Immediate Life Support

ERC GUIDELINES 2015 EDITION
Immediate Life Support
Course Manual

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Glossary

Throughout this publication the masculine pronouns he, him and his are used generically.

AED  Automated External Defibrillator
ABCDE  Airway, Breathing, Circulation, Disability, Exposure approach
CPR  Cardiopulmonary Resuscitation – this refers to chest compressions and ventilations
ECG  Electrocardiogram
EMS  Emergency Medical Service, e.g. Ambulance Service
IV  Intravenous
IO  Intraosseous
PEA  Pulseless Electrical Activity
ROSC  Return of Spontaneous Circulation
SBAR  A communication tool – Situation, Background, Assessment, Recommendation
VF  Ventricular Fibrillation
VT  Ventricular Tachycardia
VF/VT  VF/pulseless VT
<  Less than
>  Greater than
Chapter 0.

Introduction

The Immediate Life Support (ILS) course provides healthcare staff with the essential knowledge and skills needed to treat adult patients in cardiorespiratory arrest for the short time before the arrival of a resuscitation team or more experienced assistance. It also prepares healthcare staff to be members of a resuscitation team.

Many cardiorespiratory arrests are preventable. ILS teaches the recognition and treatment of the deteriorating patient using the ABCDE (Airway, Breathing, Circulation, Disability and Exposure) approach. ILS knowledge and skills will enable first responders to recognise and start treatment in patients at risk of deterioration and cardiac arrest. If cardiorespiratory arrest does occur, the skills covered are those that are most likely to resuscitate the patient.

This manual is predominantly about the resuscitation of patients in the acute hospital setting. The same principles, however, apply to the resuscitation of patients in any clinical setting, e.g. community hospital.

The interventions that contribute to survival after a cardiac arrest can be seen as a chain – the Chain of Survival.

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 – to prevent cardiac arrest
- Early cardiopulmonary resuscitation (CPR) – to buy time
- Early defibrillation – to restart the heart
- Post resuscitation care – to restore quality of life

The ILS course will teach you the important knowledge and skills for each link in the Chain of Survival.

ILS teaches how to start cardiopulmonary resuscitation in the clinical setting. This includes the importance of good quality chest compressions and ventilation with minimal interruption, and early safe defibrillation.
There is now a much greater emphasis on non-technical skills and human factors. These are the things that affect your personal performance such as situational awareness, decision making, team working and task management.

**Figure 0.1**  
Chain of Survival
Non-technical skills and quality in resuscitation

LEARNING OUTCOMES
To understand:
• the role of human factors in resuscitation
• how to use structured communication tools such as SBAR and RSVP
• the role of safety incident reporting and audit to improve patient care

1. Introduction
Skills such as defibrillation, effective chest compressions, ability to ventilate, recognition of the underlying cardiac arrest rhythm, which are all important components of successful resuscitation, are usually termed technical skills. Despite the fact that there is little disagreement that these skills are necessary for human resuscitation, recently another category of skills and factors emerged. Non-technical skills are the cognitive and interpersonal skills that underpin effective team work and it is estimated that between 70-80% of healthcare error(s) can be attributed to a breakdown in these skills1,2. Non-technical skills include the interpersonal skills of communication, leadership and followership (being a team member) and the cognitive skills of decision making, situation awareness and task management3. Non-technical skills are part of the human factors agenda1,5.

Introduction and formal training in human factors and non-technical skills has led to a significant reduction of accidents of aviation and it was only recently that medicine acknowledged the importance of these skills. There is little doubt that the pioneers in this were the anaesthesiologists with the development of formal courses but surgeons and other specialties started to take interest in these skills. There are several systems that have been developed to ensure acceptable use of non-technical skills, such as the team dimension rating form, the Oxford Non-Technical Skills Measure, just to name a few. The principles used to promote good non-technical skills in the ILS course are based on Team Emergency Assessment Measure9. The proposed Taxonomy in NTS, adopted by the ERC is illustrated in table 1.
In the medical context, a team is a group of healthcare professionals with different skills and different backgrounds working together to achieve a common goal. The leader is an integral part of the team, but each team member is equally important in the team performance. Each team member contributes to the teamwork with a certain role and skill. Therefore, the leader, as well as the team members, contribute to the completeness and integrity of a team. This requires different healthcare professions to be aware of various roles and competences throughout the team, determining the appreciation of their role and level of skills, as well as of other team members present during the process of handling the situation.

Healthcare professionals trained in immediate life support cover the period of time until the arrival of a medical emergency team. When competent with their technical skills, ILS provider should already facilitate inter-professional communication and team structure until expert help arrives on scene. Building up a team structure in the first period of a medical emergency requires ILS-trained healthcare professionals to be aware of basic principles regarding non-technical skills, involving leadership, teamwork and task management.

2. Leadership

An effective leader is the person with a global perspective of the situation s/he is facing and as a result s/he allocates roles to various team members in order to accomplish the global perspective of the leader. Medical literature agrees that leadership is not a trait, but it can be accomplished with continuous training. Leadership skills are complex and can differ in style, presenting a wide range from directional to supportive leadership. There is no gold standard for an optimal style of leadership, as it depends on context and situation. In resuscitation teams, the team leader needs to:

1. Let the team know exactly what is expected from them. This entails a high level of situational awareness and ability to allocate tasks according to the team-members’ experience and bases his/her decision making process using evidence based medicine, clearly verbalizing his/her decisions. A good team leader always knows and addresses his/her team by name and can act as a role model for the team to evolve.

2. Maintain a high level of global perspective. In reality this means putting a plan that the leader has into action. As team-members perform their tasks, the team leader carefully monitors whether these are being performed. In the setting of cardiopulmonary resuscitation, the team leader should always be able to hear what information the team-members are relaying to him/her. Consecutively, the leader should be able not just to monitor the clinical procedures as they are performed, but also to be able to provide guidance as the procedures occur, remaining “hands free”. Safe practice of all procedures is the responsibility of the leader, not just for the patient, but for the team of healthcare professionals working together. The team leader should also be empathic to other healthcare professionals and should possess inter-professional communication skills.
<table>
<thead>
<tr>
<th>LEADERSHIP</th>
<th>Not seen (✓)</th>
<th>Observed (✓)</th>
</tr>
</thead>
<tbody>
<tr>
<td>The team leader let the team know what was expected of them through direction and command.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: uses members names, allocates tasks, makes clear decisions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team leader maintained a global perspective.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: monitors clinical procedures, checks safety, plans ahead, remains ‘hands off’</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TEAMWORK</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team communicated effectively, using both verbal and non-verbal communication.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: relay findings, raise concerns, use names, appropriate body language</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team worked together to complete tasks in a timely manner.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: coordination of defibrillation, maintain chest compressions, assist each other</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team acted with composure and control.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: performed allocated roles, accept criticism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team adapted to changing situations.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: adapt to rhythm changes, patient deterioration, change of roles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team monitored and reassessed the situation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: rhythm changes, ROSC, when to terminate resuscitation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team anticipated potential actions.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: defibrillation, airway management, drug delivery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TASK MANAGEMENT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team prioritised tasks.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: continuous chest compressions, defibrillation, airway management, drug delivery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The team followed approved standards/guidelines.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COMMENTS</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Examples</strong>: What area was good? What area needs improvement?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3. **Teamwork**

Teamwork is one of the most important NTS that can contribute to the management of a cardiac arrest patient. Clinical competence and clinical experience are important for the outcome of resuscitation, but not a guarantee of success\(^{10}\). Teamwork has to be learnt and practiced in various settings to enhance resuscitation team performance\(^{11,12}\). The key elements for effective team performance are:

1. Effective verbal and non-verbal communication. The team needs to relay the findings as they occur and they should be able to understand what the leader’s plan is, carrying out the allocating tasks and always closing the communicational loop. The team members should be able to raise concerns, but they should always filter the information they provide. In cardiac arrest management, several things are happening simultaneously and effective communication needs practice within this setting. The ILS course is such a training opportunity for teams to practice effective communication.

2. Working together to complete tasks in a timely manner. Time is important in CPR and team co-ordination is extremely important for the safe delivery of defibrillation, as well as maintaining high quality chest compressions throughout the resuscitation attempt.

3. Adaptation to changing situations. Cardiac arrest management is a dynamic procedure. Cardiac arrest patients are by definition extremely unstable even if they achieve Return of Spontaneous Circulation (ROSC). During CPR the team should be comfortable to changing roles (e.g. alternation of the airway person with the compressor) and they should be able to adapt to rhythm changes, when these occur.

4. Reassessment of the situation. In CPR this means not only continuous reassessment of the patient but also a consensus on when resuscitation attempts should be terminated. The ERC guidelines provide clear guidance on when resuscitation should be terminated.

5. Anticipation of potential actions. In CPR this entails preparation for airway management, preparation and delivery of drugs, preparation and energy selection for defibrillation. Therefore, situation awareness, which requires knowledge of common algorithms and accepted guidelines, is needed, in order to anticipate potential actions as a team member.

4. **Task management**

During the resuscitation of any patient, either in the peri-arrest or full cardiac arrest situation, there are numerous tasks that should be carried out by team members. These include:

1. Prioritization of the tasks that should be performed either simultaneously or sequentially.

2. Adhering to current and approved guidelines and practices.
5. **The importance of communication when managing a sick patient**

Communication includes seeking and reporting information. During CPR, communication between team members can be verbal and non-verbal, as well as informal and structured. Of note, cardiac arrest teams may face many kinds of communication challenges at the professional, organizational, team, personal and/or patient, levels which may affect the quality of CPR.

Effective teamwork and communication skills are critical success factors during CPR; poor communication will decrease team’s effectiveness and survival rates. This usually happens due to inconsistency of team members from day to day which seriously affects communication skills. Consequently, optimization of team communication can be optimized through high-quality training, during which concepts and applications for effective team communication can be implemented, focusing on several approaches, team interaction, and relationship management.

Individual team members, regardless of the member’s position, must learn to perceive orders and accept their roles as non-intimidating. Team orientation should be built by taking steps to increase trust and cohesion and increase satisfaction, commitment, and collective efficacy. Although increasing awareness of different communication styles and possibly incorporating these skills into medical training may help teams arrive more efficiently at jointly managed efforts during CPR, precise and accurate communication through a closed-loop communication protocol should be always encouraged. Use of SBAR during written and verbal communication, active listening, body language, and tone of voice may also help team members to recognize and understand individual personal styles, preferences and temperament types. Appreciating others’ differences will enhance the approach between team members and increase team efficiency.

6. **Audit and registry reporting**

Regarding CPR, hospital-level infrastructure may comprise a coordinating resuscitation committee and resuscitation teams (1). This enables periodic, multilevel, clinical cardiac arrest audit, aimed at continuous improvement of the resuscitation service. The audit pertains to the availability and use of resuscitation/peri-arrest drugs and resuscitation equipment, the always prompt activation in case of in-hospital cardiopulmonary arrest, the documentation of management using the Utstein template and relevant audit forms, do-not-attempt resuscitation decisions and policies, outcomes, critical incidents leading to or occurring during CPR, and various safety/logistical issues (e.g. decontamination/maintenance of training/resuscitation equipment).

Local CPR management can be improved through post-CPR debriefing aimed at determining CPR quality errors and mitigating their repetition during subsequent CPR attempts. Examples of such errors include low-rate and/or shallow chest compressions, prolonged interruptions of chest compressions, and excessive ventilation. Institutions should also be encouraged to submit CPR data in standardized format to national audits and/or international registries aimed at continuous quality improvement. Such practices
### Table 1.2. SBAR and RSVP communication tools

<table>
<thead>
<tr>
<th>SBAR</th>
<th>RSVP</th>
<th>Content</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Situation</td>
<td>Reason</td>
<td>• Introduce yourself and check you are speaking to the correct person.</td>
<td>• Hello, I am Dr Smith the junior medical doctor.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Identify the patient you are calling about (who and where).</td>
<td>• I am calling about Mr Brown on acute medical admissions who I think has a severe pneumonia and is septic.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Say what you think, the current problem is, or appears to be.</td>
<td>• He has an oxygen saturation of 90% despite high-flow oxygen and I am very worried about him.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• State what you need advice about.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Useful phrases:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- The problem appears to be cardiac/respiratory/neurological/sepsis.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- I’m not sure what the problem is but the patient is deteriorating.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- The patient is unstable, getting worse and I need help.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hello, I am Dr Smith the junior medical doctor.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• I am calling about Mr Brown on acute medical admissions who I think has a severe pneumonia and is septic.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• He has an oxygen saturation of 90% despite high-flow oxygen and I am very worried about him</td>
<td></td>
</tr>
<tr>
<td>Background</td>
<td>Story</td>
<td>• Background information about the patient</td>
<td>• He is 55 and previously fit and well.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Reason for admission</td>
<td>• He has had fever and a cough for 2 days.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Relevant past medical history</td>
<td>• He arrived 15 minutes ago by ambulance.</td>
</tr>
<tr>
<td>Assessment</td>
<td>Vital</td>
<td>• Include specific observations and vital sign values based on ABCDE approach:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Signs</td>
<td>- Airway</td>
<td>• He looks very unwell and is becoming tired.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Breathing</td>
<td>• Airway - he can say a few words.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Circulation</td>
<td>• 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.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Disability</td>
<td>• Circulation - his pulse is 110, his blood pressure is 110/60.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Exposure</td>
<td>• Disability - he is drowsy but can talk a few words.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- The early warning score is…</td>
<td>• Exposure - he has no rashes.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recommendation</td>
<td>Plan</td>
<td>• State explicitly what you want the person you are calling to do.</td>
<td>• I am getting antibiotics ready and he is on IV fluids.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• What by when?</td>
<td>• I need help - please can you come and see him straight away.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Useful phrases:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- I am going to start the following treatment; is there anything else you can suggest?</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- I am going to do the following investigations; is there anything else you can suggest?</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- If they do not improve; when would you like to be called?</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- I don’t think I can do any more; I would like you to see the patient urgently</td>
<td></td>
</tr>
</tbody>
</table>
have already led to the development of validated outcome-predictive models, which may facilitate advanced care planning. In addition, a prior registry data analysis quantified the frequency of resuscitation system errors and their impact on in-hospital mortality after shockable and non-shockable cardiac arrest. Registry results have shown significant improvements in cardiac arrest outcomes within 2000-2010.

Published evidence suggests that resuscitation team-based infrastructure, multilevel institutional audit, accurate reporting of resuscitation attempts at national audit level and/or multinational registry level, and subsequent data analysis and feedback from reported results may contribute to continuous improvement of in-hospital CPR quality and cardiac arrest outcomes.

7. High-quality care

Quality care can be described as safe, effective, patient-centred, timely, efficient and equitable. Hospitals, resuscitation teams and ILS providers should ensure they deliver these aspects of quality to improve the care of the deteriorating patient and patients in cardiac arrest. Two aspects of this are safety incident reporting (also called adverse or critical incident reporting) and collecting good quality data.

7.1. Safety incident reporting

There are a number of critical incident reporting systems throughout Europe. For example, in England and Wales hospitals can report patient safety incidents to the National Patient Safety Agency (NPSA) National Reporting and Learning System (NRLS) (http://www.nrls.npsa.nhs.uk/report-a-patient-safetyincident/).

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)”. Previous reviews of this database have identified patient safety incidents associated with airway devices in critical care units and led to recommendations to improve safety. A review of NPSA safety incidents relating to cardiac arrest and patient deterioration by the Resuscitation Council (UK) shows that the commonest reported incidents are associated with equipment problems, communication, delays in the resuscitation team attending, and failure to escalate treatment.

7.2. Collecting good quality data

Most European countries have a national audit for in- and out-of-hospital cardiac arrests. These audits monitor and report on the incidence of, and outcome from, cardiac arrest in order to inform practice and policy. They aim to identify and foster improvements in the prevention, care delivery and outcomes from cardiac arrest.

Data are usually collected according to standardised definitions and entered onto secure web-based systems.

Once data are validated, participants are provided with activity reports and comparative reports, allowing a comparison of to be made not only within, but also between, systems locally, nationally and internationally.

Furthermore it also enables the effects of introducing changes to guidelines, new drugs, new techniques etc to be monitored that would not be possible on a single participant basis.
KEY LEARNING POINTS

• Human factors are important during resuscitation
• Use SBAR or RSVP for effective communication.
• Report safety incidents and collect cardiac arrest data to help improve patient care.

REFERENCES

Chapter 2.

Recognition of the Deteriorating Patient and Prevention of Cardiorespiratory Arrest

LEARNING OUTCOMES
To understand:
- the importance of early recognition of the deteriorating patient
- the causes of cardiorespiratory arrest in adults
- how to identify and treat patients at risk of cardiorespiratory arrest using the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach

1. Introduction

Early recognition of the deteriorating patient and prevention of cardiorespiratory arrest is the first link in the chain of survival. Once cardiac arrest occurs, fewer than 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.

Survivors from in-hospital cardiac arrest usually have a witnessed and monitored ventricular fibrillation (VF) arrest, primary myocardial ischaemia as the cause, and receive immediate and successful defibrillation e.g. on the coronary care unit.

Most in-hospital cardiac arrests are not sudden or unpredictable events. In approximately 80% of cases clinical signs deteriorate over the few hours before arrest. These patients often have slow, progressive physiological deterioration; often hypoxia and hypotension are either not noticed by staff, or are recognised but treated poorly. The cardiac arrest rhythm in these patients is usually non-shockable (pulseless electrical activity (PEA) or asystole) and very few of them survive and leave hospital.

Early recognition and effective treatment of the deteriorating patient might prevent cardiac arrest, death or an unanticipated intensive care unit (ICU) admission. Early recognition will also help to identify individuals for whom cardiopulmonary resuscitation is not appropriate or who do not wish to be resuscitated.
Much of this chapter is based on the deteriorating patient in the hospital setting. The same basic principles however apply to the care of the deteriorating patient in the out-of hospital setting.

2. **Prevention of in-hospital cardiac arrest: the Chain of Prevention**

The Chain of Prevention can help hospitals structure care processes to prevent and detect patient deterioration and cardiac arrest. The five rings of the chain *(figure 2.1)* represent:

- **Education** – how to observe patients; interpretation of observed signs; the recognition of signs of deterioration; and the use of the ABCDE approach and simple skills to stabilise the patient pending the arrival of more experienced help.

- **Monitoring** – patient assessment and the measurement and recording of vital signs, which may include the use of electronic monitoring 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. Continuing training of the interprofessional teams in non-technical skills leads to improve communication efficiency. 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, e.g. from an outreach or ICU team.
Table 2.1
Example of early warning scoring (EWS) system*


<table>
<thead>
<tr>
<th>Score</th>
<th>3</th>
<th>2</th>
<th>1</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse (min⁻¹)</td>
<td>≤ 40</td>
<td>41-50</td>
<td>51-90</td>
<td>91-110</td>
<td>111-130</td>
<td>≥ 131</td>
<td></td>
</tr>
<tr>
<td>Respiratory rate (min⁻¹)</td>
<td>≤ 8</td>
<td>9-11</td>
<td>12-20</td>
<td>21-24</td>
<td>≥ 25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>≤ 35.0</td>
<td>35.1-36.0</td>
<td>36.1-38.0</td>
<td>38.1-39.0</td>
<td>≥ 39.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>≤ 90</td>
<td>91-100</td>
<td>101-110</td>
<td>111-249</td>
<td>≥ 250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxygen saturation (%)</td>
<td>≤ 91</td>
<td>92-93</td>
<td>94-95</td>
<td>≥ 96</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inspired oxygen</td>
<td>Air</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVPU</td>
<td>Alert (A)</td>
<td></td>
<td></td>
<td></td>
<td>Voice (V)</td>
<td>Pain (P)</td>
<td>Unresponsive (U)</td>
</tr>
</tbody>
</table>

3. 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 among patients on general wards, but important physiological observations of acutely ill patients are not done as often as they should.

To help early detection of deteriorating patients, many hospitals use early warning scores (EWS). The 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 calling ward doctors or resuscitation teams to the patient. An example of an EWS system is shown in table 2.1.

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 help should be called for early rather than waiting for the patient to reach a specific score.

The patient’s EWS is calculated based on table 2.1.

An increased score indicates an increased risk of deterioration and death. There should be a graded response to scores according to local hospital protocols. An example of an escalation protocol is shown in table 2.2.
Alternatively, systems incorporating calling criteria are based on routine observations, which activate a response when one or more observations reach an extremely abnormal value. It is not clear which of these two systems is better.

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.

The traditional response to cardiac arrest is reactive: the name ‘cardiac arrest team’ implies that it will be called only when at least one vital function is absent or after cardiac arrest has occurred. In some hospitals the cardiac arrest team has been replaced by other resuscitation teams (e.g. rapid response team, critical care outreach team, medical emergency team). These teams can be activated according to the patient’s EWS (see above) or according to specific calling criteria. For example, the medical emergency team (MET) responds not only to patients in cardiac arrest, but also to those with acute physiological deterioration. The MET usually comprises medical and nursing staff from intensive care and general medicine and responds to specific calling criteria (table 2.3). Any member of the healthcare team 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-resuscitation [DNAR] decisions). Medical emergency team interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids. MET interventions have been associated with a reduced incidence of cardiac/respiratory arrest and improved survival rates.

Table 2.2
Example escalation protocol based on early warning score (EWS)

<table>
<thead>
<tr>
<th>EWS</th>
<th>MINIMAL observation frequency</th>
<th>Escalation</th>
<th>Doctor’s action</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-5</td>
<td>4 hourly</td>
<td>Inform nurse in charge</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>4 hourly</td>
<td>Inform doctor</td>
<td>Doctor to see within 1 hour</td>
</tr>
<tr>
<td>7-8</td>
<td>1 hourly</td>
<td>Inform doctor</td>
<td>Doctor to see within 30 minutes and discuss with senior doctor and/or outreach team</td>
</tr>
<tr>
<td>≥9</td>
<td>30 minutes</td>
<td>Inform doctor</td>
<td>Doctor to see within 15 minutes and discuss with senior doctor and ICU team</td>
</tr>
</tbody>
</table>

Critically ill patients should be admitted to a critical care area e.g. ICU, intermediate care unit, high dependency unit (HDU), or resuscitation room. These areas should be staffed by doctors and nurses experienced in advanced resuscitation and critical care skills.

There are fewer hospital staff on duty 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.
Patients discharged from ICUs to general wards at night have an increased risk of in-hospital death compared with those discharged during the day and those discharged to HDUs.

<table>
<thead>
<tr>
<th>MET calling criteria</th>
<th>Airway</th>
<th>Breathing</th>
<th>Circulation</th>
<th>Neurology</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Threatened</td>
<td>All respiratory arrests</td>
<td>All cardiac arrests</td>
<td>Sudden decrease in level of consciousness</td>
<td>Any patient causing concern who does not fit the above criteria</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Respiratory rate &lt; 5 min⁻¹</td>
<td>Pulse rate &lt; 40 min⁻¹</td>
<td>Decrease in GCS of &gt; 2 points</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Respiratory rate &gt; 36 min⁻¹</td>
<td>Pulse rate &gt; 140 min⁻¹</td>
<td>Repeated or prolonged seizures</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Systolic blood pressure &lt; 90 mmHg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### 4. Causes of deterioration and cardiorespiratory arrest

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

#### 4.1. Airway obstruction

For a detailed review of airway management see chapter 5.

#### 4.1.1. Causes

Airway obstruction can be partial or complete. Partial obstruction often precedes complete obstruction, which rapidly leads to cardiac arrest. Partial airway obstruction may lead to cerebral or pulmonary oedema, exhaustion, secondary apnoea, and hypoxic brain damage, and eventually to cardiac arrest.
Chapter 2
Recognition of the Deteriorating Patient and Prevention of Cardiorespiratory Arrest

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

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. hypoglycaemia in diabetic patients), and drugs, including alcohol, opioids and general anaesthetic agents. 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 they sleep (obstructive sleep apnoea). This is more common in obese patients and obstruction can be made worse by other factors (e.g. sedative drugs).

4.1.2. 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 but any respiratory efforts 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, with the opposite seen on expiration.
4.1.3. Treatment

The priority is to ensure that the airway remains patent. Treat any problem that places the airway at risk; for example, suction blood and gastric contents from the airway and, unless contraindicated, turn the patient on their side. Assume actual or impending airway obstruction in anyone with a depressed 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, and elective tracheal intubation or tracheostomy.

Use a nasogastric tube to empty the stomach of a patient with a decreased consciousness level.

Give oxygen as soon as possible to achieve an arterial blood oxygen saturation by pulse oximetry ($\text{SpO}_2$) in the range of 94–98%.

In case of suspected or actual cervical trauma, follow local protocols for the rachis protection and limitation of cervical motions.

4.2. Breathing problems

4.2.1. Causes

Breathing inadequacy may be acute or chronic. It may be continuous or intermittent, and severe enough to cause the person to stop breathing (apnoea or respiratory arrest). This will rapidly lead to a secondary cardiac arrest if not treated.

Respiratory arrest often arises from a combination of factors. In a patient with chronic respiratory inadequacy, a chest infection, muscle weakness, or fractured ribs may lead to exhaustion, further depressing respiratory function. If breathing is insufficient to oxygenate the blood adequately, lack of oxygen to the vital organs will lead to loss of consciousness and eventually cardiac arrest.

- **Respiratory drive**
  Central nervous system depression can 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 spinal 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**
Severe lung disease will impair gas exchange. Causes include infection, exacerbation of Chronic Obstructive Pulmonary Disease (COPD), asthma, pulmonary embolus, lung contusion, Acute Respiratory Distress Syndrome (ARDS) and pulmonary oedema. Lung function is also impaired by a pneumothorax or haemothorax.

A tension pneumothorax causes a rapid failure of gas exchange, a reduction of venous return to the heart, arrhythmias and a fall in blood pressure.

### 4.2.2. Recognition

Conscious patients will complain of shortness of breath and be distressed. The history and examination will usually point to the underlying cause. Hypoxia and hypercarbia can cause irritability, confusion, lethargy and depressed consciousness. Cyanosis is a late sign. A fast respiratory rate (> 25 min⁻¹) is a useful, simple indicator of breathing problems. Pulse oximetry is an easy, non-invasive measure of the adequacy of oxygenation (see Appendix 1). However, it is not a reliable indicator of ventilation. Arterial blood gas measurement is needed to assess adequate ventilation. A rising arterial carbon dioxide tension (PaCO₂) indicates hypoventilation.

### 4.2.3. 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, titrate the oxygen flow (use the appropriate oxygen therapy device) and aim for a SpO₂ in the range of 94-98 % (88-92 % for a COPD status). For example, give early IV antibiotics to a patient with a severe pneumonia or start bronchodilator (salbutamol nebulisers) and steroid treatment for a patient with severe asthma.

Patients who are having difficulty breathing or are becoming tired will need help with their breathing. Non-invasive ventilation using a face mask can be useful and prevent the need for tracheal intubation and ventilation. It is best to call for expert help early for patients who cannot breathe adequately as ICU admission for sedation, tracheal intubation and controlled ventilation may be needed.

### 4.3. Circulation problems

#### 4.3.1. Causes

Circulation problems may be caused by primary heart disease or by heart abnormalities secondary to other problems. The heart may stop suddenly or may produce an inadequate cardiac output for a while before stopping.
• **Primary heart problems**

Sudden cardiac arrest is most commonly caused by an arrhythmia secondary to myocardial ischaemia or myocardial infarction. The commonest initial cardiac arrest rhythm is VF.

<table>
<thead>
<tr>
<th>Causes of ventricular fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Acute coronary syndromes</td>
</tr>
<tr>
<td>• Hypertensive heart disease</td>
</tr>
<tr>
<td>• Valve disease</td>
</tr>
<tr>
<td>• Drugs (e.g. antiarrhythmic drugs, tricyclic antidepressants, digoxin)</td>
</tr>
<tr>
<td>• Inherited cardiac diseases (e.g. long QT syndromes)</td>
</tr>
<tr>
<td>• Acidosis</td>
</tr>
<tr>
<td>• Abnormal electrolyte concentration (e.g. potassium, magnesium, calcium)</td>
</tr>
<tr>
<td>• Hypothermia</td>
</tr>
<tr>
<td>• Electrocution</td>
</tr>
</tbody>
</table>

• **Acute Coronary Syndromes**

The acute coronary syndromes (ACS) comprise:

- Unstable angina
- Non-ST-segment-elevation myocardial infarction (NSTEMI)
- ST-segment-elevation myocardial infarction (STEMI)

These syndromes result from the same disease process in which, usually, thrombosis of a coronary artery is triggered by fissuring of an atheromatous plaque. The extent to which myocardial blood flow decreases determines the syndrome.

• **Secondary heart problems**

The heart is affected by changes elsewhere in the body. For example, a primary respiratory arrest will result in a secondary cardiac arrest due to lack of oxygen to the heart. Severe anaemia, hypothermia, and severe septic shock will also impair cardiac function which can eventually lead to a cardiac arrest.

4.3.2. Recognition

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

Most sudden cardiac deaths (SCDs) occur in people with pre-existing cardiac disease, which may have been unrecognised. Asymptomatic or silent cardiac disease may include hypertensive heart disease, aortic valve disease, cardiomyopathy, myocarditis, and coronary disease.
• **Acute Coronary Syndromes – recognition**

Acute myocardial infarction (AMI) typically presents with chest pain that is felt as a heaviness or tightness or indigestion-like discomfort in the chest. The pain or discomfort often radiates into the neck or throat, into one or both arms (more commonly the left), and into the back or into the epigastrium. Some patients experience the discomfort more in one of these areas than in the chest.

Sometimes discomfort is accompanied by belching, which can be misinterpreted as evidence of indigestion as the cause.

A history of sustained (i.e. 20-30 minutes or more) acute chest pain typical of AMI, with acute ST-segment elevation on a 12-lead ECG is the basis for a diagnosis of STEMI.

Some patients present with chest pain suggestive of AMI and less specific ECG abnormalities, such as ST segment depression or T wave inversion. In a patient with a history suggestive of ACS and laboratory tests showing substantial release of troponin this indicates that myocardial damage has occurred. This is referred to as NSTEMI.

Unstable angina should be considered when there is an unprovoked and prolonged episode of chest pain, raising suspicion of AMI but without definite ECG or laboratory evidence of AMI.

People with chest pain need urgent medical attention. Out of hospital they should dial 112 and call an ambulance. If they have an acute coronary syndrome they are at high risk of VF cardiac arrest and sudden cardiac death.

• **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 proportion of SCDs are caused by inherited abnormalities (e.g. long and short QT syndromes, Brugada syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy) and by congenital heart disease.

In patients with known cardiac disease, syncope is as an independent risk factor for increased risk of death. Apparently healthy children and young adults who suffer SCD may also have symptoms and signs (e.g. syncope/presyncope, chest pain, palpitations, 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 i.e. fainting when already lying down;
- Syncope occurring during or after exercise (although syncope after exercise is often vasovagal);
- Syncope with no or only brief prodromal symptoms e.g. sudden collapse without warning signs;
- 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.

4.3.3. Treatment

Treat the underlying cause of circulatory failure. In many sick patients, this means giving intravenous fluids to treat hypovolaemia as part of the ABCDE approach (see below).

- **Acute Coronary Syndromes – treatment**
  Give immediate treatment to relieve symptoms, limit myocardial damage and reduce the risk of cardiac arrest. Do a rapid clinical assessment and record a 12-lead ECG. Immediate general treatment for ACS comprises:
  - Aspirin 300 mg, orally, crushed or chewed, as soon as possible.
  - Nitroglycerine, as sublingual glyceryl trinitrate (tablet or spray – 0.4 mg) if the patient is ongoing chest pain and SBP is above 90 mmHg. Do not use nitrates in case of bradycardia and in patients with suspected right ventricular (inferior) infarction.
  - Oxygen, to achieve an arterial blood oxygen saturation of 94-98 % (or 88-92 % in the presence of COPD).
  - Relief of pain is very important and intravenous morphine (or diamorphine) should be given, titrated to control symptoms but avoiding sedation and respiratory depression.

Most patients with cardiac ischaemic pain will be more comfortable sitting up. In some instances lying flat may provoke or worsen the pain. Give an anti-emetic with analgesia or if the patient has nausea.

Further treatment depends on the type of acute coronary syndrome. Options include fibrinolytic therapy (thrombolysis) or percutaneous coronary intervention (e.g. coronary angiography and stenting). These treatments work best if they are given early so it is essential to seek expert help.

5. The ABCDE approach

5.1. 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** 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.
5. Recognise that 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 (see Chapter 1).

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. Stay calm. Remember – it can take a few minutes for treatments to work.

10. The ABCDE approach can be used irrespective of your training and experience in clinical assessment or treatment. The detail of your assessment and what treatments you give will depend on your clinical knowledge and skills. If you recognise a problem or are unsure call for help.

5.2. First steps

1. Ensure personal safety. Wear glasses, apron and gloves as appropriate.

2. Look at the patient in general to see if the patient ‘looks unwell’. Your first impression is important.

3. If he is awake, ask “How are you?” and hold his hand. If he appears unconscious or has collapsed, shake him and ask “Are you alright?” If he responds by talking normally he has a patent airway, is breathing and has brain perfusion. If he speaks only in short sentences, he may have breathing problems. If he does not respond, he is likely to be critically ill.

4. This first rapid approach of the patient should take you less of 30 second and will often tell you a patient is sick or not sick. Ask a colleague to ensure appropriate help is coming urgently.

5. If the patient is unconscious, unresponsive, and is not breathing normally (use ‘Look, Listen and Feel’ control: occasional gasps are not normal and are a sign of cardiac arrest) start CPR according to the guidance in chapter 3. If you have any doubts about the diagnosis of cardiac arrest, start CPR until expert help arrives.

6. If they are present, monitor the vital signs early. Start pulse oximetry, ECG monitoring and a non-invasive blood pressure measurement in all critically ill patients, as soon as possible. Calculate an early warning score (EWS) and call for help/escalate treatment according to your hospital protocol.

7. Insert an intravenous cannula as soon as possible. Take bloods for investigation when inserting the intravenous cannula.
5.3. **Airway (A)**

Airway obstruction is an emergency. Get expert help immediately.

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 an emergency:
   - In most cases, only simple methods of airway clearance are required (e.g. airway opening manoeuvres, suction, insertion of an oropharyngeal or nasopharyngeal airway). Tracheal intubation by an expert may be required when these fail.

3. Give oxygen at high concentration:
   - Give high-concentration oxygen using a mask with an 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.
   - 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 %.

5.4. **Breathing (B)**

**Breathing evaluation**
- *Frequency and rate*
- *Volume:* amplitude of tidal volume and symmetry
- *Work:* respiratory pattern, accessory muscles, noises
- *Oxygen*

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, massive haemothorax.

1. Look, listen and feel for the general signs of respiratory distress: sweating, central cyanosis, use of the accessory muscles of respiration, abdominal breathing.

2. Count the respiratory rate. The normal rate is 12-20 breaths min⁻¹. A high (> 25 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 and normal on both sides.
4. Note any chest deformity (this may increase the risk of deterioration in the ability to breathe normally) and 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 because the patient cannot cough or take a deep breath. Stridor or wheeze suggests partial, but important, airway obstruction.

7. Percuss the chest if you are trained to do so: hyper-resonance suggests a pneumothorax; dullness suggests consolidation or pleural fluid.

8. Auscultate the chest with a stethoscope if you are trained to do so: bronchial breathing indicates lung consolidation with patent airways; absent or reduced sounds suggest a pneumothorax or pleural fluid or lung consolidation caused by complete bronchial 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 breathing problems depends on the cause. Critically ill patients should be given oxygen. In some patients with chronic obstructive pulmonary disease (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 organ damage or cardiac arrest if their blood oxygen levels are allowed to decrease. In this group, aim for a lower than normal 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 pocket mask or two-person bag-mask ventilation to improve oxygenation and ventilation, whilst calling immediately for expert help. In cooperative patients without 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.
5.5. **Circulation (C)**

**Circulation evaluation**
- Frequency and rate
- Pulses (Peripheral and central); Peripheral perfusion (CRT, skin aspect and temperature, urine output)
- Pre-charge (Blood Volume)
- Pressure (Arterial)

In almost all medical and surgical emergencies, consider hypovolaemia to be the likeliest cause of shock, until proven otherwise. Unless there are obvious signs of a cardiac cause (e.g. chest pain, heart failure), give intravenous fluid to any patient with cool peripheries and a fast heart rate.

In surgical patients, rapidly exclude bleeding (overt or hidden). Follow your local protocols for liquid resuscitation in trauma and surgical patients. 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 fingers: are they blue, pink, pale or mottled?
2. Hold the patient’s hand: is it cool or warm?
3. Measure the capillary refill time. Apply pressure for 5 seconds 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 refill time is usually less than 2 seconds. A prolonged time suggests poor peripheral perfusion. Other factors (e.g. cold surroundings, poor lighting, old age) can prolong the time.
4. Count the patient’s pulse rate (or heart rate by listening to the heart with a stethoscope).
5. 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.
6. 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).
7. Auscultate the heart with a stethoscope if you are trained to do so. Is there a murmur or pericardial rub? Are the heart sounds difficult to hear? Does the audible heart rate correspond to the pulse rate?
8. 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 less than 0.5 ml kg⁻¹ hour⁻¹).
9. Look thoroughly for external bleeding 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.

10. The treatment of cardiovascular collapse depends on the cause, but should be directed at fluid replacement, control of bleeding 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.

11. Insert one or more large (14 or 16 G) intravenous cannulae. Use short, wide-bore cannulae, because they enable the highest flow.

12. Take blood from the cannula for routine haematological, biochemical, coagulation and microbiological investigations, and cross-matching, before infusing intravenous fluid.

13. Give a rapid fluid challenge (over 5-10 minutes) of 500 ml of warmed crystalloid solution (e.g. Hartmann’s solution or 0.9 % sodium chloride) if the patient is normotensive. Give one litre if the patient is hypotensive. Use smaller volumes (e.g. 250 ml) for patients with known cardiac failure and use closer monitoring (listen to the chest for crackles after each bolus, consider a central venous pressure (CVP) line). Follow your local protocols for liquid resuscitation in trauma and surgical patients.

14. Reassess the heart rate and BP regularly (every 5 minutes), aiming for the patient’s normal BP or, if this is unknown, a systolic pressure greater than 100 mmHg.

15. If the patient does not improve, repeat the fluid challenge.

16. If symptoms and signs of heart failure (shortness of breath, 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. Ask for expert help as other treatments to improve tissue perfusion (e.g. inotropes or vasopressors) may be needed.

17. If the patient has primary chest pain and a suspected ACS, record a 12-lead ECG early, and treat initially with aspirin, nitroglycerine and oxygen if appropriate, and morphine.

18. 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, aiming at a \( \text{SpO}_2 \) of 94–98 %; do not give supplementary oxygen if the patient’s \( \text{SpO}_2 \) is within this range when breathing air alone.
   - Morphine (or diamorphine) titrated intravenously to avoid sedation and respiratory depression.
5.6. Disability (D)

Common causes of unconsciousness or altered consciousness level include hypoxia, hypercapnia, cerebral hypoperfusion, hypoglycaemia 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.
5. Measure the blood glucose to exclude hypoglycaemia using a rapid finger-prick bedside testing method. If the blood sugar is below $4.0 \text{ mmol l}^{-1}$, give an initial dose of $50 \text{ ml}$ 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 $250 \text{ ml}$ 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 and call for expert help. Other concentrations of intravenous glucose are available and can be used according to local policy.
6. Nurse unconscious patients in the lateral position if their airway is not protected.

5.7. Exposure (E)

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

5.8. Additional information

History
- Signs and symptoms
- Allergy
- Medicines
- Pertinent medical history
- Last meal
- Events
- Risk factors

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 using SBAR or RSVP.
6. Record the patient’s response to therapy.

**KEY LEARNING POINTS**

- 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 and escalation protocols to identify and treat patients at risk of cardiorespiratory arrest.
- Airway, breathing and circulation problems can cause cardiorespiratory arrest.
- Use the ABCDE approach to assess and treat the deteriorating patient.

**FURTHER READING**

Chapter 3.

In-hospital Resuscitation

LEARNING OUTCOMES

To understand:

• how to start resuscitation in hospital
• how to continue resuscitation until more experienced help arrives
• the importance of high quality CPR with minimal interruption

1. Introduction

After in-hospital cardiac arrest, the division between basic life support and advanced life support is arbitrary. The public expect that clinical staff can undertake cardiopulmonary resuscitation (CPR). For all in-hospital cardiac arrests, ensure that:

• Cardiac arrest is recognised immediately;
• Help is summoned by dialling a standard number;
• CPR is started immediately and, if indicated, defibrillation is attempted as soon as possible (within 3 minutes at the most).

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

2. Why is in-hospital resuscitation different?

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

• 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.
2.1. Location

Patients who have a witnessed or monitored cardiac arrest in a critical care area are usually diagnosed and treated quickly.

Ideally, all patients who are at high risk of cardiac arrest should be cared for in a monitored area where staff and facilities for immediate resuscitation are available. Patients, visitors or staff may also suffer from cardiac arrest in nonclinical areas (e.g. car parks, corridors).

2.2. Skills 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, if you work in critical care and emergency medicine you may have more advanced resuscitation skills and greater experience in resuscitation than those who use resuscitation skills rarely. Hospital staff who respond to a cardiac arrest may have different levels of skill to manage the airway, breathing and circulation. Use the skills you are trained to do.

2.3. Number of responders

If you are alone, always make sure that help is coming. Usually, other staff are nearby and several actions can be undertaken simultaneously. Hospital staffing tends to be at its lowest during the night and at weekends. This may influence patient monitoring, treatment and outcomes. Studies show that survival rates from in-hospital cardiac arrest are lower during nights and weekends.

2.4. Equipment available

All clinical areas should have immediate access to resuscitation equipment and drugs to facilitate rapid resuscitation of the patient in cardiac arrest. Ideally, the equipment used for cardiopulmonary resuscitation (including defibrillators) and the layout of equipment and drugs should standardised throughout the hospital. You should be familiar with the resuscitation equipment used in your clinical area.

A review of serious patient safety incidents associated with CPR and patient deterioration reported to the UK National Patient Safety Agency showed that equipment problems during resuscitation (e.g. portable suction not working, defibrillator pads missing) are common.

Resuscitation equipment should be checked on a regular basis to ensure it is ready for use. Automated external defibrillators (AEDs) should be available in clinical and nonclinical areas where staff do not have rhythm recognition skills or rarely need to use a defibrillator.

2.5. Resuscitation team

The resuscitation team can be a traditional cardiac arrest team, which is only called when cardiac arrest is recognised.
In some hospitals a resuscitation team (e.g. medical emergency team – MET) is called if a patient is deteriorating before cardiac arrest occurs.

Resuscitation team members should meet for introductions and plan before they attend actual events. Knowing each others names, backgrounds and discussing how the team will work together during a resuscitation will improve team work during resuscitation attempts. Team members should also debrief after each event based on what they actually did during the resuscitation. This should ideally be based on data collected during the resuscitation.

**Figure 3.1**
In-hospital resuscitation algorithm

- Collapsed/sick patient
  - Shout for HELP & assess patient
    - Signs of life?
      - No
        - Call resuscitation team
        - CPR 30:2 with oxygen and airway adjuncts
        - Apply pads/monitor Attempt defibrillation if appropriate
        - Advanced Life Support when resuscitation team arrives
      - Yes
        - Assess ABCDE Recognise & treat Oxygen, monitoring, IV access
        - Call resuscitation team if appropriate
        - Handover to resuscitation team
3. **Sequence for collapsed patient in a hospital**

An algorithm for the initial management of in-hospital cardiac arrest is shown in figure 3.1.

### 3.1. 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 protective measures, such as eye protection, aprons and face masks, may be necessary.
- The risk of infection is much lower than perceived. There are isolated reports of infections such as tuberculosis (TB), and severe acute respiratory syndrome (SARS). Transmission of HIV during CPR has never been reported.
- Wear full personal protective equipment (PPE) when the victim has a serious infection such as TB or SARS. Follow local infection control measures to minimise risks.
- Be careful with sharps; a sharps box must be available.
- Use safe handling techniques for moving victims during resuscitation.
- Take care with patients exposed to poisons. Avoid mouth-to-mouth ventilation and exhaled air in hydrogen cyanide or hydrogen sulphide poisoning.
- Avoid contact with corrosive chemicals (e.g. strong acids, alkalis, paraquat) or substances such as organophosphates that are easily absorbed through the skin or respiratory tract.
- There are no reports of infection acquired during CPR training. Nevertheless, take sensible precautions to minimise potential cross-infection from manikins. Clean manikins regularly and disinfect thoroughly after each use.

### 3.2. Check the patient for a response

- If you see a patient collapse or find a patient apparently unconscious first ensure personal safety and shout for help, then assess if he is responsive (shake and shout). Gently shake his shoulders and ask loudly: “Are you all right?” *(figure 3.2).*
- If other members of staff are nearby it will be possible to undertake actions simultaneously.
3.3A. If he responds

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

3.3B. If he does not respond

The exact sequence will depend on your training and experience in assessment of breathing and circulation in sick patients.

- Agonal breathing (occasional gasps, slow, laboured or noisy breathing) is common in the early stages of cardiac arrest and is a sign of cardiac arrest and should not be confused as a sign of life.
- Shout for help (if not already).
- Turn the patient on to his back.
- Take 10 seconds at most to determine if the patient is in cardiac arrest:
  - Open the airway using head tilt and chin lift (figure 3.3).
If there is a risk of cervical spine injury, establish a clear upper airway by using jaw thrust or chin lift in combination with manual in-line stabilisation (MILS) of the head and neck by an assistant (if enough people are available). 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, oxygenation and ventilation takes priority over concerns about a cervical spine injury.

Keeping the airway open, look, listen, and feel (figure 3.4) to determine if the victim is breathing normally. This is a rapid check and should take less than 10 seconds:

- Look for chest movement (breathing or coughing).
- Look for any other movement or signs of life.
- Listen at the victim's mouth for breath sounds.
- Feel for air on your cheek.

Assessing for breathing and any other movement

Figure 3.3
Head tilt and chin lift

Figure 3.4
Assessing for breathing and any other movement
• If the patient has no signs of life (based on lack of purposeful movement, normal breathing, coughing), start CPR until more help arrives or the patient shows signs of life.

• Only those experienced in ALS should try to assess the carotid pulse whilst simultaneously looking for signs of life. This rapid assessment should take no more than 10 seconds (figure 3.5).

• If the patient has no signs of life, no pulse, or if there is any doubt, start CPR immediately.

• Diagnosing cardiac arrest can be difficult. If unsure, do not delay starting CPR. The patient is far more likely to die if there is a delay diagnosing cardiac arrest and starting CPR. Starting CPR on a very sick patient with a low blood pressure is unlikely to be harmful and may help.

• Assess the patient to confirm cardiac arrest even if the patient is monitored in a critical care area.

3.4A. If he has a pulse or other signs of life

• Urgent medical assessment is required. Depending on the local protocols, this may be a resuscitation team. While waiting for the team, assess 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.

3.4B. If he has no pulse or signs of life

• Start CPR.

• Get a colleague to call the resuscitation team (figure 3.6) 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 centre of the victim’s chest (figure 3.6).

• 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 lower half of the sternum (figure 3.7 and 3.8).

• Ensure high quality chest compressions:
  - Depth of 5 cm (max. 6 cm)
  - Rate of 100-120 compressions per minute
  - Allow the chest to recoil completely after each compression
  - Take approximately the same amount for compression and relaxation
  - Minimise any interruptions to chest compression (hands-off time)

Figure 3.6
Call the resuscitation team

Figure 3.7
Hand position for chest compressions
• If available, use a prompt and/or feedback device (e.g. metronome, smart defibrillator) to help ensure high quality chest compressions. Do not feel for pulses to assess the effectiveness of compressions.

• Each time compressions are resumed, place your hands without delay in the centre of the chest.

• The person doing chest compressions will get tired. If there are enough rescuers, this person should change about every 2 minutes or earlier if unable to maintain high quality chest compressions. This change should be done with minimal interruption to compressions.

• Use whatever equipment is available immediately for airway and ventilation. A pocket mask, which can be supplemented with an oral airway should be readily available. Alternatively, use a bag-mask, supraglottic airway device (e.g. laryngeal mask airway (LMA)) and self-inflating bag according to local policy. Maintain the airway and ventilate the lungs with the most appropriate equipment immediately to hand.

• Tracheal intubation should be attempted only if the healthcare provider is properly trained and has regular, ongoing experience with the technique.

• Use an inspiratory time of 1 second and give enough volume to produce a normal chest rise. Add supplemental oxygen to give the highest feasible inspired oxygen as soon as possible.

• Avoid rapid or forceful breaths.

• If 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 per minute, and ventilate the lungs at approximately 10 breaths per minute (i.e. do not stop chest compressions for ventilation). Avoid hyperventilation (both excessive rate and tidal volume), which may worsen outcome. If a supraglottic...
airway (e.g. LMA) device has been inserted it may also be possible to ventilate the patient without stopping chest compressions.

- Waveform capnography must be used for confirming tracheal tube placement and monitoring ventilation rate. Waveform capnography can also be used with a bag-mask device and SGA. Waveform capnography is a monitor that is always used during anaesthesia and for critically ill patients requiring mechanical ventilation. It must be used to confirm correct tracheal tube placement during resuscitation and can also help guide resuscitation interventions. Waveform capnography monitoring is available on newer defibrillators, as part of portable monitors or as a hand held device.

- 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 unwilling or unable to do this, do high quality chest compressions until help or airway equipment arrives.

- A pocket mask with filter or bag-mask should be immediately available in all clinical areas. In practice this means that mouth-to-mouth ventilation is rarely needed in clinical settings.

- When the defibrillator arrives, apply self-adhesive defibrillation electrodes (pads) to the patient and analyse the rhythm. Apply the pads whilst chest compressions are ongoing (figure 3.9). Self-adhesive electrode pads will enable faster assessment of heart rhythm than attaching ECG electrodes.

- Preferably use self-adhesive pads for defibrillation, although we recognise that defibrillator paddles are still used in some settings.

- You may have an automated external defibrillator (AED), a manual defibrillator, or a defibrillator that has both an AED and manual mode.

- If you are not skilled in rhythm recognition use an AED (or AED mode). Switch on the AED and follow the audiovisual prompts and aim to minimise pauses in chest compressions by rapidly following prompts.

- If you are experienced and confident in rhythm recognition use a manual defibrillator (or manual mode).

- If using a manual defibrillator, further treatments will depend on the cardiac arrest rhythm. See chapter 4 (Advanced Life Support Algorithm) for treatment of the different cardiac arrest rhythms.

- Continue resuscitation until the resuscitation team arrives or the patient shows signs of life.

- Once resuscitation is underway, and if there are sufficient staff present, prepare intravenous cannula and drugs likely to be used by the resuscitation team (e.g. adrenaline).

- Use a watch or clock for timing between rhythm checks. It is difficult to keep track of the number of 30:2 cycles. In practice the duration of each cycle should be about 2 minutes.
• The importance of uninterrupted chest compressions cannot be over emphasised. Even short interruptions to chest compressions are disastrous for outcome. Make every effort to ensure that continuous, effective chest compressions are maintained throughout the resuscitation attempt.

• Plan exactly what you are going to do before stopping compressions to minimise the duration of the pause in compressions.

• Identify one person to be responsible for handover to the resuscitation team leader. Use. SBAR or RSVP for handover (See chapter 1). Locate the patient’s records.

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**Figure 3.9**
Maintain chest compressions while self-adhesive pads are applied

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**3.4C. 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 10 breaths (about 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 until more experienced help arrives.

• All patients in respiratory arrest will develop cardiac arrest if the respiratory arrest is not treated rapidly and effectively.
KEY LEARNING POINTS

• 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.

• Give high quality chest compressions with a depth of 5 cm (max. 6 cm), rate of 100-120 per minute, and allow complete recoil between compressions.

• Minimise interruptions to chest compressions for other interventions – this means all interruptions must be planned before stopping compressions…

FURTHER READING


LEARNING OUTCOMES
To understand:
• the function of the advanced life support (ALS) algorithm
• the importance of minimally interrupted high quality chest compressions
• the treatment of shockable and non-shockable rhythms
• when and how to give drugs during cardiac arrest
• the potentially reversible causes of cardiac arrest

1. 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)). As you might expect, the main difference in the treatment 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 4.1) is a standardised approach to the patient with cardiac arrest. This has the advantage of enabling treatment to be delivered expediently, without protracted discussion. Each member of the resuscitation team can predict and prepare for the next stage in the patient’s treatment, making the team more efficient.

The most important interventions that improve survival after cardiac arrest are early and uninterrupted high quality chest compressions, and early defibrillation for VF/pVT. Although drugs and advanced airways are still included among ALS interventions, they have limited evidence supporting their use. Drugs and advanced airways are therefore of secondary importance to high quality, uninterrupted chest compressions and early defibrillation.
Chapter 6 deals with the recognition of cardiac arrest rhythms. If you are not experienced and trained in the recognition of cardiac arrest rhythms use an automated external defibrillator (AED). Some defibrillators have both a manual and AED capability. Once switched on, the AED will give voice and visual prompts that will guide you through the correct sequence of actions.

2. **Shockable rhythms (VF/pVT)**

The first monitored rhythm is VF/pVT in approximately 20% of cardiac arrests, both in- or out-of-hospital.

2.1. **Treatment of shockable rhythms (VF/pVT)**

1. Confirm cardiac arrest – check for signs of life or if trained to do so, breathing and pulse simultaneously.
2. Call resuscitation team.
3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads – one below the right clavicle and the other in the V6 position in the mid-axillary line.
4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.
5. Stop chest compressions not longer than 2 seconds to check rhythm, Resume chest compressions immediately.
6. Confirm VF/pVT, if in doubt use a printout rhythm strip; the designated person selects the appropriate energy on the defibrillator (150-200 J biphasic for the first shock and 150-360 J biphasic for subsequent shocks) and presses the charge button (figure 4.2).
7. While the defibrillator is charging, warn all rescuers other than the individual performing the chest compressions to ‘stand clear’ and remove any oxygen delivery device as appropriate. Ensure that the rescuer giving the compressions is the only person touching the patient.
8. Once the defibrillator is charged, tell the rescuer doing the chest compressions to "stand clear"; when clear, give the shock (figure 4.3).
9. Without reassessing the rhythm or feeling for a pulse, restart CPR using a ratio of 30:2, starting with chest compressions.
10. Continue CPR for 2 minutes; the team leader prepares the team for the next pause in CPR.
11. Pause briefly to check the monitor.
12. If VF/pVT, repeat steps 6-11 above and deliver a second shock.
**Figure 4.1**
Adult advanced life support algorithm

Unresponsive and not breathing normally?

- **CPR 30:2**
  - Attach defibrillator/monitor
  - Minimise interruptions

**Assess rhythm**

- **Shockable** (VF/Pulseless VT)
  - 1 Shock
  - Minimise interruptions

- **Non-shockable** (PEA/Asystole)
  - Return of spontaneous circulation

**IMMEDIATE POST CARDIAC ARREST TREATMENT**
- Use ABCDE approach
- Aim for $\text{SaO}_2$ of 94-98 %
- Aim for normal $\text{PaCO}_2$
- 12 Lead ECG
- Treat precipitating cause
- Targeted temperature management

**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/hyperthermia
- 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

Immediately resume CPR for 2 min
Minimise interruptions

Immediately resume CPR for 2 min
Minimise interruptions

Personal copy of Manar ELKHOLY Mahmoud (ID: 484096)
13. If VF/VT persists repeat steps 6 - 8 above and deliver a third shock. Without reassessing the rhythm or feeling for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions.

14. If IV/IO access has been obtained, during the next 2 minutes of CPR give adrenaline 1 mg and amiodarone 300 mg.

15. Repeat this 2-minute CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists.

16. Give further adrenaline 1 mg IV after alternate shocks (i.e., in practice, this will be about once every two cycles of the algorithm)

17. Give a second time of amiodarone (150 mg) if VF/pVT persists after the fifth defibrillation attempt

18. The use of waveform capnography may enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

Figure 4.2
Continuing chest compressions during charging with a manual defibrillator

Figure 4.3
Shock delivery
If signs of life return during CPR (purposeful movement, normal breathing or coughing), or there is an increase in $\text{ET}_{\text{CO}_2}$, check the monitor;

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

- Check a central pulse and end-tidal ($\text{ET}_{\text{CO}_2}$) trace if available
- If there is evidence of ROSC, start post-resuscitation care.
- If no signs of ROSC, continue CPR and switch to the non-shockable algorithm.

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

The interval between stopping compressions and delivering a shock must be minimised and, ideally, should not exceed 5 s. Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation.

Chest compressions are resumed immediately after 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 and 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.

The first dose of adrenaline is given immediately after delivery of the third shock; amiodarone 300 mg should also be given after the third shock. 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-minute loops of CPR (which equates to every 3-5 min) for as long as cardiac arrest persists. If VF/pVT persists, or recurs, give a further dose of 150 mg amiodarone. 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.

When the rhythm is checked 2 min after giving a shock, if a non-shockable rhythm is present and the rhythm is organised (complexes appear regular or narrow), try to palpate a central pulse and look for other evidence of ROSC (e.g. sudden increase in $\text{ET}_{\text{CO}_2}$, or evidence of cardiac output on any invasive monitoring equipment). Rhythm checks must be brief, and pulse checks undertaken only if an organised rhythm is observed. If an organised rhythm is seen during a 2-minute period of CPR, do not interrupt chest compressions to palpate a pulse unless the patient shows signs of life suggesting ROSC. If there is any doubt about the presence of a pulse in the presence of an organised rhythm, 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.

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.

If there is doubt about whether the rhythm is asystole or very fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Very fine VF that is difficult to distinguish from asystole is unlikely to be shocked successfully into a perfusing rhythm. 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. Delivering repeated shocks in an attempt to defibrillate what is thought to be very fine VF will increase myocardial injury both directly from the electric current and indirectly from the interruptions in coronary blood flow. If the rhythm is clearly VF, attempt defibrillation.

2.2. Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm. Its routine use is therefore not recommended. It may be appropriate therapy only when 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. There are rare reports of a precordial thump converting a perfusing to a non-perfusing rhythm.

2.3. Witnessed, monitored VF/pVT in the cardiac catheter laboratory or after cardiac surgery

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, ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for two minutes if the third shock is unsuccessful.

With respect to the ALS algorithm, these three quick, successive shocks are regarded as the first shock.

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.
3. Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as organized cardiac electrical activity in the absence of any palpable pulses. These patients often have myocardial contractions too weak to produce a detectable pulse or blood pressure. PEA may be caused by treatable conditions (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.

Remember that any rhythm check should ideally not take more than 5 seconds and that interruptions to continuous, high quality chest compressions should be given priority over detailed rhythm analysis.

3.1. Treatment for PEA and asystole

2. If asystole is displayed, without stopping CPR, check that the leads are attached correctly.
3. Once an advanced airway has been sited, continue chest compressions without pausing during ventilation.
4. Give adrenaline 1 mg as soon as venous or intraosseous access is achieved, and repeat every alternate CPR cycle (i.e. about every 3-5 minutes).
5. After 2 minutes of CPR, recheck the rhythm. If asystole is present, resume CPR immediately.
6. If a pulse and/or signs of life are present, start post resuscitation care.
7. If no pulse and/or no signs of life are present (PEA):
   - 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).
8. If VF/pVT at rhythm check, change to shockable side of algorithm.
9. If asystole or an agonal rhythm is seen at rhythm check:
   - 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).
Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves, because this may respond to cardiac pacing. There is no benefit in attempting to pace true asystole.

4. During CPR

During the treatment of persistent VF/pVT or PEA/asystole, emphasis is placed on good quality and minimal interrupted 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 during the pauses for 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-minute 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-minute period of CPR.

As soon as the airway is secured (e.g. tracheal intubation), continue chest compressions without pausing during ventilation. To reduce fatigue, change the individual undertaking compressions every 2 minutes or earlier if necessary. Use CPR feedback/prompt devices when available. Be aware that some devices may fail to compensate for compression of the underlying mattress during CPR on a bed when providing feedback.

4.1. Airway and ventilation

A bag-mask, or preferably, a supraglottic airway device (e.g. laryngeal mask airway, i-gel) should be used if no-one on the resuscitation team is skilled in tracheal intubation (chapter 5).

Once a supraglottic airway device has been inserted, attempt to deliver continuous chest compressions, uninterrupted during ventilation. Ventilate the lungs at 10 breaths per minute; do not hyperventilate the lungs. If excessive gas leakage causes inadequate ventilation of the patient’s lungs, chest compressions will have to be interrupted to enable ventilation (using a compression: ventilation ratio of 30:2).

No studies have shown that tracheal intubation increases survival after cardiac arrest. 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 seconds.

Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until after ROSC. After intubation, confirm correct tube position, ideally with waveform capnography, and secure it adequately.
Once the patient’s trachea has been intubated, continue chest compressions, at a rate of 100-120 per minute without pausing during ventilation.

4.2. Vascular access

Insert an intravenous cannula if this has not been done already. Although peak drug concentrations are higher and circulation times are 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 seconds to facilitate drug delivery to the central circulation.

If intravenous access is difficult or impossible, consider gaining intraosseous (IO) access (figure 4.4). Tibial and humeral sites are readily accessible and enable equal flow for fluids. Intraosseous delivery of resuscitation drugs will achieve adequate plasma concentrations. Several studies indicate that IO access is safe and effective for fluid resuscitation and drug delivery.

Figures 4.4
Intraosseous devices
4.3. 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 4.5).

- Hypoxia
- Hypovolaemia
- Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders
- Hypothermia, hyperthermia
- Thrombosis (coronary thrombosis or pulmonary embolism)
- Tension pneumothorax
- Tamponade - cardiac
- Toxins

**Figure 4.5**
The four Hs and four Ts
4.3.1. The four Hs

Minimise the risk of hypoxia by ensuring that the patient’s lungs are ventilated adequately with 100% oxygen. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described in Chapter 5, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by hypovolaemia is usually due to severe bleeding such as might be caused by trauma, gastrointestinal bleeding, or rupture of an aortic aneurysm. Intravascular volume should be restored rapidly with fluid and blood. Obviously, such patients need urgent control of bleeding by surgery or other means.

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. A 12-lead ECG may help diagnosis. Intravenous calcium chloride is indicated in the presence of hyperkalaemia, hypocalcaemia, and calcium channel-blocker overdose. Always measure the blood glucose to exclude hypoglycaemia.

Consider hypothermia; use a low reading thermometer.

4.3.2. The four Ts

A tension pneumothorax may cause PEA. It can follow attempts at central venous catheter insertion. The diagnosis is made clinically. Signs of tension pneumothorax include: decreased air entry, decreased expansion and hyper-resonance to percussion on affected side; tracheal deviation away from affected side. Decompress rapidly by thoracostomy or needle thoracocentesis and then insert a chest drain.

Cardiac tamponade is difficult to diagnose since the typical signs of distended neck veins and hypotension cannot be assessed during cardiac arrest. Cardiac arrest after penetrating chest trauma or after cardiac surgery should raise strong suspicion of tamponade – the need for needle pericardiocentesis or resuscitative thoracotomy should be considered in this setting.

If there is no specific history of accidental or deliberate ingestion, poisoning by therapeutic or toxic substances is difficult to detect and may only be shown by laboratory investigations. Where available, the appropriate antidotes should be used, but most often treatment is supportive.

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause cardiac arrest consider giving a thrombolytic drug immediately.
4.4. Signs of Life

If signs of life (such as regular respiratory effort, movement) or readings from patient monitors compatible with ROSC (e.g. sudden increase in exhaled carbon dioxide or arterial blood pressure waveform) appear during CPR, stop CPR briefly and check the monitor. If an organised rhythm 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. The use of waveform capnography may enable ROSC to be detected without pausing chest compressions. A significant increase in end-tidal CO₂ during CPR may be seen when ROSC occurs.

5. Discontinuing resuscitation and diagnosing death

If resuscitation is unsuccessful the cardiac arrest team leader should discuss stopping CPR with the resuscitation team. The decision to stop CPR requires clinical judgement and a careful assessment of the likelihood of achieving ROSC.

After stopping CPR, observe the patient for a minimum of 5 minutes before confirming death. The absence of mechanical cardiac function is normally confirmed by:

- 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 minutes observation from the next point of cardiorespiratory arrest. After 5 minutes of continued cardiorespiratory arrest, the absence of the pupillary responses to light, of the corneal reflexes, and of any motor response to supra-orbital pressure should be confirmed. The time of death is recorded as the time at which these criteria are fulfilled.
KEY LEARNING POINTS

- The ALS algorithm provides a framework for the standardised resuscitation of all adult patients in cardiac arrest.
- The delivery of high quality chest compression with minimal interruptions and avoidance of hyperventilation are important determinants of outcome.
- Treatment depends on the underlying rhythm.
- Look for reversible causes and, if present, treat early.
- Whenever possible, secure the airway early to enable continuous chest compressions.

FURTHER READING

Chapter 5.

Airway Management and Ventilation

LEARNING OUTCOMES
To understand:
• the causes and recognition of airway obstruction
• the treatment for choking
• techniques for airway management when starting resuscitation
• the use of simple adjuncts to maintain airway patency
• ventilation with pocket mask or self-inflating bag
• the use of supraglottic airway devices

1. Introduction

Team membership and team leadership can be taught and improved by rehearsal, reflection and direct coaching. Cross-role training - i.e. the fact that trainees take up any role within a training session, regardless of their function in real-life circumstances - helps to understand the place and role of each member within a team.

2. Causes of airway obstruction

Obstruction may be partial or complete. It may occur at any level from the nose and mouth down to the level of the trachea and bronchi. In the unconscious patient, the commonest site of airway obstruction is the pharynx. As consciousness is lost, the reduced muscle tone allows the tongue and surrounding soft tissue to occlude the airway.

Obstruction may also be caused by vomit or blood, by regurgitation of gastric contents, by trauma to the airway, or by foreign bodies. Laryngeal obstruction can be because of oedema caused by burns, inflammation or anaphylaxis.

Upper airway stimulation, or an inhaled foreign body, can cause laryngeal spasm (laryngospasm). 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.

3. **Recognition of airway obstruction**

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 is 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 there is liquid or semisolid material in the upper airways.
- Snoring arises when the pharynx is partially occluded by the tongue or palate.
- Crowing or stridor is the sound of laryngeal spasm or obstruction.

During normal breathing, the abdomen is pushed out as the chest wall expands. In contrast, if the airway is obstructed the abdomen is drawn inwards as the chest attempts to expand during inspiration. This is often described as ‘seesaw breathing’. If the airway is obstructed, accessory muscles of respiration are used: the neck and shoulder muscles contract to assist movement of the thoracic cage.

There may also be intercostal and subcostal recession. Full examination of the neck, chest and abdomen is needed to differentiate these paradoxical movements from normal breathing; it is sometimes very difficult and you must listen for the absence of breath sounds to diagnose complete airway obstruction. When listening, remember that normal breathing should be quiet; obstructed breathing will be silent; but noisy breathing indicates partial airway obstruction. Unless obstruction is relieved to allow adequate ventilation within a very few minutes, neurological and other vital organ injury will occur, leading to cardiac arrest.

Whenever possible, give high-concentration oxygen during the attempt to relieve airway obstruction. Arterial blood oxygen saturation (SaO₂) measurements (normally using pulse oximetry [SpO₂]) will guide further use of oxygen as airway patency improves. If airway patency remains poor and SaO₂ remains low, continue to give high-concentration oxygen. As airway patency improves, blood oxygen saturation values will be restored more rapidly if the inspired oxygen concentration is initially high. Inspired oxygen concentrations can then be adjusted to maintain SaO₂ at 94-98 %.
4. Choking

4.1. Recognition of choking

Foreign bodies may cause either mild or severe airway obstruction. The signs and symptoms enabling differentiation between mild and severe airway obstruction are summarized in table 5.1.

Table 5.1
Signs of choking

<table>
<thead>
<tr>
<th>General signs of choking:</th>
<th>Signs of severe airway obstruction:</th>
<th>Signs of mild airway obstruction:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Attack occurs while eating.</td>
<td>• Response to question ‘Are you choking?’</td>
<td>• Patient is unable to speak.</td>
</tr>
<tr>
<td>• Patient may clutch his neck.</td>
<td>• Patient speaks and answers yes.</td>
<td>• Patient may respond by nodding.</td>
</tr>
</tbody>
</table>

Other signs:
• Patient is unable to breathe.
• Breathing sounds wheezy.
• Attempts at coughing are silent.
• Patient may be unconscious.

4.2. Treatment of adult choking

1. If the patient shows signs of mild airway obstruction (figure 5.1):
   • Encourage him to continue coughing, but do nothing else.

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 shoulder blades (scapulae) with the heel of the other hand.
Figure 5.1
Adult choking algorithm

Assess severity

**Severe** airway obstruction (ineffective cough)
- Unconscious
  - Start CPR
- Conscious
  - 5 back blows
  - 5 abdominal thrusts

**Mild** airway obstruction (effective cough)
- Encourage cough
  - Continue to check for deterioration to ineffective cough or until obstruction relieved

- 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 (bottom of breast bone); 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 5 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.
5. **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 can be used to relieve upper airway obstruction:

- Head tilt
- Chin lift
- Jaw thrust

### 5.1. 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 5.2)*.

![Head tilt and chin lift](image)

### 5.2. Jaw thrust

Jaw thrust is another manoeuvre for bringing the mandible forward and relieving obstruction *(figure 5.3)*. It is most successful when applied with a head tilt.

#### 5.2.1. Technique for jaw thrust

- Identify the angle of the mandible.
- Apply steady upward and forward (anterior) pressure with the index and other fingers placed behind the angle of the mandible.
- Use the thumbs to open the mouth slightly by downward displacement of the chin.

Jaw thrust, or head tilt and chin lift, will usually clear the airway when obstruction is from relaxation of the soft tissues.
Check for success of the manoeuvre by using the look, listen and feel sequence described above. If the airway is still obstructed, look and remove any solid foreign body in the mouth. Remove broken or displaced dentures but leave well-fitting dentures in place, as they help to maintain the contours of the mouth, which improves the seal for ventilation by mouth-to-mouth, mouth-to-mask or bag-mask.

5.2.2. Airway manoeuvres in a patient with suspected cervical spine injury

In patients with suspected cervical spine injury (e.g. if the victim has fallen, been struck on the head or neck, or has been rescued after diving into shallow water) head tilt can worsen the injury and damage the cervical spinal cord.

When there is a risk of cervical spine injury use a jaw thrust or chin lift in combination with manual in-line 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 an open airway and ventilation takes priority over concerns about a potential cervical spine injury.
6. Adjuncts to basic airway techniques

Simple airway adjuncts are often helpful, and sometimes essential to maintain an open airway, particularly when resuscitation is prolonged. Oropharyngeal and nasopharyngeal airways overcome soft palate obstruction and backward tongue displacement in an unconscious patient, but head tilt and jaw thrust may also be necessary.

6.1. Oropharyngeal airway

The oropharyngeal (Guedel) airway is a curved plastic tube, flanged and reinforced at the oral end and flattened to fit neatly between the tongue and hard palate (figure 5.4).

There are sizes suitable for small and large adults. Estimate the size by selecting an airway with a length equal to the vertical distance between the patient’s incisors and the angle of the jaw (figure 5.5). The most common sizes are 2 for small, 3 for medium and 4 for large adults. An airway that is slightly too big will be more beneficial than one that is slightly too small.

Oropharyngeal airways are intended only for unconscious patients; attempted insertion in semi-comatose patients may provoke vomiting or laryngospasm. If a patient is intolerant of an oral airway, they do not need one.

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Figure 5.4
Oropharyngeal and nasopharyngeal airways
6.1.1. Technique for insertion of an oropharyngeal airway

Open the patient’s mouth and ensure that there is nothing in the mouth that could be pushed into the larynx; use suction if necessary.

- Introduce the airway past the teeth or gums ‘upside down’ and then rotate it through 180° as it passes beyond the hard palate and into the oropharynx (figure 5.6). This manoeuvre lessens the chance of pushing the tongue backwards and downwards. Be careful not to lever the front incisors. The patient must be sufficiently obtunded not to gag or strain. If any reflex responses are seen, remove the airway. If placement is correct, obstruction will be relieved and the flattened reinforced section will fit neatly between the patient’s front teeth or gums.

- After insertion, check the airway by the look, listen and feel approach, while maintaining alignment of the head and neck with head tilt, chin lift or jaw thrust as necessary.
6.2. Nasopharyngeal airway

This is made from soft malleable plastic, bevelled at one end and with a flange at the other (figure 5.4). In patients who are not deeply unconscious, it is tolerated better than an oropharyngeal airway. It may be life-saving in patients with clenched jaws, trismus or maxillofacial injuries. Use with caution in patients with a suspected fracture of the base of skull, and remember they often cause bleeding inside the nose.

The tubes are sized in millimetres according to their internal diameter, and the length increases with diameter. Sizes 6-7 mm are suitable for adults. If the tube is too long it may stimulate the laryngeal or glossopharyngeal reflexes and cause laryngospasm or vomiting.

6.2.1. Technique for insertion of a nasopharyngeal airway

- Some designs require a safety pin to be inserted through the flange as an extra precaution against the airway disappearing into the nose. Insert the safety pin BEFORE inserting the airway.
- Lubricate the airway thoroughly using water-soluble jelly.
- Insert the airway bevel end first, vertically along the floor of the nose with a slight twisting action (figure 5.7). Try the right nostril first. If any obstruction is met, try the left nostril.
- Once in place, check for patency and ventilation by look, listen, and feel, and if necessary maintain correct alignment of the head and neck with chin lift or jaw thrust techniques.

Figure 5.7
Nasal airway insertion
7. Oxygen

In the absence of data indicating the optimal \( \text{SaO}_2 \) 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 oxygen saturation of arterial blood (\( \text{SaO}_2 \)) can be measured reliably. A standard oxygen mask (e.g. Hudson mask) will deliver up to 50 % inspired oxygen, providing the flow of oxygen is high enough. Initially, give the highest possible oxygen concentration – a mask with a reservoir bag (non-rebreathing mask) can deliver an inspired oxygen concentration of 85 % at flows of 10-15 l min\(^{-1}\).

Monitor the oxygen saturation by pulse oximeter (\( \text{SpO}_2 \)) or arterial blood gases to enable titration of the inspired oxygen concentration. When blood oxygen saturation can be measured reliably, oxygen saturations should be maintained between 94-98 %; or between 88-92 % if the patient has chronic obstructive pulmonary disease (COPD).

8. Suction

Use a wide-bore rigid sucker (Yankauer) to remove liquid (blood, saliva and gastric contents) from the upper airway (figure 5.8). Be careful if the patient has an intact gag reflex – suction can provoke vomiting. Fine-bore flexible suction catheters can be used in patients with limited mouth opening. They can also be passed through oropharyngeal or nasopharyngeal airways. Make sure you know how to use any portable suction equipment in your clinical area. Thick vomit can be difficult to suction without a large bore sucker and good suction. Large chunks of food may have to be removed by hand or Magill’s forceps.

Figure 5.8
Suctioning
9. **Ventilation**

Patients with no or inadequate breathing require artificial ventilation. Expired air ventilation (rescue breathing) is effective but the rescuer’s expired oxygen concentration is only 16-17%; so it must be replaced as soon as possible by ventilation with oxygen-enriched air. Mouth-to-mouth ventilation does not require any equipment but is unpleasant, particularly when vomit or blood is present. There are simple adjuncts for avoiding direct person-to-person contact.

9.1. **Pocket mask**

The pocket resuscitation mask is used widely. This is similar to an anaesthetic face mask and allows mouth-to-mask ventilation. It has a unidirectional valve to direct the patient’s expired air away from the rescuer. The masks are transparent to allow vomit or blood to be seen. Some masks have a port for oxygen. This port also has a simple one-way flap-valve so there is no leak if oxygen is not attached. When using masks without an oxygen port, supplemental oxygen can be given by placing oxygen tubing underneath one side of the mask and ensuring an adequate seal. The main difficulty is maintaining an airtight seal between the mask and the face, so a two-hand technique is much better.

The risks of gastric inflation and subsequent regurgitation are increased by:

- A high proximal airway pressure because of misalignment of the head and neck obstructing the airway and/or tidal volumes that are too large.
- The incompetent oesophageal sphincter of all patients in cardiac arrest.

Give each breath over 1 second, giving a volume that corresponds to visible chest movement; this is a compromise between giving enough volume, minimising the risk of gastric inflation, and allowing enough time for chest compressions. During CPR with an unprotected airway, give 2 ventilations after every 30 chest compressions.

9.1.1. **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 (jaw thrust). At the same time, press the mask onto the face with the thumbs to make a tight seal.
- Blow through the inspiratory valve and watch the chest rise.
- Stop inflation and watch the chest fall.
- Reduce any leaks between the face and mask by adjusting the contact pressure, altering the position of the fingers and thumbs, or increasing jaw thrust.
- If oxygen is available, add via the port at a flow of 10 l min⁻¹.
9.2. Self-inflating bag

The self-inflating bag can be connected to a face mask (bag-mask), supraglottic airway device (e.g. laryngeal mask airway) or a tracheal tube. 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 only ambient air (oxygen concentration 21 %). 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 high (10-15 l min⁻¹).

A self-inflating bag enables ventilation with high concentrations of oxygen, but its use requires skill. When used with a face mask (figure 5.9), it is difficult to achieve a gas-tight seal while simultaneously performing a jaw thrust with one hand and squeezing the bag with the other. It is easy to hypoventilate because of a leak, or to push down too hard and obstruct the airway. Excessive compression of the bag when used with a face mask can inflate the stomach, further reducing ventilation and greatly increasing the risk of regurgitation and aspiration.

Bag-mask ventilation is better with two people (figure 5.10). One person holds the face mask in place, using both hands and a jaw thrust, and the other squeezes the bag. The seal will be better and the ventilation will be more effective and safer.

Figure 5.9
Bag-Mask-Valve Ventilation
10. **Supraglottic airway devices during CPR**

In comparison with bag-mask ventilation, use of supraglottic airway devices (e.g. laryngeal mask airway, i-gel) may enable more effective ventilation and reduce the risk of gastric inflation. Supraglottic airway devices sit above the larynx and are easier to insert than a tracheal tube. They can generally be inserted without having to stop chest compressions.

### 10.1. Laryngeal mask airway

The laryngeal mask airway (LMA) is a wide-bore tube with an elliptical inflated cuff, which sits above the laryngeal opening (figure 5.11). It was introduced into anaesthetic practice in the middle of the 1980s and is a reliable, safe device that can be inserted easily with a high success rate after a short period of training. The LMA does not guarantee protection of the airway, but pulmonary aspiration is uncommon. Provided tidal volumes do not generate high inflation pressures during intermittent positive pressure ventilation (> 20 cmH₂O), gastric inflation is unlikely. Inserting an LMA does not require vigorous movements to align the head and neck, so could be the best adjunct if cervical spine injury is suspected. The LMA is reliable in use during resuscitation by nursing, paramedical and medical staff. As with tracheal intubation, the patient must be deeply unconscious. The LMA is particularly useful if attempted intubation by skilled personnel has failed and bag-mask ventilation is impossible (the ‘cannot ventilate, cannot intubate’ scenario). The conventional LMA (LMA Classic™) can be sterilised and reused up to 40 times. Many single-use versions are now available and these are more practical for resuscitation use.

Many of the single-use LMAs are of a slightly different design and material to the LMA Classic™ and their performance has not been validated during CPR. Modifications of the LMA are also available. The ProSeal LMA and LMA Supreme may have some advantages but there are few data relating to their use in cardiac arrest.
10.1.1. Technique for insertion of a laryngeal mask airway

- Try to insert the LMA without stopping chest compressions. If necessary try to limit any pause in chest compressions to a maximum of 5 seconds.

- Choose a LMA of appropriate size. A size 5 is correct for most men and a size 4 for most women. Deflate the cuff fully. Apply lubricating jelly to the outer face of the cuff area (the part that will not be in contact with the larynx).

- Holding the LMA like a pen, insert it into the mouth (figure 5.12). Advance the tip 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. We recommend to insert an LMA by two-person technique for more comfort or easy-going (figure 5.13).

- Connect the inflating syringe and inflate the cuff with air (40 ml for a size 5 LMA and 30 ml for a size 4 LMA). Do not hold the LMA during inflation. The tube should lift slightly out of the mouth as the cuff finds its correct position.

- If the LMA cannot be inserted within 30 seconds, 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 seeing bilateral chest movement. A large, audible leak suggests malposition of the LMA. A small leak is acceptable if chest rise is adequate.

- Secure the LMA with a bandage or tape.
10.1.2. Limitations of the laryngeal mask airway

- If there is high airway resistance or the lungs are stiff (pulmonary oedema, bronchospasm, chronic obstructive pulmonary disease) there is a risk of a large leak around the cuff causing hypoventilation. Most of the leak usually escapes through the patient’s mouth but some may be forced into the stomach.

- We do not know whether it is possible to provide adequate ventilation by LMA without interrupting chest compressions. Uninterrupted chest compressions are likely to cause at least some gas leak around the LMA cuff during ventilation. Attempt uninterrupted compressions initially, but abandon this if there are persistent leaks and hypoventilation.

- 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 is not common in clinical practice.
• If the patient is not deeply unconscious, they may react by coughing, straining or developing laryngeal spasm.

• If a good airway is not achieved, withdraw the LMA, deflate the cuff and make a new attempt at insertion, ensuring a good alignment of the head and neck and strict adherence to the correct insertion technique.

• Uncommonly, insertion may fold the epiglottis over the laryngeal inlet. This usually causes complete obstruction. Withdraw the tube and start again.

Proficiency with the LMA requires practice on patients, which should be gained under the supervision of an anaesthetist in appropriate circumstances.

10.2. i-gel airway

The i-gel has a cuff made of jelly like material and does not require inflation. The stem of the i-gel incorporates a bite block and a narrow oesophageal drain tube that allows a gastric tube to be passed through it (figure 5.14). It is easy to insert without stopping CPR, requires only minimal training and forms a good laryngeal seal (figure 5.14). 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. Use of the i-gel during cardiac arrest has now been reported extensively for both in-hospital and out-of hospital cardiac arrest.

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 seconds.

• 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 bite block. Position the device so that the i-gel cuff outlet is facing towards the chin of the patient.

• Ensure the patient is in the ‘sniffing the morning air’ 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.

• 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 when inserting the i-gel. If there is early resistance during insertion, get an assistant to apply a jaw thrust or rotate the i-gel.

• Glide the i-gel 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.

Figure 5.14
i-gel supraglottic airway
10.3. Laryngeal tube

The laryngeal tube (LT) is another supraglottic airway device commonly used in the anaesthetic setting and out of hospital. It is a single lumen tube with both an oesophageal and pharyngeal cuff (figure 5.15). A single pilot balloon inflates both cuffs simultaneously and it is available in a variety of sizes. Successful insertion and airway pressures generated are comparable to the LMA when performed by non-anaesthetists. There are several observational studies that document successful use of the LT by nurses and paramedics during prehospital cardiac arrest. A double lumen LT with an oesophageal vent and a disposable version (LT-D) are available.

10.3.1. Technique for insertion of a laryngeal tube 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 seconds.

- Select a LT of an appropriate size for the patient and deflate the cuff fully. A size 5 will be correct when the patient’s height is >180 cm; size 4 when 155-180 cm; and a size 3 when < 155 cm. Lubricate the tip of the LT with water-soluble gel.

- Place the patient’s head and neck in the sniffing or neutral position (try to maintain neutral alignment of the head and neck if there is suspicion of cervical spine injury).
- The tip of the LT should be placed against the hard palate below the incisors. Slide the LT down the centre of the mouth until resistance is felt or the device is almost fully inserted. When the LT is inserted properly, the second bold black line on the tube should have just passed between upper and lower teeth.

- Inflate the cuff to a pressure of 60 cm H₂O. This can be done either with a cuff inflator or a 100 ml syringe with the marks for the recommended volumes for each size of the LT.

- If the LT has not been inserted successfully, oxygenate the patient using a pocket mask or bag-mask before reattempting LT 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 LT, but a small leak is acceptable provided chest rise is adequate.

- Insert a bite block alongside the tube if available and secure the LT with a bandage or tape.

10.3.2. Limitations of the LT

- 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.

- There are no data demonstrating whether or not it is possible to provide adequate ventilation via an LT without interruption of chest compressions. Uninterrupted chest compressions are likely to cause at least some gas leak from the LT 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 LT does not sit within the larynx like a tracheal tube; however, this complication has not been documented widely in clinical practice.

- If the patient is not deeply unconscious, insertion of the LT may cause coughing, straining or laryngeal spasm. This will not occur in patients in cardiorespiratory arrest.

- If an adequate airway is not achieved, deflate the cuff, withdraw the LT and attempt reinsertion ensuring a good alignment of the head and neck.

- Uncommonly, airway obstruction may be caused by pushing the tongue towards the posterior pharynx. Deflate the cuff and withdraw the LT and attempt reinsertion.

To become proficient in the insertion of a LT requires practice on patients and this should be achieved under the supervision of an appropriately experienced person (e.g. anaesthetist) in a controlled environment.
11. **Patients with tracheostomies or permanent tracheal stomas**

A patient with a tracheostomy tube or a permanent tracheal stoma (usually following a laryngectomy) can develop airway obstruction from blockage of the tracheostomy tube or stoma – airway obstruction cannot occur at the level of the pharynx in these patients. Remove any obvious foreign material from the stoma or tracheostomy tube.

When dealing with an emergency it is important to know whether the patient has a normal upper airway and a tracheostomy tube, or has had a laryngectomy:

- Some tracheal tubes can be unblocked by removing an inner tube. Otherwise, if a tracheostomy tube is blocked, remove it and ventilate the patient’s lungs by sealing the stoma (the hole at the front of the neck through which the tracheostomy was inserted). The patient will usually have a normal upper airway. Use the standard airway and ventilation techniques outlined in this chapter (e.g. bag mask ventilation) with the stoma occluded by an airtight dressing. Alternatively, if you are trained to do so replace the tracheostomy tube.

- A laryngectomee is a patient who has had his larynx (voice box) removed, usually for cancer – in lay terms they are sometimes referred to as ‘neck breathers’. He breathes through a tracheal stoma (hole in front of neck). In these patients give oxygen and, if required, assist ventilation via the stoma, and not the mouth. This can be done by mouth-to-stoma, by holding a small face mask over the stoma, or by inserting a tracheal tube into the stoma, depending on your skills.

**KEY LEARNING POINTS**

- Airway management and ventilation are essential parts of cardiopulmonary resuscitation.

- Airway obstruction can usually be relieved with simple techniques.

- Simple adjuncts make airway management more effective and acceptable.

- When the skill for tracheal intubation is not available a supraglottic airway is an acceptable alternative.

- The most experienced team member should perform the task.

**FURTHER READING**


Chapter 6.

Cardiac Arrest Rhythms – Monitoring and Recognition

LEARNING OUTCOMES
To understand:
• the reasons for ECG monitoring
• how to monitor the ECG
• how to recognise the rhythms associated with cardiac arrest

1. Introduction

ECG monitoring enables identification of the cardiac rhythm in patients in cardiac arrest. Monitoring patients at risk of developing arrhythmias can enable treatment before cardiac arrest occurs. Patients at risk of cardiac arrest include those with chest pain, collapse or syncope, palpitations, or shock (e.g. due to bleeding or sepsis). Simple, single-lead ECG monitoring will not detect cardiac ischaemia reliably. Record serial 12-lead ECGs in patients experiencing chest pain suggestive of an acute coronary syndrome.

Accurate analysis of cardiac rhythm abnormalities requires experience, but by applying basic principles most rhythms can be interpreted sufficiently to allow selection of the appropriate treatment. The inability to recognise reliably ventricular fibrillation (VF) or other rhythms likely to respond to a shock is a major drawback in the use of manual defibrillators. Automated external defibrillators (AEDs) overcome this problem by automatic analysis of the rhythm.

For a shockable rhythm, the defibrillator charges to a predetermined energy and instructs the operator that a shock is required. The introduction of AEDs has meant that more people can now apply defibrillation safely. People who lack training or confidence in recognising cardiac rhythms should use AEDs.

It may be difficult to diagnose accurately an abnormal peri-arrest rhythm. Nevertheless, by following simple rules, any arrhythmia can be classified sufficiently accurately to enable recognition that the rhythm is abnormal, to assess the effect of the rhythm on the patient’s...
Chapter 6
Cardiac Arrest Rhythms – Monitoring and Recognition

clinical condition, and thus to select appropriate and effective treatment. For example, a
precise ECG classification of a bradycardia is usually less important than recognising that
the heart rate is inappropriately slow for the patient and starting appropriate treatment
with atropine or cardiac pacing. It is equally important to assess the haemodynamic effects
of a tachycardia. In many cases the precise treatment for a tachycardia, and the urgency
for it, depends greatly on the effects of the arrhythmia on cardiac output. In turn, these
depend on the patient’s underlying cardiac function; the same arrhythmia may have
different consequences in different patients. Precise ECG classification of the tachycardia is
often less important.

Remember – treat the patient not the ECG

2. ECG monitoring

2.1. Planned monitoring

When there is time to plan ECG monitoring, attach self-adhesive ECG electrodes to the
patient’s chest. The positions described will allow records that approximate to standard
lead I, II, and III of the conventional ECG. Select the configuration that displays the most
prominent P waves (if organised atrial activity is present) with sufficient QRS amplitude.
This is usually lead II.

The ECG cables are usually colour coded. In one common pattern (figure 6.1) the red electrode
goes to the right shoulder (Red to the Right), the yellow electrode to the left shoulder
(Yellow to Left), and the green or leg electrode below the pectoral muscles or on the upper
abdominal wall (‘Green for Spleen’). Placing the electrodes over bone rather than muscle
reduces electrical interference. Leave the precordium unobstructed for chest compression
and defibrillation. If possible, shave the areas where the electrodes are attached, and clean
the skin with alcohol to dissolve skin oil. Most adhesive electrodes include an electrolyte
gel to ensure good electrical contact.

Some electrodes have a rough surface on the wrapping, which can be used to gently abrade
the skin before the electrode is attached, improving contact. In co-operative patients,
reduce movement artefact by keeping them warm and reassured.
2.2. Emergency monitoring

In an emergency, such as a collapsed patient, assess the cardiac rhythm as soon as possible by applying self-adhesive defibrillator pads, which can be used for monitoring and hands-free shock delivery (figure 6.2). The electrodes are applied beneath the right clavicle and the other over the left lower chest in the mid-axillary line. Monitor the cardiac rhythm continuously with proper ECG electrodes as soon as possible after cardiac arrest.

If self-adhesive defibrillation pads are not available, the rhythm can also be monitored with manual defibrillator paddles. This is called “quick look”. The use of self-adhesive electrode pads or a quick-look paddles technique will enable more rapid assessment of heart rhythm compared with attaching ECG electrodes. In most healthcare settings hard paddles have been replaced with hands-free self-adhesive defibrillator pads.

Figure 6.1
ECG electrode positions

Figure 6.2
Defibrillator pads
3. Diagnosis from cardiac monitors

The displays and printouts from cardiac monitors are suitable only for recognition of rhythms and not for more detailed ECG interpretation.

3.1. Basic electrocardiography

The normal adult heart rate is defined as 60-100 per minute. A rate below 60 per minute is a bradycardia and a rate of 100 per minute or more is a tachycardia.

Under normal circumstances depolarisation is initiated from a group of specialised pacemaker cells, the sino-atrial (SA) node, in the right atrium (figure 6.3). The wave of depolarisation spreads from the SA node into the atrial muscle; this is seen on the ECG as the P wave (figure 6.4). Atrial contraction is the mechanical response to this electrical impulse.

Spread to the ventricular muscle is along specialized conducting tissue the atioventricular (AV) node and His-Purkinje system. The bundle of His bifurcates to enable depolarisation to spread into the ventricular muscle along two specialised bundles of conducting tissue the right bundle branch to the right ventricle and the left bundle to the left ventricle.

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**Figure 6.3**  
Electrical conduction in the heart

**Figure 6.4**  
The normal ECG signal
Depolarisation of the ventricles is reflected in the QRS complex of the ECG. The normal sequence of cardiac depolarisation described above is known as sinus rhythm.

The T wave that follows the QRS complex represents ventricular repolarisation.

The specialised cells of the conducting tissue (the AV node and His-Purkinje system) enable coordinated ventricular depolarisation, which is more rapid than uncoordinated depolarisation. With normal depolarisation, the QRS complex is narrow, which is defined as less than 0.12 seconds. If one of the bundle branches is diseased, conduction delay causes a broad QRS complex, i.e., greater than 0.12 seconds (3 small squares on the ECG).

4. Cardiac arrest rhythms

The rhythms present during cardiac arrest are classified into 3 groups:

- Ventricular fibrillation (VF) and some cases of ventricular tachycardia (VT)
- Asystole
- Pulseless electrical activity (PEA) Larger ECG rhythm strips are included at the end of this chapter.

4.1. Ventricular fibrillation (VF)

In VF the ventricular myocardium depolarises randomly. The ECG shows rapid, bizarre, irregular waves of widely varying frequency and amplitude (figure 6.5).

VF is sometimes classified as coarse or fine depending on the amplitude (height) of the complexes. If there is doubt about whether a rhythm is asystole or very fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Very fine VF that is difficult to distinguish from asystole will not be shocked successfully into a perfusing rhythm. Continuing good quality CPR may improve the amplitude and frequency of the VF and improve the chances of successful defibrillation and a perfusing rhythm. If the rhythm is clearly VF, attempt defibrillation.

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Figure 6.5
Ventricular fibrillation (VF)
4.2. **Ventricular tachycardia (VT)**

Ventricular tachycardia, particularly at higher rates or when the left ventricle is compromised, may cause profound loss of cardiac output. Pulseless VT is managed in the same way as VF. The ECG shows a broad-complex tachycardia. In monomorphic VT, the rhythm is regular (or almost regular) at a rate of 100-300 per minute *(figure 6.6)*.

![Figure 6.6: Ventricular tachycardia (VT)](image)

4.3. **Asystole**

Usually there is neither atrial nor ventricular activity, and the ECG is a more or less straight line *(figure 6.7)*. Deflections that can be confused with fine VF can be caused by baseline drift, electrical interference, respiratory movements, or cardiopulmonary resuscitation. A completely straight line usually means that a monitoring lead has disconnected.

Whenever asystole is suspected, check that the gain on the monitor is set correctly (1 mV cm⁻¹) and that the leads are connected correctly. If the monitor has the facility, view another lead configuration.

Atrial activity, i.e. P waves, may continue for a short time after the onset of ventricular asystole: there will be P waves on the ECG but no evidence of ventricular depolarisation *(figure 6.8)*. These patients may be suitable for cardiac pacing.

![Figure 6.7: Asystole](image)
4.4. Pulseless electrical activity (PEA)

The term pulseless electrical activity, sometimes referred to as electromechanical dissociation (EMD), means normal (or near normal) electrical activity without effective cardiac output, and is treated as cardiac arrest. The diagnosis is made when cardiac arrest occurs with a rhythm that would normally be accompanied by a good cardiac output.

4.4.1. Bradycardia

The treatment of bradycardia (less than 60 per minute) depends on its haemodynamic consequences. Bradycardia may mean imminent cardiac arrest.

4.4.2. Agonal rhythm

Agonal rhythm is characterised by slow, irregular, wide ventricular complexes of varying shape (figure 6.9). It is usually seen during the late stages of unsuccessful resuscitation. The complexes slow inexorably becoming progressively broader until all recognisable electrical activity is lost.

KEY LEARNING POINTS

- Monitor the ECG in all patients in cardiac arrest.
- Automated external defibrillators (AEDs) will recognise shockable rhythms (VF/VT) and advise a shock.
5. **Rhythm Strips**

**Rhythm Strip 1**  
Normal sinus rhythm

**Rhythm Strip 2**  
Coarse ventricular fibrillation

**Rhythm Strip 3**  
Fine ventricular fibrillation

**Rhythm Strip 4**  
Ventricular tachycardia

**Rhythm Strip 5**  
Asystole
Rhythm Strip 6
P-wave asystole

Rhythm Strip 7
Sinus bradycardia

Rhythm Strip 8
Agonal rhythm
FURTHER READING

Chapter 7.

Defibrillation

LEARNING OUTCOMES

To understand:

- the mechanism of defibrillation
- the factors affecting defibrillation success
- how to deliver a shock safely using an automated external defibrillator (AED)
- how to deliver a shock safely using a manual defibrillator
- the importance of minimising interruptions of chest compressions during the defibrillation sequence.

1. Introduction

Following the onset of ventricular fibrillation or pulseless ventricular tachycardia (VF/pVT), cardiac output ceases and cerebral hypoxic injury starts within 3 minutes. For complete neurological recovery, early successful defibrillation with a return of spontaneous circulation (ROSC) is essential. The shorter the interval between the onset of VF/pVT and delivery of the shock, the greater the chance of successful defibrillation and survival.

Although defibrillation is the key to the management of patients in VF/pVT. Immediate, continuous, uninterrupted high quality chest compressions are crucial to optimise the chances of successful resuscitation. Even short interruptions in chest compressions (to deliver rescue breaths or perform rhythm analysis) reduce the chances of successful defibrillation. The aim is to ensure that chest compressions are performed continuously throughout the resuscitation attempt, pausing briefly only to enable specific interventions.

Another factor that is critical in determining the success of defibrillation is the duration of the interval between stopping chest compressions and delivering the shock: the pre-shock pause. Every 5-second increase in the pre-shock pause almost halves the chance of successful defibrillation. This can be reduced to less than 5 seconds by continuing chest compressions during charging of the defibrillator and by having an efficient teamwork coordinated by a leader who communicates effectively. These two factors will maximise the chances of successful resuscitation.
If there is any delay in obtaining a defibrillator, and while the defibrillator is applied, start chest compressions and ventilation immediately.

Some defibrillators also provide prompts and feedback on the quality of chest compressions. You need to be familiar with the defibrillators you are likely to use in a cardiac arrest.

The use of CPR feedback or prompt devices during CPR should only be considered as part of a broader system of care that should include comprehensive CPR quality improvement initiatives, rather than as an isolated intervention.

If you are not confident in rhythm recognition, use an AED (or defibrillator with an AED mode). Switch the AED on and follow the audiovisual prompts.

2. Mechanism of defibrillation

Defibrillation is defined as the termination of fibrillation or, more precisely, the absence of VF/pVT five seconds after shock delivery. To accomplish this, an electrical current is passed across the myocardium for synchronous depolarisation of a critical mass of the cardiac muscle, which allows the natural pacemaking tissue to resume synchronized control. To achieve this, all defibrillators have: a power source capable of providing direct current; a capacitor that can be charged to a pre-determined energy level; and two electrodes placed on the patient’s chest through which the capacitor is discharged.

Success depends on sufficient current (measured in amperes) being delivered through the myocardium. In reality, the actual current and the optimal energy level needed are difficult to estimate as these depend on a number of factors like biphasic waveform shape, transthoracic impedance, the position of the electrodes, and the loss of current along other pathways in the thorax away from the heart. As little as 4% of the discharged current is estimated to reach the heart.

The energy stored in the capacitor can be determined, and for a given thoracic impedance the current flow is proportional to the energy. Modern defibrillators measure the transthoracic impedance and adjust their output accordingly, which is known as impedance compensation. (biphasic defibrillation)
3. Factors affecting defibrillation success

3.1. Transthoracic impedance

Transthoracic impedance is influenced by electrode size, the coupling material, electrode-to-skin contact, metabolic status and phase of ventilation. A transdermal drug patch on the patient’s chest may prevent good contact and may cause arcing and burns if pads are placed over it: remove and wipe the area dry before applying the electrodes and attempting defibrillation. The electrodes should not be placed on generators of internal pacemakers, ICD’s (implantable cardioverter/defibrillator) or other implanted devices. Care for scars! (see chapter 7.7)

3.1.1. Shaving the chest

Very hairy chests increase impedance and can reduce defibrillation success and cause burns to the patient’s chest. If needed, shave hair from the area where the electrodes are placed. Defibrillation must not be delayed if a razor is not immediately to hand. To minimise interruptions to chest compressions, shave the chest while another rescuer continues CPR. A bi-axillary electrode position is also an option.

3.1.2. Electrode pad/size

Self-adhesive pads 8-12 cm in diameter are widely used and work well. In practice the self-adhesive pads recommended by the manufacturer for the specific defibrillator should be used.

Self-adhesive defibrillation pads have a number of advantages over manual paddles and should always be used in preference when they are available.

The electrode packages should be controlled frequently for damages because dried electrodes threaten the defibrillation success.

3.2. Electrode position

The electrodes are positioned for greatest current flow through the myocardium. The standard positions are one electrode to the right of the upper sternum below the clavicle, and the other (apical) in the mid-axillary line, approximately level with the V6 ECG electrode and clear of breast tissue. The apical electrode must be sufficiently lateral (figure 7.1). Although the electrodes are marked positive and negative, they can be placed in either position.

Other acceptable 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 standard apical position, and the other electrode on the back, over the right scapula (postero-lateral).
- The lateral chest walls, one on the right and the other on the left side (bi-axillary).
### 3.3. CPR or defibrillation first?

If you do not witness the cardiac arrest, provide high quality, uninterrupted CPR while a defibrillator is retrieved, attached and charged (or switched on in the case of an AED).

Defibrillation must be performed as soon as possible, and a specific period of CPR (e.g. 2-3 minutes) before rhythm analysis and shock delivery is not recommended.

### 3.4. Shock sequence

First-shock success of modern defibrillators exceeds 90%. Failure suggests the need for a period of CPR. Thus, immediately after giving a single shock, and without reassessing the rhythm or feeling for a pulse, resume CPR (30 compressions:2 ventilations) for 2 minutes before delivering another shock (if indicated). 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 and the delay feeling for a pulse risks further damaging the myocardium if a perfusing rhythm has not been restored. If there is a perfusing rhythm, chest compressions do not increase the chance of VF recurring. In post-shock asystole, chest compressions may induce VF.

#### 3.4.1. Witnessed, monitored VF/pVT in the cardiac catheter laboratory

Cardiac arrest from shockable rhythms VF/pVT during cardiac catheterisation should immediately be treated with up to three stacked shocks before starting chest compressions.

With respect to the ALS algorithm, these three quick, successive shocks are regarded as the first shock.

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, i.e. the patient already has the pads on – for example during transfer of unstable, high risk patients.
3.5. Shock energy

The aim is to achieve successful defibrillation and ROSC with the lowest possible energy and fewest possible shocks.

If you are using an AED, you do not need to choose the shock energy as the AED will do this for you. All you have to do is follow the AED prompts.

If you are using a manual defibrillator you will need to choose the shock energy. The recommended shock energy varies according to the defibrillator's manufacturer. On some defibrillators the recommended shock energy is readily apparent (e.g. marked on the display or preset). You should be familiar with the defibrillators you work with. If you are unaware of the effective energy dose, use the highest energy setting for the first and subsequent shocks. If the first shock is unsuccessful, second and subsequent shocks can be delivered using either fixed or increasing energies (between 150-360 J), depending on the device in use. If a shockable rhythm recurs after successful defibrillation (with or without ROSC), give the next shock with the same energy level that had previously been successful.

3.6. Importance of uninterrupted chest compressions

Early, uninterrupted chest compressions are emphasized throughout this manual. Interrupt chest compressions only for rhythm checks and shock delivery, and resume compressions as soon as a shock has been delivered. When two rescuers are present, the rescuer operating the defibrillator applies the electrodes whilst CPR is in progress.

With manual defibrillators, it is possible to perform CPR during charging thereby reducing the pre-shock pause (interval from stopping compressions to shock delivery) to less than 5 seconds.

When using manual defibrillators, the entire process of pausing chest compressions, standing clear, delivering the shock and immediately resuming chest compressions should take less than 5 seconds. A lengthy ‘top-to-toe’ safety check (e.g. “head, middle, bottom, self, oxygen away”) performed after the defibrillator has charged and before shock delivery, as commonly taught and used in clinical practice, significantly diminishes the chances of successful defibrillation.
4. **Safety**

Do not deliver a shock if anybody is touching 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. Wipe any water or fluids from the patient’s chest before attempted defibrillation. Wear gloves as they may provide some limited protection from an accidental shock.

4.1. **Safe use of oxygen during defibrillation**

Sparks in an oxygen-enriched atmosphere can cause fire and burns to the patient. Self-adhesive pads are far less likely to cause sparks than manual paddles – no fires have been reported in association with the use of self-adhesive pads.

The following precautions reduce the risk of fire:

- Take off any oxygen mask or nasal cannulae and place them at least 1 m away from the patient’s chest.

- Leave the self-inflating 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 self-inflating bag, which can be left connected or detached and removed to a distance of at least 1 m. If the ventilator tubing is disconnected, ensure that it is kept at least 1 m from the patient or, better still, switch the ventilator off; modern ventilators generate massive oxygen flows when disconnected.

5. **Automated external defibrillators**

Automated external defibrillators 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 7.2).

5.1. **Automated rhythm analysis**

It is almost impossible to shock inappropriately with an AED. Movement is usually sensed, so movement artefact is unlikely to be interpreted as a shockable rhythm.
5.2. In-hospital use of AEDs

Delayed defibrillation can occur when cardiac arrest occurs in unmonitored hospital beds or non-clinical areas. Several minutes may elapse before resuscitation teams arrive with a defibrillator and deliver shocks. AEDs allow rapid defibrillation in areas where staff have no rhythm recognition skills or where they use defibrillators infrequently. Sufficient staff should be trained to enable achievement of the goal of providing the first shock within 3 minutes of collapse anywhere in the hospital.

Training in the use of AEDs can be achieved much more rapidly and easily than for manual defibrillators. Automated equipment has made attempted defibrillation available to a much wider range of medical, nursing, paramedical, and lay workers (e.g. police and first-aiders – ‘first-responder defibrillation’). Healthcare providers with a duty to perform CPR should be trained, equipped, and authorised to perform defibrillation. First-responder attempted defibrillation is vital, as the delay to delivery of the first shock is the main determinant of survival in cardiac arrest.

5.3. 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. These programmes require an organised and practised response with rescuers trained and equipped to recognize emergencies, activate the emergency medical services (EMS) system, provide CPR, and use the AED. Lay rescuer AED programmes with very rapid response times in airports, on aircraft, or in casinos, and uncontrolled studies using police officers as first responders have achieved reported survival rates as high as 49-74%.

Recommended elements for PAD programmes include:

- a planned and practised response;
- training of rescuers in CPR and AED use;
- a link with the local ambulance service;
- a programme of continuous audit (quality improvement).
Public access defibrillation programmes are most likely to improve survival from cardiac arrest if they are established in locations where witnessed cardiac arrest is likely to occur. Suitable sites might include airports, casinos and sports facilities. Approximately 80% of out-of-hospital cardiac arrests occur in private or residential settings; this fact inevitably limits the overall impact that PAD programmes can have on survival rates.

Placement of AEDs in areas where one cardiac arrest per 5 years can be expected is considered cost-effective and comparable to other medical interventions.

### 5.4. Sequence for use of an AED

1. Make sure the victim, any bystanders, and yourself are safe.

2. If the victim is unresponsive and not breathing normally:
   - Send someone for the AED and call for an ambulance or resuscitation team.
   - If you are on your own, do this yourself.

3. Start CPR according to the guidelines (chapter 3).

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 and visual prompts. Put the pads on whilst CPR is ongoing (figure 7.3a).
   - Ensure that nobody touches the victim whilst the AED is analysing the rhythm (figure 7.3b).

5A. If a shock *IS* indicated:
   - Ensure that nobody touches the victim.
   - Push the shock button (figure 7.3c) as prompted by the AED.
   - Continue as directed by the voice and visual prompts.

5B. If NO shock is indicated:
   - Immediately resume CPR using a ratio of 30 compressions to 2 rescue breaths (figure 7.3d).
   - Continue as directed by the voice and visual prompts.

6. Continue to follow the AED prompts until:
   - Qualified help (e.g. ambulance or resuscitation team) arrives and takes over, or
   - the victim starts to wake up, i.e. moves, opens eyes and breathes normally, or
   - you become exhausted.
Figure 7.3a
Applying defibrillator pads during chest compressions

Figure 7.3b
Everyone clear and AED assessing rhythm

Figure 7.3c
Delivering shock when prompted by AED
• The carrying case with the AED must contain some strong scissors for cutting through clothing and a disposable razor for shaving excessive chest hair when necessary.

• If ALS providers are using the AED, they should implement other ALS interventions (advanced airway, ventilation, IV access, drug delivery, etc.) according to local protocols.

Figure 7.3d
Restart chest compressions

5.5. The AED algorithm

The AED algorithm is shown in Figure 7.4. It is based on the lay rescuer in the community. Depending on their training and local policy, in addition to the use of the AED, healthcare professionals should consider:

• Assessing for pulse and signs of life to diagnose cardiac arrest.
• Calling for a resuscitation team after in-hospital cardiac arrest.
• Recognising and treating the reversible causes of cardiac arrest.
• Use of other interventions (e.g. tracheal intubation, intravenous access, drug administration).

The advanced life support algorithm (chapter 4) includes these extra interventions.
6. **Manual defibrillation**

Manual defibrillators have some 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. Their main disadvantage is that the operator has to be skilled in ECG rhythm recognition.

6.1. **Sequence for use of a manual defibrillator**

This sequence is an integral part of the advanced life support treatment algorithm in chapter 4.

1. Confirm cardiac arrest (not longer than 10s) – check for signs of life or if trained to do so, breathing and pulse simultaneously.
2. Call resuscitation team.
3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads (*figure 7.5*) – one below the right clavicle and the other in the V6 apical position in the mid-axillary line.
4. Plan your actions before pausing CPR for rhythm analysis. Make sure all the team knows the plan before stopping chest compressions.
5. Stop chest compressions; confirm VF from the ECG.
6. Resume chest compressions immediately; simultaneously, the designated person selects the appropriate energy on the defibrillator and presses the charge button (*figure 7.6*).
7. While the defibrillator is charging, warn all rescuers other than the individual performing the chest compressions to ‘stand clear’ and remove any oxygen delivery device as appropriate.
8. Once the defibrillator is charged, tell the resucer doing the chest compressions to “stand clear”; when clear, give the shock.
9. Without reassessing the rhythm or feeling for a pulse, immediately restart CPR using a ratio of 30:2, starting with chest compressions.
10. Continue CPR for 2 minutes; the team leader prepares the team for the next pause in CPR. Make sure you have a plan before stopping chest compressions.
11. Pause briefly to check the monitor.
12. If VF/pVT, repeat steps 6-11 above and deliver a second shock.
13. If VF/pVT persists repeat steps 6-8 above and deliver a third shock. Resume chest compressions immediately and then give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 minutes CPR.
Figure 7.4
AED algorithm

Unresponsive?

Call for help

Open airway
Not breathing normally

Send or go for AED
Call 112*

* or national emergency number

CPR 30:2
Until AED is attached

AED assesses rhythm

Shock advised

1 Shock

Immediately resume:
CPR 30:2 for 2 min

Continue until the victim starts
to wake up: to move, opens
eyes and to breathe normally

No shock advised

Immediately resume:
CPR 30:2 for 2 min

Until AED is attached
14. Repeat this 2-minute CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists.

15. Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3-5 minutes).

16. Give amiodarone after three defibrillation attempts irrespective of whether them being consecutive or interrupted.

17. If organised electrical activity is seen during the pause to check the monitor, feel for a pulse:
   a. If a pulse is present, start post-resuscitation care.
   b. If no pulse is present, continue CPR and switch to the non-shockable algorithm.

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

---

**Figure 7.5**
Applying defibrillator pads

---

**Figure 7.6**
Charging a manual defibrillator during chest compressions
7. **Synchronised cardioversion**

If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronised to occur with the R wave (not the T wave) of the electrocardiogram. By avoiding the relative refractory period, the risk of inducing VF is minimised. 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.

With some defibrillators, the synchronised mode has to be reset if a second shock is 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.

8. **Cardiac pacemakers and implantable cardioverter defibrillators**

If the patient has a cardiac pacemaker or implantable cardioverter-defibrillator (ICD), be careful when placing the electrodes. Although modern pacemakers are fitted with protection circuits, the current may travel along the pacemaker wire or ICD lead causing burns where the electrode tip makes contact with the myocardium. This may increase resistance at the contact point and gradually increase the threshold for pacing. Place the defibrillator electrodes at least 8 cm from the pacemaker unit to minimise the risk. Alternatively place the pads in the antero-posterior or postero-lateral position as described above. If resuscitation is successful following defibrillation, check the pacemaker threshold regularly over the next two months.

Recent case reports have documented rescuers receiving shocks from ICDs when in contact with the patient during CPR. It is particularly important to wear gloves and avoid skin-to-skin contact with the patient while performing CPR as there is no warning before the ICD discharges.

**KEY LEARNING POINTS**

- For the patient in VF, early defibrillation is the only effective means of restoring a spontaneous circulation.
- When using a defibrillator, minimise interruptions in chest compressions.
- Use an AED if you are not confident in rhythm recognition or manual defibrillation.
FURTHER READING

LEARNING OUTCOMES
To understand:
• the need for continued resuscitation after return of spontaneous circulation
• how to treat the post-cardiac arrest syndrome
• how to facilitate transfer of the patient safely
• the role and limitations of assessing prognosis after cardiac arrest

1. Introduction

Immediate Life Support (ILS) skills may be successful before expert help arrives. Return of a spontaneous circulation (ROSC) is an important first step, but the ultimate goal of resuscitation 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. 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, transfer the patient to the most appropriate high care area (e.g. intensive care unit (ICU), coronary care unit (CCU)) for further treatment. You will need expert help for the post-resuscitation care of survivors from cardiac arrest.

2. The post-cardiac arrest syndrome

The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and persistence of the 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 neurological dysfunction and brain death. Significant myocardial dysfunction is common after cardiac arrest but typically recovers by 2-3 days. The whole body ischaemia/reperfusion that occurs with resuscitation from cardiac arrest activates immunological and coagulation pathways contributing to multiple organ failure and increasing the risk of infection.
Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion and vasodilation.

3. Continued resuscitation

In the immediate post-resuscitation phase treat the patient by following the ABCDE approach (figure 8.1).

3.1. Airway and breathing

Aim: to ensure a clear airway, adequate oxygenation and ventilation.

Patients who have had a brief period of cardiac arrest that 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 if their arterial blood oxygen saturation is less than 94 %.

Other patients may not be immediately neurologically normal, even after a rapid successful resuscitation. Hypoxia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Recent studies suggest that high levels of oxygen in the blood after resuscitation from cardiac arrest may also be harmful. 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 %.

Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration.

Consider tracheal intubation, sedation and controlled ventilation in patients with obtunded cerebral function. This requires expert help.

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 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 to 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, is cooperative and breathing normally, consider immediate extubation: coughing on the tracheal tube may provoke arrhythmias and, or hypertension. If immediate or early extubation is not possible, sedate the patient to ensure the tracheal tube is tolerated, and provide ventilatory support.
A sedation protocol is highly recommended. Bolus doses of a neuromuscular blocking drug may be required, particularly if using targeted temperature management (TTM).

Figure 8.1
The ABCDE approach to post-resuscitation care

3.2. Circulation

Aim: the maintenance of normal sinus rhythm and a cardiac output adequate for perfusion of vital organs.

Cardiac rhythm and haemodynamic function are likely to be unstable following a cardiac arrest. Continuous monitoring of the ECG is essential. Record the pulse and blood pressure and assess peripheral perfusion: warm, pink fingers 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. 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), either with thrombolytic therapy or by emergency percutaneous coronary intervention (PCI). Primary PCI is the preferred treatment for ST-elevation myocardial infarction (STEMI) if it can be performed by an experienced team in a timely manner.

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 in all post-cardiac arrest patients who are suspected of having coronary artery disease as the cause of their arrest, even if they are sedated and mechanically ventilated. Several studies indicate that the combination of therapeutic hypothermia (see below) and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction.
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.

### 3.3. Disability and exposure

Aim: to evaluate the neurological function and ensure that cardiac arrest has not been associated with other medical or surgical conditions requiring immediate treatment.

Although cardiac arrest is caused frequently 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 is appropriate for 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 8.1). The maximum score possible is 15; the minimum score possible is 3.

<table>
<thead>
<tr>
<th><strong>Glasgow Coma Scale score</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
</tr>
<tr>
<td>Spontaneously</td>
</tr>
<tr>
<td>To speech</td>
</tr>
<tr>
<td>To pain</td>
</tr>
<tr>
<td>Nil</td>
</tr>
<tr>
<td><strong>Verbal</strong></td>
</tr>
<tr>
<td>Orientated</td>
</tr>
<tr>
<td>Confused</td>
</tr>
<tr>
<td>Inappropriate words</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
</tr>
<tr>
<td>Nil</td>
</tr>
<tr>
<td><strong>Best motor response</strong></td>
</tr>
<tr>
<td>Obeys commands</td>
</tr>
<tr>
<td>Localises</td>
</tr>
<tr>
<td>Normal flexion</td>
</tr>
<tr>
<td>Abnormal flexion</td>
</tr>
<tr>
<td>Extension</td>
</tr>
<tr>
<td>Nil</td>
</tr>
</tbody>
</table>

Consider the need for inducing targeted temperature management (TTM) in any patient that remains comatose after initial resuscitation from cardiac arrest (see below). The prevention of fever is very important.
4. Further assessment

4.1. History

Aim: To establish the patient’s state of health and regular drug therapy before the cardiac arrest.

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, ward staff, and relatives). Ask specifically about symptoms of cardiac disease. If primary cardiac disease seems unlikely, consider other causes of cardiac arrest (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.

4.2. Monitoring

Aim: to enable continuous assessment of vital organ function and to identify trends.

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). This will require expert help.

4.3. Investigations

Several physiological variables may be abnormal immediately after a cardiac arrest and urgent biochemical and cardiological investigations should be undertaken (table 8.2).
### Table 13.2
Investigations after restoration of circulation

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Purpose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Full blood count</strong></td>
<td>To exclude anaemia as contributor to myocardial ischaemia and provide baseline values</td>
</tr>
<tr>
<td><strong>Biochemistry</strong></td>
<td>To assess renal function</td>
</tr>
<tr>
<td></td>
<td>To assess electrolyte concentrations ($K^+$, $Mg^{2+}$ and $Ca^{2+}$)</td>
</tr>
<tr>
<td></td>
<td>To ensure normoglycaemia</td>
</tr>
<tr>
<td></td>
<td>To commence serial cardiac troponin measurements</td>
</tr>
<tr>
<td></td>
<td>To provide baseline values</td>
</tr>
<tr>
<td><strong>12-lead ECG</strong></td>
<td>To record cardiac rhythm</td>
</tr>
<tr>
<td></td>
<td>To look for evidence of acute coronary syndrome</td>
</tr>
<tr>
<td></td>
<td>To look for evidence of old myocardial infarction</td>
</tr>
<tr>
<td></td>
<td>To detect and monitor abnormalities (e.g. QT prolongation)</td>
</tr>
<tr>
<td></td>
<td>To provide a baseline record</td>
</tr>
<tr>
<td><strong>Chest radiograph</strong></td>
<td>To establish the position of a tracheal tube, a gastric tube, and/or a central venous catheter</td>
</tr>
<tr>
<td></td>
<td>To check for evidence of pulmonary oedema</td>
</tr>
<tr>
<td></td>
<td>To check for evidence of pulmonary aspiration</td>
</tr>
<tr>
<td></td>
<td>To exclude pneumothorax</td>
</tr>
<tr>
<td></td>
<td>To detect unintended CPR sequelae (e.g. sternal, rib fracture)</td>
</tr>
<tr>
<td></td>
<td>To assess cardiac contour (accurate assessment of heart size requires standard PA erect radiograph – not always practicable in the post-resuscitation situation)</td>
</tr>
<tr>
<td><strong>Arterial blood gases</strong></td>
<td>To ensure adequacy of ventilation and oxygenation</td>
</tr>
<tr>
<td></td>
<td>To ensure correction of acid/base imbalance</td>
</tr>
<tr>
<td><strong>Echocardiography</strong></td>
<td>To identify contributing causes to cardiac arrest.</td>
</tr>
<tr>
<td></td>
<td>To assess size/function of cardiac structures (chambers, valves), presence of pericardial effusion Cranial Computed tomography</td>
</tr>
<tr>
<td></td>
<td>If the immediate cause of cardiorespiratory arrest is not obvious</td>
</tr>
<tr>
<td></td>
<td>To identify causes to cardiac arrest (subarachnoid/subdural haemorrhage, intracerebral bleeding, tumour)</td>
</tr>
<tr>
<td></td>
<td>To identify cardiac arrest associated changes (e.g. oedema)</td>
</tr>
</tbody>
</table>
5. Patient transfer

Aim: to transfer the patient safely between the site of resuscitation and a place of definitive care.

Following the period of initial post-resuscitation care and stabilisation, the patient will need to be transferred to an appropriate critical care setting (e.g., ICU or CCU). The decision to transfer should be made only after discussion with senior members of the admitting team. Handover care using SBAR or RSVP (chapter 1). 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 and 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.

6. Optimising organ function

Aim: to optimise vital organ function and limit secondary organ damage.

The extent of secondary organ injury after ROSC depends on the ability to minimise the harmful consequences of post cardiac arrest syndrome. There are several ways to limit the insult to organs following cardiac arrest.

6.1. Heart and cardiovascular system

Post-cardiac arrest myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, a low cardiac output and arrhythmias. Early echocardiography will enable the degree of myocardial dysfunction to be quantified. Treatment with fluid, inotropes and vasopressors may be guided by blood pressure, heart rate, urine output, and rate of plasma lactate clearance and central venous oxygen saturations. Fluids, inotropes and vasopressors may be needed.

In the absence of definitive data supporting a specific goal for blood pressure, target the mean arterial blood pressure to achieve an adequate urine output (1 ml kg\(^{-1}\) 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.

6.1.1. Referral for implantable cardioverter defibrillator

Consider the need for an implantable cardioverter defibrillator (ICD) in any patient who has been resuscitated from cardiac arrest in a shockable rhythm and without a proven acute STEMI. Refer before discharge from hospital to a cardiologist with expertise in heart rhythm disorders.
6.2. **Brain: optimising neurological recovery**

6.2.1. **Cerebral perfusion**

Immediately after ROSC there is a period of cerebral hyperaemia followed by low blood flow. Normal cerebral autoregulation is lost, leaving cerebral perfusion dependent on mean arterial pressure. Under these circumstances, hypotension will compromise cerebral blood flow severely and will worsen any neurological injury. Try to maintain mean arterial pressure at the patient’s normal level.

6.2.2. **Control of seizures**

Seizures or myoclonus or both occur in 5-15 % of adult patients who achieve ROSC and 10-40 % of those who remain comatose. Seizures increase cerebral metabolism by up to three-fold and may cause cerebral injury: treat promptly and effectively with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate. Clonazepam is the most effective antimyoclonic drug, but sodium valproate, levetiracetam and propofol may also be effective.

6.2.3. **Glucose control**

A high blood glucose after resuscitation from cardiac arrest is associated with a poor neurological outcome. However, severe hypoglycaemia is also associated with increased mortality in critically ill patients. Following ROSC, blood glucose should be maintained at ≤10 mmol l⁻¹. Hypoglycaemia (< 4.0 mmol l⁻¹) must be avoided.

6.2.4. **Temperature control**

- **Treatment of hyperpyrexia**
  
  A period of hyperthermia (hyperpyrexia) is common in the first 48 hours after cardiac arrest and associated with a worse outcome. Treat any hyperthermia occurring after cardiac arrest with antipyretics or active cooling.

- **Targeted Temperature Management (TTM)**
  
  Mild induced hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia as occurs during cardiac arrest. Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death).

- **Which post-cardiac arrest patients should be cooled?**
  
  Consider Targeted temperature management (TTM) for any mechanically ventilated patient admitted to the ICU for post resuscitation organ support. There is good evidence supporting the use of mild induced hypothermia (36°C for 24 hours) in comatose survivors of out-of-hospital cardiac arrest caused by VF.
  
  Hypothermia may also benefit comatose survivors after in-hospital cardiac arrest or a cardiac arrest with a non-shockable rhythm.
• How to cool
Use of Targeted temperature management is divided into three phases: induction, maintenance, and rewarming. External and/or internal cooling techniques can be used to initiate and maintain TTM.

Other methods of inducing and/or maintaining hypothermia include:

- Ice packs and/or wet towels.
- Cooling blankets or pads.
- Water or air circulating blankets.
- Water circulating gel-coated pads.
- Intravascular heat exchanger, placed usually in the femoral or subclavian veins.
  - Transnasal evaporative cooling.
  - Extracorporeal circulation (e.g. cardiopulmonary bypass, ECMO).

Initial cooling is helped by neuromuscular blockade and sedation, which will prevent shivering.

Maintenance 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 or oesophagus. There are no data indicating that any specific cooling technique increases survival when compared with any other cooling technique. Once the temperature is in the target range (36°C), maintain this temperature for 24 hours. Rewarming must be at about 0.25-0.5°C of warming per hour with strict avoidance of hyperthermia.

• Contraindications to hypothermia
Generally recognised contraindications to therapeutic hypothermia include: severe systemic infection, established multiple organ failure, and pre-existing medical coagulopathy (fibrinolytic therapy is not a contraindication to therapeutic hypothermia).

7. Assessment of prognosis
Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury. A quarter of those dying after admission to ICU following in-hospital cardiac arrest die from neurological injury. A means of predicting neurological outcome that can be applied to individual patients immediately after ROSC is required. Any prognostic tests must a have 100 % specificity or zero false positive rate, i.e. no individuals should have a good long term outcome if predicted to have a poor outcome.

There are no clinical neurological signs that predict reliably poor outcome (severe cerebral disability or death) in the first 24 hours after cardiac arrest. In adult patients who are comatose after cardiac arrest, and who have not been treated with hypothermia and who do not have confounding factors (such as hypotension, sedatives or muscle relaxants), the
absence of both pupillary light and corneal reflex at more than 72 hours predicts poor outcome reliably.

Potentially reliable predictors of poor outcome in patients treated with therapeutic hypothermia after cardiac arrest include the absence of both corneal and pupillary reflexes 3 or more days after cardiac arrest.

Electrophysiological tests can also be helpful. Given the limited available evidence, decisions to limit care should not be based on the results of a single assessment method.

8. Organ donation

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

9. Care of the resuscitation team

Audit all resuscitation attempts and, ideally, send these data to a national audit (chapter 1). Whether the resuscitation attempt was successful or not, the patient’s relatives will require considerable support. Consider the pastoral needs of all those associated with the arrest.

KEY LEARNING POINTS

• After cardiac arrest, return of spontaneous circulation is just the first stage in a continuum of resuscitation.
• The patient’s final outcome depends on the quality of post-resuscitation care.
• These patients require appropriate monitoring, safe transfer to a critical care environment, and continued organ support.
• Our ability to predict the final neurological outcome for those patients remaining comatose after cardiopulmonary resuscitation remains very poor.

FURTHER READING

APPENDIX 1.

Pulse Oximetry and Oxygen Therapy

1. Introduction

Pulse oximetry is used to assess the patient’s arterial oxygen saturation. Without pulse oximetry, you may not notice the patient has a decreased arterial oxygen saturation of haemoglobin \(\text{SaO}_2\) until the saturation is between 80-85%.

Pulse oximetry is simple to use, relatively cheap, non-invasive and provides an immediate, objective measure of arterial blood oxygen saturation.

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 \(\text{SpO}_2\) displayed as a digital reading, waveform, or both.

Most pulse oximeters have an audible tone related to the \(\text{SpO}_2\), with a decreasing tone reflecting increasing hypoxaemia. The pulse rate is also usually displayed. A poor signal indicates a low blood pressure or poor tissue perfusion – reassess the patient.

Pulse oximeter readings must not be used in isolation: it is vital to interpret them in light of the clinical picture and alongside other investigations, and potential sources of error.

Pulse oximetry provides only a measure of oxygen saturation, not content, and thus gives no indication of actual tissue oxygenation. Furthermore, it provides no information on adequacy of ventilation. A patient may be breathing inadequately and have a high carbon dioxide level despite a normal oxygen saturation. Arterial blood gases are needed in critically ill patients to assess oxygenation and ventilation.

1.1. Limitations

The relationship between oxygen saturation and arterial oxygen partial pressure \(\text{PaO}_2\) is demonstrated by the oxyhaemoglobin dissociation curve (figure A.1). The sinusoid shape of the curve means that an initial decrease from a normal \(\text{PaO}_2\) is not accompanied by a drop of similar magnitude in the oxygen saturation of the blood, and early hypoxaemia may be masked. At the point where the \(\text{SpO}_2\) reaches 90-92%, the \(\text{PaO}_2\) will have decreased to around 8 kPa. In other words, the partial pressure of oxygen in the arterial blood will have decreased by almost 50% despite a reduction in oxygen saturation of only 6-8%.

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.

Presence of other haemoglobins: carboxyhaemoglobin (carbon monoxide poisoning), methaemoglobin (congenital or acquired), fetal haemoglobins and sickling red cells (sickle cell disease), haemoglobin lansing:

- Surgical and imaging dyes: methylene blue, indocyanine green and indigo carmine cause falsely low saturation readings.
- Nail varnish (especially blue, black and green) and artificial nails with natural look.
- 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 (Inaccurate oximetry readings have been observed in pigmented patients. In critically ill patients, a bias of more than 4 % has been observed to occur more frequently in black (27 %) than in white patients (11 %)

**Pulse oximetry does not provide a reliable signal during CPR.**

### 1.2. Uses

Pulse oximetry has four main uses:

1. Detection of/screening for hypoxaemia;
2. Targeting oxygen therapy;
3. Routine monitoring during anaesthesia;
4. Diagnostic (e.g. sleep apnoea).

### 2. Targeted oxygen therapy

In critically ill patients, those presenting with acute hypoxaemia (initial $\text{SpO}_2 < 85 \%$), or in the peri-arrest situation, give high-concentration oxygen immediately. 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 % oxygen to maximise arterial oxygen content and delivery to the tissues.

Once return of spontaneous circulation has been achieved and the oxygen saturation of arterial blood can be monitored reliably, adjust the inspired oxygen concentration to maintain a $\text{SpO}_2$ 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 both hypoxaemia and hyperoxaemia ($\text{PaO}_2 > 20 \text{ kPa}$) in the post-resuscitation phase may lead to worse outcomes than those in whom normoxaemia is maintained.

### 2.1. Special Clinical Situations

Patients with respiratory failure can be divided into two groups:

- **Type I:** low $\text{PaO}_2$ (<8 kPa), normal $\text{PaCO}_2$ (<6-7 kPa). In these patients it is safe to give a high concentration of oxygen initially with the aim of returning their $\text{PaO}_2$ to normal and then once clinically stable, adjusting the inspired oxygen concentration to maintain an $\text{SpO}_2$ of 94-98 %.

- **Type II:** low $\text{PaO}_2$ (<8 kPa), increased $\text{PaCO}_2$ (>7 kPa). This is often described as hypercapnic respiratory failure and is usually caused by chronic obstructive
pulmonary disease (COPD). If given excessive oxygen, these patients may develop worsening respiratory failure with further increases in PaCO₂ 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 should be 88-92 %. However, when critically ill, 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 SpO₂ 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 SpO₂ of 94-98 % (or 88-92 % if the patient is at risk of hypercapnic respiratory failure). This may be possible without supplementary oxygen therapy, and represents a change from previously accepted practice.

FURTHER READING
APPENDIX 2.

Drugs used in the treatment of Cardiac Arrest

<table>
<thead>
<tr>
<th>Drug</th>
<th>Shockable (VF/Pulseless VT)</th>
<th>Non-Shockable (PEA/Asystole)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>• Dose: 1 mg (10 ml 1:10,000 or 1 ml 1:1,000) IV</td>
<td>• Dose: 1 mg (10 ml 1:10,000 or 1 ml 1:1,000) IV</td>
</tr>
<tr>
<td></td>
<td>• Given after the 3rd shock if iv/io access is obtained</td>
<td>• Given as soon as iv/io access is obtained</td>
</tr>
<tr>
<td></td>
<td>• Repeated every 3-5 min (alternate loops)</td>
<td>• Repeated every 3-5 min (alternate loops)</td>
</tr>
<tr>
<td></td>
<td>• Give without interrupting chest compressions</td>
<td>• Give without interrupting chest compressions</td>
</tr>
</tbody>
</table>

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 macrovascular 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 acidaemia), transient hypoxaemia because of pulmonary arteriovenous shunting, impaired microcirculation, and increased post cardiac arrest myocardial dysfunction may offset these benefits. Adrenaline use is associated with more rhythm transitions during ALS, both during VF and PEA. 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.

<table>
<thead>
<tr>
<th>Amiodarone</th>
<th>• Dose: 300 mg bolus IV</th>
<th>• Not indicated for PEA or asystole</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Given after the 3rd shock if iv/io access is obtained</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Further dose of 150 mg after the 5th shock if VF/pVT persists</td>
<td></td>
</tr>
</tbody>
</table>

Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Atioventricular 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. The hypotension that occurs with intravenous amiodarone is related to the rate of delivery and is caused by the solvent, rather than the drug itself.

When amiodarone is unavailable, consider an initial dose of 100 mg (1-1.5 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.
## Drugs used in the treatment of Cardiac Arrest

### Appendix 2

### Magnesium
- **Shockable (VF/Pulseless VT)**
  - Dose: 2 g given peripherally IV
  - May be repeated after 10-15 min
  - Indicated for VT, torsade de pointes, or digoxin toxicity associated with hypomagnesaemia

- **Non-Shockable (PEA/Asystole)**
  - Dose: 2 g given peripherally IV
  - May be repeated after 10-15 min
  - Indicated for supraventricular tachycardia or digoxin toxicity associated with hypomagnesaemia

Magnesium facilitates neurochemical transmission: it decreases acetylcholine release and reduces the sensitivity of the motor endplate. Magnesium also improves the contractile response of the stunned myocardium, and may limit infarct size.

### Calcium
- **Shockable (VF/Pulseless VT)**
  - Not indicated for shockable rhythms

- **Non-Shockable (PEA/Asystole)**
  - 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
- **Shockable (VF/Pulseless VT)**
  - 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 because pulmonary gas exchange ceases and cellular metabolism becomes anaerobic. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. 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. During resuscitation, there are no clear advantages in using colloid, so use 0.9 % sodium chloride or Hartmann’s solution. Avoid dextrose, which is redistributed away from the intravascular space rapidly and causes hyperglycaemia, which may worsen neurological outcome after cardiac arrest.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Shockable (VF/Pulseless VT)</th>
<th>Non-Shockable (PEA/Asystole)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombolytics</td>
<td>• Tenecteplase 500-600 mcg kg⁻¹ IV bolus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Alteplase (r-tPA) 6 mg kg⁻¹ IV bolus</td>
<td></td>
</tr>
</tbody>
</table>

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.
APPENDIX 3.

Useful websites

www.erc.edu  European Resuscitation Council
www.resus.org.uk  Resuscitation Council UK
www.ilcor.org  International Liaison Committee on Resuscitation
www.americanheart.org  American Heart Association
www.bestbets.org  Best evidence topics in emergency medicine
www.escardio.org  European Society of Cardiology
www.escim.org  European Society of Intensive Care Medicine
www.euroanesthesia.org  European Society of Anaesthesiology
www.eusem.org  European Society for Emergency Medicine