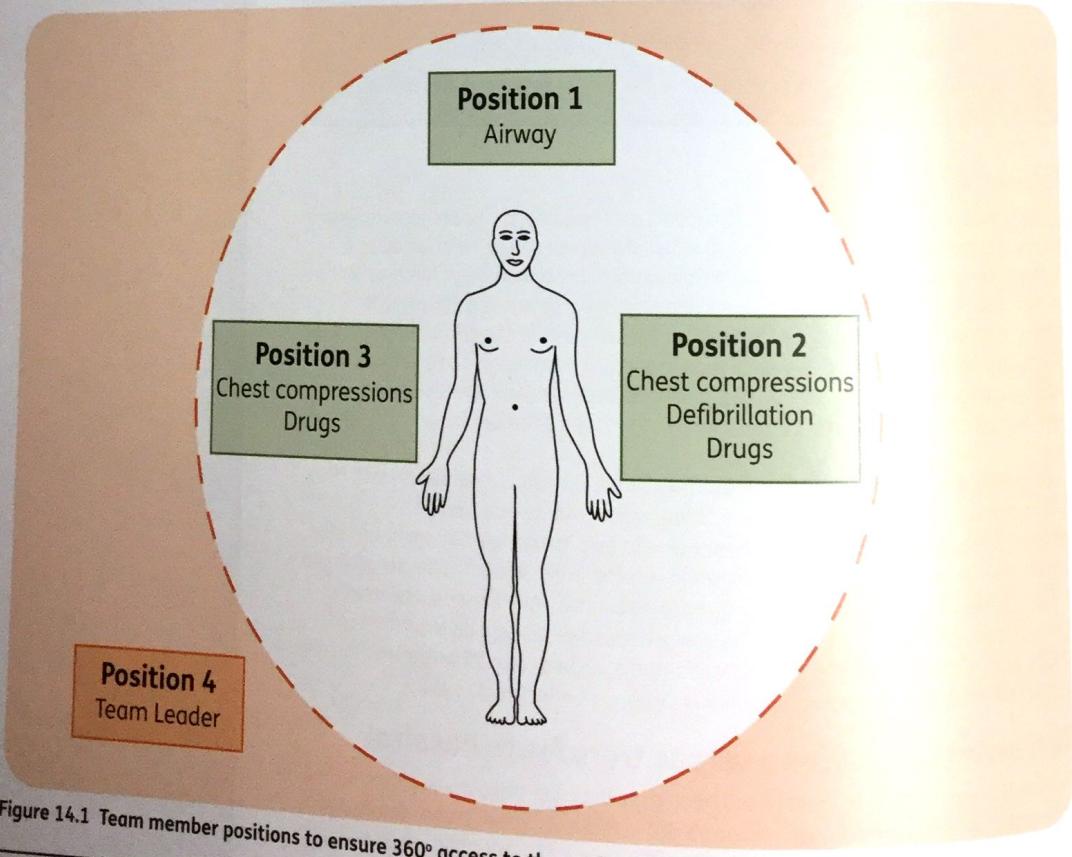


Figure 14.1 Team member positions to ensure 360° access to the patient

ALS

Stepwise approach to airway care

In practice, several airway interventions (e.g. no airway interventions, mouth-to-mouth, bag-mask, supraglottic airway, tracheal intubation) and devices are used in a stepwise manner during CPR and after ROSC. Rescuers should use the skills in which they are proficient. Stepwise airway care provides a progressive approach to obtaining and maintaining an open airway. During a cardiac arrest, it is very important to maintain high quality chest compressions with minimal interruption for airway interventions. Start with a head tilt/chin lift or jaw thrust, using the latter in patients at risk of cervical spine injury. Simple airway adjuncts (e.g. oropharyngeal airway or nasopharyngeal airway (Chapter 7)) can assist with opening the airway at this stage. In unresponsive patients, particularly those in respiratory arrest or where ventilation is required, a supraglottic airway (SGA) insertion may be most appropriate.

Tracheal intubation

The tracheal tube is the most challenging of all airway devices to insert successfully and requires adequate initial training and ongoing practice. There is no evidence that outcome from out-of-hospital cardiac arrest is improved by tracheal intubation and several studies have suggested that simpler airway techniques during CPR (i.e. bag-mask, SGA) result in at least as good, if not better, patient outcomes.

Prehospital tracheal intubation – practical considerations

Views of the larynx are often suboptimal because of restricted neck movement/jaw opening, poor lighting, vomit and other airway secretions, poor patient positioning, and difficult access to the patient. The following can help achieve safe and successful prehospital tracheal intubation.

- Ensure 360° access around the patient where possible.
- Ensure all the necessary equipment is placed by the patient as a 'kit dump' (Figure 14.2).



Figure 14.2 An example of a prehospital 'kit dump'.

- Use a checklist to check all equipment is available, allocate roles and make a rescue plan if initial attempts at intubation or ventilation fail.
- Patient positioning: Whenever possible, place the patient supine on an ambulance trolley (preferably one that tips head-down in case of vomiting). Intubation is easier when the trolley is at knee level of the airway person.
- Do not routinely use cricoid pressure for tracheal intubation during CPR. Many patients have already aspirated and cricoid pressure can make tracheal intubation more difficult.
- Use a bougie, if necessary, over which a tracheal tube can be rail-roaded. A bougie should always be available.
- Choose the correct-sized laryngoscope blade; generally a size 4 MAC blade can be used for most adults. Videolaryngoscopy can help with difficult intubation – a variety of devices are available and require specific training in their use. A good view of the cords on the video screen does not mean tube placement will be easy.
- Secure the tracheal tube immediately after insertion (adult males – 22–23 cm, adult females 21–22 cm), using a tie, commercial clamp or tape. A tie around the patient's head or neck can decrease venous drainage of blood from the head and brain and increase the intracranial pressure – do not over tighten the tie and consider using tape in patients where intracranial pressure may be raised (e.g. head injury). The same is true for securing SGAs. When moving the patient, hold the tracheal tube, even when it is secured. Regularly check the tracheal tube length during transit to ensure that it has not dislodged.
- During CPR aim to minimise the interruption to chest compressions for tube placement to less than 5 s – this means doing the initial laryngoscopy whilst compressions are ongoing, a brief pause to optimise the view and pass the tube and then restarting compressions. Plan actions and pauses whilst compressions are ongoing.

Tracheal intubation – complications

Complications associated with tracheal intubation include:

1. Hypoxaemia – oxygenate the lungs between intubation attempts.
2. Unrecognised oesophageal intubation – use waveform capnography to exclude oesophageal intubation (Chapter 7).
3. Endobronchial intubation – insert the tracheal tube to no more than the appropriate length, listen to breath sounds in both axillae, and ensure both lungs are ventilated. Monitor the position of the tube during transit.

Breathing

Initial management

Having opened the airway, the first priority is to rapidly assess, identify and treat reversible causes of impaired breathing. Assessment of breathing follows the look, listen and feel approach. In sick patients, expose the chest sufficiently to look for signs of injury, chest wall expansion and asymmetry between each side. Listening with a stethoscope is particularly difficult in the noisy prehospital setting. Feeling for equal chest wall expansion, bony injury or surgical emphysema is useful; the latter in most cases indicates a pneumothorax or rupture of a large airway is likely. It can be difficult to measure oxygen saturation reliably with a pulse oximeter when patients are cold, or have a low blood pressure. If a pulse oximeter placed on the finger fails to read adequately, other sites that may work include the toes, nose, ear lobes, tongue or lip.

Gastric distension

Positive pressure ventilation using a bag-mask can lead to gastric insufflation, respiratory compromise, regurgitation of stomach contents and aspiration. Gentle ventilation with a bag-mask (sufficient to just enable the chest wall to rise) minimises airway pressure and reduces the fraction of air forced down the oesophagus and into the stomach. Low tidal volumes also minimise adverse haemodynamic effects on the circulation. SGAs minimise the risk of gastric distension and should be considered in preference to a bag-mask.

Tension pneumothorax

A tension pneumothorax requires immediate treatment by decompression (Chapter 12). The aim of prehospital management is to relieve the tension and convert a tension pneumothorax to a simple pneumothorax; the latter is generally not life-threatening and will enable transfer to hospital for definitive care. In patients very close to a destination hospital, needle decompression can be sufficient to transport the patient before the tension pneumothorax recurs. In patients with longer transit times or requiring air transfer, who are breathing spontaneously, a chest drain with a one-way valve will need to be inserted, usually in the 5th intercostal space in the midaxillary line. In patients receiving positive pressure ventilation, a thoracostomy (5th intercostal space in the midaxillary line) is preferable to a chest drain as it is less invasive, a quicker procedure and avoids complications associated with the chest drain.

Oxygenation and ventilation

Give high-flow oxygen to correct and avoid hypoxia. As soon as the oxygen saturation can be reliably measured, adjust the inspired oxygen concentration to achieve a target oxygen saturation (e.g. 94–98% after ROSC from cardiac arrest – Chapter 13).

Hyperventilation (from excessive tidal volumes, respiratory rate, or both) is common and can cause haemodynamic instability. Hypocapnia induced by hyperventilation can cause excessive cerebral vasoconstriction and ischaemia. The use of mechanical ventilators may prevent hyperventilation, as well as freeing a rescuer for other tasks.

Circulation

Defibrillation

Defibrillation is one of the few interventions that improves outcome from sudden cardiac arrest, but its application is time critical, with mortality increasing 10% for every minute's delay in defibrillation. Attach the defibrillation electrodes and deliver the first shock as soon as possible. When attending as a solo responder, immediate assessment of the rhythm and defibrillation when indicated, should take precedence over airway or breathing interventions (Chapter 9).

A few patients who present in VF are difficult to cardiovert, (i.e. they either remain in VF or keep developing recurrent VF). In such cases, consider a change in electrode position, (e.g. move from the standard anterior-lateral to an anterior-posterior or bi-axillary position). Ventricular fibrillation recurs in 50% of patients within 2 min of successful termination and in 75% of patients during the entire cardiac arrest. Give amiodarone (300 mg IV) after three defibrillation attempts, irrespective of whether those episodes are concurrent or separate.

Defibrillating and performing effective chest compressions whilst moving these patients to the ambulance and transporting to hospital is difficult. If necessary, put the patient down or stop the ambulance while further therapy is given.

Vascular access

Obtain IV access if this has not been done already. Peripheral venous cannulation is quick and relatively easy and is preferable to IO access as the initial cannulation choice.

If IV access is considered too difficult or cannot be established, consider gaining IO access (Chapter 6). Some prehospital services recommend a maximum of two attempts at IV access or a maximum of 2 min for attempts before considering IO access. During CPR effective chest compressions and early defibrillation is the first priority and the role of drugs is uncertain, so do not delay other more useful interventions for vascular access. Tibial and humeral sites are readily accessible. Follow any drug injection (IV or IO) by a flush of at least 20 mL of fluid to facilitate drug delivery to the central circulation.

Chest compressions

Start chest compressions as soon as cardiac arrest is confirmed. Whenever possible, perform CPR on a firm surface because soft surfaces (e.g. mattress) make it difficult to estimate how deep the chest is being compressed and can lead to under-compression.

CPR monitoring, feedback and prompt devices

Some defibrillators and specific feedback/prompt devices monitor CPR quality (e.g. compression rate, depth, leaning, ventilation rate) during CPR. These devices give the rescuer real-time objective feedback on the quality of CPR. If these devices are used they should be part of an overall quality improvement programme that monitors CPR quality and provides rescuers with post-event feedback on performance.

Mechanical chest compression devices

The routine use of automated mechanical chest compression devices is not superior to manual chest compressions. However, automated mechanical chest compression devices can give high quality manual chest compressions in situations where sustained high quality manual chest compressions are impractical or compromise rescuer safety (e.g. during transport, enclosed spaces). Mechanical devices should only be used within a structured, monitored programme, which includes comprehensive competency-based training and regular opportunities to refresh skills. Improper use of mechanical compression devices can delay life-saving interventions such as defibrillation, and cause harmful pauses in chest compressions when switching from manual to mechanical compressions.

Examples of scenarios where mechanical chest compressions are suitable include transport of patients to hospital for correction of a potentially reversible cause:

- Patients with VF or pulseless VT (pVT) after a myocardial infarction, refractory to 5 shocks (i.e. persistence or recurrence of fibrillation) in whom a decision is made to transport whilst the arrhythmia persists, and enable coronary angiography and percutaneous coronary angiography.
- The patient with hypothermia in whom prolonged resuscitation is considered.

Continuous waveform capnography as a guide to circulatory status

Continuous waveform capnography is a useful guide for managing the circulation and should be used for all patients immediately following tracheal intubation, during the resuscitation attempt and following ROSC. It has several roles in addition to monitoring tracheal tube placement and ventilation rate (Chapter 6):

- early indication of ROSC
- monitoring the quality of CPR
- helping with prognostication.

Recognition of life extinct

The Association of Ambulance Services Chief Executives (through the Joint Royal Colleges Ambulance Liaison Committee, UK Ambulance Service - Clinical Practice Guidelines), and the Resuscitation Council (UK) have both provided clinical guidelines around both the futility of starting resuscitation in certain circumstances and when it may be appropriate to stop a resuscitation attempt. In the presence of cardiorespiratory arrest, and in the absence of a documented do not attempt cardiopulmonary resuscitation (DNACPR) decision or refusal of CPR in an Advance Decision to Refuse Treatment (ADRT) ('living will'), ambulance clinicians should start CPR unless the patient has a condition unequivocally associated with death.

When not to start resuscitation

The UK Ambulance Service Clinical Practice Guidelines list the following situations as unequivocally associated with death:

- decapitation
- massive cranial and cerebral disruption
- hemicorporectomy (or similar massive injury)
- incineration (> 95% full thickness burns)
- decomposition/putrefaction
- rigor mortis and hypostasis.

Resuscitation should also not be started in patients where a valid DNACPR decision has been made (Chapter 16). These decisions are difficult to confirm in an out-of-hospital setting and it may be appropriate to start and continue resuscitation whilst the facts are clarified.

There are situations where an experienced practitioner clearly identifies that the patient is in the final stages of a terminal illness and resuscitation is unlikely to be appropriate or successful. In these circumstances the practitioner must be able to explain the grounds for the decision not to start resuscitation, and these must be documented.

When to stop resuscitation

In patients where ROSC is not achieved on scene, despite appropriate ALS and treatment of potentially reversible causes, little is to be gained from transporting these patients to hospital.

The asystolic patient

The UK Ambulance Service Clinical Practice Guidelines (2013) state that where a patient has had persistent asystole for greater than 20 min, despite ALS and where drowning, hypothermia, poisoning or overdose and pregnancy have been excluded, it is appropriate to stop resuscitation.

Pulseless electrical activity

The decision about when to stop resuscitation when pulseless electrical activity (PEA) persists is less clear and is not currently within the UK Ambulance Service Clinical Practice Guidelines (2013). There is limited evidence to support when one should stop resuscitation in a PEA cardiac arrest. When making this decision take into consideration the length of time in cardiac arrest before CPR was started, the absence of treatable reversible causes, and patient co-morbidities.

Ventricular fibrillation

Where practical, patients with persistent VF or pulseless VT should be taken to a cardiac arrest centre ('heart attack centre') with ongoing CPR, because further treatment (e.g. coronary angiography and primary percutaneous intervention (PCI)) may be successful.

Communication with relatives

Although priorities during the management of a cardiac arrest are focused on the patient, it is important to consider the relatives who are often present. Relatives are also patients and treating them sensitively can help with the mourning process if resuscitation is unsuccessful (Chapter 17). Allow relatives to be present during the resuscitation attempt, providing they do not interfere with clinical care.

Prehospital post-resuscitation care

General principles

The focus of post-resuscitation care is directed at optimising perfusion of the brain and heart (Chapter 13). Use the ABCDE approach (Chapter 3).

Monitor the patient with:

- pulse oximetry
- continuous waveform capnography to monitor end-tidal CO₂
- blood pressure
- ECG
- 12-lead ECG where appropriate
- blood glucose measurement
- temperature.

Haemodynamic management

Following ROSC, hypotension and arrhythmias are common. Aim for a systolic blood pressure of 100 mmHg. Treat hypotension with a 250 mL IV/IO bolus of 0.9% saline, repeated as necessary. Treat arrhythmias according to the peri-arrest guidelines (Chapter 11). In the event of severe hemodynamic instability unresponsive to atropine and/or fluids and/or pacing, consider using inotropic support. Boluses of adrenaline 0.05–0.1 mg IV/IO can be titrated against blood pressure. This is undertaken only in the setting of robust clinical governance with medical support (e.g. by telephone). If the 12-lead ECG shows an acute STEMI, transport the patient to a hospital capable of delivering primary PCI, regardless of the patient's conscious level.

Ventilation

Change from hand ventilation to mechanical ventilation of the patient's lungs, ensuring that there is adequate rise and fall of the chest. Waveform capnography is mandatory for intubated patients.

Aim to maintain the SpO₂ at 94–98% and the end-tidal CO₂ at 4.6–6 kPa (35–45 mmHg). End-tidal CO₂ values may be lower in patients with a poor cardiac output.

Targeted temperature management

The benefits of therapeutic hypothermia for post-arrest patients are unclear, but immediate temperature management aims to achieve a core temperature no higher than 36.0°C. Passive cooling is appropriate for these patients. Do not cover the patient in blankets and maintain ambulance temperature no higher than ambient. Do not use rapid high-volume (2 L) cold intravenous fluid infusion immediately after ROSC for rapid cooling – it is associated with an increased incidence of re-arrest and pulmonary oedema. Cooling with cold IV fluids must only be done with close monitoring.

The combative patient

Following ROSC, patients may be cerebrally irritated and combative. Exclude hypoglycaemia. These patients can benefit from formal anaesthetic management, but incremental doses of IV diazepam or midazolam may be indicated with appropriate on-line medical support if advanced medical care is not available on scene. If sedation is used ensure that oxygenation and ventilation are adequate.

Transporting the patient to hospital

Attempts to provide resuscitation whilst moving the patient to the ambulance and transporting to hospital are likely to be sub-optimal. Aim to achieve ROSC on scene. In patients who achieve ROSC, and in those who have not achieved ROSC within 20 min but in whom resuscitation is being continued, aim to leave the scene as soon as possible. When the decision is made to transport the patient with ROSC, maintain the patient in a position most likely to optimise cerebral perfusion:

- supine
- feet-first if coming down stairs
- slightly head-up (30°) once in the vehicle.

Assess using the ABCDE approach before leaving the scene and transport the patient to the nearest appropriate hospital.

Patient destination

Cardiac arrest centres

Ambulance services should transfer patients following cardiac arrest with ST elevation on their ECG to a regional 'heart attack centre' so they can undergo urgent coronary angiography and PCI if indicated. This should be the case irrespective of the patient's conscious level. Other patients who are likely to have a primary cardiac cause for their cardiac arrest should also be considered for direct transfer to a regional heart attack centre.

Pre-alerting

Alert the receiving hospital as soon as possible whenever transporting a patient in cardiac arrest or once ROSC has been achieved. When pre-alerting, ensure that you identify the hospital, department and name of the person receiving your message.

Use a structured method to give the key information. Typically, this is done using 'ATMIST' (Table 14.1).

ATMIST	
MEDICAL	TRAUMA
Age	Age
Time of onset	Time of incident
Medical complaint/history	Mechanism of injury
Investigations (brief examination of findings)	Injuries (top to toe)
Vital Signs (first set and significant changes)	Vital Signs (first set and significant changes)
Treatment, including ETA and any specialist resources needed on arrival	Treatment, including ETA and any specialist resources needed on arrival

ETA – expected time of arrival

Table 14.1 A structured approach to providing key information – the ATMIST mnemonic

Most patients will be transported to an emergency department resuscitation room where a handover from the ambulance to hospital clinician will take place. Ensure that the receiving hospital is aware of the patient's conscious level – heart attack centres often do not have anaesthetic support as part of their receiving team and will need to summon such personnel if the patient is unconscious.

Team handover and debriefing

Handover

Use a handover template to transfer information rapidly during resuscitation. A commonly used EMS handover template is ATMIST (**A**ge, **N**ame, **T**ime of onset, **M**edical history, **I**nvestigations, **S**igns, **T**reatment – see above). Speak loudly and clearly using the template headings; pauses will enable important points to be understood and assimilated. Be concise and try and limit the handover. Adjuncts to handover such as pictures from the scene can be extremely useful. If you have taken them show them to the hospital team leader.

Debriefing

Following an out-of-hospital resuscitation attempt, an immediate debrief with the attending EMS team is valuable. This 'Hot' debrief can provide a valuable learning

opportunity and also ensure the welfare of the prehospital resuscitation team. Debriefing in a structured manner is particularly useful to explore non-technical elements of resuscitation such as communication, leadership and team working. Ideally, the debrief is led by a person trained and capable of leading the session in a non-threatening, constructive manner, and should include review of records and data from the resuscitation attempt.

Audit

Quality of CPR/defibrillator downloads

Modern defibrillators include the ability to obtain downloads of data related to the quality of CPR. The systematic analysis of such data and provision of feedback to ambulance clinicians is recommended and has been shown to improve the quality of CPR.

Out-of-Hospital Cardiac Arrest Outcomes (OHCAO) database

Ongoing, systematic collection and analysis of data about out-of-hospital cardiac arrest and bystander CPR is essential to the planning, implementation, and evaluation of effective CPR programs. The British Heart Foundation and Resuscitation Council (UK) established a national OHCAO registry in partnership with the National Association of Ambulance Medical Directors and University of Warwick. The OHCAO registry (www.warwick.ac.uk/go/ohcao) collects process and outcome information about patients who are treated by ambulance services for cardiac arrest and which is based on the international Utstein template. The registry will provide a tool to support local quality improvement initiatives and will facilitate measuring the impact of resuscitation interventions.

Blood gas analysis and pulse oximetry

Chapter
15

Contents

- The components of an arterial blood gas result
- Interpreting arterial blood gas values
- Example case studies
- Venous blood gases
- Pulse oximetry
- Targeted oxygen therapy

Learning outcomes

To enable you to:

- Understand the terms used to describe the results of arterial blood gas analysis
- Know how respiration and metabolism are linked
- Use the 5-step approach to analyse arterial blood gas results
- Understand the principles of pulse oximetry
- Understand how to use oxygen safely and effectively

Introduction to blood gas analysis

Interpreting the results of an arterial blood sample to determine a patient's acid-base status and respiratory gas exchange is a key component in the management of any ill patient. Although it is tempting to try and analyse the numerical data in isolation, it is essential to have a system to ensure that nothing is overlooked or misinterpreted. The first question you should always ask is: "how is the patient?" You must also be aware of their past medical history, current drug regimen and current oxygen therapy.

Analysing blood gas results

There are usually five key pieces of information contained in the results of an arterial blood sample:

- pH
- PaCO_2 (partial pressure of carbon dioxide in arterial blood)
- Bicarbonate
- Base excess
- PaO_2 (partial pressure of oxygen in arterial blood).

To interpret these results, we first need to understand what each means. Normal ranges are given in the text; however, these will vary slightly between institutions.

pH

The acidity or alkalinity of the blood (or any solution) is determined by the concentration of hydrogen ions (H^+); the greater the concentration, the more acid the solution. In the body, the concentration of hydrogen ions is extremely low, normally around 40 **nanomoles per litre** (nmol L^{-1}).

- A mole is the molecular weight of a substance in grams.
- A mole of hydrogen (H_2) is 2 g.
- A nanomole is 1 billionth of a mole (0.000000002 g).
- In arterial blood, the $[\text{H}^+]$ is 40 nmol L^{-1} or $0.00000080 \text{ g L}^{-1}$.
- Sodium (Na^+) is present in a concentration of 135 **millimoles per litre** (mmol L^{-1}) (i.e. 3 million times greater).

In order to make dealing with such small concentrations of hydrogen ions easier, we use the pH scale; this is a logarithmic scale expressing the hydrogen ion concentration between 1 and 14.

The pH of a normal arterial blood sample lies between 7.35–7.45, or $[\text{H}^+]$ 44–36 nmol L^{-1} .

There are two key points to remember about the pH scale:

1. The numerical value of pH changes inversely with hydrogen ion concentration; a **decrease in blood pH below 7.35** indicates an increase in $[\text{H}^+]$ above normal, a condition referred to as an **acidaemia**. Conversely, an **increase in blood pH above 7.45** indicates a reduction in $[\text{H}^+]$ below normal, a condition referred to as an **alkalaemia**. Clinicians often use the terms acidosis and alkalosis respectively to describe these situations, however these terms refer to the processes that lead to the changes in pH, and it is in this context that they will be used in this manual.
2. Small changes in pH represent big changes in hydrogen ion $[\text{H}^+]$ concentration; a pH change from 7.4 to 7.1 means that the $[\text{H}^+]$ has increased from 40 nmol L^{-1} to 80 nmol L^{-1} (i.e. it has doubled for a pH change of 0.3).

Many of the complex reactions within cells are controlled by enzymes that function only within a very narrow pH range; hence, normal pH is controlled tightly between 7.35–7.45. Each day, during normal activity, we produce massive amounts of hydrogen ions (approximately 14 500 000 000 nmol), which if unchecked would cause a substantial decrease in pH (acidaemia) before they could be excreted. To prevent this happening the body has a series of substances known as buffers that take-up hydrogen ions and thereby prevent the development of an acidaemia. The major intracellular buffers are proteins, phosphate and haemoglobin (within red blood cells) and the extracellular buffers are plasma proteins and bicarbonate (see below).

The buffering system is only a temporary solution to the production of acids and once they are all consumed acids will start to accumulate. A system is therefore required to eliminate the acids and thereby regenerate the buffers. This is achieved by both the lungs and the kidneys.

Partial pressure

We normally use percentages to describe the composition of a mixture of gases, for example air is: 21% oxygen, 78% nitrogen, 0.04% carbon dioxide. A better indication of the number of different molecules of gases in a mixture is its partial pressure, measured in kiloPascals (kPa) or millimetres of mercury (mmHg). The partial pressure is the contribution each gas in a mixture makes to the total pressure. The importance of using this measure is best demonstrated by the fact that if we double the total pressure of a mixture, the partial pressures of the constituents are doubled, but the percentages remain the same. We breathe gases at atmospheric pressure, which is very close to a pressure of 101 kPa or 760 mmHg (1 kPa = 7.5 mmHg). As a result, in air, the contribution (partial pressure) of nitrogen is 78 kPa (78% of 100 kPa) and oxygen 21 kPa (21% of 100 kPa). When breathing 40% oxygen, the partial pressure of the oxygen in the inspired gas is 40 kPa.

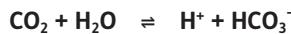
At atmospheric pressure, the partial pressure of a gas in a mixture in kPa is numerically the same as the percentage (%) of the gas by volume.

When a gas is dissolved in a liquid (e.g. blood) the partial pressure within the liquid is the same as in the gas in contact with the liquid. This enables us to measure the partial pressure of oxygen and carbon dioxide in blood.

In summary, the partial pressure of a gas is a measure of the concentration of the gas in the medium it is in and is expressed as $P_{\text{medium}}\text{Gas}$ (e.g. PaCO_2 is the partial pressure (P) in arterial blood (a) of carbon dioxide (CO_2)).

PaCO₂

Carbon dioxide is an important waste product of metabolism. Under normal circumstances, it is transported in the blood to the lungs where it is excreted during expiration. During transport to the lungs, it is combined either with protein or haemoglobin, or dissolved in plasma where it reacts with water to form hydrogen ions and bicarbonate (HCO_3^-):



The normal PaCO₂ is 5.3 kPa with a range of 4.7–6.0 kPa.

In the lungs, the reaction proceeds in reverse: CO₂ is generated and expired. From this reaction, we can see that CO₂ behaves as an acid: any increase in PaCO₂ will cause the reaction to move to the right and increase the hydrogen ion concentration with the subsequent development of an acidaemia. There is the same increase in bicarbonate concentration but, as this is only

nanomoles, it has little effect on the overall total bicarbonate concentration of 22–26 mmol L⁻¹. If the metabolic production of CO₂ is constant, the only factor that affects the amount in the blood is the rate at which it is removed by alveolar ventilation. A **decrease in alveolar ventilation** will reduce excretion of CO₂ causing an increase in PaCO₂ and the production of more hydrogen ions. If the pH decreases below 7.35 an acidaemia has been produced. As the primary cause of the acidaemia is a problem with the respiratory system, we call this process a **respiratory acidosis**.

Conversely, an **increase in alveolar ventilation** that removes CO₂ faster than it is generated reduces PaCO₂ and moves the reaction to the left, reducing the concentration of hydrogen ions. As a result the pH will increase and if it exceeds 7.45 an alkalaemia has been produced. Again, the primary cause is the respiratory system and we call this process a **respiratory alkalosis**.

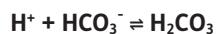
From this, it can be seen how even a brief period of apnoea, as may occur during cardiac arrest, will result in a respiratory acidosis. However, under normal circumstances, the respiratory centre in the brain stem is very sensitive to blood [H⁺] and within a few minutes rapidly increases alveolar ventilation. This increases CO₂ excretion, reduces PaCO₂, decreases [H⁺] and returns pH towards normal.

The lungs are the primary mechanism by which [H⁺] is adjusted by regulating PaCO₂.

Bicarbonate and base excess

Bicarbonate

Bicarbonate (HCO₃⁻) is the most important buffer and is generated by the kidneys. It can be thought of as the opposite of an acid and as such is also called a base. Acids not immediately eliminated by the respiratory system can be buffered by HCO₃⁻ as shown below. The reaction below moves to the right and bicarbonate “neutralises” the effect of the H⁺ and prevents a decrease in plasma pH.



Under normal circumstances, the concentration of bicarbonate is 22–26 mmol L⁻¹.

In the kidneys, the reaction now proceeds to the left; the H⁺ is excreted in the urine and bicarbonate filtered and returned to the plasma. Depending on the acid load, the kidneys will excrete either acid or alkaline urine.

Following an acute increase in the acid load, as the respiratory system tries to increase the excretion of CO₂, bicarbonate will also buffer the extra H⁺ and its concentration will decrease. Once the reserves of bicarbonate are used, H⁺ will accumulate, decreasing the pH (an acidaemia). Unlike the respiratory system, the kidneys respond slowly and it takes many hours or even days for

additional bicarbonate to be produced to meet the demand to buffer the extra acid. If the kidneys fail to produce sufficient bicarbonate the resultant **metabolic acidosis** will lead to a decrease in pH below 7.35 (acidaemia).

Occasionally, the situation arises where there is an excess of bicarbonate. This is most commonly caused by loss of acid, such as from persisting vomiting. This will have the effect of excessive buffering of hydrogen ions and will produce a **metabolic alkalosis** and increase the pH above 7.45 (alkalaemia).

Base excess

This is a measure of the amount of excess acid or base is in the blood as a result of a metabolic derangement. It is calculated as the amount of strong acid or base that would have to be added to a blood sample with an abnormal pH to restore it to normal (pH 7.4).

Consequently, a patient with a **base excess** of 8 mmol L⁻¹ would require 8 mmol L⁻¹ of **strong acid** to return their pH to normal; that is they have a metabolic alkalosis.

Conversely, a patient with a **base deficit** of 8 mmol L⁻¹ will require the addition of 8 mmol L⁻¹ of **strong base** to normalise their pH (again, compare with bicarbonate which would be reduced). Unfortunately, the term ‘negative base excess’ is used instead of base deficit and in the example above, the patient would have a negative base excess of -8 mmol L⁻¹.

The normal values of base excess are +2 to -2 mmol L⁻¹.

A base excess more negative than -2 mmol L⁻¹ indicates a metabolic acidosis.

A base excess greater than +2 mmol L⁻¹ indicates a metabolic alkalosis.

The respiratory – metabolic link

From the above we can see that the body has two systems for ensuring a stable internal environment and preventing the development of an acidosis. Additional protection is provided by the fact that the two systems are linked and can compensate for derangements in each other. This link is provided by the presence of carbonic acid (H₂CO₃), which is dependent on the presence of an enzyme called carbonic anhydrase, present in both red blood cells and the kidneys, and ideally situated to facilitate the link between the two systems.



Although this link exists, the ability of each system to compensate for the other is not instantaneous, but becomes more marked when the initial disturbance in one system is prolonged. A typical example demonstrating the link between the two systems is a patient with chronic obstructive pulmonary disease (COPD). This condition diminishes capacity to excrete carbon dioxide and a respiratory acidosis results, which if uncompensated,

would cause a persistent acidemia. The increase in carbon dioxide however, drives the reaction above to the right, which increases H^+ . The kidneys excrete the acid in the urine while at the same time the bicarbonate produced buffers the H^+ in the plasma. As a result the patient has a respiratory acidosis (increased $PaCO_2$) with a compensatory metabolic alkalosis (increased bicarbonate) and the pH will return close to normal.

Another good example is a diabetic in ketoacidosis (strictly speaking ketoacidemia). The excess ketoacids rapidly exceed the kidney's ability to excrete them, but a decrease in pH is initially minimised as they are buffered by bicarbonate, which reduces plasma bicarbonate. However, the reaction above moves to the left in effect, converting the H^+ to CO_2 that can be removed by increasing ventilation. This is seen clinically as an increased respiratory rate. Consequently, the patient has a metabolic acidosis (reduced bicarbonate) with a compensatory respiratory alkalosis (reduced $PaCO_2$).

PaO₂

The concentration of oxygen in inspired air is 21%, or a partial pressure of 21 kPa. As the air passes down the respiratory tract this is gradually reduced; firstly because of the addition of water vapour and, in the alveoli, by the addition of carbon dioxide. In the alveoli the partial pressure of oxygen (P_{AO_2}) is around 13 kPa. However, the partial pressure of oxygen in arterial blood (PaO_2) is always lower than in the alveoli, which creates a gradient, the magnitude of which is determined largely by the presence of any lung disease. In a healthy individual breathing air, the PaO_2 is normally higher than 11 kPa (i.e. about 10 kPa lower than the inspired partial pressure). This can be used as a 'rule of thumb' to estimate the PaO_2 for any given inspired concentration; it should be numerically about 10 less than the inspired concentration (%). For example, 40% inspired oxygen should result in a $PaO_2 \approx 30$ kPa. With increasing lung injury, the gradient between inspired concentration and PaO_2 increases. This is important because for someone breathing 50% oxygen a PaO_2 of 13 kPa is not 'normal'.

Interpreting the results

Interpretation of the result of blood gas analysis is achieved by following closely five steps. For clarity, only changes in base excess are discussed; however, bicarbonate will also change numerically in the same direction.

Step 1

How is the patient?

This will often provide useful clues to help with interpretation of the results. For example, one might reasonably predict that analysis of arterial blood shortly after successful resuscitation would show signs of a respiratory acidosis, caused by a period of inadequate ventilation, and a metabolic acidosis due to the period of anaerobic respiration during the arrest producing lactic

acid. Consequently, we would expect the patient to have a very low pH, a high $PaCO_2$ and significant base deficit. A patient with well-compensated COPD will usually display clues about the primary cause (respiratory acidosis) and secondary compensation (metabolic alkalosis). Without the clinical history, the results of a blood gas analysis from a patient with COPD could be misinterpreted as a primary metabolic alkalosis with a compensatory respiratory acidosis.

Step 2

Is the patient hypoxaemic?

While breathing room air the PaO_2 should be 10.0–13.0 kPa. However, if the patient is receiving supplemental oxygen, the PaO_2 must be interpreted in light of the inspired oxygen concentration. As discussed above, the inspired partial pressure of oxygen can be regarded as the numerical equivalent of the inspired concentration (%). If there is a difference of greater than 10 between the two values, there is a defect in oxygenation, proportional to the magnitude of the difference.

Step 3

Is the patient acidaemic ($pH < 7.35$) or alkalaemic ($pH > 7.45$)?

If the pH is within or very close to the normal range then this suggests normality or a chronic condition with compensation. In principle, the body never over compensates and this should enable the primary problem to be determined. If necessary, seek more clinical information about the patient.

Step 4

What has happened to the $PaCO_2$?

In other words, is the abnormality wholly or partially due to a defect in the respiratory system?

If the pH is < 7.35 (acidaemia):

4a. Is the $PaCO_2$ increased (> 6.0 kPa)?

If so, there is a **respiratory acidosis** that may be accounting for all or part of the derangement. There could also be a metabolic component, see Step 5a.

If the pH is > 7.45 (alkalaemia):

4b. Is the $PaCO_2$ reduced (< 4.7 kPa)?

If so, there is a **respiratory alkalosis**, but this is an unusual isolated finding in a patient breathing spontaneously, with a normal respiratory rate. It is seen more often in patients who are being mechanically ventilated with excessively high rates and/or tidal volumes. As a result, $PaCO_2$ decreases, there is a reduction in H^+ and an alkalois develops. It may also be seen when patients are in severe pain or suffering from acute anxiety.

Step 5

What has happened to the base excess or bicarbonate?

In other words, is the abnormality wholly or partially due to a defect in the metabolic system?

If the pH is < 7.35 (acidaemia):

5a. Is there a base deficit (< -2 mmol L⁻¹) (i.e. more negative), and/or the bicarbonate reduced (< 22 mmol L⁻¹)? If so, there is a **metabolic acidosis** accounting for all or part of the derangement. There could be a respiratory component if the PaCO₂ is also increased – see Step 4a, a situation commonly seen after a cardiac arrest.

If the pH is > 7.45 (alkalaemia):

5b. Is there a base excess (> +2 mmol L⁻¹) and/or the bicarbonate increased (> 26 mmol L⁻¹)? If so, there is a **metabolic alkalosis** accounting for all or part of the derangement. There could be a respiratory component if the PaCO₂ is also decreased – see Step 4b, but this would be unusual.

Examples

Using the above we can now work through some examples, based on clinical cases to highlight key points.

Case 1: 21 year-old woman, thrown from her horse at a local event. On the way to hospital, she has become increasingly drowsy and the paramedics have inserted an oropharyngeal airway and given high-flow oxygen via a face mask with a reservoir. On arrival at hospital, following a rapid ABCDE assessment, an arterial blood sample is taken and shows:

PaO ₂	18.8 kPa (FiO ₂ ≈ 85%)
pH	7.26
PaCO ₂	8.2 kPa
Bicarbonate	26.0 mmol L ⁻¹
Base excess	-2.4 mmol L ⁻¹

Step 1: From the history we would predict the reduction in level of consciousness to reduce ventilation, increasing PaCO₂, causing a respiratory acidosis. It may also impair oxygenation. There is unlikely to be much compensation because the situation is acute.

Step 2: Although the PaO₂ is just above the ‘normal’ range, breathing 85% oxygen we would expect a PaO₂ around 75 kPa. Therefore there is a significant impairment in oxygenation.

Step 3: The patient clearly has an acidaemia with a pH well below normal.

Step 4: The PaCO₂ is increased, consistent with the low pH, so the patient has a *respiratory acidosis*.

Step 5: The base excess is just below the normal limit and the bicarbonate is within normal limits. This confirms that there is no significant metabolic contribution or compensation.

In summary, the patient has an acute respiratory acidosis with impaired oxygenation

Case 2: A 19 year-old man with asthma is brought to the emergency department (ED) by his parents. Over the past 4 h he has become increasingly wheezy with no response to his inhalers. He is now very distressed, tachypnoeic and has audible wheeze. He is receiving oxygen at 15 L min⁻¹ via a face mask with reservoir. Following a rapid ABCDE assessment, an analysis of an arterial blood sample shows:

PaO ₂	23.6 kPa (FiO ₂ ≈ 85%)
pH	7.57
PaCO ₂	3.4 kPa
Bicarbonate	23.1 mmol L ⁻¹
Base excess	+1.8 mmol L ⁻¹

Step 1: From the history we would predict the bronchospasm to impair oxygenation and the hyperventilation to reduce his PaCO₂ causing a respiratory alkalosis. There is unlikely to be much compensation because the situation is acute.

Step 2: Although the PaO₂ is above the normal range, breathing 85% oxygen we would expect a PaO₂ around 75 kPa. Therefore there is a significant impairment in oxygenation.

Step 3: The patient clearly has a pH above the normal range, an alkalaemia.

Step 4: The PaCO₂ is decreased, consistent with a raised pH, the patient has a respiratory alkalosis.

Step 5: The base excess and bicarbonate are within normal limits. This confirms that there is no significant metabolic contribution or compensation.

In summary, the patient has an acute respiratory alkalosis with impaired oxygenation (Type I respiratory failure – see below) and requires continued medical therapy for acute severe asthma.

Case 3: A 52 year-old man, complaining of crushing central chest pain, is brought to the emergency department by his wife. He is attached to an ECG monitor, given oxygen 40% via a facemask, sublingual GTN and aspirin to chew. An intravenous cannula is inserted and he is given intravenous morphine. During an ABCDE assessment, he suddenly has a cardiac arrest. After 4 minutes of resuscitation he has a palpable pulse and starts to breathe spontaneously. Analysis of an arterial blood sample shows:

PaO ₂	8.9 kPa (FiO ₂ ≈ 40%)
pH	7.11
PaCO ₂	7.2 kPa
Bicarbonate	14 mmol L ⁻¹
Base excess	-10.6 mmol L ⁻¹

Step 1: From the history we would predict the impaired ventilation to result in hypoxia, an increased PaCO₂ and respiratory acidosis. The impaired circulation will cause an increase in anaerobic respiration, production of lactate and a metabolic acidosis.

This will also decrease bicarbonate as it is used to buffer the excess acid. The failure of circulation is likely to prevent any degree of compensation.

Step 2: The patient is hypoxaemic and breathing 40% oxygen we would expect a PaO_2 around 30 kPa. Therefore there is a significant impairment in oxygenation.

Step 3: The patient clearly has a very low pH; a severe acidaemia.

Step 4: The PaCO_2 is increased, consistent with the low pH and the patient has a respiratory acidosis.

Step 5: The base excess and bicarbonate are both reduced. This is consistent with a metabolic acidosis.

In summary, the patient has a mixed respiratory and metabolic acidosis with impaired oxygenation following the cardiac arrest. He will still require assisted ventilation and treatment of the underlying cause of the arrest.

Derangements of both PaCO_2 and base excess or bicarbonate - compensation

In addition to the combined changes seen in case 3, where both the respiratory and metabolic changes are additive, some conditions may show changes in both the respiratory and metabolic components in ‘opposite directions’, with minimal disturbance of the pH. This is the result of compensation; both the respiratory and metabolic systems are capable of reacting to changes in the other, the aim being to minimise long term changes in pH.

Example 1

- $\text{pH} < 7.40$
- $\text{PaCO}_2 > 6.0 \text{ kPa}$
- base excess $> +2 \text{ mmol L}^{-1}$ or bicarbonate $> 26 \text{ mmol L}^{-1}$

The tendency towards an acidaemia indicates that this is the primary problem and the increased PaCO_2 indicates that it is primarily a **respiratory acidosis**. The increased base excess/bicarbonate represents a **compensatory metabolic alkalosis**, bringing the pH back towards normality. This pattern occurs in a variety of chronic respiratory diseases.

Example 2

- $\text{pH} < 7.40$
- $\text{PaCO}_2 < 4.7 \text{ kPa}$
- base excess $< -2 \text{ mmol L}^{-1}$ or bicarbonate $< 22 \text{ mmol L}^{-1}$

The tendency towards an acidaemia indicates that this is the primary problem and the decreased base excess/bicarbonate suggests that it is primarily a **metabolic acidosis**. The decrease in PaCO_2 represents a **compensatory respiratory alkalosis**, bringing the pH back

towards normality. This very common abnormality has many causes including sepsis and dehydration.

Example 3

- $\text{pH} > 7.40$
- $\text{PaCO}_2 > 6.0 \text{ kPa}$
- base excess $> +2 \text{ mmol L}^{-1}$ or bicarbonate $> 26 \text{ mmol L}^{-1}$

The tendency towards an alkalaemia indicates that this is the primary problem and the increase in base excess/bicarbonate suggests that it is primarily a **metabolic alkalosis**. The increased PaCO_2 is **respiratory compensation** bringing the pH back towards normality. This picture may be seen where there is loss of acid from the body (e.g. prolonged vomiting of gastric contents (hydrochloric acid)) and also occurs in chronic hypokalaemia. In this case, the body compensates by moving potassium from intracellular to extracellular in exchange for hydrogen ions. The pH increases and CO_2 is retained to try and compensate.

Example 4

- $\text{pH} > 7.40$
- $\text{PaCO}_2 < 4.7 \text{ kPa}$
- base excess $< -2 \text{ mmol L}^{-1}$ or bicarbonate $< 22 \text{ mmol L}^{-1}$

The tendency towards an alkalaemia indicates that this is the primary problem and the decrease in PaCO_2 suggests that this is primarily a **respiratory alkalosis**. The decrease in base excess/bicarbonate is the **metabolic compensation** bringing the pH back towards normality.

This is not a common finding, but may be seen after a few days when hyperventilation is used to help control intracranial pressure in patients with brain injury. It is also seen due to hyperventilation at altitude due to low PaO_2 .

Using the above we can now work through some further examples:

Case 4: A 68 year-old man with a long history of COPD is reviewed on the medical ward before discharge. Analysis of an arterial blood sample shows:

PaO_2	8.9 kPa
pH	7.34
PaCO_2	7.3 kPa
Bicarbonate	30.2 mmol L $^{-1}$
Base excess	5.3 mmol L $^{-1}$

Step 1: From the history we would predict the patient to have a chronically raised PaCO_2 causing a respiratory acidosis. However, there is likely to be significant compensation in the form of a metabolic alkalosis. Oxygenation is also likely to be impaired.

Step 2: The PaO_2 is significantly reduced. Breathing air we would expect a PaO_2 around 11 kPa. Therefore there is impairment in oxygenation, although this is likely to be chronic.

Step 3: The patient has a borderline acidaemia with a pH just below the normal range.

Step 4: The PaCO₂ is increased, causing a respiratory acidosis. In a normal person, we would expect this degree of hypercapnia to cause a pH < 7.34, suggesting that there is a degree of compensation.

Step 5: The base excess and bicarbonate are both increased confirming that there is a metabolic alkalosis. This compensation has helped minimise or compensate for the pH disturbance caused by the respiratory acidosis identified in Step 4.

In summary, the patient has a chronic respiratory acidosis with a compensatory metabolic alkalosis, with significantly impaired oxygenation (Type II respiratory failure – see below). He will require optimisation of medical management of his COPD prior to discharge.

Case 5: A 22 year-old male, recently diagnosed with insulin dependent diabetes mellitus (IDDM) presents to the emergency department, having been unwell for 48 hours, with a gradually increasing blood sugar concentration, despite taking his insulin. He is tachypnoeic, tachycardic and a point-of-care measurement of his blood glucose is 23 mmol L⁻¹. Following a rapid ABCDE assessment, analysis of an arterial blood sample while breathing oxygen, 6 L min⁻¹ via a facemask shows:

PaO ₂	22.2 kPa (FiO ₂ ≈ 40%)
pH	7.14
PaCO ₂	3.8 kPa
Bicarbonate	19.1 mmol L ⁻¹
Base excess	-7.9 mmol L ⁻¹

Step 1: From the history the most likely problem is that the patient is developing a diabetic ketoacidosis (DKA) (i.e. a metabolic acidosis). However, the fact that he is tachypnoeic suggests that he is trying to compensate by reducing his PaCO₂ that effectively reduces the [H+] causing a respiratory alkalosis.

Step 2: There is a reduction in the PaO₂ compared to what might be expected (30 kPa), which may indicate the possibility of an underlying chest infection. Alternatively, it may be that the facemask is delivering less than 40% oxygen and his oxygenation is unimpaired.

Step 3: The patient's pH is well below the lower limit of normality, he has an acidaemia.

Step 4: The PaCO₂ is decreased. This will cause a respiratory alkalosis and therefore is not the cause of the primary disturbance.

Step 5: The base excess and bicarbonate are both decreased as they have been consumed buffering the ketoacids. This also confirms that there is a metabolic

acidosis. The change in pH would have been greater without the concurrent respiratory changes.

In summary, the patient has a metabolic acidosis (as a result of impaired glucose metabolism and the development of ketoacids) with a compensatory respiratory alkalosis. He will require careful IV fluid and electrolyte replacement along with a fixed rate insulin infusion according to local DKA protocols.

There is one final situation that deserves mention and is important to identify. An ill patient with:

pH	< 7.4
PaCO ₂	4–6.0 kPa
base excess	< -2 mmol L ⁻¹ or bicarbonate < 22 mmol L ⁻¹

This is most likely to represent the situation of a metabolic acidosis in a patient with chronic carbon dioxide retention. The patient is trying to compensate by lowering their CO₂ (to cause a compensatory respiratory alkalosis), but they are starting from a higher PaCO₂. Their lung disease will limit the amount of CO₂ they can excrete, thereby preventing it decreasing any further. Once again it illustrates the importance of having information about the patient as identified at the beginning.

Practical aspects of blood gas analysis during resuscitation

During cardiac arrest, arterial blood gas values are of limited use because they correlate poorly with the severity of hypoxaemia, hypercapnia and acidosis in the tissues. Indeed, during cardiac arrest, venous blood gases may reflect more accurately the acid-base state of the tissues. These are interpreted using the same 5-step approach, however the normal range of values will be different to arterial blood and reference should be made to local values.

Once return of spontaneous circulation (ROSC) is achieved arterial blood gas analysis will provide a useful guide to post cardiac arrest treatment, such as the optimal FiO₂ and minute ventilation. Arterial lactate concentration can also be used to indicate adequacy of tissue oxygenation, normal arterial blood lactate concentration being 0.5–2.0 mmol L⁻¹. Immediately after cardiac arrest, concentrations are high, reflecting the lactic acidosis that has been caused by inadequate oxygenation of the tissues during the period of cardiac arrest. After ROSC a progressively decreasing lactate value is another indicator of adequate tissue oxygenation.

In the peri-arrest setting, it may be easiest to obtain a sample of arterial blood (into a heparinised syringe) from the femoral artery. The radial artery may be preferable once the patient has an adequate cardiac output and blood pressure. The radial artery is also the best site for insertion of an arterial cannula; this allows continuous monitoring of blood pressure and frequent blood sampling in the post cardiac arrest period.

Venous blood gases

Venous blood gases (VBGs) are often used in the assessment of acutely unwell patients instead of ABGs. They are particularly useful for monitoring metabolic disturbances when there is no clinical indication of any respiratory compromise. A specific example is in DKA, where serial VBGs enable the clinician to monitor for a gradual resolution of the acidosis whilst also providing a point of care test for essential electrolytes (specifically potassium in DKA).

The use of a tourniquet to take a peripheral venous sample can alter VBG values, so they are best taken from large veins without a tourniquet (e.g. from a femoral vein or a central venous catheter).

VBGs from a central venous line provide a better indication of the acid-base status of tissues because, unlike ABGs, the blood has just passed through them. This is particularly useful in the cardiac arrest setting, where the lack of effective circulation means that ABGs do not accurately reflect tissue metabolism. A typical VBG result during cardiac arrest would show:

- pH < 7.35
- base deficit < -2 and a bicarbonate < 22 mmol L⁻¹.

It will also show a low PvO₂ and normal/high PvCO₂ although these are not clinically useful in this instance.

The same five-stage approach is needed to interpret VBGs; however, the oxygen reference range is significantly lower and the carbon dioxide values are highly variable, making the PvO₂ and PvCO₂ values difficult to interpret. If there are any concerns about oxygenation or if a VBG demonstrates an acidosis that is not metabolic, an ABG is essential.

Pulse oximetry

Role of pulse oximetry

Pulse oximetry is a vital adjunct to the assessment of hypoxaemia. Clinical recognition of decreased arterial oxygen saturation of haemoglobin (SaO₂) is subjective and unreliable: a person may not appear to be cyanosed until their arterial oxygen saturations are as low as 80–85%. Pulse oximetry is simple to use, relatively cheap, non-invasive and provides an immediate, objective measure of arterial blood oxygen saturation. It is now used widely in all in-hospital settings, in both primary care and the pre-hospital environment. Oxygen saturation of arterial blood, ‘the fifth vital sign’, now also forms a component of many physiological early warning systems (e.g. National Early Warning Score (NEWS)).

Principles of pulse oximetry

The pulse oximeter probe containing light-emitting diodes (LEDs) and a photoreceptor situated opposite, is placed across tissue, usually a finger or earlobe. Some of the light

is transmitted through the tissues while some is absorbed. The ratio of transmitted to absorbed light is used to generate the peripheral arterial oxygen saturation (SpO₂) displayed as a digital reading, waveform, or both. As a result of rapid sampling of the light signal, the displayed reading will alter every 0.5–1 s, reflecting the average SpO₂ over the preceding 5–10 s. Most pulse oximeters are accurate to within +/- 2% above an SaO₂ of 90%.

Tissue thickness should be optimally between 5–10 mm. Poor readings may be improved by trying different sites, warming sites or applying local vasodilators.

Pulse oximeters often provide an audible tone related to the SpO₂ value, with a decreasing tone reflecting increasing hypoxaemia. Information about pulse rate and waveform (plethysmographic waveform) may also be provided. A poor signal may indicate a low blood pressure or poor tissue perfusion – reassess the patient.

Pulse oximetry provides only a measure of oxygen saturation, and thus gives no indication of actual tissue oxygenation. Furthermore, it provides no information on the PaCO₂ in the body and is not a measure of adequacy of ventilation. In cases of critical illness, or when type II respiratory failure (see below) is suspected (e.g. known COPD, congenital heart disease) arterial blood gas sampling must be performed. Do not use readings from a pulse oximeter in isolation – they must be interpreted in light of the clinical picture and alongside other investigations and potential sources of error.

Limitations of pulse oximetry

The relationship between oxygen saturation and PaO₂ is demonstrated by the oxyhaemoglobin dissociation curve (Figure 15.1). The sinusoid shape of the curve means that an initial decrease from a normal PaO₂ is not accompanied by a decrease of similar magnitude in the oxygen saturation of the blood, and early hypoxaemia may be masked. At the point where the SpO₂ reaches 90–92%, the PaO₂ will have decreased from 12–14 kPa to 8 kPa. In other words, the arterial blood will have lost more than 25% of its oxygen content for a fall of only 6–8% in SpO₂. Further reductions in SpO₂ reflect the more rapid desaturation that occurs with diminishing oxygen partial pressure.

The output from a pulse oximeter relies on a comparison between current signal output and standardised reference data derived from healthy volunteers. Readings provided are thus limited by the scope of the population included in these studies, and become increasingly unreliable with increasing hypoxaemia. Below ~70% the displayed values are highly unreliable.

Oxygen dissociation curve

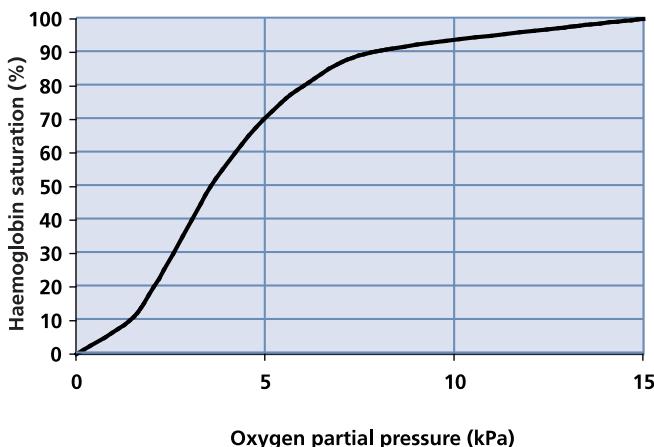


Table 15.1 Haemoglobin oxygen dissociation curve

There are several acknowledged sources of error with pulse oximetry:

- Presence of other haemoglobins: carboxyhaemoglobin (carbon monoxide poisoning), methaemoglobin (congenital or acquired), fetal haemoglobins and sickling red cells (sickle cell disease).
- Surgical and imaging dyes (e.g. methylene blue) can cause falsely low saturation readings.
- Nail varnish (especially blue, black and green).
- High-ambient light levels (fluorescent and xenon lamps).
- Motion artefact.
- Reduced pulse volume:
 - hypotension
 - low cardiac output
 - vasoconstriction
 - hypothermia.

Pulse oximeters are not affected by:

- Anaemia (reduced haemoglobin).
- Jaundice (hyperbilirubinaemia).
- Skin pigmentation.

Pulse oximetry does not provide a reliable signal during CPR.

Uses

Pulse oximetry has four main uses:

- i) detection of/screening for hypoxaemia
- ii) targeting oxygen therapy
- iii) routine monitoring during anaesthesia
- iv) diagnostic (e.g. sleep apnoea).

Targeted oxygen therapy

Give high-concentration oxygen immediately to critically ill patients with acute hypoxaemia (initial $\text{SpO}_2 < 85\%$) or in the peri-arrest situation. Give this initially with an oxygen mask and reservoir ('non-rebreathing' mask) and an oxygen flow of 15 L min^{-1} . During cardiac arrest use 100% inspired oxygen concentration to maximise arterial oxygen content and delivery to the tissues.

Once ROSC has been achieved and the oxygen saturation of arterial blood can be monitored reliably, adjust the inspired oxygen concentration to maintain a SpO_2 of 94–98%. If pulse oximetry (with a reliable reading) is unavailable, continue oxygen via a reservoir mask until definitive monitoring or assessment of oxygenation is available. All critically ill patients will need arterial blood gas sampling and analysis as soon as possible. Evidence suggests that in the post-resuscitation phase both hypoxaemia and hyperoxaemia ($\text{PaO}_2 > 20 \text{ kPa}$) are associated with worse outcomes compared with those in whom normoxaemia is maintained.

Special clinical situations

Patients with respiratory failure can be divided into two groups:

- **Type I:** low PaO_2 ($< 8 \text{ kPa}$), normal PaCO_2 ($< 7 \text{ kPa}$). In these patients it is safe to give a high concentration of oxygen initially with the aim of returning their PaO_2 to normal and then once clinically stable, adjusting the inspired oxygen concentration to maintain an SpO_2 of 94–98%.
- **Type II:** low PaO_2 ($< 8 \text{ kPa}$), increased PaCO_2 ($> 7 \text{ kPa}$). This is often described as hypercapnic respiratory failure and is often caused by COPD. If given excessive oxygen, these patients may develop worsening respiratory failure with further increases in PaCO_2 and the development of a respiratory acidosis. If unchecked, this will eventually lead to unconsciousness, and respiratory and cardiac arrest. **The target oxygen saturation in this at risk population is 88–92%.** However, when critically ill and their arterial blood oxygen saturation is unknown, give these patients high-flow oxygen initially; then analyse the arterial blood gases and use the results to adjust the inspired oxygen concentration. When clinically stable and a reliable pulse oximetry reading is obtained, adjust the inspired oxygen concentration to maintain an SpO_2 of 88–92%.

In patients with a myocardial infarction or an acute coronary syndrome, and who are not critically or seriously ill, aim to maintain an SpO_2 of 94–98% (or 88–92% if the patient is at risk of hypercapnic respiratory failure). This may be achievable without supplementary oxygen, and represents a change from previously accepted practice.

Summary learning

- Arterial blood gas analysis results are interpreted systematically using the 5-step approach.
- Pulse oximetry enables arterial blood oxygen saturation to be monitored continuously.
- Use an inspired oxygen concentration of 100% until return of spontaneous circulation (ROSC) is achieved.
- After ROSC is achieved, and once the SpO₂ can be monitored reliably, titrate the inspired oxygen concentration to keep the SpO₂ in the range 94–98%.

My key take-home messages from this chapter

Further reading

A Simple Guide to Blood Gas Analysis. Eds. Driscoll P, Brown T, Gwinnutt C, Wardle T. BMJ Publishing Group. London 1997.

Berend K, de Vries AP, Gans RO. Physiological approach to assessment of acid-base disturbances. N Engl J Med 2014;371:1434–45.

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Making decisions about CPR

Chapter 16

Contents

- The ethical principles that underpin decisions about CPR
- What constitutes good decision-making, effective communication and clear documentation of discussions and decisions
- Legal aspects of decisions about CPR
- When to withhold CPR and when to stop a CPR attempt
- When and when not to consider withdrawal of other treatments

Learning outcomes

To enable you to:

- Understand when it is and when it is not appropriate to attempt CPR
- Know when to stop CPR
- Consider the ethical and legal principles involved
- Consider who should make decisions about CPR
- Know what you and others should do to achieve good decisions about CPR
- Understand the importance of effective communication with all involved
- Understand the importance of effective documentation of decisions and discussions

and to feel more confident in:

- Making anticipatory decisions about CPR with your patients
- Encouraging others to follow best practice in decision-making

Introduction

Although only a minority of people survives and makes a complete recovery after receiving CPR, successful resuscitation attempts bring extended, useful and precious life to many individuals. If it is to be successful it is important that CPR is started without delay in a person who may have a reversible cause for cardiac arrest. Used inappropriately, as with any treatment, CPR has the potential to cause harm. When someone is dying from an irreversible condition CPR is unlikely to work, but is likely to subject them to an undignified death or possibly cause suffering and prolong the process of dying. Prolonging life at all costs is not an appropriate goal of medicine.

In people who are at risk of dying or suffering cardiac arrest it is important, whenever possible, to make decisions in advance about whether or not it would be appropriate to start CPR in such circumstances. Ideally, such decisions are made as shared decisions with patients and as part of a wider consideration of other care and treatments that may or may not be appropriate for each individual. Detailed guidance has been published by the British Medical Association (BMA), Resuscitation Council (UK) (RC (UK)) and Royal College of Nursing (RCN) (Figure 16.1). As an ALS provider, you should read and be familiar with that guidance and follow the principles that it sets out.

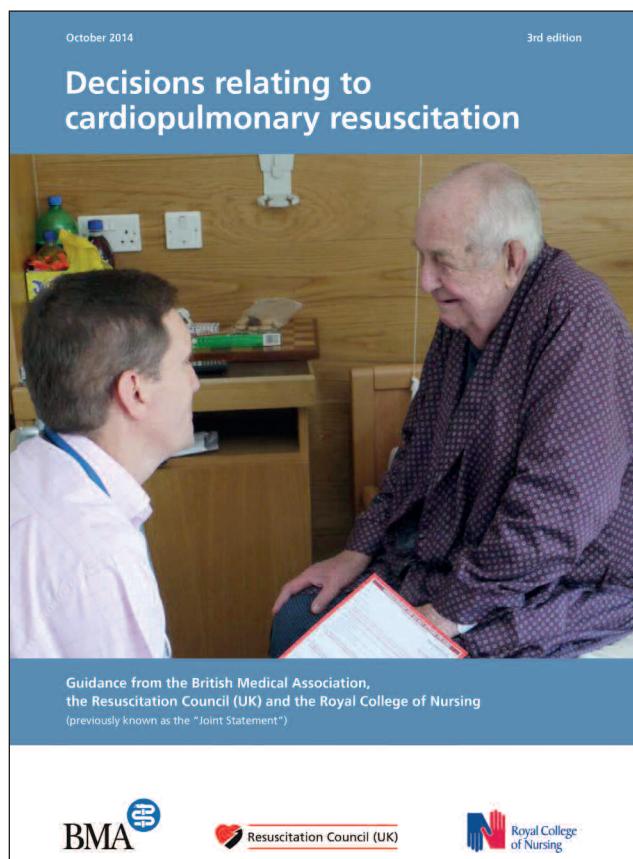


Figure 16.1 Decisions relating to cardiopulmonary resuscitation

All healthcare professionals must practise within the law. The laws relevant to CPR, including those on matters such as capacity and consent, vary from country to country. Within the UK there are some differences in such laws between nations. These are summarised within the guidance by the BMA, RC (UK) and RCN. As an ALS provider you should be familiar with the relevant aspects of law in the nation where you live and work. The RC (UK) has published guidance on the legal status of those who attempt resuscitation.

Discussing decisions about CPR can be difficult for patients, their families or carers and health professionals. Many people welcome the opportunity to discuss their wishes, once they realise that the purpose is to plan with them what treatment would be best for them, not to deny them beneficial treatment. Such discussions should not be forced on people who don't want them, but should not be withheld from those who do. They should be undertaken by health professionals who are trained and competent in having such sensitive conversations and, whenever possible, who know the person well. Some people with capacity decide that they do not want certain types of treatment and record those wishes in an advance decision to refuse treatment.

Every decision about CPR must be made on the basis of a careful assessment of each individual's situation at any particular time. These decisions should never be dictated by 'blanket' policies. Individual decisions about CPR may be influenced by many factors including personal beliefs and

opinions, cultural or religious influences, ethical or legal considerations and social or economic circumstances. As an ALS provider you should understand the ethical and legal principles as well as the clinical aspects involved before undertaking discussions or making a decision about CPR.

Principles

The four key principles of medical ethics are summarised in the box:

Autonomy requires people to be allowed and helped to make their own informed decisions, rather than having decisions made for them. A person with capacity must be adequately informed about the matter to be decided and free from undue pressure in making their decision. Autonomy allows an informed person to make a choice, even if that choice is considered illogical or incorrect by others, including health professionals.

Beneficence involves provision of benefit to an individual, while balancing benefit and risks. Commonly this will involve attempting CPR for cardiac arrest. If CPR will offer no benefit or the risks clearly outweigh any likely benefit it will mean withholding CPR. Beneficence includes also actions that benefit the wider community, such as providing a programme of public access defibrillation.

Non-maleficence means doing no harm. CPR should not be attempted in people in whom it will not succeed, where no benefit is likely but there will be a clear risk of doing harm.

Justice requires spreading benefits and risks equally within a society. If CPR is provided, it should be available to all who may benefit from it. There should be no discrimination purely on the grounds of factors such as age or disability. Justice does not imply an entitlement to expect or demand CPR for everyone. If limited resources are engaged in attempting CPR on people with no chance of benefit those resources may not be available when needed by others who are likely to benefit.

Communication: discussing decisions about CPR with patients

Effective communication is essential to ensure that decisions about CPR are made well and understood clearly by all those involved. The courts have made clear that there should be a presumption in favour of involving patients in discussions about whether or not CPR will be attempted. This upholds the principle of autonomy and the provisions of the Human Rights Act (1998). If discussing a decision

about CPR with a person is likely to cause them physical or psychological harm, such discussion may be omitted; the reasons for doing so should be documented clearly.

If a patient has capacity, discussions about CPR should usually involve shared decision-making, following the principles outlined in ‘Decisions relating to CPR’ and in guidance from the General Medical Council. Requirements for effective communication include providing information in a format that the person can understand, and checking that they have understood it. They may require time and more than one discussion before they can reach a shared decision that they are comfortable with. They should be offered opportunities for further discussion and be made aware that they may change their decision if they wish to.

It is not necessary to have a discussion about CPR with a patient if there is no reason to expect them to suffer cardiac arrest or die. However, if such a patient wants to discuss CPR they must be given full opportunity to do so.

If the healthcare team is as certain as it can be that a person is dying as an inevitable result of underlying disease or a catastrophic health event, and that CPR would not re-start the heart and breathing for a sustained period, CPR should neither be offered nor attempted. Making a decision not to attempt CPR that has no realistic prospect of success does not require the consent of the patient (or of those close to the patient). However, there is still a presumption in favour of informing a patient of such a decision and explaining the reasons for making it. A patient (or someone representing them) is not entitled to insist on receiving treatment that is clinically inappropriate. Healthcare professionals are not obliged to offer or deliver treatment that they believe to be inappropriate. Explaining these matters requires sensitive discussion.

Communication: discussing decisions about CPR with those close to patients

Effective communication with those close to a patient (often but not always family members) and keeping them fully informed is an important component of high-quality health care. You must respect a patient’s wishes regarding confidentiality, but most patients want family members or others close to them to be involved in discussions about treatment such as CPR and to have their support in making these decisions.

Discussion with those close to a patient must be used to guide any decision made in the best interests of a person who lacks capacity. The question is what the patient would have wanted rather than what the family or carers may want. Those close to the patient must not be burdened with feeling that they are being asked to make a decision about CPR as this responsibility rests with the senior clinician.

Communication: discussing decisions about CPR when a patient lacks capacity

If a person lacks capacity, they can still be involved in decision-making to the limit of their ability. Formal assessment of their capacity should be undertaken and recorded. Decisions about their treatment, including about CPR, must be made in their best interests. If a person who lacks capacity has a representative (e.g. a ‘welfare attorney’) with power to make such decisions on their behalf, that person must be involved in the decision-making process. Because of the slight differences in capacity law in England & Wales, in Scotland and in Northern Ireland it is essential to be familiar with and follow the law that applies in your locality.

Deciding whether or not to attempt CPR

Whether or not to attempt CPR at the time of a cardiac arrest requires immediate decision by the person or persons present. That decision will be influenced by their role, training and experience, as well as by the immediate circumstances. Where a decision has been considered and recorded in advance it can help those present at the moment of crisis to make the best possible immediate decision (see below).

The overall responsibility for a decision about CPR, whether made in advance or at the time of an arrest, rests with the senior health professional in charge of the person’s care at the time of the decision. When making a decision in advance there should be appropriate consultation with other health professionals involved, as well as appropriate discussion with the person themselves and those close to them.

In some circumstances the law requires you to involve others in the decision-making process when a patient lacks capacity. For example the Mental Capacity Act (2005), which applies in England and Wales, requires an Independent Mental Capacity Advocate (IMCA) to act on behalf of a person who lacks capacity and has no other representatives, guiding a decision by the senior clinician. When an emergency decision is needed there may not be time to appoint or contact an IMCA; such a decision should not be delayed and must be made in the patient’s best interests. The basis for the decision should be documented clearly and fully.

If a difference of opinion occurs between the healthcare team and the patient or their representatives this can usually be resolved by careful discussion and explanation. If not, a second clinical opinion should be offered. Seeking a decision by legal authorities may involve delay and uncertainty. Formal legal judgement may be needed if there are irreconcilable differences between the parties. In difficult cases, the senior clinician may wish to seek legal advice from his/her indemnity provider or other professional organisation.

Recording decisions about CPR

It is important that any decision made in advance about whether or not to attempt CPR and the reasons for that decision are documented clearly and that (as far as is reasonably possible) the record is available immediately if it is needed in a crisis. For example, if a do-not-attempt-CPR (DNACPR) decision is recorded on a paper form this should be readily available to help a paramedic to decide not to attempt CPR in a person's home.

Previously it has been common practice to record only DNACPR decisions, using a 'DNACPR form'. There is increasing recognition that anticipatory decisions about CPR are best made in the context of other care and treatment choices, and that it is important to record decisions to attempt CPR as well as decisions not to do so. Various forms have been developed in different places to record people's treatment decisions in advance. The RC (UK) favours the use of a standard document that is used and accepted by all health and care provider organisations, so that it is effective across all geographical and organisational boundaries. As an ALS provider you should ensure that you record decisions about CPR fully, clearly and accurately.

You should make sure also that you understand the status of any such document. Most such forms are not legally binding and are intended to guide immediate clinical decision-making at a moment of crisis. It would be unwise to ignore such a document that records an agreed, shared decision and/or the person's express wishes. However, if you encounter an arrest in circumstances that you believe were not those envisaged when the decision was recorded it may be appropriate to attempt CPR (e.g. to relieve choking or a blocked tracheostomy tube).

Some recorded advance decisions are legally binding. For example, if a person with capacity has recorded refusal of CPR in an Advance Decision to Refuse Treatment (ADRT) (Mental Capacity Act (2005) – England & Wales) you must respect this, provided the document is valid and that the immediate circumstances are those that were envisaged when the ADRT was recorded.

The term 'advance decision' may apply to any expression of preferences by a patient. Refusal of treatment does not have to be in writing in order to be valid. If an informed person has expressed clear and consistent refusal verbally, this is likely to have the same status as a written advance refusal. People should be encouraged and helped to make their healthcare team and those close to them aware of their wishes. Occasionally people have taken the more extreme step of having a DNACPR instruction tattooed on their chest. This is not considered a legally binding refusal; its expression of their wishes at the time must be balanced against the possibility that they may not have been fully informed, that they may have since changed their mind, or that the current circumstances are not those that they had envisaged when the tattoo was created.

Where no explicit advance decision has been made and the express wishes of the patient are unknown there is a presumption that health professionals will make all reasonable and appropriate efforts to resuscitate someone from cardiac arrest. In sudden out-of-hospital cardiac arrest, those present may not know the victim's situation or wishes. Even if an ADRT has been recorded, it may not be available. In these circumstances CPR can be started immediately and further information obtained when possible. There is no ethical difficulty in stopping a resuscitation attempt if the health professionals performing CPR are then presented with a valid, recorded ADRT refusing CPR or a valid DNACPR decision.

Communicating decisions to health and care teams

Good communication within the team is an essential component of high-quality, safe health care. When an advance decision is made about whether or not to attempt CPR it should be communicated clearly to all those who may need to know about it and act on it. The basis for the decision, details of those involved in making it, and details of discussions with patients and those close to them should be recorded. The decision itself should be recorded in a way that is immediately available and recognisable to those present, should the patient suffer sudden cardiac arrest.

There should be effective verbal communication with all those caring for the person and robust written and/or electronic documentation to ensure that such decisions are known about and the records remain available if the person travels to a different location, however briefly. Within a hospital that might involve, for example, attending a radiology or physiotherapy department for investigation or treatment. In the community it might involve, for example, attending a healthcare appointment or going out with or visiting friends or family.

When to withhold CPR

When an informed person with capacity refuses CPR as a potential treatment option it should be withheld. If CPR would not re-start the heart and breathing for a sustained period because a person is dying as an inevitable result of underlying disease or a catastrophic health event CPR should be withheld. Although these statements seem relatively straightforward, people may interpret them in different ways. In particular, what constitutes 'futility' in relation to CPR, what constitutes 'successful resuscitation' and what constitutes 'a sustained period' may be viewed differently by individual patients and by individual health professionals.

Many out-of-hospital cardiac arrests are attended by emergency medical technicians or paramedics, who face dilemmas about when CPR will not succeed and when it should be stopped. In general CPR will be started in out-of-hospital cardiac arrest unless there is a valid ADRT refusing it or a record of a valid DNACPR decision. Ambulance service

guidelines allow trained personnel to refrain from starting CPR in defined situations, for example in people with mortal injuries such as decapitation or hemicolectomy, known submersion for more than 1.5 hours, incineration, rigor mortis and hypostasis. In such cases, the ambulance clinician may identify that death has occurred but cannot certify the cause of death (which in most countries can be done only by a doctor or coroner).

Similar recognition that death has occurred and is irreversible and a resulting decision not to start CPR may be made by experienced nurses working in the community or in settings that provide care for people who are terminally or chronically ill. Whenever possible, decisions about CPR should be considered as part of advance care planning before they are needed in a crisis.

Defining ‘success’ and ‘futility’

Achieving return of spontaneous circulation (ROSC) does not mean that CPR has been successful. A resuscitation attempt can only be regarded as successful if it restores a patient to a duration and quality of life that they themselves regard as worth having. Attempted resuscitation can only be regarded as being truly futile if it has no chance of achieving that outcome. These statements emphasise the importance, whenever possible, of knowing a person’s wishes and beliefs in advance.

Predicting outcome

Predicting the outcome from CPR for cardiac arrest is far from easy. The outcome is dependent on many factors, including the prior state of health of the patient and the times from arrest to starting CPR and from arrest to defibrillation. Predictors of non-survival after attempted resuscitation have been published, but do not have sufficient predictive value to be used in general clinical practice.

Avoiding discrimination

Inevitably, clinical judgements have to be made. There will be uncertainties when decisions are needed for patients with conditions such as heart failure, chronic respiratory disease, asphyxia, major trauma, head injury and neurological disease. It is crucial that decisions are non-discriminatory and, for example, do not deprive people of CPR purely on the grounds of factors such as age or a disability. The age of the patient may be considered in the decision-making process but is only a relatively weak independent predictor of outcome. However, many elderly people have significant comorbidity, which influences outcome.

Decisions about implanted cardioverter-defibrillators

When a person who is approaching the end of their life has an implanted cardioverter-defibrillator (ICD), a discussion with them about CPR should prompt also a sensitive discussion about whether and when they may wish to

have the shock function of their ICD deactivated. A proportion of people who die with an active ICD in place will receive shocks from the device in the last hours or days of their life. These are usually painful and can be distressing for the patient and for those close to them. However, it must not be assumed that a DNACPR decision automatically warrants deactivation of an ICD. Some people may wish to have prompt treatment from an ICD, but choose not to have CPR for cardiac arrest, which is much more traumatic, would have a lower chance of success and greater risk of harm. Detailed guidance on this topic has been published by the Resuscitation Council (UK), British Cardiovascular Society and National Council for Palliative Care, together with a short clinical guide and an information leaflet for patients (see reference below).

DNACPR decisions in the context of overall treatment plans

A DNACPR decision means that it is not appropriate to start CPR for cardiorespiratory arrest (unless the circumstances of the arrest are not those envisaged when the decision was recorded). All other care should be given in accordance with the person’s treatment plan and should be of the highest standard. This may include elements of care such as giving nutrition or recording physiological observations, and treatments such as pain relief or sedation. As an ALS provider you should make sure that a properly made and recorded DNACPR decision does not (through your actions or those of others) lead to withholding from a patient of other care or treatment.

It is increasingly common to agree an overall treatment plan with the patient and those close to them that includes not just decisions about CPR but also other treatments such as emergency admission to hospital or to an intensive care unit, non-invasive respiratory support, dialysis, antibiotics, oxygen, and intravenous fluid therapy. When a person’s wishes about these other aspects of their care and treatment are made alongside advance decisions about CPR these other recorded decisions will help to guide decision-making at times of acute illness, should the patient have lost capacity to make a decision at the time of the acute event. These overall treatment plans have a variety of names and approaches for documentation (e.g. Treatment Escalation Plan, Universal Form of Treatment Options).

Deciding to stop CPR

Many resuscitation attempts do not succeed and in those at some point a decision has to be made to stop CPR. This decision can be made when it is clear that continuing CPR will not achieve ROSC or would be contrary to the patient’s previously expressed wishes. Factors influencing the decision will include the patient’s clinical history and prognosis, the cardiac arrest rhythm that is present, the response or absence of response to initial resuscitation measures, and the duration of the resuscitation attempt (particularly if the rhythm is asystole - see below). Sometimes during a resuscitation attempt, information

becomes available that was not known when CPR was started, and indicates that CPR will not succeed. It is appropriate to stop CPR in those circumstances.

In general, CPR should be continued as long as a shockable rhythm or other potentially reversible cause for cardiac arrest persists. It is accepted that asystole for more than 20 min, in the absence of a reversible cause and with all ALS measures applied, is unlikely to be corrected by further CPR and is a reasonable basis for stopping CPR. In some countries, including the UK, paramedics may cease a resuscitation attempt in this situation. Their strict protocol requires that conditions that might indicate a remote chance of survival (e.g. hypothermia) are absent, and the presence of asystole must also be established beyond reasonable doubt and documented on an ECG recording.

A decision to stop CPR should be made by the leader of the resuscitation attempt after consultation with other team members. Ultimately, the decision is based on a clinical judgement that further ALS will not re-start the heart and breathing.

Special considerations

Certain circumstances at the time of cardiac arrest (e.g. hypothermia) enhance the chances of recovery without neurological damage. In such situations do not use prognostic criteria (such as asystole persisting for more than 20 min); continue CPR until the reversible problem has been corrected (e.g. re-warming has been achieved).

Withdrawal of other treatment during the post-resuscitation period

It is difficult to predict the clinical and neurological outcome in people who remain unconscious during the first three days after ROSC. In general, other supportive treatment should be continued during this period, after which the prognosis can be assessed and predicted with greater confidence. This topic is covered in more detail in Chapter 13.

Recording and auditing cardiac arrests and decisions about CPR

Every cardiac arrest is best regarded as a critical clinical event, irrespective of the setting. The decisions made and actions taken by those present must be recorded clearly and accurately. Local audit of such events should take place routinely, allowing recognition of good practice and allowing corrective action where system failures have led to poor or absent decision-making or inappropriate responses. Recording all arrests in one of the national databases (in-hospital: National Cardiac Arrest Audit, out-of-hospital: Out of Hospital Cardiac Arrest Outcomes) will help to identify variation in practice and outcome to try to ensure equality of access to and delivery of treatment.

Health Service Circular 2000/028 states 'NHS Trust chief executives are asked to ensure that appropriate resuscitation policies which respect patients' rights are in

place, understood by all relevant staff, and accessible to those who need them, and that such policies are subject to appropriate audit and monitoring arrangements.' As making anticipatory decisions about CPR is an integral part of good clinical care the decision-making process and the documentation of discussions and decisions about CPR should be audited routinely in all healthcare settings.

Summary learning

- **In the event of cardiac arrest, attempt CPR immediately and effectively unless there is a clear reason to withhold it.**
- **Good communication is essential to ensure that anticipatory decisions about CPR are made well and understood clearly by all those involved.**
- **There should be a presumption in favour of involving patients in discussions about whether or not CPR will be attempted, but there is no requirement to offer to attempt CPR when it will not succeed.**
- **Communicate effectively and sensitively about CPR with patients and with those close to patients.**
- **Communicate decisions about CPR effectively to all health and care professionals involved.**
- **Whenever possible, anticipatory decisions about CPR are best made early (rather than at a time of crisis) and in the context of other choices about a person's care and treatment.**
- **If a valid ADRT refusing CPR has been made this is legally binding; do not attempt CPR unless the circumstances of the arrest are not those envisaged when the ADRT was made.**
- **If a valid DNACPR decision has been recorded do not attempt CPR unless the circumstances of the arrest are not those envisaged when the decision was made.**
- **If CPR will not re-start the heart and breathing do not start CPR.**
- **If continuing CPR will not be successful make the decision to stop.**
- **Record anticipatory decisions about CPR carefully and try to ensure that the record will available when needed.**
- **Do not allow a DNACPR decision to deprive a patient from receiving any other appropriate care or treatment.**

My key take-home messages from this chapter

Further reading

Bossaert L, Perkins GD, Askitopoulou H, et al. European Resuscitation Council Guidelines for Resuscitation 2015 Section 11 The Ethics of Resuscitation and End-of-Life Decisions. *Resuscitation* 2015;95:301-10.

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UK Government. Mental Capacity Act 2005 Code of Practice. <https://www.gov.uk/government/collections/mental-capacity-act-making-decisions>

Supporting relatives in resuscitation practice

Chapter
17

Contents

- The involvement of relatives and friends
- Caring for the recently bereaved
- Staff support and debriefing

Learning outcomes

To enable you to:

- Know how to support relatives witnessing attempted resuscitation
- Know how to care for the recently bereaved
- Consider the religious and cultural requirements when a patient has died
- Consider the legal and practical arrangements following a recent death

Throughout this chapter, the term 'relatives' includes close friends/other people important to the patient.

Introduction

In many cases of out-of-hospital cardiac arrest, the person who performs CPR will be a close friend or relative and they may wish to remain with the patient. Many relatives find it more distressing to be separated from their family member during these critical moments than to witness attempts at resuscitation. In keeping with the move to more open clinical practice, healthcare professionals should take the preferences of patients and relatives into account. If the resuscitation attempt fails, relatives perceive a number of advantages of being present during resuscitation:

- It helps them come to terms with the reality of death, reducing the severity or duration of grief.
- The relative can speak while there is still a chance that the dying person can hear.
- They are not distressed by being separated from a loved one at a time when they feel the need to be present.
- They can see that everything possible was done for the dying person, which helps with their understanding of the reality of the situation.
- They can touch and speak with the deceased while the body is warm.

There are also potential disadvantages to relatives being present:

- The resuscitation attempt may be distressing, particularly if the relatives are not kept informed.
- Relatives may physically or emotionally hinder the staff involved in the resuscitation attempt. Actions or remarks by medical or nursing staff may offend grieving family members.

- Relatives may be disturbed by the memory of events, although evidence indicates that fantasy (about unwitnessed events) is worse than fact (about events that have been seen). The staff should take into account the expectations of the bereaved and their cultural background during and following death.
- Relatives may demonstrate their emotions vocally or physically whilst others may wish to sit quietly or read religious text. The staff must have sufficient insight, knowledge and skills to anticipate individual needs and identify potential problems.

The involvement of relatives and friends

Care and consideration of relatives during resuscitation becomes increasingly important as procedures become more invasive. Support should be provided by an appropriately qualified healthcare professional whose responsibility is to care for family members witnessing cardiopulmonary resuscitation. Adopt the following safeguards:

- Acknowledge the difficulty of the situation. Ensure that they understand that they have a choice of whether or not to be present during resuscitation. Avoid provoking feelings of guilt whatever their decision.
- Explain that they will be looked after whether or not they decide to witness the resuscitation attempt. Ensure that introductions are made and names are known.
- Give a clear explanation of what has happened in terms of the illness or injury and what they can expect to see when they enter the resuscitation area.
- Ensure that relatives understand that they will be able to leave and return at any time, and will always be accompanied.
- Ask relatives not to interfere with the resuscitation process but offer them the opportunity to touch the patient when they are told that it is safe to do so.
- Explain the nature of procedures in simple terms. If resuscitation is unsuccessful, explain why the attempt has been stopped.
- If the patient dies, advise the relatives that there may be a brief interval while equipment is removed, after which they can return to be together in private.

Under some circumstances, the Coroner or other appropriate authority may require certain equipment to be left in place. Offer the relatives time to think about what has happened and the opportunity for further questions.

Hospitals should develop policies to enable relatives to observe attempted resuscitation of their loved one.

Caring for the recently bereaved

Caring for the bereaved compassionately will ease the grieving process. Adopt the following considerations to the

individual family and their cultural needs:

- early contact with one person, usually a nurse
- provision of a suitable area for relatives to wait (e.g. relatives' room)
- breaking bad news sympathetically and supporting the grief response appropriately
- arranging for relatives to view the body
- religious and pastoral care requirements
- legal and practical arrangements
- follow up and team support.

Early contact with one person

Ideally this should be the person who has supported the relatives during the resuscitation attempt. If the resuscitation attempt was not observed allocate a member of the care team specifically to support the relatives. Communication between the emergency services and the receiving hospital should ensure that the arrival of relatives is anticipated for an out-of-hospital arrest. A warm, friendly and confident greeting will help to establish an open and honest relationship.

Provision of a suitable room

This should provide the appropriate ambience, space and privacy for relatives to ask questions and to express their emotions freely.

Breaking bad news and supporting the grief response

An uncomplicated and honest approach will help avoid mixed messages. The most appropriate person (not necessarily a doctor) should break the bad news to the relatives.

It may be more appropriate for the nurse who has been accompanying the relatives to break the news, although relatives may take comfort from talking to a doctor as well and this opportunity should always be offered. When preparing to talk to relatives, consider the following:

- Prepare yourself mentally and physically. Check your clothing (e.g. for blood stains), wash your hands and tidy your clothing.
- Confirm that you are talking with the correct relatives and establish their relationship to the deceased. Briefly establish what they know and use this as the basis for your communication with them.
- Use tone of voice and non-verbal behaviour to support what you are saying. Smiles, nods, eye contact, the use of touch, facial expression and gestures can help support verbal communication.

- Use simple words and avoid platitudes that will be meaningless.
- Sit or position yourself next to the relative on the same level.
- Do not enter into a long preamble about issues such as what the relative wants to know immediately whether alive or dead.
- Introduce the word 'dead', 'died' earliest moment and reinforce further occasion, so that there is no confusion.
- When breaking bad news allow relatives to absorb and think what has been told.
- Anticipate the different types of response you may experience.

Possible responses to grief include:

- acute emotional distress/shock
- anger
- denial/disbelief
- guilt
- catatony.

Individuals may move from one response to another, returning to some repeatedly. The individual's age and cultural background will influence their response to grief. Respect cultural requirements and provide written guidelines for interpretation.

Arranging viewing of the body

Many newly bereaved relatives want to see their loved ones. Their emotional state will be affected by whether the deceased is in an acceptable or presentable condition. Advise the relatives before they view the body, particularly if the deceased has suffered any mutilating injuries or damage. Advise about medical devices and equipment used during resuscitation that may be believed. Being in the physical presence of the deceased will help them work through their grief. Offer the opportunity to touch/hold the body. A family member should accompany relatives on the journey to the mortuary and they should remain near the body until the information is given as required.

Religious requirements and practical arrangements

Variations in handling the body and funerals are influenced by a patient's religion. The resuscitation team should take into account the patient's religious values and rituals of the patient.

- Use simple words and avoid medical jargon and platitudes that will be meaningless to relatives.
- Sit or position yourself next to the relative so that you are on the same level.
- Do not enter into a long preamble or start to question the relative about issues such as premorbid health. They want to know immediately whether their loved one is alive or dead.
- Introduce the word 'dead', 'died' or 'death' at the earliest moment and reinforce this on at least one further occasion, so that there is no ambiguity.
- When breaking bad news allow periods of silence for relatives to absorb and think about what they have been told.
- Anticipate the different types of reaction/emotional response you may experience after breaking bad news.

Possible responses to grief include:

- acute emotional distress/shock
- anger
- denial/disbelief
- guilt
- catatony.

Individuals may move from one stage to another, returning to some repeatedly. An individual's gender, age and cultural background will influence the response to grief. Respect cultural requirements and, where possible, provide written guidelines for individual ethnic groups.

Arranging viewing of the body

Many newly bereaved relatives value the opportunity to view their loved ones. Their experience is likely to be affected by whether the deceased appears in a presentable condition. Advise relatives what to expect before they view the body, particularly if the deceased has suffered any mutilating injuries. People are less concerned about medical devices and equipment than is generally believed. Being in the physical presence of their loved one will help them work through the grieving process. Ensure the opportunity to touch/hold the deceased is given. Staff should accompany relatives during the viewing process and they should remain near by to offer support or provide information as required.

Religious requirements, legal and practical arrangements

Variations in handling the body and expressions of grief are influenced by a patient's religious convictions. The resuscitation team should take into account the beliefs, values and rituals of the patient and the family. There is an

increasing emphasis on the need for care practice to be culturally sensitive, as a way of valuing and respecting the cultural and religious needs of patients. Religious representatives from the patient's denomination or faith are usually available to attend in hospital. Hospital chaplains/spiritual care teams are a great source of strength and information to families and staff. Prayers, blessings, religious acts and procedure are all important in ensuring that relatives are not distressed further. It is also important to respect the views and wishes of those who have no religious belief.

Legal and practical arrangements following death are equally important. These include:

- notification of the coroner or other appropriate authority
- notification of the patient's family doctor
- organ and tissue donation decisions
- provision of information about what to do in the event of death
- involvement of religious advisors
- adherence to hospital procedure about the return of patients' property and valuables
- information concerning social services that are available
- information concerning post-mortem examination where indicated
- follow-up arrangements for relatives, which may involve long-term counselling
- provision of a telephone contact number for relatives to use and a named staff member who they can call should they have any further questions.

Staff support and debriefing

When possible, make arrangements for staff to discuss with the team leader and the rest of the team any issues that may have emerged from the resuscitation event. This can be done individually or as a group and provides a valuable opportunity for reflection. Supporting staff through feedback and reflection can be an extremely powerful learning tool.

Summary learning

- Many relatives want the opportunity to be present during the attempted resuscitation of their loved one. This may help the grieving process.
- Communication with relatives during resuscitation and after bereavement should be honest, simple, and supportive.

My key take-home messages from this chapter

Further reading

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Drug
treat

Drug

Adrenaline

Amiodarone

Drugs used during the treatment of cardiac arrest

Appendix

A

Drug	Shockable (VF/pVT)	Non-Shockable (PEA/Asystole)
Adrenaline	<ul style="list-style-type: none">Dose: 1 mg (10 mL 1:10,000 or 1 mL 1:1,000) IVGiven after the 3rd shock once compressions have been resumedRepeated every 3–5 min (alternate loops)Given without interrupting chest compressions	<ul style="list-style-type: none">Dose: 1 mg (10 mL 1:10,000 or 1 mL 1:1,000) IVGiven as soon as circulatory access is obtainedRepeated every 3–5 min (alternate loops)Given without interrupting chest compressions
Adrenaline has been the primary sympathomimetic drug for the management of cardiac arrest for 45 years. Its alpha-adrenergic effects cause systemic vasoconstriction, which increases coronary and cerebral perfusion pressures. The beta-adrenergic actions of adrenaline (inotropic, chronotropic) may increase coronary and cerebral blood flow, but concomitant increases in myocardial oxygen consumption and ectopic ventricular arrhythmias (particularly in the presence of acidemia), transient hypoxaemia because of pulmonary arteriovenous shunting, impaired microcirculation, and increased post-cardiac arrest myocardial dysfunction may offset these benefits. Although there is no evidence of long-term benefit from the use of adrenaline, the improved short-term survival documented in some studies warrants its continued use pending the outcome of ongoing clinical trials.		
Amiodarone	<ul style="list-style-type: none">Dose: 300 mg bolus IV diluted in 5% dextrose (or other suitable solvent) to a volume of 20 mLGiven during chest compressions after three defibrillation attemptsFurther dose of 150 mg if VF/pVT persists after five defibrillation attempts	<ul style="list-style-type: none">Not indicated for PEA or asystole
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 slowed, and a similar effect is seen with accessory pathways. Amiodarone has a mild negative inotropic action and causes peripheral vasodilation through non-competitive alpha-blocking effects. Although there is no evidence of long-term benefit from the use of amiodarone, it may improve short-term survival warranting its continued use pending the outcome of ongoing clinical trials. Amiodarone should be flushed with 0.9% sodium chloride or 5% dextrose. When amiodarone is unavailable, consider an initial dose of 100 mg ($1\text{--}1.5 \text{ mg kg}^{-1}$) of lidocaine for VF/pVT refractory to three shocks. Give an additional bolus of 50 mg if necessary. The total dose should not exceed 3 mg kg^{-1} during the first hour.		

Drug

Calcium

- Dose: 10 mL 10% calcium chloride (6.8 mmol Ca²⁺) IV
- Indicated for PEA caused specifically by hyperkalaemia, hypocalcaemia or overdose of calcium channel blocking drugs

Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. High plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may impair cerebral recovery. Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.

Sodium Bicarbonate

- Dose: 50 mmol (50 mL of an 8.4% solution) IV
- Routine use not recommended
- Consider sodium bicarbonate in shockable and non-shockable rhythms for
 - cardiac arrest associated with hyperkalaemia
 - tricyclic overdose.
- Repeat the dose as necessary, but use acid-base analysis to guide therapy.

Cardiac arrest results in combined respiratory and metabolic acidosis as 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. Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. This 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.

Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.

Fluids

Infuse fluids rapidly if hypovolaemia is suspected. Use 0.9% sodium chloride or Hartmann's solution, or blood for major haemorrhage. Avoid dextrose, which is redistributed away from the intravascular space rapidly and causes hyperglycaemia, which may worsen neurological outcome and survival after cardiac arrest.

Fibrinolytics

No fibrinolytic drugs are licensed for use in acute pulmonary embolism; however, use of the following drugs is well accepted in cardiac arrest likely to be caused by pulmonary embolism. Drugs are given as a bolus dose during CPR:

- Tenecteplase 500–600 mcg kg⁻¹ IV bolus
- Alteplase (r-tPA) 10 mg IV bolus. Give further doses to a total dose of 50 mg at 15 min and 100 mg by 2 h.

Fibrinolytic therapy should not be used routinely in cardiac arrest. Consider fibrinolytic therapy when cardiac arrest is caused by proven or suspected acute pulmonary embolus. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts. Ongoing CPR is not a contraindication to fibrinolysis.

Drugs used in the peri-arrest period

Patients given these drugs should be closely monitored and managed by clinicians experienced in their use.

Appendix

B

Drug	Indication	Dose
Adenosine	<ul style="list-style-type: none">Paroxysmal SVT with re-entrant circuits that include the atrioventricular (AV) node (AVNRT and AVRT)	<ul style="list-style-type: none">6 mg IV bolusIf unsuccessful, give up to two further doses of 12 mg after 1-2 min intervals
Adenosine is a naturally occurring purine nucleotide. It blocks transmission through the AV node but has little effect on other myocardial cells or conduction pathways. It has an extremely short half-life of 10–15 s and, therefore, is given as a rapid bolus into a fast running intravenous infusion or followed by a saline flush. Warn patients of transient unpleasant side effects; in particular, nausea, flushing, and chest discomfort.		
Adrenaline	<ul style="list-style-type: none">Second-line treatment for cardiogenic shockBradycardia (alternative to external pacing)Anaphylaxis	<ul style="list-style-type: none">0.05–1 mcg kg⁻¹ min⁻¹2–10 mcg min⁻¹0.5 mg IM for anaphylaxis and repeated every 5 min (Chapter 12)50 mcg IV bolus doses titrated effect.
An adrenaline infusion is indicated in the post-resuscitation period when less potent inotropic drugs (e.g. dobutamine) have failed to increase cardiac output adequately. It is indicated also for bradycardia associated with adverse signs and/or risk of asystole, which has not responded to atropine, if external pacing is unavailable or unsuccessful.		
Amiodarone	<ul style="list-style-type: none">Control of haemodynamically stable monomorphic VT, polymorphic VT and wide-complex tachycardia of uncertain originTo control a rapid ventricular rate caused by accessory pathway conduction in pre-excited atrial arrhythmias (e.g. atrial fibrillation (AF)) and/or achieve chemical cardioversionAfter unsuccessful electrical cardioversion, to achieve chemical cardioversion or to increase the likelihood of further electrical cardioversion succeeding	<ul style="list-style-type: none">300 mg IV over 10–60 min (depending on haemodynamic stability of patient)Followed by 900 mg over 24 h
Intravenous amiodarone has effects on sodium, potassium and calcium channels as well as alpha- and beta-adrenergic blocking properties. In patients with severely impaired heart function, intravenous amiodarone is preferable to other anti-arrhythmic drugs for atrial and ventricular tachyarrhythmias. Major adverse effects (caused by the solvent, not the active drug) are hypotension and bradycardia, which can be minimised by slowing the rate of drug infusion. Whenever possible, intravenous amiodarone should be given via a central venous catheter; it causes thrombophlebitis when infused into a peripheral vein, but in an emergency it can be injected into a large peripheral vein.		

Drug	Indication	Dose
Aspirin	<ul style="list-style-type: none"> Acute coronary syndromes <p>Aspirin improves the prognosis of patients with acute coronary syndromes, significantly reducing cardiovascular death. The efficacy of aspirin is achieved by anti-platelet activity and preventing early platelet thrombus formation.</p>	<ul style="list-style-type: none"> 300 mg orally, crushed or chewed, as soon as possible
Atropine	<ul style="list-style-type: none"> Sinus, atrial, or nodal bradycardia or AV block, when the haemodynamic condition of the patient is unstable because of the bradycardia. <p>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 AV node, increasing sinus automaticity and facilitating AV node conduction. Side effects of atropine are dose-related (blurred vision, dry mouth and urinary retention). It can cause acute confusion, particularly in elderly patients. Asystole during cardiac arrest 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.</p>	<ul style="list-style-type: none"> 500 mcg IV Repeated doses to maximum of 3 mg
Beta-blockers	<ul style="list-style-type: none"> Narrow-complex regular tachycardias by vagal manoeuvres or adenosine in patients with preserved ventricular function To control rate in atrial AF and atrial flutter when ventricular function is preserved. <p>Beta-blocking drugs reduce the effects of circulating catecholamines and decrease heart rate and blood pressure. They also have cardioprotective effects for patients with acute coronary syndromes. Side effects of beta blockade include bradycardia, AV conduction delay, hypotension and bronchospasm. Contraindications to the use of beta-adrenoceptor blocking drugs include second- or third-degree heart block, hypotension, severe congestive heart failure and lung disease associated with bronchospasm.</p>	<p>Atenolol (β_1)</p> <ul style="list-style-type: none"> 5 mg IV over 5 min, repeated if necessary after 10 min <p>Metoprolol (β_1)</p> <ul style="list-style-type: none"> 2–5 mg IV at 5-min intervals to a total of 15 mg <p>Propranolol (β_1 and β_2 effects)</p> <ul style="list-style-type: none"> 100 mcg kg⁻¹ IV slowly in three equal doses at 2–3 min intervals <p>Esmolol</p> <ul style="list-style-type: none"> short-acting (half-life of 2–9 min) β_1-selective beta-blocker IV loading dose of 500 mcg kg⁻¹ over 1 min followed by an infusion of 50–200 mcg kg⁻¹ min⁻¹
Verapamil	<ul style="list-style-type: none"> Stable regular narrow-complex tachycardias uncontrolled or unconverted by vagal manoeuvres or adenosine. To control ventricular rate in patients with AF or atrial flutter and preserved ventricular function when the duration of the arrhythmia is less than 48 h. <p>Verapamil is a calcium channel blocking drug that slows conduction and increases refractoriness in the AV node. These actions may terminate re-entrant arrhythmias and control the ventricular response rate in patients with atrial tachycardias (including AF and atrial flutter). Intravenous verapamil should be given only to patients with narrow-complex paroxysmal SVT or arrhythmias known with certainty to be of supraventricular origin. Giving calcium channel blockers to a patient with ventricular tachycardia may cause cardiovascular collapse. Verapamil may decrease myocardial contractility and critically reduce cardiac output in patients with severe LV dysfunction.</p>	<ul style="list-style-type: none"> 2.5–5 mg IV given over 2 min In the absence of a therapeutic response or induced adverse event, give repeated doses of 5–10 mg every 15–30 min to a maximum of 20 mg

Drug	Indication	Dose
Digoxin	<ul style="list-style-type: none"> Atrial fibrillation with fast ventricular response <p>Digoxin is a cardiac glycoside that slows ventricular rate by increasing vagal tone, decreasing sympathetic activity by suppression of baroreceptors, and prolonging AV node refractory period.</p>	<ul style="list-style-type: none"> 500 mcg IV over 30 min
Positive inotropic drugs	<ul style="list-style-type: none"> Hypotension in the absence of hypovolaemia Cardiogenic shock 	<p>Dobutamine 5-20 mcg kg⁻¹ min⁻¹</p> <p>Dopamine 1-10 mcg kg⁻¹ min⁻¹</p> <p>Noradrenaline 0.05-1 mcg kg⁻¹ min⁻¹</p>
	<p>Dobutamine is often the positive inotropic drug of choice in the post-resuscitation period. Its beta agonist activity also causes vasodilation and an increase in heart rate. It is indicated when poor cardiac output and hypotension cause significantly reduced tissue perfusion. It is useful particularly when pulmonary oedema is present and hypotension prevents the use of other vasodilators.</p> <p>Dopamine is the precursor of the naturally occurring catecholamines adrenaline and noradrenaline. It has a dose dependent positive inotropic effect.</p> <p>Noradrenaline is a potent vasoconstrictor but also has a positive inotropic effect. It is indicated in the post resuscitation period when hypotension and poor cardiac output cause reduced tissue perfusion.</p>	
Magnesium	<ul style="list-style-type: none"> Polymorphic ventricular tachycardia (torsade de pointes) Digoxin toxicity 	<ul style="list-style-type: none"> Dose: 2 g IV over 10 min May be repeated once if necessary Correction of hypomagnesaemia
	Magnesium facilitates neurochemical transmission: it decreases acetylcholine release and reduces the sensitivity of the motor endplate.	
Nitrates	<ul style="list-style-type: none"> Prophylaxis or relief of angina Unstable angina pectoris Myocardial infarction Acute and chronic left ventricular failure 	<ul style="list-style-type: none"> Glyceryl trinitrate (GTN): 1-2 doses of sublingual 300 mcg tablet or 400 mcg spray GTN IV infusion at 10-200 mcg min⁻¹ Isosorbide dinitrate IV infusion at 2-10 mg h⁻¹
	<p>After conversion to nitric oxide, nitrates cause vascular smooth muscle relaxation. The resultant dilation is more marked on the venous than the arterial side of the circulation, and it is this venodilation, reducing left ventricular diastolic pressure, that is mainly responsible for relieving angina. Nitrates also dilate the coronary arteries and relieve spasm in coronary smooth muscle. Nitrates are contraindicated in hypotensive patients (systolic blood pressure < 90 mmHg).</p>	