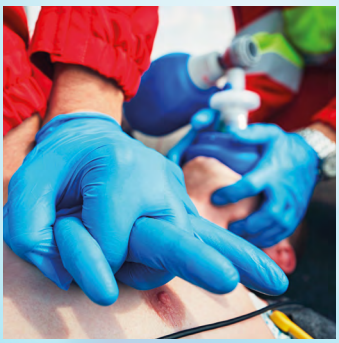
Advanced Life Support

ERC GUIDELINES 2021 EDITION



EUROPEAN RESUSCITATION COUNCIL

Advanced Life Support

ERC GUIDELINES 2021 EDITION

Edition 1

Advanced Life Support
Course Manual

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Contents

About the course

The advanced courses in adult resuscitation (ILS and ALS) aim at training all healthcare providers caring for adult patients. ILS focuses at the provider who is not regularly part of a resuscitation team, to recognise and manage critically ill patients and patients in cardiorespiratory arrest whilst awaiting the arrival of a resuscitation team **(first 5 minutes)** and then further provide support once the team arrives.

ALS aims at training all healthcare provider who might be part of the resuscitation team and specifically on the resuscitation team leader.

Where the 'hands-on' session will focus on performance, integrating both knowledge and skills about resuscitation, *the virtual learning environment* will optimise your cognitive preparation. Please take sufficient time to go through each of the lessons (allow at least 12 hours)."

When finished, we ask you to complete the final questionnaire as a mandatory test of readiness before attending the practical session.

This manual should thus be read in conjunction with the virtual learning materials (VLE) available at the European Resuscitation Council (ERC) **'CoSy'** website. As it is, the ERC e-learning english version is considered the most complete and up to date, including a lot of background information, whereas this manual only contains the core learning content as it is known at the date of publication. *For this reason, you will find **QR codes** in this manual that directly link to the chapters and/or audiovisual materials within the VLE. You can click on them or scan these codes with for instance your mobile phone.*

If you receive this manual as part of your preparation for an ERC course, you also have access to all relevant VLE e-learning materials via your CoSy account.

If you however bought this manual without being registered for a relevant ERC course, you might still gain access to the virtual materials by using the links and QR codes within this manual. The first time you do so, we will ask you to register to the platform and enter the following redeem code:

Sticker with redeem code to be added here.

To really train CPR and life support, you obviously need the knowledge (as provided by the given learning materials) but also should receive appropriate skill training. We therefore invite you – if not already done – to consider enrolling in a hands-on course. You can find a relevant course in your region via https://CoSy.erc.edu/en/sessions/calendar. Those completing a hands-on course also will receive an official ERC certificate.

Recertification & Lifelong Learning

With the launch of the new CoSy 2020, ERC has decided to better take into account the rapid decay in performance yet appreciating the difficult circumstances in which providers might work and the rules and regulations they need to comply with. ERC therefore decided to replace the end date of the certificate by a **deadline for recertification** via the ERC recertification program. This deadline for recertification is standard *2 year for advanced courses* (with a flexibility of up to 3 months). After this deadline, the only way to update your certificate is by redoing a full provider course.

To keep your certificate 'healthy', within the recertification program, ERC will demand you to do specific **'recertification'** activities at regular intervals. What these activities are is best found by going to your certificates in CoSy 2020 and clicking on the 'how to keep my certificates healthy' link.

! It might be that such recertification trajectory is not yet available in your country or for your course type and then the only way to update your certificate (date of last course) and keep access to all ERC learning materials is by doing a full provider course anew.

Glossary

ACS	acute coronary syndrome
AED	automated external defibrillator
ALS	Advanced Life Support
ARDS	adult respiratory syndrome
BLS	basic life support
CBP	cardiopulmonary bypass
COPD	chronic obstructive pulmonary disease
CPR	cardiopulmonary resuscitation
CRT	capillary refill time
CTPA	computed tomographic pulmonary angiography
DVT	deep venous thrombosis
ECLS	extracorporeal life support
ECMO	extra-corporeal membrane oxygenation
ECPR	extracorporeal CPR
EEG	electroencephalography
ELSO	extracorporeal life support organisation
EMS	Emergency medical services
$ETCO_2$	end-tidal CO_2
EWS	early warning score
GOLD	global initiative for chronic obstructive lung disease
HD	haemodialysis
HDU	high dependency unit
HEMS	helicopter emergency medical services
ICD	implanted cardioverter defibrillator
ICU	intensive care unit
IEC	International Electrotechnical Committee
IHCA	in-hospital-cardiac arrest
ILS	Immediate Life Support
IPCS	International program on Chemical Safety
JVP	jugular venous pulse
LLL	lifelong learning
LMA	laryngeal mask airway
MET	medical emergency team
MILS	manual in-line stabilisation
NIV	non-invasive ventilation
NSE	neuron-specific enolase
OHCA	out-of-hospital-cardiac arrest
PAD	public access defibrillation
PCI	percutaneous coronary intervention
PE	pulmonary embolism
PEA	pulseless electrical activity
PEEP	positive end-expiratory pressure
POCUS	point of care ultrasound
pVT	pulseless ventricular tachycardia
REBOA	resuscitative endovascular balloon occlusion of the aorta
ROSC	return of spontaneous circulation
RRT	rapid response team
RSVP	reason, story, vital signs, plan

RT	resuscitative thoracotomy
SBAR	Situation, Background, Assessment, and Recommendation
SCA	sudden cardiac arrest
SCD	sudden cardiac death
SGA	supraglottic airway device
SSEP	somatosensory evoked potential
STEMI	ST-elevation myocardial infarction
TCA	traumatic cardiac arrest
TTM	targeted temperature management
VF	ventricular fibrillation
VLE	virtual learning environment
WLST	withdrawal of life-sustaining treatment

In times of a pandemic...

Assuring the safety of the rescuer has always been a priority in ERC guidelines but lack of evidence has made it difficult to precisely define the associated risks. Rescuers may value the benefit for the patient more highly than their personal risk but should equally be aware of their responsibility towards their relatives, colleagues, and the wider community. In general, when there is a risk of transmission of a severe disease, *rescuers should use appropriate **PPE** (personal protection equipment) before providing life support.* Systems should be in place to facilitate this, and if extra time is required to achieve safe care this should be considered an acceptable part of the resuscitation process. Procedures and techniques that limit the risk of disease transmission (for instance by aerosol spread) are to be preferred.

A dedicated e-learning on CPR during the COVID-19 pandemic can be found in your CoSy account. The specific COVID-19 guideline text is also provided as background within the VLE.

CHAPTER 1
ALS IN PERSPECTIVE

Sudden cardiac arrest (SCA) is the third leading cause of death in Europe. Considerable effort has been made to understand the background and causes for cardiac arrest, and the differences in the incidence within and between countries. Factors influencing survival after out-of-hospital-cardiac arrest (OHCA) and in-hospital-cardiac arrest (IHCA) are well established but there remains considerable variation in incidence and outcome.

OUT OF HOSPITAL CARDIAC ARREST

The annual incidence of OHCA in Europe is between 67 to 170 per 100 000 inhabitants. Resuscitation is attempted or continued by emergency medical services (EMS) personnel in about 50-60% of cases (between 19 to 97 per 100 000 inhabitants). The rate of bystander cardiopulmonary resuscitation (CPR) varies between and within countries (average 58%, range 13% to 83%). The use of automated external defibrillators (AEDs) remains low in Europe (average 28%, range 3.8% to 59%). 80% of European countries provide dispatch assisted CPR and 75% have an AED registry. Most (90%) countries have access to cardiac arrest centres for post resuscitation care. Survival rates at hospital discharge are on average 8%, varying from 0% to 18%. Differences EMS in Europe account for at least some of the differences observed in OHCA incidence and survival rates.

IN HOSPITAL CARDIAC ARREST

The annual incidence of IHCA in Europe is between 1.5 and 2.8 per 1,000 hospital admissions. Factors associated with survival include the initial rhythm, the location, the degree of monitoring at the time of collapse (including witnessed status), and other patient factors (e.g. age and comorbidities). Survival rates at 30 days/ hospital discharge range from 15% to 34%.

LONG TERM OUTCOMES

In European countries where withdrawal of life sustaining treatment is routinely practiced, a good neurological outcome is seen in over 90% of patients. Most patients are able to return to work. In countries where withdrawal of life sustaining treatment is not practiced, poor neurological outcomes are more common (50% and 33% of patients remain in a persistent vegetative state).

Exposure to resuscitation, rather than years of experience, is associated with survival following OHCA. Whether a paramedic or physician-based EMS system effects outcomes is uncertain. EMS agencies with high survival rates often have:

- treatment by more than 6 EMS personnel within 15 min
- a short EMS call-response interval
- advanced airway attempts
- treatment from an ALS-BLS tiered system.

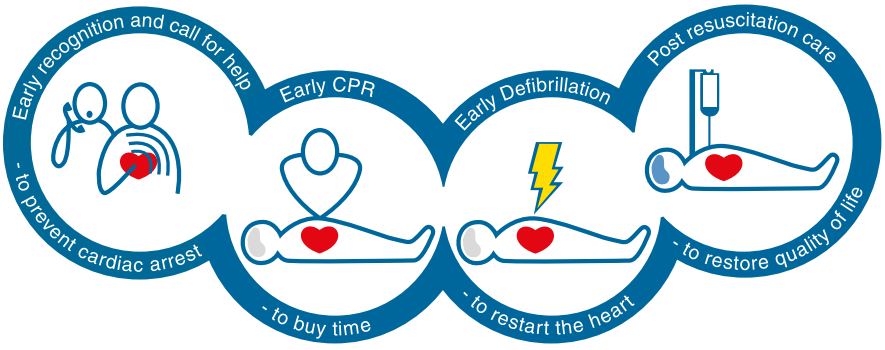
Team training in CPR involving all EMS personnel is reported for some parts of 26 European countries. Real-time CPR performance data collection for feedback and debriefing purposes is used in parts in 16 countries, in all areas only in Cyprus (ERC survey 2020).

THE CHAIN OF SURVIVAL

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

- early recognition and call for help
- early CPR
- early defibrillation
- post-resuscitation care

Figure 1.1 Chain of survival



Early recognition and call for help

In-hospital, early recognition of the critically ill patient who is at risk of cardiac arrest and a call for the resuscitation team or medical emergency team (MET) will enable treatment to prevent cardiac arrest. Hospitals should use a track-and-trigger early warning score system (EWS) for the early identification of patients who are critically ill or at risk of clinical deterioration. The EWS should have a predefined graded and escalating response according to the patient's score. The choice of system depends on local circumstances and should align with national guidelines.

All staff should be empowered to call for help and also trained to use structured communication tools such as SBAR or others to ensure effective communication.

SBAR:

- Situation
- Background
- Assessment
- Recommendation

A universal number for calling the resuscitation team or MET should be adopted in all European hospitals (2222).

Out of hospital, early recognition of the importance of chest pain will enable the victim or a bystander to call the EMS so that the victim can receive treatment that may prevent cardiac arrest. For OHCA, immediate access to the EMS is vital. In most countries, access to the EMS is achieved by means of a single telephone number (e.g. 112 or 999).

Early CPR

Chest compressions and ventilation will slow down the rate of deterioration of the brain and heart.

After cardiac arrest, bystander CPR extends the period for successful resuscitation and at least triples the chance of survival after ventricular fibrillation (VF) cardiac arrest. Performing chest-compression-only CPR is better than giving no CPR at all.

After IHCA, chest compressions and ventilation must be undertaken immediately, but should not delay attempts to defibrillate those patients in VF/pulseless ventricular tachycardia (pVT). Interruptions to chest compressions must be minimised and should occur only very briefly during defibrillation attempts and rhythm checks.

Early defibrillation

Several patient and CPR factors affect outcome from OHCA. Community programs of lay bystander CPR and AED use improve outcome from OHCA. Chest compressions and early defibrillation are the cornerstones of CPR in OHCA. The only definitive treatment for VF remains prompt defibrillation. In many areas, achievement of this goal will require the introduction of Public Access Defibrillation programs using AEDs.

Hospital systems should aim to recognise cardiac arrest, start CPR immediately, and defibrillate rapidly (<3 minutes) when appropriate. All hospital staff should be able to rapidly recognise cardiac arrest, call for help, start CPR and defibrillate (attach an AED and follow the AED prompts, or use a manual defibrillator).

Post-resuscitation care

Return of a spontaneous circulation (ROSC) is an important phase in the continuum of resuscitation; however, the ultimate goal is to return the patient to a state of normal cerebral function, a stable cardiac rhythm, and normal haemodynamic function, so that they can leave hospital in reasonable health at minimum risk of a further cardiac arrest. The quality of treatment in the post-resuscitation period influences the patient's ultimate outcome. The post-resuscitation phase starts at the location where ROSC is achieved. The ALS provider must be capable of providing high-quality post-resuscitation care until the patient is transferred to an appropriate high-care area. In OHCA, patients who achieve ROSC should be transferred to a designated centre of care such as a cardiac arrest centre.

LIFE SUPPORT COURSES

The ERC life support courses Immediate Life Support (ILS) and Advanced Life Support (ALS) provide a standardised approach to cardiopulmonary resuscitation in adults. The courses are targeted at physicians, nurses, and other healthcare professionals who are expected to provide ALS in and out of hospital. The multidisciplinary nature of the courses encourages efficient teamwork. By training together, all participants are given the opportunity to gain experience as both resuscitation team members and team leaders. ILS has its focus on management of the critically ill patient, recognition of cardiac arrest, start of resuscitation and team membership in the resuscitation team. ALS adds additional information on peri-arrest situations and special circumstances and has its focus on team leadership skills.

Resuscitation competences deteriorate with time and therefore recertification is required. Recertification provides the opportunity to refresh resuscitation competences and to be updated on resuscitation guidelines. There are different activities available for recertification. Information on these activities is available via the **ERC VLE**.

Going to your certificates in COSY 3.0 and clicking on the 'how to keep my certificates healthy' link.

Possible activities include:

- **ONLINE MODULES:** new modules will become available. In general, depending on the course type, you will have to complete one every two years.
- **HANDS-ON RECERTIFICATION MODULES:** these will be organised by your local course organiser or can also be followed at national or international congresses. In general, depending on the course type, you will also have to complete one of these every two years.
- **ASSESSMENT MODULES:** depending on the course type, official re-assessment is mandatory at a certain moment in your lifelong learning (LLL) trajectory (e.g. for ALS this is after 5 years). These assessment modules will also be organised by your local course organiser or at national or international congresses.

CHAPTER 2
NON-TECHNICAL SKILLS IN RESUSCITATION

LEARNING OUTCOMES
To understand:

- **the role of non-technical skills in resuscitation medicine**
- **the role of briefing and debriefing**
- **how to use structured communication tools such as SBAR and RSVP**

INTRODUCTION

Resuscitation requires teamwork which is evident throughout the patient care pathway: the pre-hospital resuscitation team, the in-hospital resuscitation team, and finally the prognostication resuscitation team. Effective team dynamics in resuscitation is of paramount importance in the delivery of safe patient care practice. Given its interdisciplinary nature, the excellence in CPR cannot be achieved by individual competence alone. Human factors and non-technical skills have been widely recognised as an essential part of high-quality ALS.

Human factors provide information on how healthcare professionals interact with clinical guidelines and procedures, with the resources available and with other health care providers taking behavioral factors, like management of stress, into account.

Non-technical skills examine the co-operation between team members by focusing on the interpersonal skills of leadership and team membership, in combination with the cognitive/ mental skills of teamwork, task management, situational awareness, and decision-making, all closely coupled with communication. Used integrally with medical knowledge and clinical skills, non-technical skills enclose safe and effective performance of CPR.

Often undesirable outcomes in resuscitation are due to underlying vulnerabilities and breakdown in human factors. Therefore, training and assessment of health care professionals in non-technical skills during formal hands-on courses and hospital training cannot be overemphasized. The principles of non-technical skills used in ALS courses are based on the "Team Emergency Assessment Measure". The proposed taxonomy in non-technical skills, adopted by the ERC is illustrated in table 2.1.

Table 2.1. Taxonomy of Non-Technical Skills adapted and modified from Cooper et al (2010) to be used in ALS training courses. See http://medicalemergencyteam.com/ for full details.

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

LEADERSHIP

Leadership during resuscitation is the art of guiding and motivating a group of healthcare professionals to act toward achieving a common objective – better survival. Leadership is not a trait, but rather an area for life-long improvement with continuous hands-on training. An effective resuscitation team leader is the person with a global perspective of the situation, who:

- Plans ahead.
- Allocates roles and distributes tasks by clear communication.
- Motivates team members by creating a positive atmosphere.
- Establishes clear decision-making process, summarises the situation and creates a “shared mental model”.
- Carefully monitors actions, remain "hands off".

TEAMWORK

Teamwork during resuscitation is the collaborative effort of two or more healthcare professionals with different backgrounds and complimentary skills to achieve the common goal or to complete a task in the most effective and efficient way. Typically, it encompasses the leader who distributes the workload to other team members by the means of:

- Effective verbal and non-verbal communication.
- Working together to complete tasks in a timely manner.
- Acting with composure and control.
- Adaptation to changing situations.
- Reassessment of the situation.
- Anticipation of potential actions.

It is important to specify the individual competences and to allocate appropriate roles accordingly.

TASK MANAGEMENT

Task management during resuscitation requires planning, tracking and executing relevant tasks by team members during the entire resuscitation effort. These include:

- Prioritization of the tasks that should be performed either simultaneously or sequentially.
- Effective use of all available resources.
- Adhering to current and approved guidelines and practices.
- Ensuring high-quality peri- and post-resuscitation care.

STRUCTURED COMMUNICATION

During resuscitation, precise and accurate communication using a closed-loop communication protocol is encouraged. That allows the sender to know that their requests have been heard and understood by the responder. Closed-loop communication and use of names and eye contact can reduce error rates by removing ambiguity from instructions. The Stop-Start sequence in chapter 4 is an example for this kind of communication. SBAR (Situation, Background, Assessment, and Recommendation) and RSVP (Reason, Story, Vital signs, plan) are acronyms that help in facilitating health professional communication ensuring clarity and completeness of information throughout verbal communication. The proposed taxonomy in SBAR and RSVP, adopted by the ERC is illustrated in table 2.2.

Table 2.2 SBAR and RSVP communication tools

SBAR	RSVP	Content	Example
Situation	**R**eason	• Introduce yourself and check you are speaking to the correct person. • Identify the patient you are calling about (who and where). • Say what you think, the current problem is, or appears to be. • State what you need advice about. • Useful phrases: - The problem appears to be cardiac/respiratory/neurological/sepsis. - I'm not sure what the problem is but the patient is deteriorating. - The patient is unstable, getting worse and I need help.	• Hello, I am Dr Smith the junior medical doctor. • I am calling about Mr Brown on acute medical admissions who I think has a severe pneumonia and is septic. • He has an oxygen saturation of 90 % despite high-flow oxygen and I am very worried about him
Background	**S**tory	• Background information about the patient • Reason for admission • Relevant past medical history	• He is 55 and previously fit and well. • He has had fever and a cough for 2 days. • He arrived 15 minutes ago by ambulance.

SBAR	RSVP	Content	Example

Assessment	**V**ital Signs	• Include specific observations and vital sign values based on ABCDE approach:
		  • Airway
		  • Breathing
		  • Circulation
		  • Disability
		  • Exposure
		  • The early warning score is…
			• He looks very unwell and is becoming tired.
			• Airway - he can say a few words.
			• Breathing - his respiratory rate is 24, he has bronchial breathing on the left side. His oxygen saturation is 90 % on high-flow oxygen. I am getting a blood gas and chest X-ray.
			• Circulation - his pulse is 110, his blood pressure is 110/60.
			• Disability - he is drowsy but can talk a few words.
			• Exposure - he has no rashes.

Recommendation	**P**lan	• State explicitly what you want the person you are calling to do.
		• What by when?
		• Useful phrases:
		  - I am going to start the following treatment; is there anything else you can suggest?
		  - I am going to do the following investigations; is there anything else you can suggest?
		  - If they do not improve; when would you like to be called?
		  - I don't think I can do any more; I would like you to see the patient urgently
			• I am getting antibiotics ready and he is on IV fluids.
			• I need help - please can you come and see him straight away.

BRIEFING AND DEBRIEFING

Communication during resuscitation is an interpersonal skill that results in effective information exchange among team leader and team members. Of note, whether verbal and/ or non-verbal, as well as informal and/ or structured, resuscitation teams may face various communication challenges at the personal and/or patient, professional, organizational, and team level which may affect the quality of resuscitative efforts. Effective resuscitation depends on every team member to work together. Communication represents the glue that holds the disparate members together.

With respect to resuscitation teams, it is useful to consider three phases of communication:

- Initial team briefing.
- Communication during resuscitation.
- Final team debriefing.

Ideally, the resuscitation team should meet at the beginning of each shift to outline a plan for potential resuscitation attempts. The aim of this briefing is to:

- Allocate individual roles according to the competence levels in the team.
- Allocate tasks to be completed.
- Create a same-shared mental model of the resuscitation modality.
- Share the anticipated plan of resuscitative efforts.
- Create awareness of potential challenges.
- Facilitate the team to ask questions and clarify any issues.

Debriefing after resuscitation takes place within a team following a clinical or simulated training case, with the intention of narrowing the gap between actual and desired performance. Debriefing interventions generally take two different formats:

- Hot debriefing, where individuals or teams are provided with debriefing immediately after the event. Ideally, there should be a quick hot debriefing following each resuscitation attempt.
- Cold debriefing, where individuals or teams are provided with feedback at a convenient time after the event.

The effective debriefing should:

- become a standard component of every resuscitation attempt (real-life or simulated).
- encourage team's own perceptions of their performance and their ideas for improvement

- ensure psychological safety while sharing emotions and experiences.
- utilize whenever possible, objective performance data, such as defibrillator downloads or videotape recordings.
- clearly state the performance criteria/ learning objectives.
- focus on specific behaviors rather than on general resuscitation team performance.
- emphasise what was directly observed during resuscitative efforts in a nonjudgmental language.
- be offered it at the time of a resuscitation event or shortly afterwards.
- be limited to the relevant events encountered throughout the course of resuscitation.
- focus on events to be improved or serving as learning models.
- summarise key points for learning and future implementation and name responsibilities for actions.

AUDIT AND REGISTRY

KEY LEARNING POINTS

- **non-technical skills are essential in resuscitation medicine.**
- **briefing and debriefing represent a standard of resuscitation care.**
- **utilize SBAR and RSVP for effective communication.**

see the CoSy VLE for more information

CHAPTER 3

RECOGNITION OF THE DETERIORATING PATIENT AND PREVENTION OF CARDIORESPIRATORY ARREST

LEARNING OUTCOMES
To understand:

- **The importance of early recognition of the deteriorating patient.**
- **How to identify and treat patients at risk of cardiorespiratory arrest using the Airway, Breathing, Circulation, Disability, Exposure (ABC-DE) approach.**

INTRODUCTION

Early recognition of the deteriorating patient and prevention of cardiac arrest is the first link in the chain of survival. Prevention of cardiac arrest requires education, monitoring of patients, recognition of patient deterioration, a system to call for help, and an effective response.

Most cardiorespiratory arrests in hospital are not sudden or unpredictable events: in approximately 80% of cases there is deterioration in clinical signs during the preceding few hours. These patients often have slow and progressive physiological deterioration, particularly hypoxia and hypotension (e.g. Airway-Breathing-Circulation problems).

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 cardiorespiratory resuscitation is not appropriate or who do not wish to be resuscitated.

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)*. The assessment of very simple vital signs, such as respiratory rate, may help to predict cardiorespiratory arrest. To help early detection of critical illness, many hospitals use early warning scores (EWS) or calling

criteria. Early warning scoring systems allocate points to measurements of routine vital signs on the basis of their derangement from an arbitrarily agreed 'normal' range. The weighted score of one or more vital sign observations, or the total EWS, indicates the level of intervention required, e.g. increased frequency of vital signs monitoring, or calling ward doctors or resuscitation teams to the patient.

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.

An increased score indicates an increased risk of deterioration and death. There should be a graded response to scores according to local hospital protocols.

Alternatively, systems incorporating calling criteria are based on routine observations, which activate a response when one or more variables reach an extremely abnormal value. It is not clear which of these two systems is better. Some hospitals combine elements of both systems.

RESPONSE TO CRITICAL ILLNESS

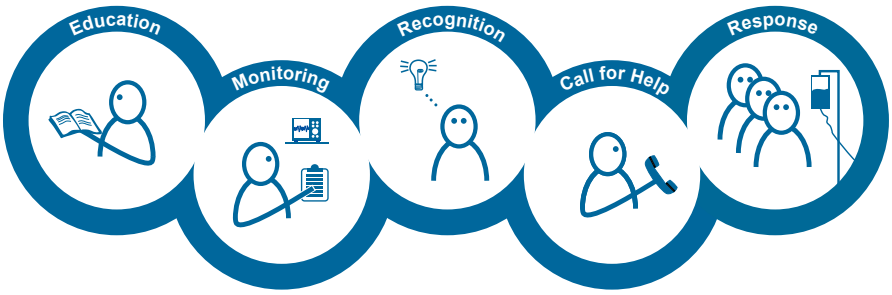
The traditional response to cardiac arrest is reactive: the name 'cardiac arrest team' implies that it will be called only after cardiac arrest has occurred. In some hospitals the cardiac arrest team has been replaced by other resuscitation teams (e.g. rapid response team, critical care outreach team, 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.

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

THE ABCDE APPROACH

The patient's assessment using the Airway-Breathing-Circulation-Disability-Exposure approach is the standard of care used by the European Resuscitation Council for immediate and advanced life support. The ERC Science and Education Committee for Advanced Life Support prepared and validated a cognitive aid for patient assessment *(figure 3.1)*.

Figure 3.1 Chain of Prevention



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 as soon as detected.
4. Assess the effects of treatment.
5. Recognise when you 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 2)*.
8. The aim of the initial treatment is to manage the life-threatening problems of the patient.
9. Remember - it can take a few minutes for treatments to work.

First steps

1. Ensure personal and patient safety. Organise a safe setting and secure environment. Wear apron, gloves, face masks/shields and glasses as appropriate.
2. First impression: Check the patient to see if the patient appears unwell.
3. If the patient is awake, ask "How are you?". If the patient appears unconscious or has collapsed, shake him and ask "Are you alright?" If he responds normally, he has a patent airway, is breathing and has brain perfusion. If he speaks only in short sentences, he may have breathing problems. Failure of the patient to respond is a clear marker of critical illness.

4. This first rapid "Look, Listen and Feel" of the patient should take no longer than 30 seconds.
5. If the patient is unconscious, unresponsive, and is not breathing normally start CPR according to the guidance in chapter 4. If you are confident and trained to do so, feel for a pulse to determine if the patient has a cardiac arrest. If there are any doubts about the presence of a pulse start CPR.
6. If the patient is not in cardiac arrest start the ABCDE approach.

Airway (A)

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

1. Look for the signs of airway obstruction:

- Airway obstruction causes paradoxical chest and abdominal movements *('see-saw' respirations)* and the use of the accessory muscles of respiration. Central cyanosis is a late sign of airway obstruction. In complete airway obstruction, there are no breath sounds at the mouth or nose. In partial obstruction, air entry is diminished and often noisy.
- In the critically ill patient, depressed consciousness often leads to airway obstruction.

2. Treat airway obstruction as a medical emergency:

- Obtain expert help immediately.
- In most cases, simple methods of airway clearance are required (e.g. airway opening manoeuvres, airways suction, insertion of an oropharyngeal or nasopharyngeal airway). Tracheal intubation may be required if these fail.

3. Give oxygen at high concentration:

- Provide 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.
- In acute respiratory failure, aim to maintain an oxygen saturation of 94–98%. In patients at risk of hypercapnic respiratory failure *(see below)* aim for an oxygen saturation of 88–92 %.

Breathing (B)

Life-threatening breathing problems comprise a number of conditions including acute severe asthma, exacerbation in COPD patients, pulmonary oedema, tension pneumothorax, massive pleural effusion, and massive haemothorax.

1. Look, listen and feel for the general signs of respiratory distress: sweating, central cyanosis, use of the accessory muscles of respiration, and abdominal breathing.
2. Count the respiratory rate. The normal rate is 12–20 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 on both sides.
4. Note any chest deformity (this may increase the risk of deterioration in the ability to breathe normally); look for a raised jugular venous pulse (JVP) (e.g. in acute severe asthma or a tension pneumothorax); note the presence and patency of any chest drains; remember that abdominal distension may limit diaphragmatic movement, thereby worsening respiratory distress.
5. Record the inspired oxygen concentration (%) and the SpO_2 reading of the pulse oximeter. If the patient is receiving supplemental oxygen, the SpO_2 may be normal in the presence of a very high $PaCO_2$.
6. Listen to the patient's breath sounds a short distance from his face: rattling airway noises indicate the presence of airway secretions, usually caused by the inability of the patient to cough sufficiently or to take a deep breath. Stridor or wheeze suggests partial, but significant, airway obstruction.
7. Percuss the chest: hyper-resonance may suggest a pneumothorax; dullness usually indicates consolidation or pleural fluid.
8. Auscultate the chest: bronchial breathing indicates lung consolidation with patent airways; absent or reduced sounds suggest a pneumothorax or pleural fluid or lung consolidation caused by complete bronchial obstruction.
9. Check the position of the trachea in the suprasternal notch: deviation to one side indicates mediastinal shift (e.g. tension pneumothorax).
10. Feel the chest wall to detect surgical emphysema or crepitus (suggesting a pneumothorax until proven otherwise).
11. The specific treatment of respiratory disorders depends upon the cause. Nevertheless, all critically ill patients should be given oxygen. In a subgroup of patients with 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 end-organ damage or cardiac arrest if their blood oxygen tensions are allowed to decrease.

In this group, aim for a lower than normal PaO_2 and oxygen saturation. Aim for target SpO_2 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).

12. If the patient's depth or rate of breathing is inadequate, or absent, use bag-mask or pocket mask ventilation to improve oxygenation and ventilation, whilst calling immediately for expert help.

In cooperative patients who do not have airway obstruction consider the use of non-invasive ventilation (NIV). In patients with an acute exacerbation of COPD, the use of NIV is often helpful and prevents the need for tracheal intubation and invasive ventilation.

Circulation (C)

Circulation problems may be caused by primary heart disease or by heart and circulation abnormalities secondary to other problems. Most often, circulation problems in acutely ill patients are due to hypovolaemia. In surgical patients, rapidly exclude haemorrhage (overt or hidden).

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

8. Auscultate the heart. Is there a murmur or pericardial rub? Are the heart sounds difficult to hear? Does the audible heart rate correspond to the pulse rate?

9. Look for other signs of a poor cardiac output, such as reduced conscious level and, if the patient has a urinary catheter, oliguria (urine volume < 0.5 ml kg^{-1} h^{-1}).

10. Look thoroughly for external haemorrhage from wounds or drains or evidence of concealed haemorrhage (e.g. thoracic, intra-peritoneal, retroperitoneal or into gut). Intra-thoracic, intraabdominal or pelvic blood loss may be significant, even if drains are empty.

11. The specific treatment of cardiovascular collapse depends on the cause, but should be directed at fluid replacement, haemorrhage control and restoration of tissue perfusion. Seek the signs of conditions that are immediately life threatening, e.g. cardiac tamponade, massive or continuing haemorrhage, septicaemic shock, and treat them urgently.

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

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

14. If there is no suspected injury lift the legs of the patient or put the patient into the Trendelenburg position. If the heart rate decreases and the blood pressure improves give a rapid fluid challenge (over 5-10 min) of 500 ml of warmed crystalloid solution (e.g. Ringers lactate 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 or trauma and use closer monitoring (listen to the chest for crackles after each bolus).

15. Reassess the heart rate and BP regularly (every 5 min), aiming for the patient's normal BP or, if this is unknown, a target > 100 mmHg systolic.

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

17. If symptoms and signs of cardiac failure (dyspnoea, increased heart rate, raised JVP, a third heart sound and pulmonary crackles on auscultation) occur, decrease the fluid infusion rate or stop the fluids altogether. Seek alternative means of improving tissue perfusion (e.g. inotropes or vasopressors).

18. If the patient has primary chest pain and a suspected ACS, record a 12-lead ECG early. Treat ACS according to the guidance in chapter 6.

Disability (D)

Common causes of unconsciousness include profound hypoxia, hypercapnia, cerebral hypoperfusion, intoxication, 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 mmol l^{-1}, give an initial dose of 50 ml of 10% glucose solution intravenously. If necessary, give further doses of intravenous 10% glucose every minute until the patient has fully regained consciousness, or a total of 250 ml of 10% glucose has been given. Repeat blood glucose measurements to monitor the effects of treatment. If there is no improvement consider further doses of 10% glucose.
6. Consider other causes of reduced levels consciousness like electrolyte disorders or metabolic disorders (elevated plasma ammonia in patients with liver disease).
7. Nurse unconscious patients in the lateral position if their airway is not protected.
8. Recognise neurologic deficits e.g. aphasia and other signs of stroke.

Exposure (E)

To examine the patient properly full exposure of the body may be necessary. Assess the patient from head to toe. Respect the patient's dignity, minimise heat loss and measure the body temperature.

Additional information

1. Take a full clinical history from the patient (e.g. SAMPLE), any relatives or friends, and other staff.
    - Signs and symptoms
    - Allergies
    - Medication
    - Past medical history / Pregnancy
    - Last oral intake
    - Events leading up to present problems

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 structured communication.
6. Record the patient's response to therapy.
7. Consider definitive treatment of the patient's underlying condition.

KEY LEARNING POINTS

- **It is important to recognise a deteriorating patient as early as possible**
- **The ABCDE approach allows identification and treatment of patients at risk of cardiorespiratory arrest**
- **The ABCDE approach is a structured tool to identify and manage life threatening problems in patients**

ABCDE chart

	EXAMINATION	INTERVENTION	GOAL
A 	• **airway noises** • **position of head** • **foreign body** • **fluid, secretions** • **oedema**	• open • suction • secure • O_2	**Patent airway**
B 	• **look - listen - feel approach** • **respiratory rate and effort** • **breath and added sounds** • **subcutaneous emphysema** • **symmetry of chest movement** • **tracheal deviation** • **jugular vein distention** • **cyanosis** SpO_2 - $ETCO_2$ - POCUS - X-ray - CT	• O_2 according to SpO_2 • pneumothorax therapy • inhalation therapy • ventilation	**Sufficient oxygenation and ventilation**
C 	• **heart rate** • **blood pressure** • **capillary refill time** • **bleeding** • **skin colour** • blood samples • diuresis ECG - POCUS - CT - X-ray	• I.V. / I.O. access • control of bleeding • massive haemorrhage protocol • fluids • drugs • transfusion	**Stabilization of circulation**
D 	• **AVPU / GCS** • **reactivity and symetry of pupils** • **blood glucose level** • **basic neurological examination** • posture • toxicological examination	• glucose • antidotes	**Evaluation of neurological state**
E 	• **head to toe examination** • **medical history** • temperature • injuries • oedemas • scars • signs of drug abuse • skin changes • signs of infection/sepsis	• identified cause therapy • thermomanagement • trauma treatment • insertion of NGT, IUC	**Revealing other symptoms and thermomanagement**

CHAPTER 4
ADVANCED LIFE SUPPORT

LEARNING OUTCOMES
To understand:

- **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**
- **specific strategies in IHCA and OHCA patients**
- **ethical considerations in cardiac arrest**

INTRODUCTION

Heart rhythms associated with cardiac arrest are divided into two groups:

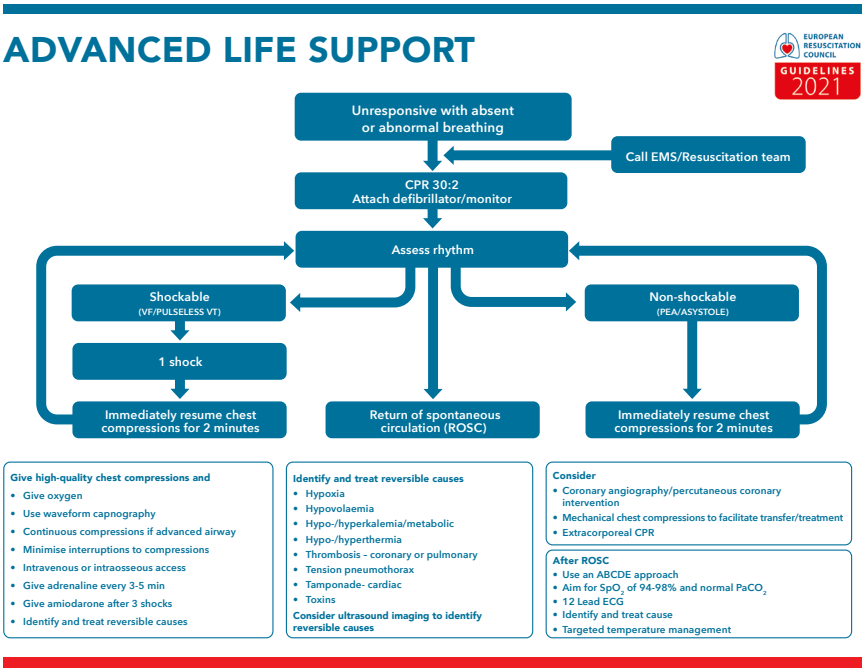
- shockable rhythms (ventricular fibrillation (VF)/ pulseless ventricular tachycardia (pVT))
- non-shockable rhythms (asystole and pulseless electrical activity (PEA)).

The difference in the management of these two groups of arrhythmias is the need for attempted defibrillation in patients with VF/pVT. Subsequent actions, including chest compressions, airway management and ventilation, venous access, injection of adrenaline and the identification and correction of reversible factors, are common to both groups.

The ERC ALS algorithm *(figure 4.1)* is a standardised approach to cardiac arrest management. This has the advantage of enabling treatment to be delivered expediently, without protracted discussion. It enables each member of the resuscitation team to predict and prepare for the next stage in the patient's treatment, further enhancing efficiency of the team using the shared mental model. Although the ALS algorithm is applicable to most cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances *(see chapter 6)*.

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander cardiopulmonary resuscitation (CPR), uninterrupted, high-quality chest compressions, and early defibrillation for VF/ pVT. All other interventions are of secondary importance.

Figure 4.1 The Advanced Life Support algorithm



MAINTAIN HIGH-QUALITY, UNINTERRUPTED CHEST COMPRESSIONS

The quality of chest compressions and ventilations are important determinants of outcome. Avoid interruptions in chest compressions because pauses cause coronary perfusion pressure to decrease substantially. Ensure that compressions are of adequate depth (at least 5 cm but not more than 6 cm) and rate (100-120 min^{-1}), and release pressure from the chest completely between compressions.

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

TREATMENT OF SHOCKABLE RHYTHMS (VF/ PVT)

For management of cardiac arrest patients during the COVID-19 pandemic see the ERC COVID guidelines: https://erc.edu/covid

1. Confirm cardiac arrest - check for signs of life or if trained to do so, breathing and pulse simultaneously.
2. Call the resuscitation team (or EMS for OHCA).
3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/ monitoring pads - one below the right clavicle and the other in the V6 position in the midaxillary line.
4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.
5. Use the "Stop – Start" sequence – with the "Stop" command chest compressions are interrupted, with the "Start" command immediately resumed.
6. Stop chest compressions not longer than 2 seconds to check the rhythm, resume compressions immediately.
7. Confirm VF/ pVT, if in doubt use a printout rhythm strip.
8. Hand over to an assigned team member for defibrillation.
9. Select the appropriate energy on the defibrillator (150-200 J biphasic for the first shock and 150-360 J biphasic for subsequent shocks) and press the charge button.
10. 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 if not using a closed system. Ensure that the rescuer giving the compressions is the only person touching the patient.
11. Once the defibrillator is charged, tell the rescuer doing the chest compressions to" Stop"; when clear, give the shock.
12. Use the "Start" command without reassessing the rhythm or feeling for a pulse, restart CPR using a ratio of 30:2, starting with chest compressions. The interruption for shock delivery should not exceed 3 seconds.
13. The command goes back to the team leader.
14. Continue CPR for 2 min; prepare the team for the next pause in CPR.
15. Use the "Stop – Start" sequence.
16. Stop chest compressions not longer than 2 seconds to check the rhythm, resume chest compressions immediately.
17. If VF/ pVT, repeat steps 8-13 above and deliver a second shock.

18. If VF/ pVT persists deliver a third shock as above.
19. If IV/ IO access has been obtained, give adrenaline 1 mg IV and amiodarone 300 mg IV as bolus during the next 2 minutes of CPR. Lidocaine 100 mg IV (IO) may be used as an alternative if amiodarone is not available or a local decision has been made to use lidocaine instead of amiodarone.
20. A rapid increase in end-tidal CO_2 on 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.
21. Give further adrenaline 1 mg IV after alternate shocks (i.e., in practice, this will be about once every two cycles of the algorithm).
22. Repeat amiodarone 150 mg once following the 5th shock if in persistent VT/ pVT.
23. Check for reversible causes (4 Hs, 4Ts) and manage where appropriate.
24. If signs of life return during CPR (purposeful movement, normal breathing or coughing) perform a rhythm check.
25. If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of ROSC.
26. If there is evidence of ROSC, start post-resuscitation care.
27. If no signs of ROSC, continue CPR and switch to the non-shockable algorithm.
28. If asystole is confirmed during a rhythm check continue CPR and switch to the non-shockable algorithm.

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 $ETCO_2$ or evidence of cardiac output on any invasive monitoring equipment). Rhythm checks must be brief, and pulse checks undertaken only if the rhythm has changed to an organised rhythm. 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.

It is important in shock-refractory VF/ pVT to check the position and contact of the defibrillation pads.

NON-SHOCKABLE RHYTHMS (PEA AND ASYSTOLE)

For management of cardiac arrest patients during the COVID-19 pandemic see the ERC COVID guidelines: https://erc.edu/covid

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 midaxillary line.
4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.
5. Use the "Stop – Start" sequence – with the "Stop" command chest compressions are interrupted, with the "Start" command immediately resumed.
6. Stop chest compressions not longer than 2 seconds to check rhythm, resume compressions immediately.
7. Confirm asystole or PEA and, without stopping CPR, check that the leads are attached correctly.
8. 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).
9. Continue CPR for 2 min; prepare the team for the next pause in CPR.
10. Use the "Stop – Start" sequence.
11. Stop chest compressions not longer than 2 seconds to check rhythm, Resume chest compressions immediately.
12. Check for reversible causes (4 Hs, 4Ts) and manage where appropriate.
13. If signs of life return during CPR (purposeful movement, normal breathing or coughing) perform a rhythm check.
14. If VF/ pVT at rhythm check, change to shockable side of algorithm.
15. If adrenaline has already been given before changing to the shockable side of the algorithm give further adrenaline 1 mg IV every 2-5 min following the initial application (i.e., in practice, this will be about once every two cycles of the algorithm).

If there is doubt about whether the rhythm is asystole or very fine VF, go for early defibrillation as VF should be shocked irrespective of the amplitude.

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves (p-wave asystole), because this may respond to cardiac pacing. There is no benefit in attempting to pace true asystole.

EXTRACORPOREAL CPR (ECPR)

Consider ECPR as a rescue therapy for selected patients with cardiac arrest when conventional ALS measures are failing or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI), pulmonary thrombectomy for massive pulmonary embolism, rewarming after hypothermic cardiac arrest) in settings in which it can be implemented.

REVERSIBLE CAUSES

Potential causes or aggravating factors for which specific immediate 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. *More details on these conditions are given in chapter 6.*

- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia and other metabolic disorders
- Hypothermia
- Thrombosis (pulmonary embolism or coronary thrombosis)
- Tamponade
- Tension pneumothorax
- Toxic agents

H

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. Check that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Hypovolaemic cardiac arrest will be caused in the majority of cases by haemorrhage, anaphylaxis or sepsis. Evidence might be obvious, e.g. trauma or active bleeding. Intravascular volume should be restored rapidly with fluid and blood or direct intervention if applicable. *Other measures are described in detail in chapter 6.*

Hypo-/hyperkalaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history e.g. renal failure *(chapter 6).*

Use a low reading thermometer to detect hypothermia.

T

The commonest cause of thromboembolic cardiac arrest is coronary thrombosis. Mechanical circulatory obstruction might be caused by massive pulmonary embolism. If pulmonary embolism is thought to be the cause cardiac arrest, consider giving a thrombolytic drug immediately.

Cardiac tamponade is difficult to diagnose because 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.

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

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

IN-HOSPITAL CARDIAC ARREST (IHCA)

In the hospital setting, the immediate availability of trained clinical staff and equipment provides an opportunity for the rapid identification of cardiac arrest and initiation of treatment. For IHCA, BLS and ALS interventions can often start and take place at the same time.

Where a telephone system is used to activate the emergency team, the standard European number (2222) should be implemented and be used.

Following the completion of initial actions, staff should collect ALS equipment and prepare to handover to the resuscitation team using either the SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital Signs, Plan) systems.

Each clinical area in a hospital should consider patient acuity, risk of cardiac arrest, and geographical location (e.g. distance for the resuscitation team to travel) in determining the specific training needs of staff and equipment (e.g. AED).

The resuscitation team may take the form of a traditional cardiac arrest team that responds only to cardiac arrest events or a MET/RRT (medical emergency team/ rapid response team) that responds to both cardiac arrests and critically unwell patients. Resuscitation teams often include individuals from a range of specialties (e.g. emergency medicine, cardiology, critical care) and the team members often arrive to the scene at different time points without knowing each other beforehand (ad hoc team). Lack of knowledge of team member roles, including who is acting as team

leader can lead to errors during CPR for IHCA. A team meeting at the beginning of each shift for introductions and allocation of roles may support effective team-working during resuscitation *(see chapter 2)*. Team and leadership training should be provided on a regular basis as it is associated with improved patient and process-outcomes.

Hospitals should ensure that clinical areas have immediate access to resuscitation equipment and drugs to facilitate rapid resuscitation of the patient in cardiac arrest. Equipment should be standardised throughout the hospital and should be checked regularly. A data-driven debriefing should be implemented after each resuscitation attempt *(see chapter 2)*.

OUT-OF-HOSPITAL CARDIAC ARREST (OHCA)

In most cases pre-hospital resuscitation has to be managed by fewer practitioners than would normally be present at an in-hospital arrest also, transportation to a receiving centre (ideally to a dedicated cardiac arrest center) adds an extra dimension. This emphasises the need for a structured and disciplined approach.

Chest compressions and early defibrillation are the cornerstones of CPR in OHCA. The only definitive treatment for VF remains prompt defibrillation.

The probability of successful defibrillation and subsequent survival to hospital discharge declines rapidly with time and the ability to deliver early defibrillation is one of the most important factors in determining survival from cardiac arrest.

EMS personnel should provide high-quality CPR while a defibrillator is retrieved, applied and charged. Defibrillation should not be delayed longer than needed to establish the need for defibrillation and charging.

Mechanical CPR, if available might maintain high-quality CPR during transfer by ambulance or helicopter.

If a cardiac arrest victim is transported to hospital, clear and accurate communication and documentation are essential elements of the handover to hospital staff.

A pre-alert message should be routine and is essential to ensure that emergency department staff and/or the hospital resuscitation team are ready to receive the patient. This gives time for the hospital resuscitation team to elect a team leader and assign roles to team members. Specific interventions to treat potentially reversible causes or specialist intervention can be arranged.

EMS should monitor exposure of their clinical personnel to resuscitation and implement strategies to address low exposure or ensure that treating teams have members with recent exposure or possibilities of simulated case-based training sessions.

DECISIONS RELATING TO RESUSCITATION

Systems, clinicians, and the public should consider CPR a conditional therapy. Clinicians should start CPR in patients who do not meet local criteria for withholding CPR. Treatments may then be tailored as more information becomes available.

No single factor, such as cardiac standstill on echocardiography, is suggested to be used for termination of CPR in isolation. For IHCA, there is no clinical decision rule that can predict death reliably. For OHCA, systems may consider implementing validated criteria for the termination of CPR, taking into consideration the specific local legal, organisational, and cultural context. In general, the ERC suggests that CPR may be terminated after 20 minutes of ongoing ALS with asystole and no reversible cause to treat. The following criteria may be considered for the withholding and termination of CPR:

Unequivocal criteria:

- When the safety of the rescuer cannot be adequately assured.
- When there is obvious mortal injury or irreversible death.
- When a valid and relevant advance directive becomes available that recommends against the provision of CPR.

Further criteria to inform decision making:

- Unwitnessed cardiac arrest with an initial non-shockable rhythm where the risk of harm to the patient from ongoing CPR likely outweighs any benefit e.g. absence of return of spontaneous circulation (ROSC), severe co-morbidity, very poor quality of life prior to cardiac arrest.
- Other strong evidence that further CPR would not be consistent with the patient's values and preferences, or in their best interests.
- In general, CPR should be continued as long as there is a shockable rhythm, considering e-CPR.

DEBRIEFING

Every resuscitation should be followed by a structured debriefing *(chapter 2)*. Where available, a data-driven performance-focused debriefing should be performed as this is associated with improved patient outcomes.

KEY LEARNING POINTS

- **The advanced life support (ALS) algorithm summarises management strategies in cardiac arrest.**
- **Minimally interrupted, high-quality chest compressions are of utmost importance.**
- **There are different treatment strategies for shockable and non-shockable rhythms.**
- **Adrenaline and amiodarone are relevant drugs during cardiac arrest.**
- **The potentially reversible causes of cardiac arrest must be considered.**
- **There are specific management strategies in IHCA and OHCA patients.**
- **Don't forget about the ethical considerations in cardiac arrest patients.**

CHAPTER 5

CARDIAC MONITORING, RHYTHM RECOGNITION, AND PERI-ARREST ARRHYTHMIAS

LEARNING OUTCOMES
To understand:

- **the reasons for ECG monitoring**
- **how to monitor the ECG**
- **how to recognise and treat rhythms associated with cardiac arrest**

INTRODUCTION

Treat the patient, not the ECG!

The analysis of cardiac rhythm abnormalities requires experience and expertise. However, the non-cardiologist can select the appropriate management strategy based on rhythm analysis. The priority is to recognise if the rhythm is abnormal and if the heart rate is inappropriately slow or fast. The need for immediate treatment will be determined mainly by assessing the patient with the ABCDE approach.

During cardiac arrest, immediate identification of the cardiac rhythm is paramount to determine the correct treatment. Cardiac monitoring and determination of the rhythm are one of the primary goals of the resuscitation team. The introduction of automated external defibrillators (AEDs) has enabled defibrillation to be performed by people who do not have skill in rhythm recognition, both in hospitals and in the community.

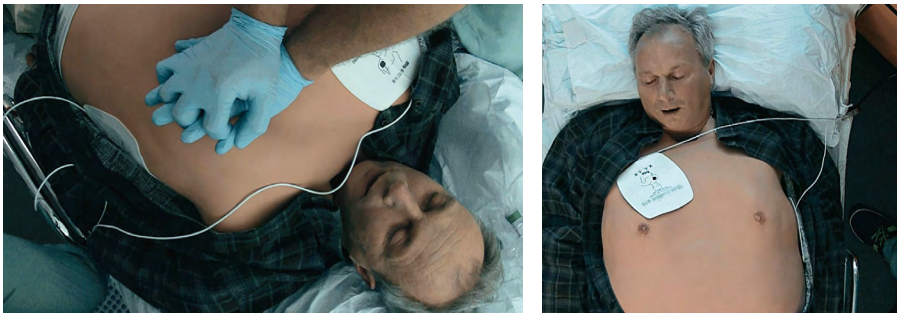
Some patients present with an arrhythmia that may lead to severe deterioration or even cardiac arrest. Early detection, treatment, and monitoring may prevent such complications. Monitoring alone will not always allow accurate rhythm recognition, and single-lead monitoring is not a reliable technique for detecting myocardial ischemia. A good-quality (serial) 12-lead ECG should be recorded whenever possible.

TECHNIQUES FOR ECG MONITORING

Cardiac monitors

Emergency monitoring

Figure 5.1 Defibrillator pads



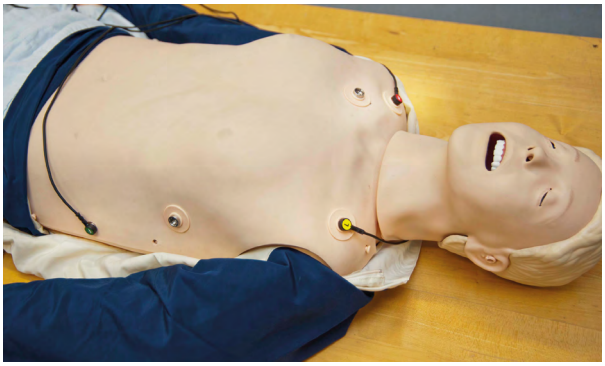
In a collapsed, non-responsive patient assess the cardiac rhythm as soon as possible by applying adhesive defibrillator pads. These can be used for single-lead monitoring and hands-free shock delivery. Apply the pads below the right clavicle and in the left mid-axillary line approximately level with the cardiac apex. Either pad can be placed in either position (apical or sternal).

Three-leads monitoring

Attach the ECG electrodes using the positions shown in figure 5.2. Ensure skin is dry, not greasy, shave off dense hair, and place electrodes over bone rather than muscle. Different electrode positions may be used when necessary.

Begin by monitoring lead II but switch to another lead if needed to obtain better ECG signals.

Figure 5.2 Position of electrodes for monitoring the ECG using modified limb leads



Diagnosis from cardiac monitors

Use displays and printouts from cardiac monitors only for rhythm recognition. When an arrhythmia is detected on a monitor, record a rhythm strip and preferably a diagnostic 12-lead ECG. Do not attempt to interpret ST-segment abnormalities from monitors or 3-leads ECG but use changes in the monitor signal as a guide to make a 12-lead ECG.

Valuable information can also be obtained by recording responses to treatment (e.g., carotid sinus massage, adenosine). Whenever possible, the effect of any such intervention should be recorded on a continuous ECG recording, preferably using multiple leads.

BASIC ELECTROCARDIOGRAPHY

The electrical signal that travels through the heart and triggers depolarisation of myocardial cells can be observed on an ECG *(figure 5.3)*. This depolarisation results in mechanical contraction. The repolarisation (returning to the negative resting membrane potential) is also visible on the ECG.

Figure 5.3 Components of the normal ECG signal

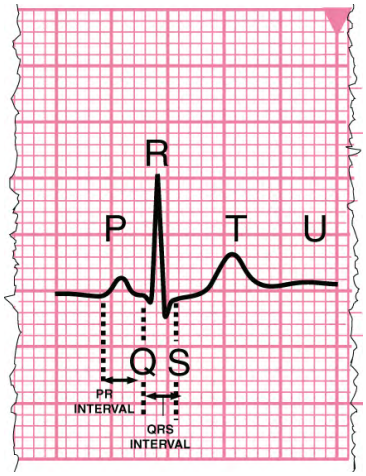


Table 5.1 Normal intervals on the ECG

Component	**Normal interval**
PR-interval	<0.2 s
QRS	<0.12 s
QT-interval	0.44-0.46 s
Ventricular rate	60-100 beats min^{-1}

HOW TO READ A RHYTHM STRIP

A simple, structured approach to interpreting the rhythm on any ECG recording will define the rhythm in sufficient detail to enable the most appropriate treatment.

Apply the following 6-step approach to the analysis of any rhythm on an ECG:

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

CARDIAC ARREST RHYTHMS

Rhythms present during cardiac arrest can be classified into four groups:

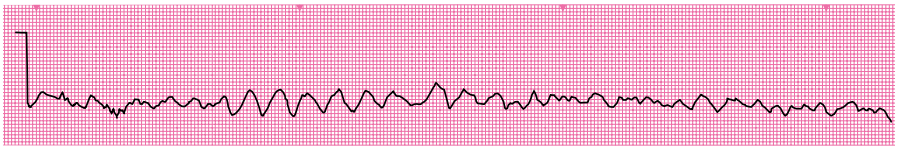
- Ventricular fibrillation (VF)
- Pulseless ventricular tachycardia (pVT)
- Asystole
- Pulseless electrical activity (PEA)

Extreme bradycardia and rarely very fast supraventricular tachyarrhythmia may cause such a severe fall in cardiac output to effectively cause cardiac arrest.

Ventricular fibrillation

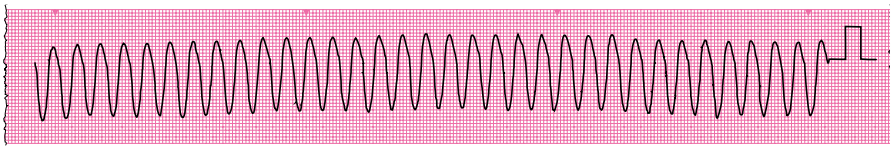
VF *(figure 5.4)* is usually easily recognisable and does not need the systematic rhythm analysis described earlier. In unresponsive patients, VF requires immediate defibrillation.

Figure 5.4 Fine ventricular fibrillation



Ventricular tachycardia

Figure 5.5 (Monomorphic) Ventricular tachycardia



VT *(figure 5.5)* may cause loss of cardiac output, particularly at faster rates or in the presence of structural heart disease, and may degenerate suddenly into VF. Pulseless VT is treated in the same way as VF with immediate defibrillation. VT with a pulse should follow the broad complex tachycardia algorithm described later in this chapter.

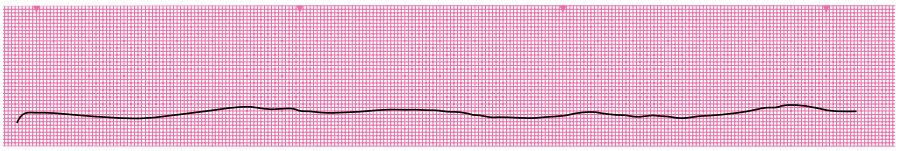
In the presence of a bundle branch block, a supraventricular tachycardia will produce a broad complex tachycardia. The safest approach is to regard all broad complex tachycardia as VT and call for expert help.

One important type of polymorphic VT is torsade de pointes, in which the axis of the electrical activity changes rotationally. The ECG resembles a sinusoidal pattern. It usually arises in a patient with a prolonged QT interval and can occur as an inherited or acquired phenomenon (drugs, ischemia).

Asystole

Asystole requires the absence of ventricular electrical activity (QRS). In rare circumstances, P-waves may be visible without QRS complexes; a P-wave asystole.

Figure 5.6 Asystole



Pulseless electrical activity

PEA *(figures 5.7 and 5.8)* does not refer to a specific rhythm but defines the absence of palpable pulse despite an electrical rhythm that should produce a palpable pulse.

Figure 5.7 PEA

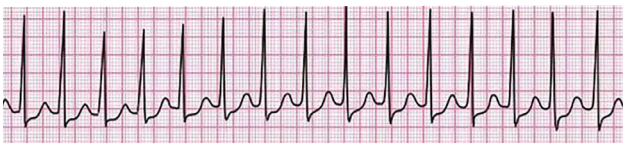
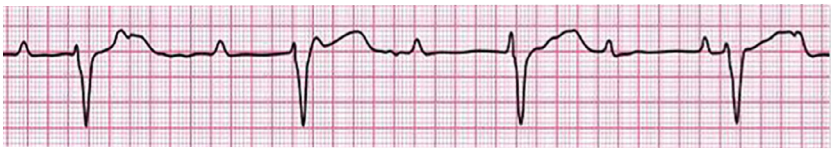


Figure 5.8 PEA



PERI-ARREST ARRHYTHMIAS

The treatment algorithms described in this section have been designed to enable the non-specialist ALS provider to treat a patient effectively and safely in an emergency. For this reason, they have been kept as simple as possible.

The patient's status determines management strategies for peri-arrest arrhythmias. Unstable patients showing adverse signs as described below, need early treatment to prevent deterioration rather than prolonged attempts to identify the precise rhythm.

In stable patients, there might be different treatment options. These include the use of drugs (oral or parenteral) that will be less familiar to the non-expert. For this patient group, it is recommended to seek expert advice.

Adverse features

The presence or absence of adverse signs or symptoms will dictate the urgency and choice of treatment for most arrhythmias. The following adverse features indicate that a patient is unstable and at risk of deterioration, wholly or partly because of the arrhythmia:

- **Shock.**
  Hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold extremities.
- **Syncope.**
  Transient loss of consciousness because of a global reduction in blood flow to the brain.
- **Heart failure.**
  Pulmonary oedema or raised jugular venous pressure (with or without peripheral oedema).
- **Myocardial ischaemia.**
  Typical ischaemic chest pain or evidence of myocardial ischaemia on a 12-lead ECG.
- Extremes of heart rate – in addition to the above adverse features, it may be appropriate to consider extremes of heart rate as adverse signs in themselves,

1. Extreme tachycardia. Rhythm abnormalities that cause very high heart rates (> 150 min^{-1}) reduce cardiac output dramatically and potentially cause myocardial ischaemia. The faster the heart rate, the less well it will be tolerated.

2. Extreme bradycardia. In general, the slower the bradycardia, the less well it will be tolerated. Heart rates below 40 min^{-1} are often tolerated poorly.

Treatment options

Depending on the clinical status of the patient (i.e., the presence or absence of adverse features) and the nature of the arrhythmia, immediate treatments can be categorised under five headings:

1. Eliminate or correct relevant triggers like ischaemia, hypoxia, acidaemia, hypo-, hyperkalaemia, drugs, stress, and pain.
2. Electrical (cardioversion for tachyarrhythmia or pacing for bradyarrhythmia).
3. Simple clinical intervention (e.g., vagal manoeuvres, percussion pacing).
4. Pharmacological (drug treatment).
5. No treatment needed.

Most drugs act slowly and less reliably than electrical treatments, so electrical treatment is preferred for an unstable patient with adverse features. If treating patients primarily with drugs, be aware of possible deterioration due to the drugs or the arrhythmia's natural course. Be prepared for immediate electrical treatment (defibrillation, cardioversion, or pacing).

If a patient develops an arrhythmia as a complication of some other condition (e.g., infection, AMI, heart failure), ensure that the underlying condition is assessed and treated appropriately, involving relevant experts if necessary.

Bradyarrhythmia

Bradycardia is present with a ventricular rate <60 min^{-1}. It may be a physiological state in very fit people, during sleep, or as the result of treatment. Several problems may cause symptomatic bradycardia.

Table 5.2 Types of bradyarrhythmia

Level of problem	
SA node	Sinus bradycardia
Sinus arrest	
AV node and bundle of His	(First degree AV-block)[a]
Second degree AV-block	Type I (Mobitz I) Type II (Mobitz II) 2:1 AV-block (Indetermined)
Third degree AV-block	

[a]Not a type of bradyarrhythmia per se since all atrial beats are conducted

If the normal cardiac impulse or conduction fails, depolarisation may be initiated from a "subsidiary" pacemaker in the myocardium. This pacemaker can be situated in the atria, AV conduction system, or ventricles. The resulting escape rhythm will be slower as its origin is more distal.

The emergency treatment of most bradycardias is atropine or cardiac pacing. Sympathomimetic drugs such as isoprenaline or adrenaline may be necessary.

Initial assessment

Assess the patient with bradycardia using the ABCDE approach. Consider the potential cause of the bradycardia and look for adverse signs. Treat any reversible causes of bradycardia identified in the initial assessment. In case of adverse signs, start treatment of the bradycardia. Initial treatment is pharmacological, with pacing being reserved for patients unresponsive to pharmacological therapy or at risk for asystole *(figure 5.6)*.

Pharmacological treatment for bradycardia

If adverse signs are present, give atropine (500 micrograms) intravenously. If necessary, repeat every 3-5 minutes to a total of 3 mg. Doses of atropine less than 500 micrograms may, paradoxically, cause further slowing of the heart rate. Use atropine cautiously in the presence of acute coronary ischaemia or myocardial infarction; increased heart rate may worsen ischaemia or increase the zone of infarction.

If treatment with atropine is ineffective, consider second-line drugs. These include isoprenaline (5 micrograms min^{-1} starting dose), adrenaline (2-10 micrograms min^{-1}), and dopamine (2-10micrograms kg^{-1} min^{-1}). Theophylline (100-200 mg slow intravenous injection) can be considered if the bradycardia is caused by inferior myocardial infarction, cardiac transplant, or spinal cord injury. Consider giving intravenous glucagon if beta-blockers or calcium channel blockers are a potential cause of bradycardia. Do not give atropine to patients with cardiac transplants – it can cause a high-degree AV-block or even sinus arrest.

Cardiac pacing for bradycardia

Initiate transcutaneous pacing immediately if there is no response to atropine or effect is unlikely.

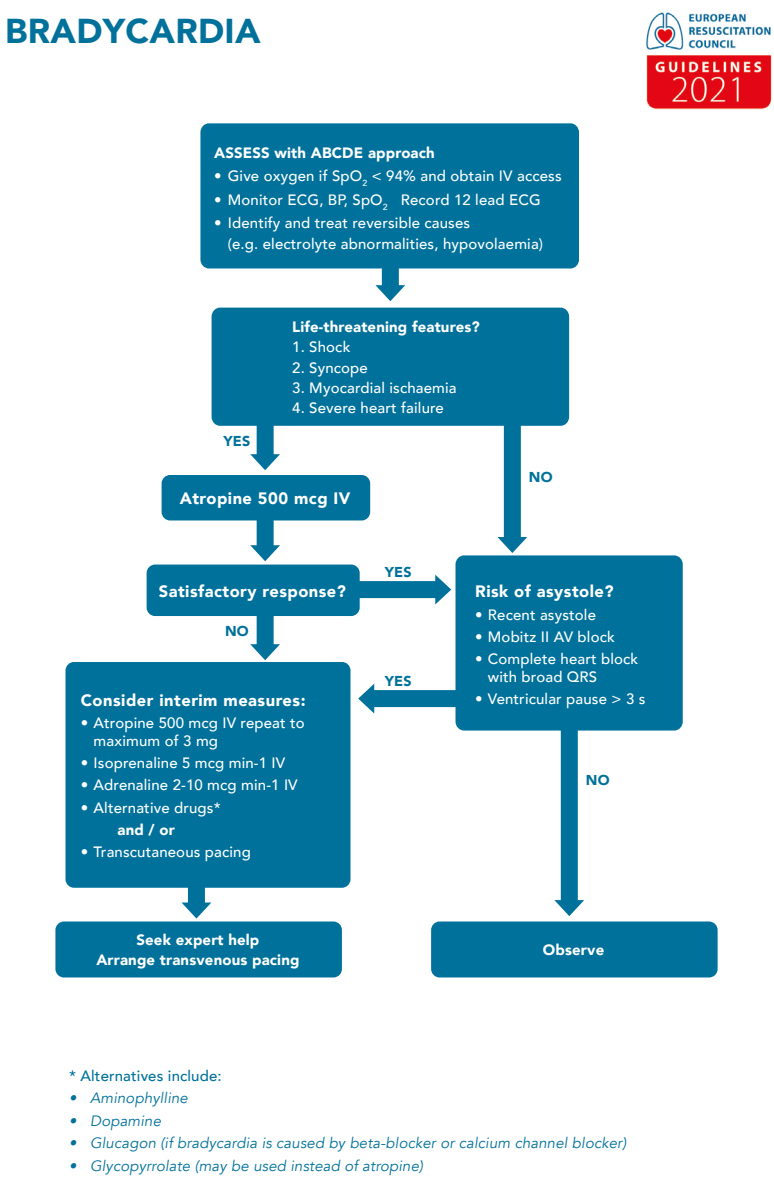
Transcutaneous pacing can be painful and may fail to produce effective mechanical capture. Verify mechanical capture and reassess the patient's condition. Use analgesia and sedation to control pain, and attempt to identify the cause of the bradyarrhythmia.

Seek expert help to assess the need for temporary transvenous pacing. Temporary transvenous pacing should be considered if there is a history of recent asystole, Mobitz type II AV-block, complete (third-degree) heart block (especially with broad QRS or initial heart rate < 40 beats min^{-1}), or evidence of ventricular standstill of more than 3 seconds.

If the patient has no adverse features

No immediate treatment is necessary for a patient with bradycardia and no adverse features or high risk of progression to asystole. Continue to monitor the patient. Assess the patient to identify the cause of the bradycardia. If the cause is physiological or reversible (e.g., by stopping suppressant drug therapy), no further treatment may be needed. Seek expert help to arrange appropriate further assessment and treatment for those with other causes of bradycardia.

Figure 5.9 Bradycardia algorithm



* Alternatives include:

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

Tachyarrhythmia

Tachycardia may arise from the atrial myocardium, the AV-junction, or ventricular myocardium. Sinus tachycardia is not an arrhythmia per se and usually represents a response to some other physiological or pathological state.

Tachycardia with adverse signs

Assess the patient with tachycardia using the ABCDE approach. Consider the potential cause of the tachycardia and look for the adverse signs. Treat any reversible causes of tachycardia identified in the initial assessment. If adverse signs are present, start treatment of the tachycardia. Initial treatment is synchronised cardioversion immediately *(figure 5.10)*. In patients with otherwise normal hearts, adverse signs are unlikely if the ventricular rate is < 150 beats min^{-1}. Patients with impaired cardiac function or significant comorbidity may be symptomatic and unstable at lower heart rates. If repeated cardioversion fails to restore sinus rhythm and the patient remains unstable, give amiodarone 300 mg intravenously over 10-20 minutes (or procainamide 10-15 mg kg^{-1}) and re-attempt electrical cardioversion afterwards. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 hours.

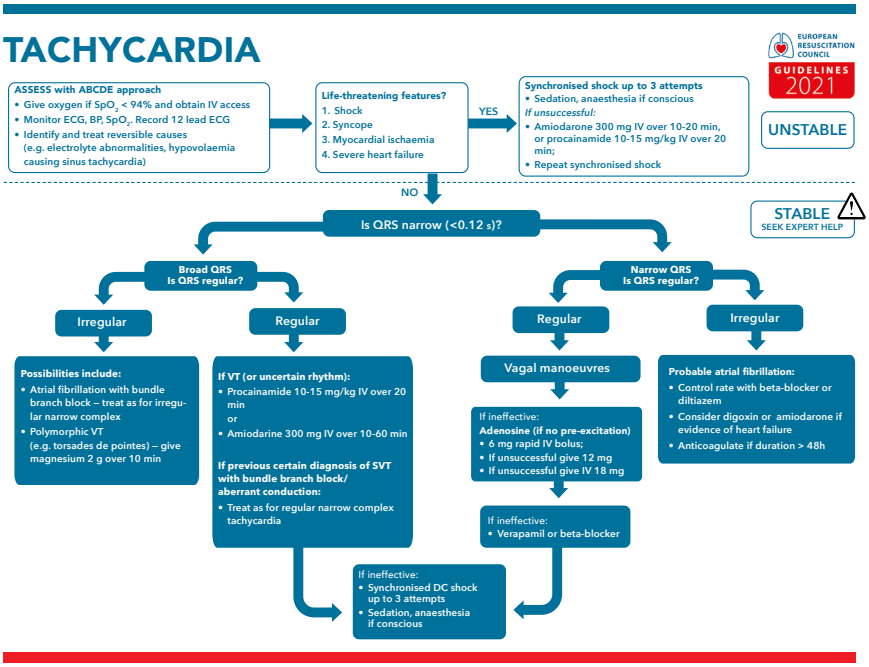
Repeated electrical cardioversions are not appropriate for recurrent (within hours or days) paroxysms (self-terminating episodes) of atrial fibrillation. This is relatively common in critically ill patients who may have ongoing precipitating factors causing the arrhythmia (e.g., metabolic disturbance, sepsis). Cardioversion does not prevent subsequent arrhythmias. If there are recurrent episodes, treat them with drugs and seek expert help.

Tachycardia without adverse signs

If the patient with tachycardia is stable, pharmacological treatment may be possible. Evaluate the rhythm using a 12-lead ECG and assess the QRS duration. If the QRS duration is equal to or greater than 0.12 seconds, it is classified as a broad-complex tachycardia. If the QRS duration is less than 0.12 seconds, it is a narrow-complex tachycardia.

All anti-arrhythmic treatments (physical manoeuvres, drugs, or electrical treatment) can also be pro-arrhythmic. Clinical deterioration may be caused by the treatment rather than a lack of effect. The use of multiple anti-arrhythmic drugs or high doses of a single one can cause myocardial depression and hypotension. This may cause a deterioration of the cardiac rhythm and the patient's condition. Expert help should be sought before using repeated doses or combinations of anti-arrhythmic drugs.

Figure 5.10 Tachycardia algorithm



Narrow-complex tachycardia

Regular narrow-complex tachycardia

Start with vagal manoeuvres such as carotid sinus massage or a Valsalva manoeuvre.

If the arrhythmia persists and is not atrial flutter, use adenosine. Give 6 mg (5-6 mg – depending on the preparation available) as a rapid intravenous bolus. Record an ECG (preferably multi-lead) during each injection; adenosine response can be therapeutic and diagnostic. If the ventricular rate slows transiently but the arrhythmia persists, look for atrial activity and treat accordingly. If there is no response to adenosine 6 mg, give a 12 mg bolus; if there is no response, consider an 18 mg bolus. This strategy will terminate 90-95% of supraventricular arrhythmias.

If adenosine is contraindicated or fails to terminate a regular narrow-complex tachycardia, seek expert help and consider a calcium channel blocker (e.g., verapamil or diltiazem).

Irregular narrow-complex tachycardia

An irregular narrow-complex tachycardia is most likely to be AF with an uncontrolled ventricular response or, less commonly, atrial flutter with variable AV-block. Record a 12-lead ECG to identify the rhythm.

Do not use adenosine if the rhythm is obviously atrial fibrillation/flutter. If there are no adverse features, treatment options include:

- Rate control by drug therapy (with beta-blocker or diltiazem, consider digoxin or amiodarone if evidence of heart failure).
- Rhythm control using drugs to encourage pharmacological cardioversion.
- Rhythm control by electrical cardioversion.

Obtain expert help to determine the most appropriate treatment for the individual patient. The longer a patient remains in AF, the greater the likelihood of developing atrial thrombus. In general, patients who have been in AF for > 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for three weeks. Seek expert advice on the duration of anticoagulation.

Broad complex tachycardia

Regular broad-complex tachycardia

Start with vagal manoeuvres such as carotid sinus massage or a Valsalva manoeuvre.

If broad complex tachycardia is present and vagal manoeuvres were ineffective, seek expert help and consider giving amiodarone 300 mg IV over 10-60 min.

If the treatment above is ineffective, consider up to 3 attempts of synchronised cardioversion.

Irregular broad-complex tachycardia

Irregular broad-complex tachycardia without adverse signs is most likely to be AF with bundle branch block. Another possible cause is AF with ventricular pre-excitation (Wolff–Parkinson–White (WPW) syndrome). In this case, there is more variation in the appearance and width of the QRS complexes than in AF with bundle branch block. A third possible cause is polymorphic VT (e.g., torsade de pointes), although this rhythm is relatively unlikely to be present without adverse features.

Seek expert help with the assessment and treatment of irregular broad-complex tachyarrhythmia.

Treat torsade de pointes VT immediately by stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate, 2 g, intravenously over 10 minutes. Obtain expert help, as other treatment (e.g., overdrive pacing) may be indicated to prevent relapse once the arrhythmia has been corrected. If adverse features develop (which is usual), arrange immediate synchronised cardioversion. If the patient becomes pulseless, attempt defibrillation immediately *(ALS algorithm)*.

KEY LEARNING POINTS

- **Monitor the ECG in patients at high risk of deterioration and cardiac arrest.**
- **Use a systematic approach to analyse the ECG**
- **Accurate rhythm recognition is essential for applying effective treatment**

CHAPTER 6
CARDIAC ARREST IN SPECIAL CIRCUMSTANCES

LEARNING OUTCOMES
To understand:
- **when and how to prioritise reversible causes in managing cardiac arrest in special circumstances**
- **how to modify the ALS algorithm in managing cardiac arrest in special circumstances**

INTRODUCTION

Irrespective of the special circumstances in cardiac arrest the most important interventions are universal and according to the chain of survival. These include:

- Early recognition and calling for help.
- Management of the deteriorating patient to prevent cardiac arrest.
- Prompt defibrillation and high-quality cardiopulmonary resuscitation (CPR) with minimal interruption of chest compressions.
- Treatment of reversible causes.
- Post-resuscitation care.

In certain conditions, however, basic and advanced life support interventions may require modification. This chapter provides guidelines on the modifications required to basic and advanced life support for the prevention and treatment of cardiac arrest in special circumstances:

- **Special causes**
  - Hypoxia
  - Hypovolaemia
  - Hypo-/ Hyperkalaemia and other electrolyte disorders
  - Hypo-/ Hyperthermia
  - Thrombosis
  - Cardiac tamponade
  - Tension pneumothorax
  - Toxic agents

- **Special settings**
  - Operating room
  - Cardiac surgery
  - Catheter laboratory
  - Dialysis unit
  - Dental clinics
  - Transportation
  - Sport
  - Drowning
  - Mass casualties
- **Special patient groups**
  - Asthma and COPD
  - Neurological disease
  - Obesity
  - Pregnancy

SPECIAL CAUSES

Hypoxia

Cardiac arrest solely due to hypoxaemia is uncommon. It is seen more commonly as a consequence of asphyxia, which accounts for most of the non-cardiac causes of cardiac arrest.

Modifications to CPR:

- Treat the cause of the asphyxia/ hypoxaemia as the highest priority because this is a potentially reversible cause of the cardiac arrest.
- Follow the European Resuscitation Council (ERC) Advanced Life Support (ALS) algorithm when resuscitating patients with asphyxial cardiac arrest.
- Effective ventilation with the highest feasible inspired oxygen is a priority in patients with asphyxial cardiac arrest.

Hypovolaemia

Hypovolaemia is a potentially treatable cause of cardiac arrest that usually results from a reduced intravascular volume (e.g. haemorrhage), but relative hypovolaemia may also occur in patients with severe vasodilation (e.g., anaphylaxis, sepsis, spinal cord injury).

Modifications to CPR:

- Depending on the suspected cause, initiate volume therapy with warmed blood products and/ or crystalloids, in order to rapidly restore intravascular volume.
- In the initial stages of resuscitation use any crystalloid solution that is immediately available, if haemorrhage is likely aim for early blood transfusion and vasopressor support.
- At the same time, initiate immediate intervention to control haemorrhage (e.g. surgery, endoscopy, endovascular techniques) or treat the primary cause (e.g. anaphylactic shock).
- Follow the ERC ALS algorithm when resuscitating patients with hypovolaemic cardiac arrest.
- If there is a qualified sonographer able to perform ultrasound with minimum interruption to chest compressions, it may be considered as an additional diagnostic tool in hypovolaemic cardiac arrest.

Traumatic cardiac arrest

Traumatic cardiac arrest (TCA) carries a very high mortality, but in those where return of spontaneous circulation (ROSC) can be achieved, neurological outcome in survivors appears to be much better than in other causes of cardiac arrest. The response to TCA is time-critical and success depends on a well-established chain of survival, including advanced prehospital and specialised trauma centre care.

Potentially reversible causes of TCA are:

- Hypoxaemia
- Hypovolaemia
- Tension pneumothorax
- Cardiac tamponade

Modifications to CPR:

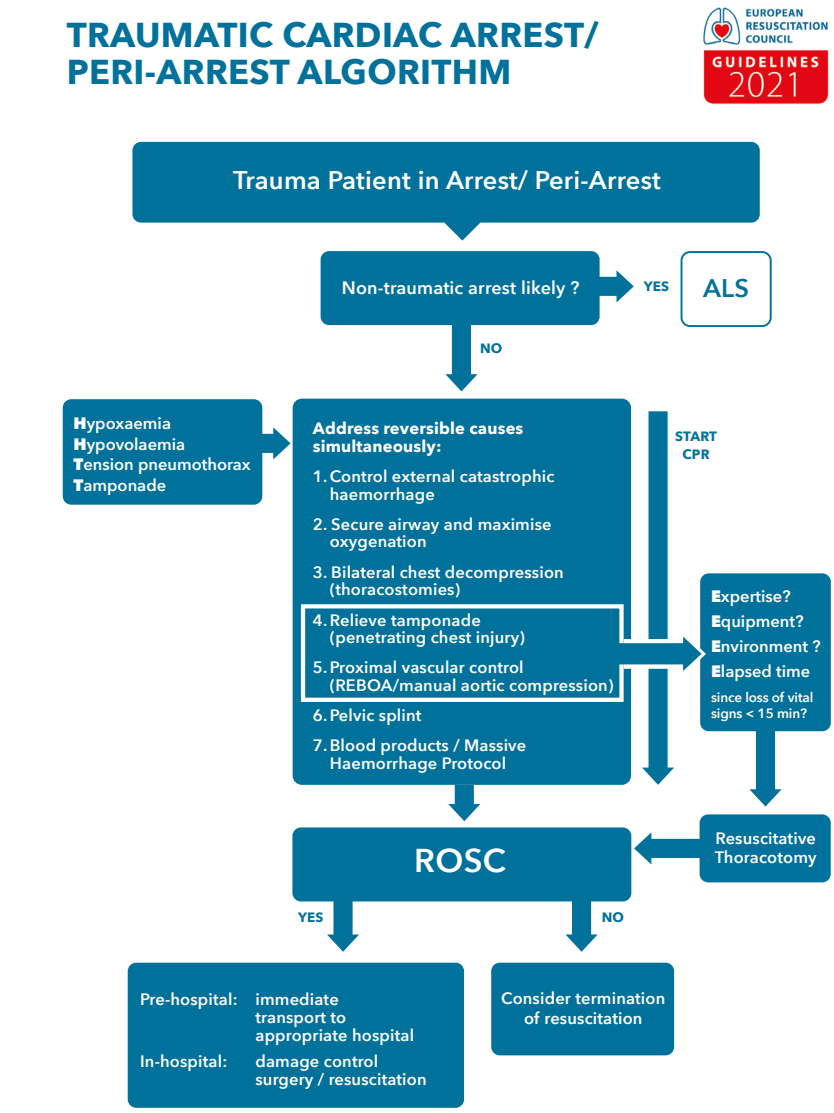
Follow the ERC traumatic cardiac (peri-) arrest algorithm when resuscitating patients with TCA *(Figure 6.1.)*.

- Immediate resuscitative efforts in TCA focus on simultaneous treatment (e.g., thoracotomy, controlling haemorrhage with external pressure, haemostatic

gauze, tourniquets and pelvic binder, etc.) of reversible causes, which takes priority over chest compressions.

- Immediate aortic occlusion is recommended as a last resort measure in patients with exsanguinating and uncontrollable infra-diaphragmatic torso haemorrhage. This can be achieved through Resuscitative Thoracotomy (RT) and cross-clamping the descending aorta or Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA).
- Use ultrasound to identify the underlying cause of cardiac arrest and target resuscitative interventions.
- Consider withholding resuscitation in TCA in any of the following conditions:
  - No signs of life within the preceding 15 min.
  - Massive trauma incompatible with survival (e.g., decapitation, penetrating heart injury, loss of brain tissue).
- Consider termination of resuscitative efforts if there is:
  - No ROSC after reversible causes have been addressed.
  - No detectable ultrasonographic cardiac activity in PEA after reversible causes have been addressed.

Figure 6.1 Traumatic cardiac (peri-) arrest algorithm



Anaphylaxis

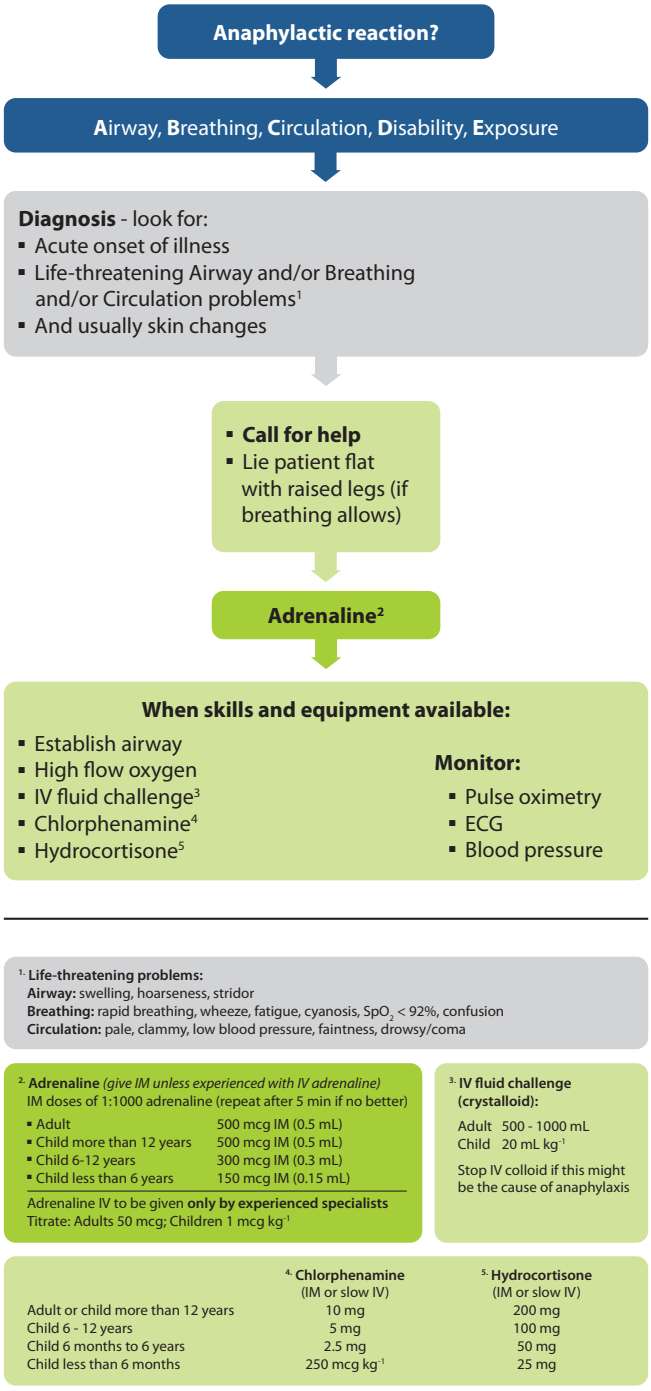
Anaphylaxis is a serious systemic allergic reaction that is rapid in onset and may cause death. Anaphylaxis causes life threatening airway (swollen lips, tongue, uvula), breathing (dyspnoea, wheeze, bronchospasm, stridor, reduced peak flow, hypoxaemia) and circulation problems (hypotension, cardiac arrest) with or without skin or mucosal changes (generalised urticaria, flushing or itching) as part of an allergic reaction. This can be in the context of a known trigger in a patient with an allergy, or suspected anaphylaxis in a patient with no previous history of allergy.

Modifications to CPR:

The key steps are described in the anaphylaxis algorithm *(Figure 6.2.)*:

- Call for help early.
- Remove or stop the trigger if feasible.
- Give intramuscular (IM) adrenaline (0.5 mg (which is 0.5 mL of a 1mg in 1 mL ampoule of adrenaline)) into the anterolateral thigh as soon as anaphylaxis is suspected. Repeat the IM adrenaline if there is no improvement in the patient's condition after about 5 min.
- Ensure the patient is lying and do not suddenly sit or stand the patient up. Use an ABCDE approach and treat problems early (oxygen, fluids, monitoring).
- Give an intravenous (IV) crystalloid fluid bolus early and monitor the response – large volumes of fluids may be needed.
- Consider IV adrenaline as a bolus (20-50 mcg) or infusion for refractory anaphylaxis or in specialist care settings where the skills are available.
- Consider alternative vasopressors (vasopressin, noradrenaline, metaraminol, phenylephrine) in refractory anaphylaxis.
- Consider IV glucagon in patients taking beta-blockers.
- Start chest compressions and ERC ALS algorithm as soon as cardiac arrest is suspected and follow standard guidelines.
- Consider Extracorporeal Life Support (ECLS) or Extracorporeal Cardiopulmonary Resuscitation (ECPR) for patients who are peri-arrest or in cardiac arrest as a rescue therapy in those settings where it is feasible.
- Follow existing guidelines for the investigation and follow-up care of patients with suspected anaphylaxis and confirmed anaphylaxis.

Figure 6.2 Anaphylaxis algorithm



Sepsis

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Septic shock is a subset of sepsis with circulatory and cellular/ metabolic dysfunction associated with a higher risk of mortality.

Modifications to CPR:

Key steps in the initial treatment and management of severe sepsis to prevent cardiac arrest in adults are summarized in the Surviving Sepsis Guidelines Hour-1 bundle *(Figure 6.3.)*:

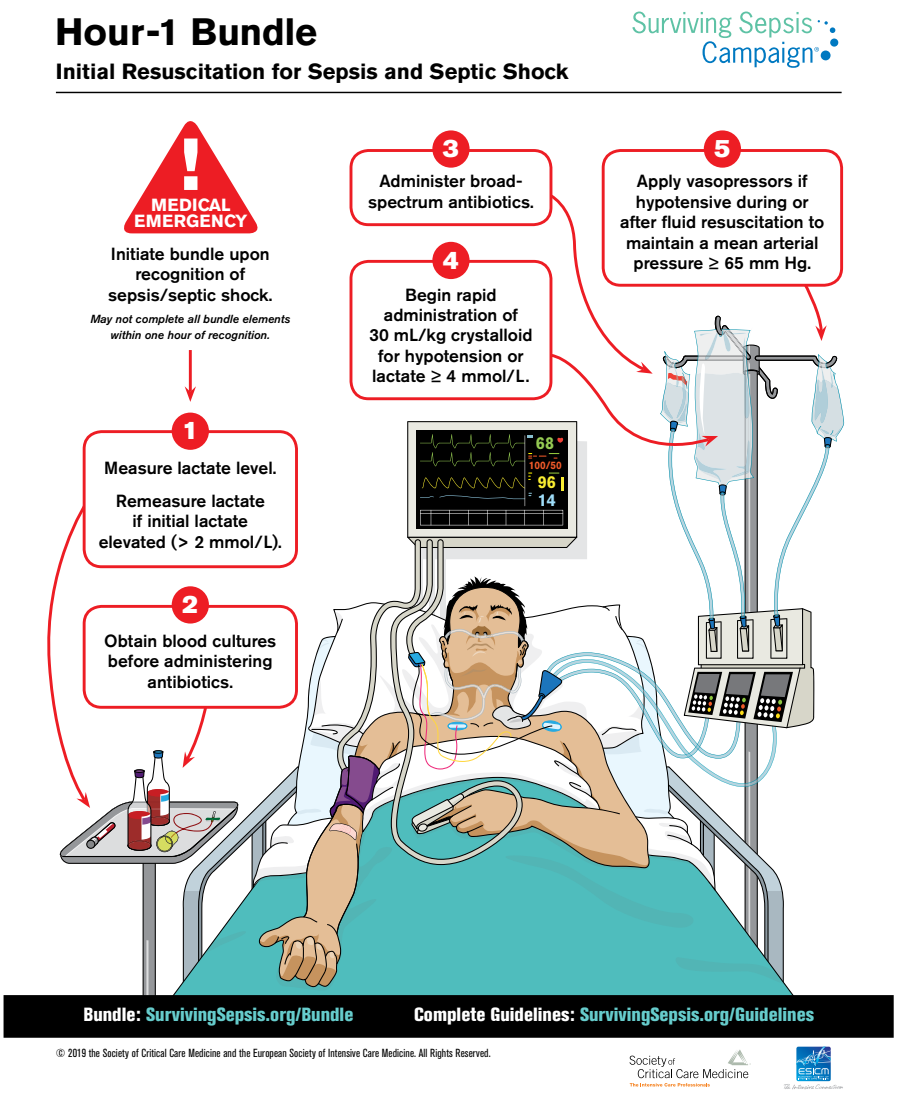
Prevention of cardiac arrest:

- Assess the patient using the ABCDE approach.
- Control the underlying source of infection to prevent shock, multi-organ failure and cardiorespiratory arrest.
- Administer high-flow oxygen to optimize oxygen delivery to tissues.
- Measure lactate levels.
- Obtain blood cultures prior to administration of broad-spectrum antibiotics.
- Begin rapid administration of 30 ml/kg crystalloid for hypotension or a lactate ≥4 mmol L^{-1}.
- Measure hourly urine output to help guide IV fluid therapy.
- Apply vasopressors if the patient is hypotensive during or after fluid resuscitation to maintain mean arterial pressure ≥65 mmHg.

Management of cardiac arrest

- Intubate the trachea if able to do so safely.
- Intravenous (IV) crystalloid fluid resuscitation with a 500ml initial bolus. Consider administering further boluses.
- Venepuncture for venous blood gas/ lactate/ electrolytes.
- Control the source of sepsis, if feasible, and give antibiotics early.

Figure 6.3 Surviving Sepsis Guidelines Hour-1 bundle



Hypo-/ hyperkalaemia and other electrolyte disorders

Electrolyte abnormalities are recognised causes of arrhythmias and cardiac arrest. Potassium disorders, hyperkalaemia and hypokalaemia are the most common electrolyte disturbances associated with life-threatening arrhythmias, whilst calcium and magnesium disorders occur less commonly.

Modifications to CPR:

- Assess the patient using the ABCDE approach.
- Utilize point-of-care testing (e.g. blood gas analyser) for electrolyte disorders if available.
- Obtain IV access and correct any electrolyte abnormalities.
- Use ECG, if already available as diagnostic tool, and look for abnormalities.

Hyperkalaemia

There is no universal definition of hyperkalaemia. We have defined hyperkalaemia as a serum potassium (K^+) concentration greater than 5.5 mmol/l, although in clinical practice, hyperkalaemia is a continuum. Hyperkalaemia may be categorised as:

- Mild (K^+ 5.5 – 5.9 mmol/l)
- Moderate (K^+ 6.0 – 6.4 mmol/l)
- Severe ($K^+ \geq 6.5$ mmol/l)

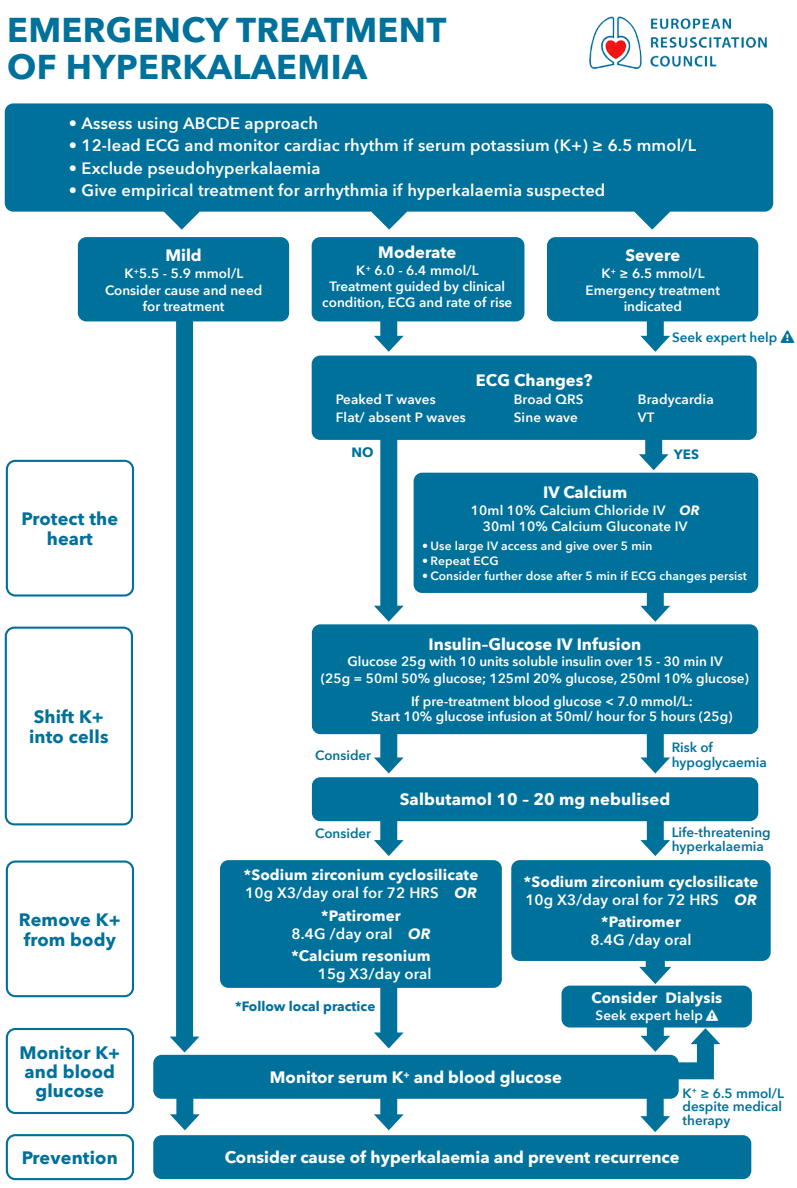
The severity of hyperkalaemia guides response to treatment. Treat the cause of the hyperkalaemia as the highest priority because this is a potentially reversible cause of the cardiac arrest.

There are five key steps in the treatment of hyperkalaemia:

- Protect the heart.
- Shift K^+ into cells.
- Remove K^+ from the body.
- Monitor serum K^+ and glucose levels.
- Prevent the recurrence of hyperkalaemia.

Follow a systematic approach, guided by the severity of hyperkalaemia and ECG changes, as outlined in the hyperkalaemia treatment algorithm *(Figure 6.4.)*.

Figure 6.4 Hyperkalaemia treatment algorithm



Emergency treatment of hyperkalaemia. ECG – electrocardiogram; VT ventricular tachycardia.

Modifications to CPR:

- Confirm hyperkalaemia using blood gas analyser if available.
- Protect the heart: Give 10 ml calcium chloride 10% IV by rapid bolus injection. Consider repeating dose if cardiac arrest is refractory or prolonged.
- Shift K^+ into cells: Give 10 units soluble insulin and 25g glucose IV by rapid injection. Monitor blood glucose. Administer 10% glucose infusion guided by blood glucose to avoid hypoglycaemia.
- Shift K^+ into cells: Give 50 mmol sodium bicarbonate (50 ml 8.4% solution) IV by rapid injection.
- Remove K^+ from the body: Consider dialysis for refractory hyperkalaemic cardiac arrest.
- Consider the use of a mechanical chest compression device if prolonged CPR is needed.
- Consider ECLS or ECPR for patients who are peri-arrest or in cardiac arrest as a rescue therapy in those settings where it is feasible.

Hypokalaemia

Hypokalaemia is a common electrolyte disorder in clinical practice. It is associated with higher in-hospital mortality and an increased risk of ventricular arrhythmias. The risk of adverse events is increased in patients with pre-existing heart disease and in those treated with digoxin. Hypokalaemia is defined as a serum $K^+ < 3.5$ mmol/l. The severity of hypokalaemia guides response to treatment. Treat the cause of the hypokalaemia as the highest priority because this is a potentially reversible cause of the cardiac arrest.

There are four key steps in the treatment of hypokalaemia:

- Restore K^+ level (rate and route of replacement guided by clinical urgency).
- Check for any potential exacerbating factors (e.g., digoxin toxicity, hypomagnesaemia).
- Monitor serum K^+ (adjust replacement as needed depending on level).
- Prevent recurrence (assess and remove cause).

Modifications to CPR:

- Slow replacement of potassium is preferable, but in an emergency, more rapid IV replacement is required. The standard rate of infusion of potassium is 10 mmol/hr. The maximum rate is 20 mmol/hr, but more rapid infusion (e.g. 2 mmol/ min for 10 min, followed by 10 mmol over 5-10 min) is indicated for unstable arrhythmias when cardiac arrest is imminent.

- Continuous ECG monitoring is essential, ideally in a high dependency area.
- Monitor potassium level closely and titrate rate of replacement according to the level.
- Magnesium is important for potassium uptake and for the maintenance of intracellular potassium concentration, particularly in the myocardium. Magnesium deficiency is common in patients with hypokalaemia. Repletion of magnesium will facilitate more rapid correction of hypokalaemia. If hypokalaemia occurs concurrently with hypomagnesaemia, give 4ml magnesium sulphate 50% (8 mmol) diluted in 10ml NaCl 0.9% over 20 minutes, followed by potassium replacement (40 mmol KCL in 1000ml 0.9% NaCl at a rate guided by urgency for correction as above). Follow with further magnesium replacement.

Hypothermia/ Hyperthermia

Accidental Hypothermia

Accidental hypothermia is the involuntary drop in core temperature <35°C. Primary hypothermia is induced by exposure to cold, while secondary hypothermia is induced by illness and other external causes.

Modifications to CPR:

Follow a systematic approach as outlined in the management of accidental hypothermia algorithm *(Figure 6.5.)*.

- Assess core temperature with a low reading thermometer (e.g., tympanic in spontaneously breathing patients, oesophageal in patients with a tracheal tube or a supraglottic device with an oesophageal channel in place).
- Check for the presence of vital signs for up to one minute.
- Prehospital insulation, triage, fast transfer to a hospital and rewarming are key interventions.
- Hypothermic patients with risk factors for imminent cardiac arrest (e.g., core temperature <30°C, ventricular arrhythmia, systolic blood pressure <90 mmHg) and those in cardiac arrest should ideally be directly transferred to an extracorporeal life support (ECLS) centre for rewarming.
- Hypothermic cardiac arrest patients should receive continuous CPR during transfer.
- Chest compression and ventilation rate should not be different to CPR in normothermic patients.
- If ventricular fibrillation (VF) persists after three shocks, delay further attempts until the core temperature is >30°C.

- Withhold adrenaline if the core temperature is <30°C.
- Increase administration intervals for adrenaline to 6-10 min if the core temperature is >30°C.
- If prolonged transport is required or the terrain is difficult, use of a mechanical CPR device is recommended.
- In hypothermic arrested patients < 28°C delayed CPR may be used when CPR on site is too dangerous or not feasible, intermittent CPR can be used when continuous CPR is not possible.
- In-hospital prognostication of successful rewarming should be based on the Hypothermia Outcome Prediction after ECLS (HOPE) score *(Table 6.6.)*. The traditional in-hospital serum potassium prognostication is less reliable.
- In hypothermic cardiac arrest rewarming should be performed with ECLS, preferably with extra-corporeal membrane oxygenation (ECMO) over cardiopulmonary bypass (CPB).
- Non-ECLS rewarming should be initiated in a peripheral hospital if an ECLS centre cannot be reached within hours (e.g. 6 hours).

Figure 6.5 Management of accidental hypothermia algorithm

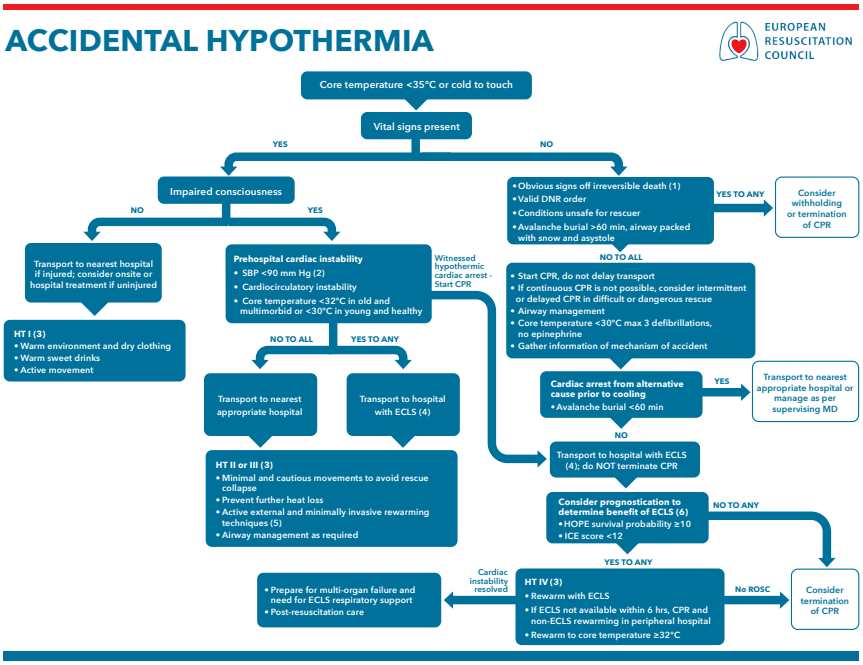


Table 6.1 HOPE score for hypothermic cardiac arrest patients, description of parameters affecting HOPE with regard to estimation of the survival probability. CPR denotes cardiopulmonary resuscitation, ECLS extracorporeal life support.

	Definition
Age (yrs)	On site or in hospital
Sex	On site or in hospital
Core temperature (°C/°F)	First measurement at hospital admission
Serum potassium (mmol/L)	First measurement at hospital admission
Presence of asphyxia	Asphyxia (head fully covered by water or snow) AND in cardiac arrest at extrication. No asphyxia: immersion, outdoor or indoor exposure. Data recorded on site
Duration of CPR (min)	From initiation of manual CPR until expected start of ECLS. Data recorded prehospitally and in-hospital once establishment of ECLS can be expected.

Avalanche Rescue

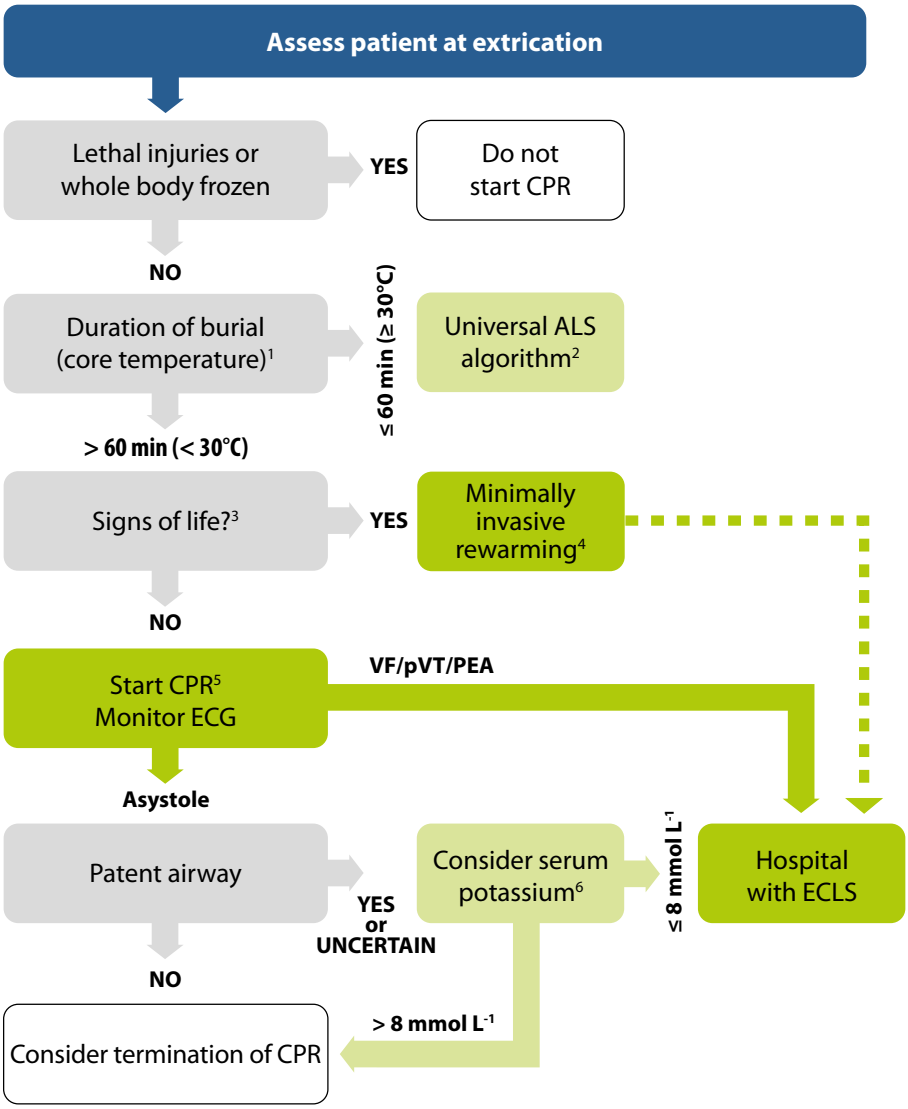
Most avalanche victims die from asphyxia. Avalanche victims in an unwitnessed cardiac arrest have a poor chance of survival. The chances of a good outcome are improved if there is a ROSC in the first minutes of CPR.

Modifications to CPR:

Follow a systematic approach as outlined in the avalanche accident algorithm for completely buried victims *(Figure 6.6.)*.

- Start with five ventilations in cardiac arrest, as hypoxia is the most likely cause of cardiac arrest.
- Perform standard ALS if burial time is <60 minutes.
- Provide full resuscitative measures, including ECLS rewarming, for avalanche victims with duration of burial >60 minutes without evidence of an obstructed airway or additional un-survivable injuries.
- Consider CPR to be futile in cardiac arrest with a burial time >60 minutes and additional evidence of an obstructed airway.
- In-hospital prognostication of successful rewarming should be based on the HOPE score *(Table 6.1)*. The traditional triage with serum potassium and core temperature (cut-offs 7mmol/L and 30°C, respectively) is less reliable.

Figure 6.6. The avalanche accident algorithm for completely buried victims.



[1] Core temperature may substitute if duration of burial is unknown
[2] Transport patients with injuries or potential complications (e.g. pulmonary oedema) to the most appropriate hospital
[3] Check for spontaneous breathing and pulse for up to 1 min
[4] Transport patients with cardiovascular instability or core temperature < 28°C to a hospital with ECLS (extracorporeal life support)
[5] Withold CPR if risk to the rescue team is unacceptably high
[6] Crush injuries and depolarizing neuromuscular blocking agents may elevate serum potassium

Hyperthermia

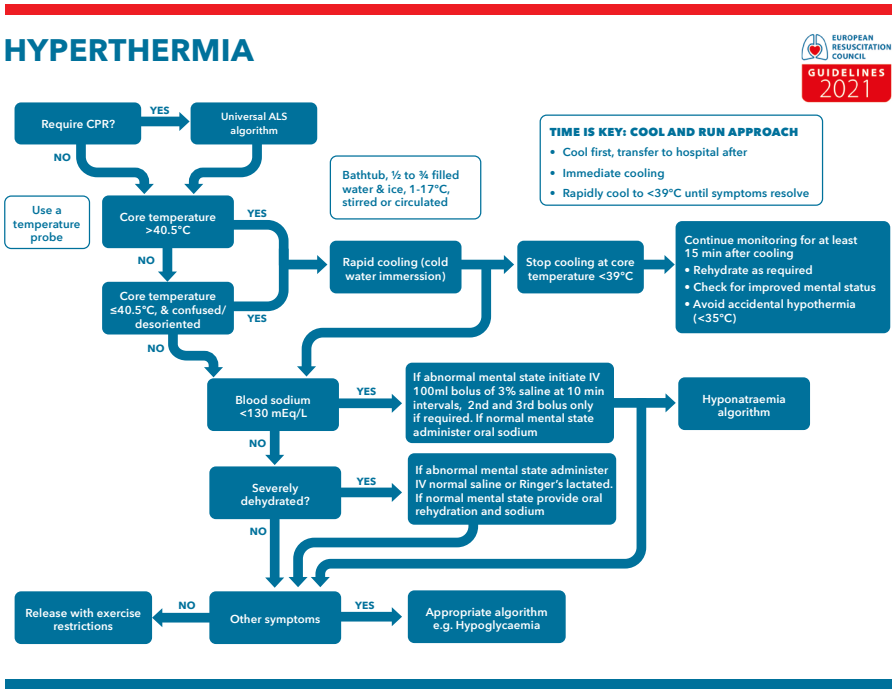
Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds values normally maintained by homeostatic mechanisms. Hyperthermia may be primarily induced by environmental conditions, or secondary due to endogenous heat production.

Modifications to CPR:

Follow a systematic approach for hyperthermia treatment as outlined in the hyperthermia algorithm *(Figure 6.7.)*.

- Measurement of core temperature should be available to guide treatment.
- Simple external cooling measures are usually not required but may involve conductive, convective and evaporative measures.
- **Heat syncope:** remove patient to a cool environment, cool passively and provide oral isotonic or hypertonic fluids.
- **Heat exhaustion:** remove patient to a cool environment, lie them flat, administer IV isotonic or hypertonic fluids, consider additional electrolyte replacement therapy with isotonic fluids. Replacement of 1-2 L crystalloids at 500 mL/h is often adequate.
- **Heat stroke:** a 'cool and run' approach is recommended:
  - Remove patient to a cool environment.
  - Lie them flat.
  - actively cool immediately using whole body (from neck down) water immersion technique (1-26°C) until core temperature <39°C.
  - Where water immersion is not available immediately use any active or passive technique that provides the most rapid rate of cooling.
  - Administer IV isotonic or hypertonic fluids (with blood sodium ≤ 130 mmol/L up to 3x 100 mL NaCl 3%).
  - Consider additional electrolyte replacement with isotonic fluids. Substantial amounts of fluids may be required.
  - In exertional heat stroke a cooling rate faster than 0.10°C/min is safe and desirable.
  - Follow the ABCDE approach in any patient with deteriorating vital signs.

Figure 6.7 Hyperthermia algorithm



Thrombosis

Pulmonary embolism

Cardiac arrest prevention:

- Treat life-threatening hypoxia with high-flow oxygen.
- Consider pulmonary embolism (PE) in all patients with sudden onset of progressive dyspnoea and absence of known pulmonary disease (always exclude pneumothorax and anaphylaxis).
- Obtain 12-lead ECG (exclude acute coronary syndrome, look for right ventricle strain).
- Identify haemodynamic instability and high-risk PE.
- Perform bedside echocardiography.
- Initiate anticoagulation therapy (heparin 80 IU/kg IV) during diagnostic process, unless signs of bleeding or absolute contraindications.

- Confirm diagnosis with computed tomographic pulmonary angiography (CTPA).
- Set-up a multidisciplinary team for making decisions on management of high-risk PE (depending on local resources).
- Give rescue thrombolytic therapy in rapidly deteriorating patients.
- Consider surgical embolectomy or catheter-directed treatment as alternative to rescue thrombolytic therapy in rapidly deteriorating patients.
- Request information about past medical history, predisposing factors, and medication that may support diagnosis of pulmonary embolism:
  - Previous pulmonary embolism or deep venous thrombosis (DVT).
  - Surgery or immobilisation within the past four weeks.
  - Active cancer.
  - Clinical signs of DVT.
  - Oral contraceptive use or hormone replacement therapy.
  - Long-distance flights.

Modifications to CPR:

- Cardiac arrest commonly presents as PEA.
- Low $ETCO_2$ readings (below 1.7 kPa/13 mmHg) while performing high-quality chest compressions may support a diagnosis of pulmonary embolism, although it is a non-specific sign.
- Consider emergency echocardiography performed by a qualified sonographer as an additional diagnostic tool.
- Administer thrombolytic drugs for cardiac arrest when PE is the suspected cause of cardiac arrest.
- When thrombolytic drugs have been administered, consider continuing CPR attempts for at least 60-90 minutes before termination of resuscitation attempts.
- Use thrombolytic drugs or surgical embolectomy or percutaneous mechanical thrombectomy for cardiac arrest when PE is the known cause of cardiac arrest.
- Consider ECPR as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it can be implemented.

Coronary thrombosis

Prevent and be prepared:

- Encourage cardiovascular prevention to reduce the risk of acute events.
- Endorse health education to reduce delay to first medical contact.
- Promote layperson basic life support to increase the chances of bystander CPR.

- Ensure adequate resources for better management.
- Improve quality management systems and indicators for better quality monitoring.

Detect parameters suggesting coronary thrombosis and activate the ST-elevation myocardial infarction (STEMI) network:

- Chest pain prior to arrest.
- Known coronary artery disease.
- Initial rhythm: VF, pulseless ventricular tachycardia (pVT).
- Post-resuscitation 12-lead ECG showing ST-elevation.

Resuscitate and treat possible causes (establish reperfusion strategy):

- Patients with sustained ROSC
  - STEMI patients:
    - Primary percutaneous coronary intervention (PCI) strategy ≤120 min from diagnosis: activate catheterisation laboratory and transfer patient for immediate PCI.
    - Primary PCI not possible in ≤120 min: perform pre-hospital thrombolysis and transfer patient to PCI centre *(Figure 6.8)*.
  - Non STEMI patients: individualise decisions considering patient characteristics, OHCA setting and ECG findings.
    - Consider quick diagnostic work-up (discard non-coronary causes and check patient condition).
    - Perform urgent coronary angiography (≤120 min) if ongoing myocardial ischaemia is suspected or the patient is hemodynamically/ electrically instable.
    - Consider delayed coronary angiography if there is no suspected ongoing ischaemia and the patient is stable.
- Patients with no sustained ROSC: Assess setting and patient conditions and available resources
  - Futile: Stop CPR.
  - Not-futile: Consider patient transfer to a percutaneous coronary intervention (PCI) centre with on-going CPR *(Figure 6.8)*.
    - Consider mechanical compression and ECPR.
    - Consider coronary angiography.

Cardiac tamponade

Cardiac tamponade is a frequent cause of cardiac arrest in penetrating chest trauma and immediate resuscitative thoracotomy (RT) via a clamshell or left anterolateral incision, is indicated to restore circulation. The chance of survival is about 4 times higher in cardiac stab wounds than in gunshot wounds.

Modifications to CPR:

- Decompress the pericardium immediately.
- Point of care echocardiography supports the diagnosis.
- Perform resuscitative thoracotomy or ultrasound guided pericardiocentesis.

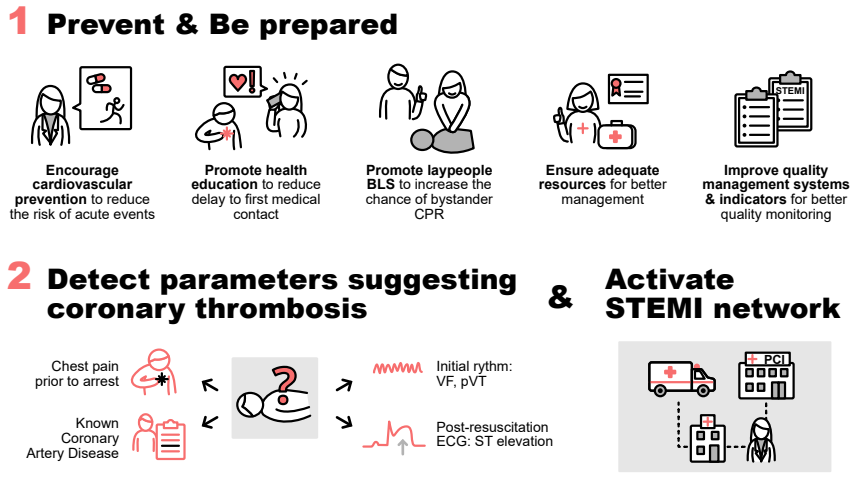
The prerequisites for a successful RT can be summarized as "four E rule" (4E):

- Expertise: teams that perform RT must be led by a highly trained and competent healthcare practitioner. These teams must operate under a robust governance framework.
- Equipment: adequate equipment to carry out RT and to deal with the intrathoracic findings is mandatory.
- Environment: ideally RT should be carried out in an operating theatre. RT should not be carried out if there is inadequate physical access to the patient, or if the receiving hospital is not easy to reach.
- Elapsed time: the time from loss of vital signs to commencing a RT should not be longer than 15 minutes.

If any of the four criteria is not met, RT is futile and exposes the team to unnecessary risks. RT is also a viable therapeutic option in the prehospital environment.

Figure 6.8 Management of out-of-hospital cardiac arrest in the setting of suspected coronary thrombosis. *Note that prolonged or traumatic resuscitation is a relative contraindication for fibrinolysis. **Individualised decision based on careful evaluation of the benefit/ futility ratio, available resources and team expertise.

Abbreviations: OHCA, out-of-hospital cardiac arrest; STEMI, ST-elevation myocardial infarction; ROSC, return of spontaneous circulation; PCI, percutaneous coronary intervention; CPR: cardiopulmonary resuscitation.



Tension pneumothorax

Modifications to CPR:

- Diagnosis of tension pneumothorax in a patient with cardiac arrest or haemodynamic instability must be based on clinical examination or point of care ultrasound (POCUS).
- Decompress chest immediately by open thoracostomy when a tension pneumothorax is suspected in the presence of cardiac arrest or severe hypotension.
- Needle chest decompression serves as rapid treatment, it should be carried out with specific needles (longer, non-kinking).
- Any attempt at needle decompression under CPR should be followed by an open thoracostomy or a chest tube if the expertise is available.

Chest decompression effectively treats tension pneumothorax and takes priority over other measures.

Toxic agents

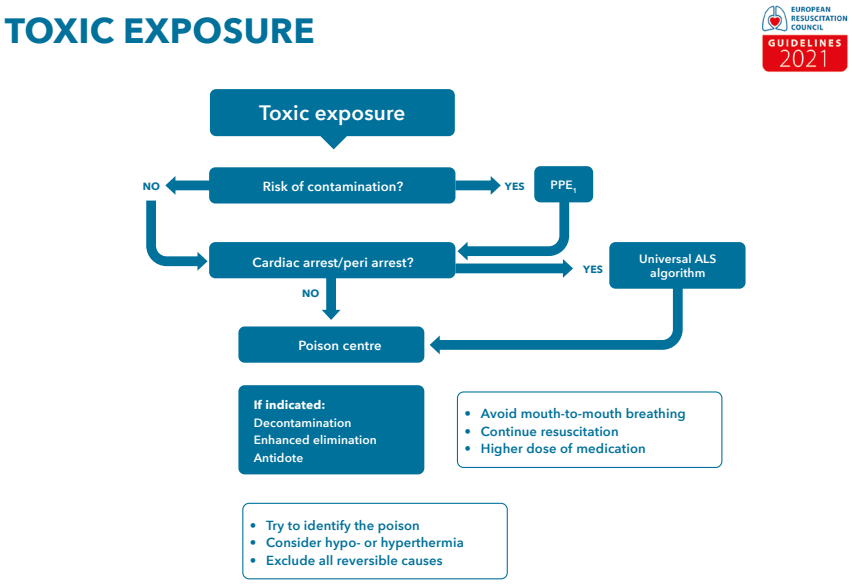
Overall, poisoning rarely causes cardiac arrest or death.

Modifications to CPR *(figure 6.9.)*:

- Have a low threshold to ensure your personal safety.
- Consider using specific treatment measures as antidotes, decontamination and enhanced elimination.
- Do not use mouth-to-mouth ventilation in the presence of chemicals such as cyanide, hydrogen sulphide, corrosives and organophosphates.
- Exclude all reversible causes of cardiac arrest, including electrolyte abnormalities, which can be indirectly caused by a toxic agent.
- Measure the patient's temperature because hypo- or hyperthermia may occur during drug overdose.
- Be prepared to continue resuscitation for a prolonged period of time. The toxin concentration may fall as it is metabolized or excreted during extended resuscitation measures.
- Consult regional or national poison centres for information on treatment of the poisoned patient.
- Consider ECPR as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it can be implemented.

Regional or national poison centres for information on treatment of the poisoned patient and on-line databases for information on toxicology and hazardous chemicals are available for consultation. The International program on Chemical Safety (IPCS) lists poison centres on its website: https://www.who.int/gho/phe/chemical_safety/poisons_centres/en/

Figure 6.9. Management of cardiac arrest caused by toxic agents



SPECIAL SETTINGS

Cardiac arrest in the operating room

Cardiac arrest in the operating room is a rare but a potentially life-limiting event with a mortality rate of more than 50%. Strong predictors of Intraoperative Cardiac Arrest are associated with:

- Higher American Society of Anesthesiologists physical status.
- Current sepsis.
- Urgent/ emergency case.
- Anaesthetic technique.
- Age.

In addition, there are also several factors such as hypoxia, acute blood loss with shock, pulmonary embolism, myocardial infarction, arrhythmia or electrolyte disturbances, which all can be the cause or confounding factors in an intraoperative cardiac arrest.

Modifications to CPR:

- Recognise cardiac arrest by continuous monitoring.
- Inform the surgeon and the theatre team. Call for help and the defibrillator.
- Initiate high-quality chest compressions and effective ventilation.
- Follow the ALS algorithm with a strong focus on reversible causes, especially hypovolaemia (anaphylaxis, bleeding), hypoxia, tension-pneumothorax, thrombosis (pulmonary embolism).
- Use ultrasound to guide resuscitation.
- Adjust the height of the OR table to enable high-quality CPR.
- Check the airway and review the $ETCO_2$ tracing.
- Administer oxygen with a FiO_2 1.0.
- Open cardiac compression should be considered as an effective alternative to closed chest compression.
- Consider ECPR as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing.

Cardiac arrest during cardiac surgery

The incidence of cardiac arrest following cardiac surgery has been reported around 2-5%, with higher survival rates (around 50%) compared to other scenarios. Major causes of cardiac arrest in this setting include ventricular fibrillation (VF), accounting for up to 50% of cases, followed by cardiac tamponade and major bleeding, which often present as PEA.

Modifications to CPR:

Follow a systematic approach for cardiac arrest treatment as outlined in the cardiac arrest during cardiac surgery algorithm *(Figure 6.10.)*.

Prevent and be prepared:

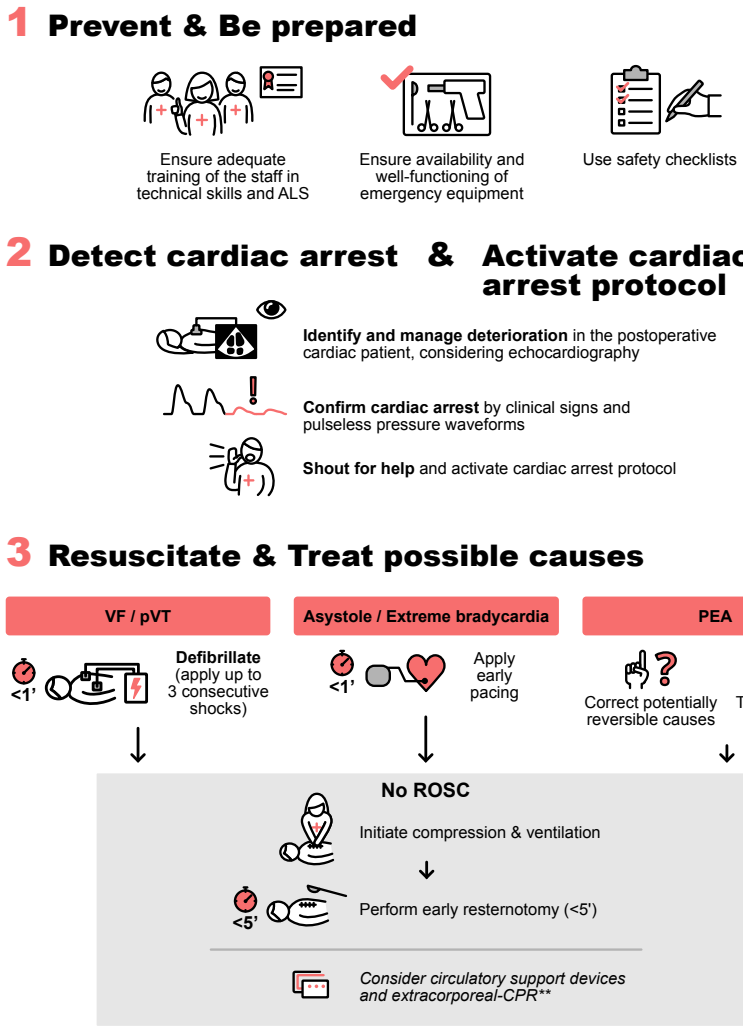
- Ensure adequate training of the staff in resuscitation technical skills and ALS.
- Ensure equipment for emergency re-sternotomy is available in the ICU.
- Use safety checklists.
- Detect cardiac arrest and activate cardiac arrest protocol.
- Identify and manage deterioration in the postoperative cardiac patient.
- Consider echocardiography.
- Confirm cardiac arrest by clinical signs and pulseless pressure waveforms.
- Shout for help and activate cardiac arrest protocol.

Resuscitate and treat possible causes:

- Resuscitate according to ALS MODIFIED algorithm:
  - VF/ pVT: Defibrillate. Apply up to 3 consecutive shocks (< 1 min).
  - Asystole/ extreme bradycardia: Apply early pacing (< 1 min).
  - PEA: Correct potentially reversible causes. If paced rhythm, turn off pacing to exclude VF.
  - No ROSC:
    - Initiate chest compression and ventilation.
    - Perform early resternotomy (< 5 min).
    - Consider circulatory support devices and ECPR.

Figure 6.10 Cardiac arrest during cardiac surgery algorithm

Advanced Life Support (ALS) algorithm for postoperative cardiac arrest following cardiac surgery. ALS, advanced life support, VF, ventricular fibrillation; PVT: pulseless ventricular tachycardia; CPR, cardiopulmonary resuscitation; ROSC, return of spontaneous circulation; PEA: pulseless electrical activity. ** Consider IABP to support CPR or extracorporeal life support as an alternative if resternotomy is not feasible or fails to revert cardiac arrest.



Cardiac arrest in the catheterisation laboratory

The type of patients treated and procedures performed in the catheterisation laboratory has evolved over the last years towards greater complexity. More critically ill patients now undergo percutaneous coronary intervention (PCI) or implant of ventricular assist devices, and the volume of structural heart interventions, mostly offered to high-risk patients who are unfit for surgery, is rapidly increasing (i.e. percutaneous valve replacement or repair, leaks, septal defects or left atrial appendage closure). As a result, cardiac arrest in the catheterisation laboratory may occur in critically ill patients (i.e. cardiogenic shock due to extensive myocardial infarction), but also in stable patients undergoing planned procedures, which carry inherent potential hazards and are extremely sensitive to both technical and human factors.

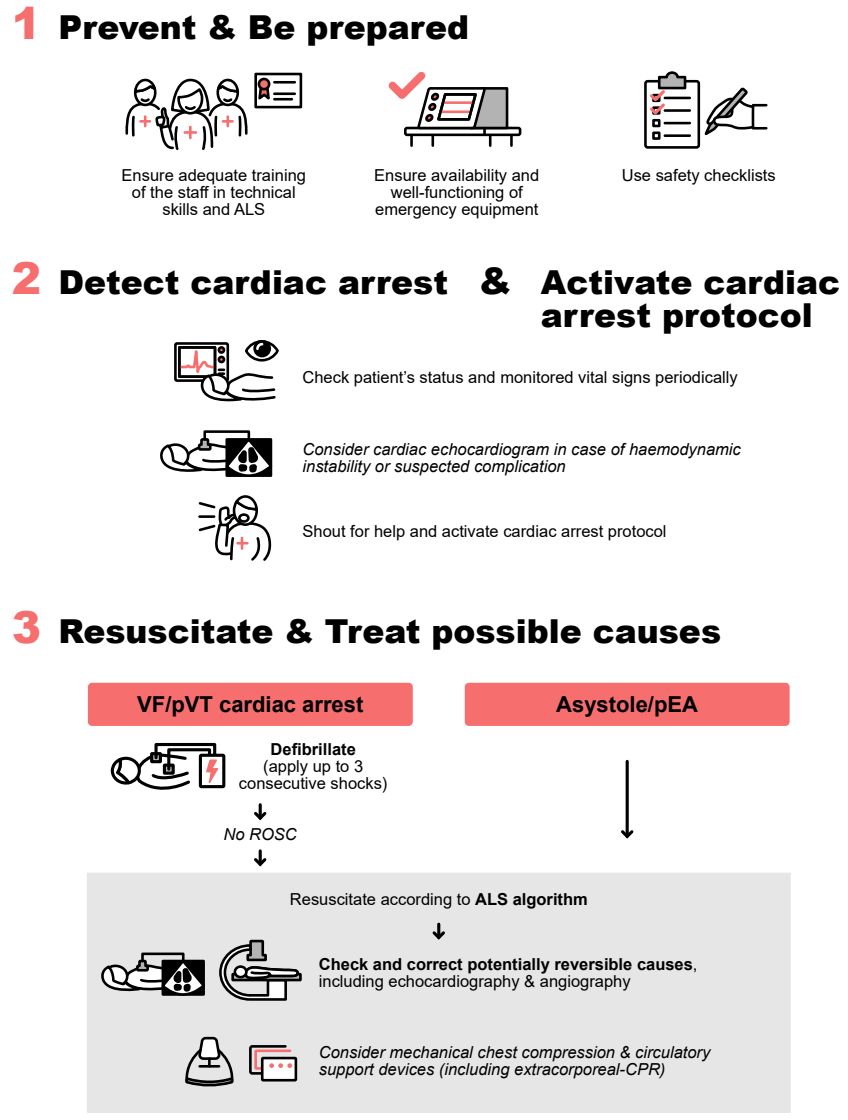
Modifications to CPR:

Follow a systematic approach for cardiac arrest treatment as outlined in the cardiac arrest in the catheterisation laboratory algorithm *(figure 6.11.)*.

Prevent and be prepared:

- Ensure adequate training of the staff in resuscitation technical skills and ALS.
- Use safety checklists.
- Detect cardiac arrest and activate cardiac arrest protocol.
- Check patient's status and monitored vital signs periodically.
- Consider cardiac echocardiography in case of haemodynamic instability or suspected complication.
- Shout for help and activate cardiac arrest protocol.
- Resuscitate and treat possible causes.
- Resuscitate according to ALS MODIFIED algorithm:
  - VF/ pVT: Defibrillate. Apply up to 3 consecutive shocks (< 1 min). If no ROSC resuscitate according to ALS algorithm *(Figure 4.1 - page 34)*.
  - Asystole/ PEA: Resuscitate according to ALS algorithm *(Figure 4.1 - page 34)*.
  - Check and correct potentially reversible causes, including the use of echocardiography and angiography.
  - Consider mechanical chest compression and circulatory support devices (including ECPR).

Figure 6.11 Management of cardiac arrest in the catheterisation laboratory. ALS, advanced life support, VF, ventricular fibrillation; PVT: pulseless ventricular tachycardia; CPR, cardiopulmonary resuscitation; ROSC, return of spontaneous circulation; PEA: pulseless electrical activity.



Cardiac arrest in the dialysis unit

Patients receiving long-term HD are one of the highest risk groups for out-of-hospital cardiac arrest (OHCA), which includes events occurring within dialysis clinics. OHCA occurs 20 times more frequently in dialysis patients compared with the general population. Cardiac arrests occurring within a dialysis clinic are predominantly witnessed events and may occur before, during or after dialysis treatment. Several risk factors for cardiac arrest in patients receiving long-term HD have been postulated:

- Hyperkalaemia
- Excessive volume shifts during dialysis
- 2-day inter-dialytic interval
- Low potassium dialysate fluid
- Cardiac disease
- Non-compliance with diet and dialysis regimen

Although HD patients are susceptible to cardiac arrest in the first 12 hours from the start of the HD session, the highest risk period is the latter end of the 2-day inter-dialytic interval (e.g. weekend break) as potassium level rises and fluid accumulates.

Modifications to CPR:

- Follow the universal ALS algorithm.
- Assign a trained dialysis nurse to operate the haemodialysis (HD) machine.
- Stop dialysis and return the patient's blood volume with a fluid bolus.
- Disconnect from the dialysis machine (unless defibrillation-proof) in accordance with the International Electrotechnical Committee (IEC) standards.
- Leave dialysis access open to use for drug administration.
- Dialysis may be required in the early post resuscitation period.
- Provide prompt management of hyperkalaemia.
- Avoid excessive potassium and volume shifts during dialysis.

Cardiac arrest in dentistry

Medical emergencies in a dental office include a variety of situations ranging from psychosomatic disorders precipitated by fear and anxiety to life-threatening situations. Life-threatening emergencies commonly arise from myocardial infarction, seizures or exacerbation of asthma. Cardiac arrest in primary dental practice is a rare event with an incidence of 0.002–0.011 cases per dentist per year.

Modifications to CPR:

- All dental care professionals should undergo annual practical training in the recognition and management of medical emergencies, including the delivery of CPR, incl. basic airway management and the use of an AED.
- Check patient's mouth and remove all solid materials from the oral cavity (e.g., retractor, suction tube, tampons). Prevention of foreign body airway obstruction should precede positioning.
- Recline the dental chair into a fully horizontal position. If reduced venous return or vasodilation has caused loss of consciousness (e.g., vasovagal syncope, orthostatic hypotension), cardiac output can be restored.
- Place a stool under the backrest for stabilisation.
- Start chest compressions immediately while patient lying flat on the chair.
- Consider the over-the-head technique of CPR if access to either side of chest is limited.
- Basic equipment for a standard CPR including a bag-valve-mask device should be available immediately.

Inflight cardiac arrest

Although air travel is safe in general, passenger demographics, pre-existing medical conditions as well as the number of passengers aboard larger aircraft and flights over very long distances raise the probability of in-flight emergencies per flight. Between 1 out of 14,000 and 50,000 passengers will experience acute medical problems/emergencies during a flight with cardiac arrest accounting for 0.3% of all in-flight medical emergencies.

Modifications to CPR:

- Medical professional help should be sought (in-flight announcement).
- The rescuer should kneel in the leg-space in front of the aisle seats to perform chest compressions if the patient cannot be transferred within a few seconds to an area with adequate floor space (galley).
- Over-the-head-CPR is a possible option in limited space environments.
- Airway management should be based on the equipment available and the expertise of the rescuer.

- If the flight plan is over open-water with high possibility of ROSC during an ongoing resuscitation consider an early diversion.
- Consider risks of diversion if ROSC is unlikely and give appropriate recommendations to the flight crew.
- If CPR is terminated (no ROSC) a flight diversion should not usually be performed.

Helicopter emergency medical services (HEMS) and air ambulances

Air ambulance services operate either a helicopter or a fixed wing aircraft that routinely transport critically ill or injured patients directly to specialty centers. They also perform secondary transfers between hospitals. Cardiac arrest may occur during flight, both in patients being transported from an accident site (primary missions) and also in critically ill patients being transported between hospitals (secondary missions).

Modifications to CPR:

- Proper pre-flight-evaluation of the patient, early recognition and communication within the team, early defibrillation, high-quality CPR with minimal interruption of chest compressions, and treatment of reversible causes before flight are the most important interventions for the prevention of CPR during HEMS missions.
- Check the patient status properly before flight. Sometimes ground-based transport might be a suitable alternative, especially for patients with high-risk of cardiac arrest.
- Check security of the airway and ventilator connections prior to flight. For a cardiac arrest in an unventilated patient during flight consider a SGA for initial airway management.
- Pulse oximetry (SpO_2) monitoring and oxygen supplementation should be available immediately if not already attached.
- CPR should be performed as soon as possible, OTH-CPR might be possible depending on the type of helicopter.
- If cabin size does not allow high-quality CPR, consider immediate landing.
- Always consider attaching a mechanical CPR device before flight.
- Consider three stacked shocks in case of shockable rhythm during flight.
- Defibrillation during flight is safe.

Cardiac arrest in sport

The incidence of sudden cardiac death (SCD) associated with sport or exercise in the general population is 0.46 per 100,000 person-years. The incidence is markedly higher in those susceptible to cardiac arrythmias during or shortly after participating in sport.

Modifications to CPR:

Prevention:

- Do not undertake exercise, especially extreme exercise or competitive sport, if feeling unwell.
- Follow medical advice in relation to the levels of exercise or sport competition.
- Consider cardiac screening for young athletes undertaking high level competitive sport.

Cardiac arrest management:

- Recognise collapse.
- Gain immediate and safe access to the Field of Play.
- Call for help and activate EMS.
- Assess for signs of life.
- If no signs of life:
  - Commence CPR.
  - Access an AED and defibrillate if indicated.
- If ROSC occurs, carefully observe and monitor the casualty until advanced medical care arrives.
- If there is no ROSC:
  - Continue cardio-pulmonary resuscitation and defibrillation until advanced medical care arrives.
  - In a sport arena, consider moving patient to a less exposed position and continue resuscitation. This should be accomplished with minimal interruption to chest compressions.

Drowning

Care of a submersion victim in high-resource countries often involves a multiagency approach, with several different organizations being independently responsible for different phases of the patient's care, beginning with the initial aquatic rescue, through on-scene resuscitation and transfer to hospital and in-hospital and rehabilitative care. Attempting to rescue a submerged patient has substantial resource implications and may place rescuers at risk themselves. The major sequalae of drowning is hypoxia caused by respiratory impairment secondary to the aspiration of fluid into the lungs. If severe or prolonged, this can cause cardiac arrest.

Modifications to CPR:

Initial rescue:

- Undertake a dynamic risk assessment considering feasibility, chances of survival and risks to the rescuer:
  - Submersion duration is the strongest predictor of outcome.
  - Salinity has an inconsistent effect on outcome.
- Assess consciousness and breathing:
  - If conscious and/ or breathing normally, aim to prevent cardiac arrest.
  - If unconscious and not breathing normally, start resuscitation.

Cardiac arrest treatment:

- Start resuscitation as soon as safe and practical to do so. If trained and able this might include initiating ventilations whilst still in the water or providing ventilations and chest compressions on a boat.
- Start resuscitation by giving 5 rescue breaths/ ventilations using 100% inspired oxygen if available.
- If the person remains unconscious, without normal breathing, start chest compressions.
- Alternate 30 chest compressions to 2 ventilations.
- Apply an AED if available and follow instructions.
- Intubate the trachea if able to do so safely.
- Consider ECPR in accordance with local protocols if initial resuscitation efforts are unsuccessful.

SPECIAL PATIENTS

Asthma / COPD

Evidence based recommendations for the management of acute life threatening asthma are provided by the British Thoracic Society, Scottish Intercollegiate Guidelines Network (https://www.sign.ac.uk/our-guidelines/british-guideline-on-the-management-of-asthma/).

Modifications to CPR:

Cardiac arrest prevention:

- Ensure a patent airway.
- Treat life threatening hypoxia with high flow oxygen.
- Titrate subsequent oxygen therapy with pulse oximetry (SpO_2 94-98% for asthma; 88-92% for chronic obstructive pulmonary disease (COPD)).
- Assess respiratory rate, accessory muscle use, ability to speak in full sentences, pulse oximetry, percussion and breath sounds; request chest x-ray.
- Look for evidence of pneumothorax / tension pneumothorax.
- Provide nebulised bronchodilators (oxygen driven for asthma, consider air driven for COPD).
- Administer steroids (Prednisolone 40-50mg or hydrocortisone 100mg IV).
- Consider IV magnesium sulphate for asthma.
- Obtain vascular access.
- Consider IV fluids.

Cardiac arrest treatment:

- Administer high concentration oxygen.
- Ventilate with respiratory rate (8-10 min^{-1}) and sufficient tidal volume to cause the chest to rise.
- Intubate the trachea if able to do so safely.
- Check for signs of tension pneumothorax and treat accordingly.
- Disconnect from positive pressure ventilation if relevant and apply pressure manually to reduce hyper-inflation.
- Consider IV fluids.
- Consider E-CPR in accordance with local protocols if initial resuscitation efforts are unsuccessful.

Obesity

Delivery of effective CPR in obese patients may be challenging due to a number of factors:

- patient access and transportation
- vascular access
- airway management
- quality of chest compressions
- efficacy of vasoactive drugs
- efficacy of defibrillation

Modifications to CPR:

- Provide chest compressions up to a maximum depth of 6 cm.
- Obese patients lying in a bed do not necessarily need to be moved down onto the floor.
- Change the rescuers performing chest compression more frequently.
- Consider escalating defibrillation energy to maximum for repeated shocks.
- Manual ventilation with a bag-mask should be minimised and be performed by experienced staff using a two-person technique.
- An experienced provider should intubate the trachea early so that the period of bagmask ventilation is minimised.

Pregnancy

Maternal mortality remains high with an estimated 295,000 deaths in 2017, the majority (94%) occurring in low and lower middle-income countries. A maternal cardiac arrest is a cardiac arrest that occurs at any stage in pregnancy and up to 6 weeks after birth.

Modifications to CPR:

- Call for expert help early (including an obstetrician and neonatologist).
- Start basic life support according to standard guidelines.
- Use the standard hand position for chest compressions on the lower half of the sternum if feasible.
- If over 20 weeks pregnant or the uterus is palpable above the level of the umbilicus:
  - Manually displace the uterus to the left to remove aortocaval compression.
  - If feasible, add left lateral tilt – the chest should remain on supported on a firm surface (e.g. in the operating room). The optimal angle of tilt is unknown. Aim for a tilt between 15 and 30 degrees. Even a small amount of tilt may be

better than no tilt. The angle of tilt used needs to enable high-quality chest compressions and if needed allow caesarean delivery of the fetus.

- Prepare for early emergency hysterostomy – the fetus will need to be delivered if immediate (within 4 minutes) resuscitation efforts fail.
- If over 20 weeks pregnant or the uterus is palpable above the level of the umbilicus and immediate (within 4 min) resuscitation is unsuccessful, deliver the fetus by emergency caesarean section aiming for delivery within 5 min of collapse.
- Place defibrillator pads in the standard position as far as possible and use standard shock energies.
- Consider early tracheal intubation by a skilled operator.
- Identify and treat reversible causes (e.g. haemorrhage). Focused ultrasound by a skilled operator may help identify and treat reversible causes of cardiac arrest.
- Consider extracorporeal CPR (ECPR) as a rescue therapy if ALS measures are failing.

Preparation for cardiac arrest in pregnancy:

- Healthcare settings dealing with cardiac arrest in pregnancy should:
  - Have plans and equipment in place for resuscitation of both the pregnant woman and the newborn.
  - Ensure early involvement of obstetric, anaesthetic, critical care and neonatal teams.
  - Ensure regular training in obstetric emergencies.

KEY LEARNING POINTS

- **Use the ABCDE approach for early recognition and treatment to prevent cardiac arrest.**
- **High-quality CPR and treatment of reversible causes is the mainstay of treatment of cardiac arrest from any cause.**
- **Call for expert help early when specialist procedures are needed.**

CHAPTER 7
POST-RESUSCITATION CARE

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**

INTRODUCTION

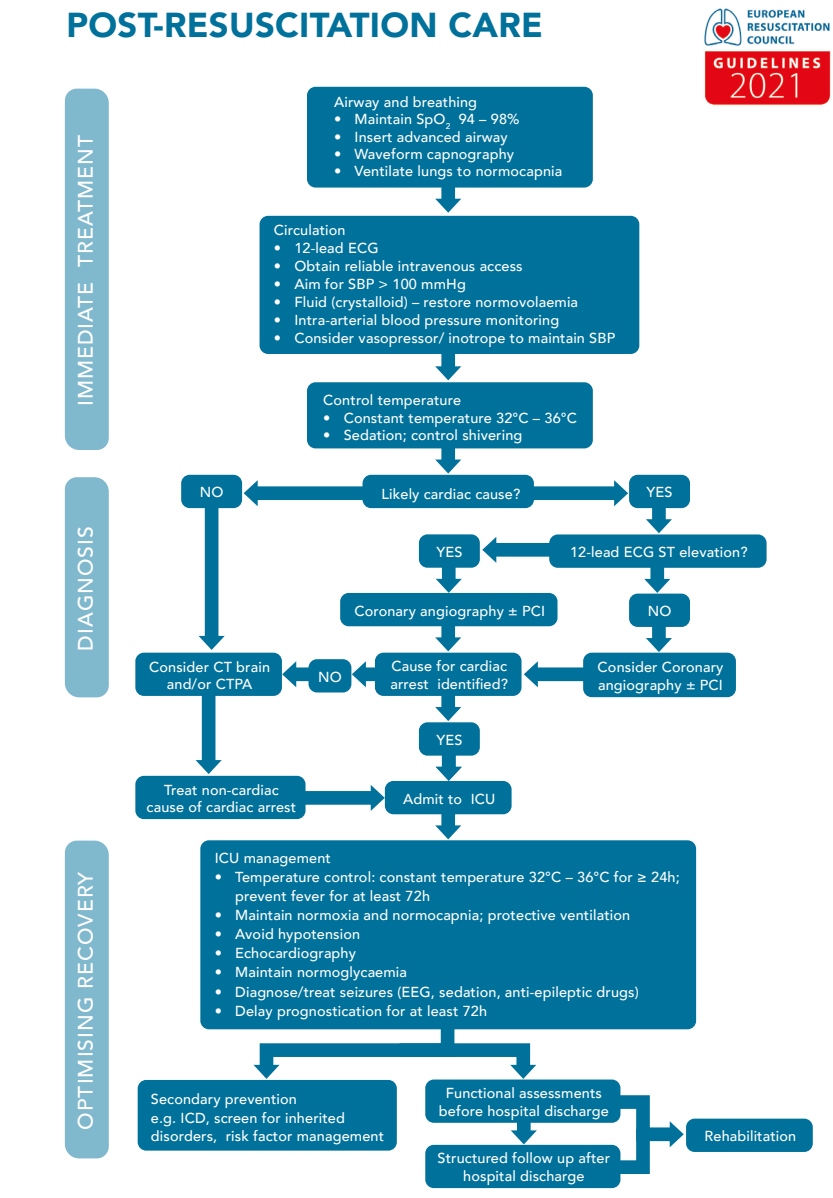
Successful return of spontaneous circulation (ROSC) is the first step toward the goal of complete recovery from cardiac arrest. The post-cardiac arrest syndrome is the complex of pathophysiological processes that occur following whole-body ischaemia during cardiac arrest. Depending on the cause of arrest and the severity of the post-cardiac arrest syndrome, many patients require multiple organ support during the post-resuscitation phase. This phase starts at the location where ROSC is achieved. The post-resuscitation care algorithm outlines some of the key interventions required to optimise outcome *(Figure 7.1)*.

THE POST-CARDIAC ARREST SYNDROME

The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischemia/reperfusion response, and the persistent precipitating pathology. It may not occur at all if the cardiac arrest is brief and varies with the duration and cause of cardiac arrest.

Post-cardiac arrest brain injury manifests as coma, seizures, myoclonus, varying degrees of neurocognitive dysfunction and brain death. Significant myocardial dysfunction is common after cardiac arrest but typically starts to recover within 2-3 days. The whole-body ischemia/reperfusion activates immunological and coagulation pathways, contributing to multiple organ failure. Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion and vasodilation.

Figure 7.1 Post-resuscitation care algorithm



CONTINUED RESUSCITATION

In the immediate post-resuscitation phase, pending transfer to an appropriate area of high-level care (or cardiac arrest centre), treat the patient by following the ABCDE approach.

Airway and breathing

Control of oxygenation

Patients who have had a brief cardiac arrest may achieve an immediate return of normal cerebral function. These patients do not require tracheal intubation and ventilation but should be given oxygen via facemask if their arterial oxygen saturation is less than 94%. Hypoxaemia and hypercarbia increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. As soon as arterial blood oxygen saturation can be monitored reliably, titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94-98%. Avoid hyperoxaemia, which is also harmful.

Control of ventilation

Consider tracheal intubation, sedation and controlled ventilation in any patient with compromised cerebral function. Ensure the tracheal tube is positioned correctly, well above the carina. After cardiac arrest, hypocapnia induced by hyperventilation causes cerebral ischemia. Adjust ventilation to achieve normocapnia ($PaCO_2$ 4.5-6.0 kPa or 35-45 mmHg) and monitor this using the end-tidal CO_2 and arterial blood gas values. Apply lung-protective ventilation: tidal volume 6-8 ml kg^{-1} ideal body weight and positive end-expiratory pressure 4-8cm H_2O. Insert a gastric tube to decompress the stomach and perform a chest x-ray to check for complications after resuscitation and confirm the correct placement of tubes and lines.

Circulation

Coronary reperfusion

Acute coronary syndrome (ACS) is a frequent cause of out-of-hospital cardiac arrest (OHCA). Cardiac catheterisation should be performed immediately in the presence of ST-elevation and considered as soon as possible (less than two hours) in other patients in the absence of an apparent non-coronary cause if they are hemodynamically or electrically unstable.

Haemodynamic management

Post-resuscitation myocardial dysfunction causes hemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias. Perform early echocardiography in all patients to detect and quantify the degree of myocardial dysfunction.

Vasoplegia and severe vasodilatation post-cardiac arrest may require noradrenaline and relatively large volumes of fluid. Treatment may be guided by blood pressure, heart rate, urine output, plasma lactate clearance rate, and central venous oxygen saturation. An arterial line for continuous blood pressure monitoring may be needed. Goals should be considered on an individual patient basis and are likely influenced by post-cardiac arrest status and pre-existing comorbidities. If inotropic support is necessary, dobutamine is the most conventional treatment in this setting.

Hypokalaemia may predispose to ventricular arrhythmias. Give potassium to maintain the serum potassium concentration between 4.0 and 4.5 mmol l^{-1}.

Bradycardia can be left untreated as long as there are no signs of hypoperfusion and has even been shown to be associated with a good outcome.

Disability

Although cardiac arrest is frequently caused by primary cardiac disease, other precipitating conditions must be excluded, particularly in hospital patients (e.g. massive blood loss, respiratory failure, pulmonary embolism). Assess the different body systems rapidly so that further resuscitation can be targeted at the patient's needs.

Cerebral perfusion

Often autoregulation of cerebral blood flow is impaired after cardiac arrest, which means that cerebral perfusion varies with the perfusion pressure. Maintain mean arterial pressure near the patient's normal level.

Sedation

Many post-cardiac arrest patients will require appropriate sedation and pain management, particularly those treated with "targeted temperature management" (TTM). Shivering can be common during TTM and is managed with opioids and sedation. TTM influences the metabolism of several drugs. Short-acting drugs will enable reliable and earlier neurologic assessment. Sedation stops are best initiated after TTM and rewarming have been complete.

Control of seizures

Seizures are common after cardiac arrest. They may increase the cerebral metabolic rate and have the potential to exacerbate brain injury caused by cardiac arrest. Clinically observed convulsions (or myoclonus) may or may not be of epileptic origin. Use intermittent electroencephalography (EEG) to detect epileptic activity in patients with clinical seizure manifestations. Consider continuous EEG to monitor patients with a diagnosed status epilepticus and effects of treatment. Prophylactic antiepileptic drugs are not recommended but treat seizures after the first event with levetiracetam or sodium valproate. Increasing the dose of propofol or benzodiazepine may suppress myoclonus and electrographic seizures. Thiopental or phenobarbital may be considered in selected patients.

Glucose control

Maintain the blood glucose at 5-10 mmol l^{-1} (90-180 mg dl^{-1}) and avoid hypoglycaemia. Do not implement strict glucose control in adult patients with ROSC after cardiac arrest because it increases the risk of hypoglycaemia.

Exposure

Targeted Temperature Management

Treat hyperthermia occurring after cardiac arrest with antipyretics and consider active cooling in unconscious patients. Induction of maintenance of TTM can be done with a wide range of external or internal methods.

- Select and maintain a constant target temperature between 32 – 36°C for patients in whom TTM is used for at least 24 hours.
- Use TTM for adults who remain unresponsive after ROSC.
- Avoid fever (T > 37.7 0C, core temperature) for at least 72 hours in patients who remain unresponsive after completion.
- Avoid standard use of neuromuscular blocking drugs in patients undergoing TTM.
- Do not use rapid infusion of large volumes of cold IV fluid for prehospital cooling.

General ICU management

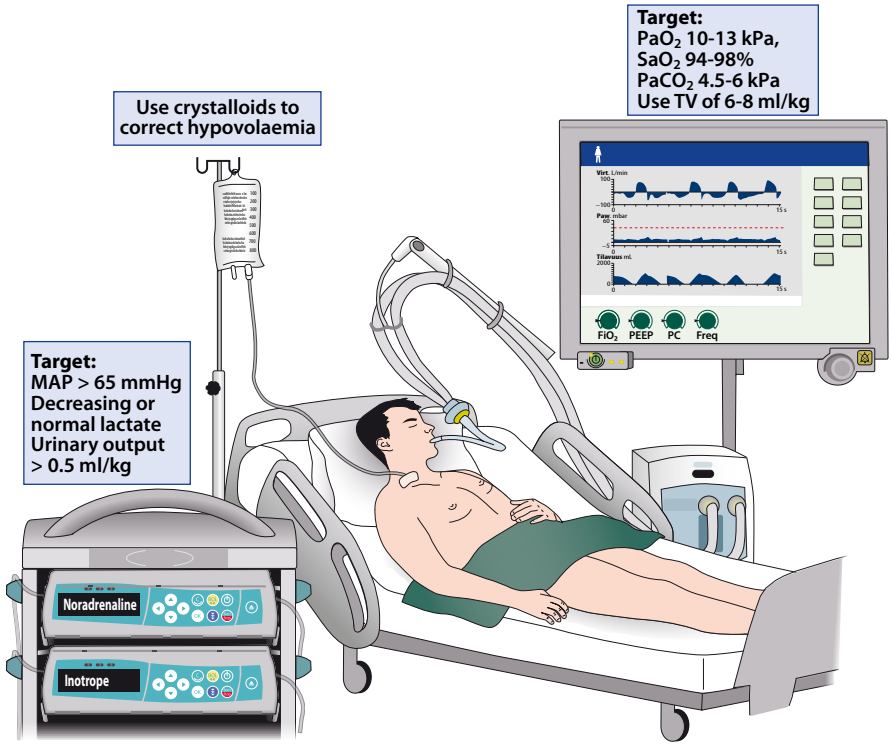
Most aspects of post-cardiac arrest care follow general ICU practices. Some differences and nuances are inherent.

Prophylactic antibiotics are not required following ROSC.

Appropriate sedation and pain management is required, particularly in patients treated with TTM. Standard neuromuscular block is not required but may be used in uncontrollable shivering or severe adult respiratory syndrome (ARDS) following cardiac arrest.

A summary of appropriate treatment targets in the ICU is shown in *figure 7.2*.

Figure 7.2. Possible targets in ICU management



Patient transfer

The decision to transfer a patient from the place where stabilisation has been achieved should be made only after discussion with senior members of the admitting team. Continue all established monitoring during the transfer and secure all cannula, catheters, tubes and drains. Make a complete re-assessment immediately before the patient is transferred using the ABCDE approach. Ensure that portable suction apparatus, an oxygen supply and a defibrillator/monitor accompany the patient and transfer team.

PROGNOSTICATION

About two-thirds of in-hospital deaths in patients admitted to an ICU in a coma after OHCA are caused by hypoxic-ischaemic brain injury. In a minority of cases, these deaths occur due to hypoxic-ischaemic brain injury, resulting in an irreversible loss of all brain function (brain death). However, most of these neurological deaths result from the active withdrawal of life-sustaining treatment (WLST) in patients where the severity of hypoxic-ischaemic brain injury indicates that survival with a poor neurological outcome is very likely. Therefore, accurate prognostication is essential to avoid inappropriate WLST in patients who still have a chance of a neurologically meaningful recovery and avoid futile treatment in patients with a severe and irreversible neurological injury.

Clinical examination

No single predictor is 100% accurate; a multimodal neuroprognostication strategy is recommended.

- Pupillary light reflex or quantitative pupillometry at 72 hours or later after ROSC predicts the neurological outcome of comatose adults after cardiac arrest.
- Bilateral absence of corneal reflex at 72 h or later after ROSC predicts poor outcome.

The presence of myoclonus or status myoclonus within 96 h after ROSC, combined with other tests, predicts poor neurological outcome in adults who are comatose after cardiac arrest.

Neurophysiology

Somatosensory evoked potentials (SSEPs)

When performing SSEP, the median nerve is electrically stimulated and the ascending signals are recorded from the peripheral plexus brachialis, cervical level, subcortical level and the sensory cortex (N20-potential). SSEPs may be depressed by barbiturate coma but are preserved with other sedative drugs such as propofol and midazolam. A bilateral absence of the short-latency N20-potentials over the sensory cortex is a reliable sign of a poor prognosis after cardiac arrest with high specificity and limited sensitivity both early and late after cardiac arrest.

Electroencephalography

Electroencephalography (EEG) is one of the most widely used and studied methods to assess brain function and prognosis after cardiac arrest. EEG is also important for diagnosing and treating seizures. The main aspects when evaluating EEG are the background activity, superimposed discharges and reactivity. The EEG background continuity is most important for the prognosis and is commonly categorised as continuous, discontinuous, burst suppression (>50% suppression periods) or suppression (all activity <10 μV amplitude).

Immediately after a cardiac arrest the EEG is suppressed in many patients, returning to a continuous normal voltage EEG within the first 24 h in most patients who ultimately achieve a good outcome. The time for this restitution is correlated with the outcome. The EEG-background is often discontinuous and of low frequency on its first appearance. Sedative drugs affect background continuity and can induce discontinuous or burst-suppression background in a dose-dependent manner.

In patients who remain comatose after cardiac arrest, the absence of EEG background reactivity, unequivocal seizures on EEG, or burst-suppression are indicators of a poor outcome. It can be used in conjunction with other indices to predict poor outcome.

Biomarkers

Protein biomarkers that are released following the injury to neurons and glial cells may be measured in blood and are likely to correlate with the extent of brain injury and with neurological outcome. Neuron-specific enolase (NSE), combined with other tests, is the only biomarker recommended for assessing brain injury and helping prognostication after cardiac arrest.

Imaging

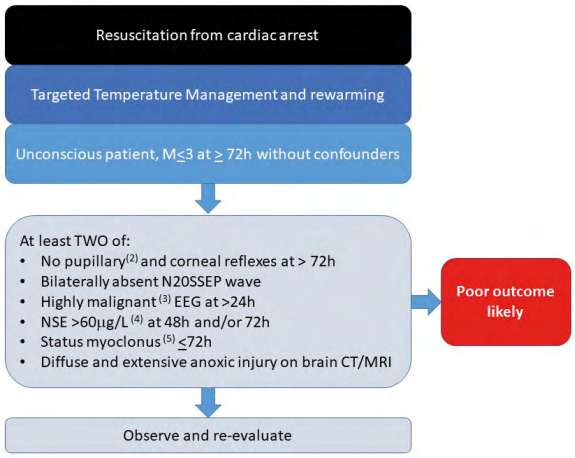
Many imaging modalities have been studied, but none will reliably predict the outcome of comatose cardiac arrest survivors. In experienced centres they can be used in a multimodal neuroprognostication strategy. Generalised brain oedema on brain CT (grey matter/white matter ratio) or extensive diffusion restriction on brain MRI predict a poor neurological outcome.

Suggested prognostication strategy

A careful clinical neurological examination remains the foundation for prognostication of the comatose patient after cardiac arrest. The process of brain recovery following global post-anoxic injury is completed within 72 h from arrest in most patients. Before a decisive assessment is performed, major confounders must be excluded. Suspend sedatives and neuromuscular blocking drugs for long enough to avoid interference and preferably use short-acting drugs.

The prognostication strategy algorithm *(figure 7.3)* applies to all patients who remain comatose with an absent or extensor motor response to pain at ≥ 72 h from ROSC.

Figure 7.3 Multimodal prognostication algorithm



ORGAN DONATION

Organ donation should be considered in those who have achieved ROSC and who fulfil the criteria for death using neurological criteria. Organ donation can also be considered in individuals where CPR is not successful in achieving ROSC. All decisions concerning organ donation must follow local legal and ethical requirements, as these vary in different settings.

KEY LEARNING POINTS

- **After cardiac arrest, return of spontaneous circulation is just the first stage in a continuum of resuscitation.**
- **The quality of post-resuscitation care will influence significantly the patient's final outcome.**
- **These patients require appropriate monitoring, safe transfer to a critical care environment, and continued organ support.**
- **The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and persistence of precipitating pathology.**
- **A multimodal neuroprognostication strategy is needed for patients remaining comatose after cardiac arrest.**

CHAPTER 8
AIRWAY MANAGEMENT AND VENTILATION

SECTION 1
BASIC AIRWAY MANAGEMENT, VENTILATION AND ALTERNATIVE AIRWAY DEVICES

LEARNING OUTCOMES
To understand:

- **the causes and recognition of airway obstruction**
- **techniques for airway management when starting resuscitation**
- **the use of simple adjuncts to maintain airway patency**
- **the use of simple devices for ventilating the lungs**
- **the role of supraglottic airway devices during CPR**

Please consult the VLE chapter 8 on airway management and ventilation for more detailed information in CoSy.

INTRODUCTION

Loss of consciousness causes airway obstruction, sometimes it may be the primary cause of cardiorespiratory arrest. Prompt assessment, with control of airway patency and ventilation of the lungs are essential to prevent secondary hypoxic brain damage. However, the priority is immediate defibrillation of a cardiac arrest victim followed by attention to the airway.

RECOGNITION OF AIRWAY OBSTRUCTION

The 'look, listen and feel' approach is a simple, systematic method of detecting airway obstruction.

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

Inspiratory stridor, expiratory wheeze, gurgling, snoring, crowing or stridor are noisy signs of diminished air entry in partial airway obstruction. Patients with complete

airway obstruction make respiratory efforts causing paradoxical chest and abdominal movement, described as *'see-saw breathing'*.

Patients with tracheostomies or permanent tracheal stomas

In case of blockage of the tracheostomy tube or stoma remove any obvious foreign material. If necessary, remove the tracheostomy tube or, if present, exchange the tracheostomy tube liner. After removal ventilate the patient's lungs by sealing the stoma and by bag face-mask ventilation, or by intubating the trachea orally. Assisted ventilation via the stoma is possible in patients with a permanent tracheal stoma.

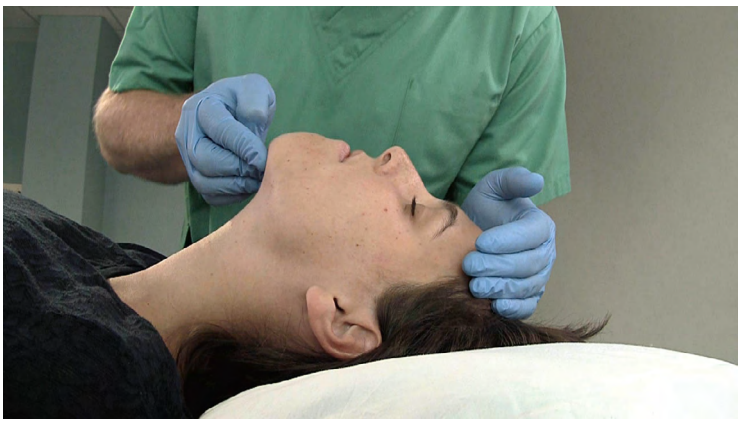
BASIC TECHNIQUES FOR OPENING THE AIRWAY

Three manoeuvres can be used immediately to relieve upper airway obstruction and maintain a clear airway: head tilt and chin lift, or jaw-thrust

Head tilt and chin lift

Place one hand on the patient's forehead and tilt the head back gently; place the fingertips of the other hand under the point of the patient's chin, and gently lift to stretch the anterior neck structures *(figure 8.1)*.

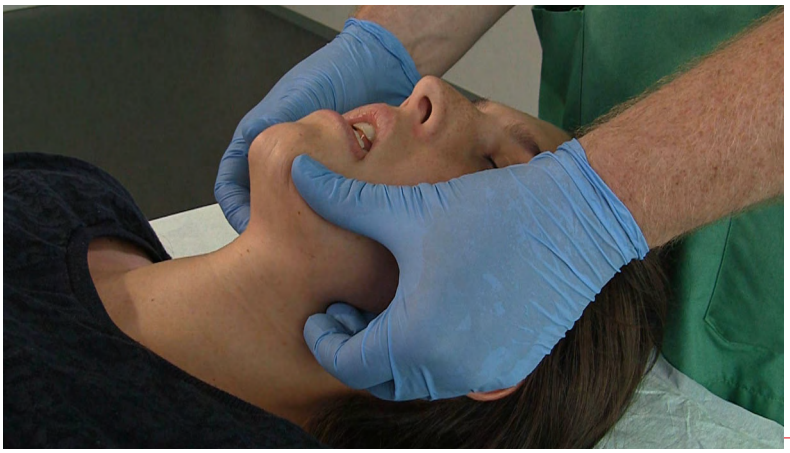
Figure 8.1 Head tilt and chin lift



Jaw-thrust

brings the mandible forward and relieving obstruction by the tongue, soft palate and epiglottis *(figure 8.2)* and is most successful when applied with a head tilt. Place the index and other fingers behind the angle of the mandible and apply steady upwards and forward pressure to lift the mandible. The thumbs slightly open the mouth by downward displacement of the chin.

Figure 8.2 Jaw-thrust



After each manoeuvre, check for success using the look, listen and feel sequence. If a clear airway cannot be achieved, look for other causes of airway obstruction and remove any foreign material visible.

Airway manoeuvres in a patient with suspected cervical spine injury

Maintain the head, neck, chest, and lumbar region in the neutral position during resuscitation. Establish a clear upper airway by using jaw-thrust or chin lift in combination with manual in-line stabilisation (MILS) by an assistant. In life-threatening airway obstruction, add head tilt a small amount at a time until the airway is open. A patent airway takes priority over concerns about a potential cervical spine injury.

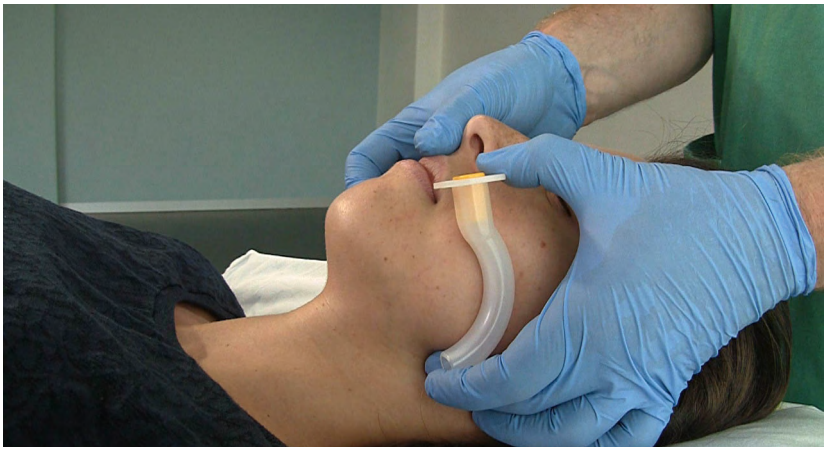
Adjuncts to basic airway techniques

Nasopharyngeal and oropharyngeal airways during CPR are often helpful to maintain an open airway. These airway devices overcome backward displacement of the soft palate and tongue in unconscious patients, but head tilt and jaw-thrust may also be required.

Oropharyngeal airway

are available for newborn to adults. A size estimate is displayed in Fig. 8.3. Attempt insertion only in unconscious patients to avoid vomiting or laryngospasm if laryngeal reflexes are still present.

Figure 8.3 Sizing an oropharyngeal airway



Nasopharyngeal airway

Not deeply unconscious patients tolerate nasopharyngeal airways better than oropharyngeal airways and may be lifesaving when insertion of an oral airway is impossible. In the presence of a known or suspected basal skull fracture an oral airway is preferred.

Oxygen

During CPR and airway management, give the maximal feasible inspired oxygen concentration and connect it to the reservoir system of a self-inflating bag or use a demand valve system. After ROSC, titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94-98 % *(see Chapter 7, post resuscitation care)*.

Ventilation

is started as soon as possible when spontaneous ventilation is inadequate or absent. Mouth-to-mouth ventilation does not require any equipment, but the technique is aesthetically unpleasant, and rescuers may be reluctant to perform it with unknown victims or in situations of possible infection. Simple adjuncts are available to avoid direct person-to-person contact. The pocket resuscitation mask enables mouth-to-mask ventilation.

the detailed technique for mouth-to-mask ventilation is described in the airway chapter at CoSy

Deliver each breath over approximately 1 second, giving a volume that corresponds to normal chest movement; this represents a compromise between giving an adequate volume, minimising the risk of gastric inflation, and allowing adequate time for chest compressions. During CPR with an unprotected airway, give two ventilations after each sequence of 30 chest compressions. During continuous chest compressions a ventilation rate of 10 min^{-1} with an advanced airway is recommended.

Self-inflating bag

can be connected to a facemask, tracheal tube, or supraglottic airway device. An inspired oxygen concentration of approximately 85 % is achieved if a reservoir system is attached and the oxygen flow is maximally increased. Using a demand valve allows application of up to 100% oxygen. The two-person technique for bag-mask ventilation is recommended *(figure 8.4)* as this technique provides sufficient gas-tight seal between the mask and the patient's face and the patient's lungs can be ventilated more effectively and safely. One person holds the facemask in place using a jaw-thrust with both hands and an assistant squeezes the bag.

Figure 8.4 The two-person technique for bag-mask ventilation

Alternative airway devices

Effective bag-mask ventilation requires skill and experience. Tracheal tubes are optimal to manage an airway during cardiac arrest, but without adequate training and experience complications are unacceptably high. Prolonged attempts at tracheal intubation are harmful. The cessation of chest compressions compromises coronary and cerebral perfusion and with each attempt intubation gets more difficult. Compared to bag-mask ventilation, supraglottic airway devices (SGAs) enable ventilation that is more effective and reduce the risk of gastric inflation. SGAs are easier to insert than a tracheal tube.

The best technique depends on the cardiac arrest circumstances and the rescuers' competence.

KEY LEARNING POINTS

- **Airway patency and ventilating the lungs are important components of CPR.**
- **Simple airway manoeuvres, with or without basic adjuncts, often achieve a patent airway.**
- **Give all patients high-concentration oxygen until the arterial oxygen saturation is measurable.**
- **Supraglottic airway devices (SGA) are good alternatives to bag-mask devices.**
- **SGA should be used instead of tracheal intubation unless individuals highly skilled in intubation are immediately available and must be used if attempted intubation is unsuccessful.**

The techniques for insertion of an oropharyngeal or a nasopharyngeal airway, of SGAs, specifically the "classic Laryngeal Mask Airway" (cLMA), the "i-gel airway", and the "Laryngeal tube", as well as the limitations of SGAs are described in details in the airway chapter in CoSy.

SECTION 2
TRACHEAL INTUBATION, CRICOTHYROIDOTOMY, AND BASIC MECHANICAL VENTILATION

LEARNING OUTCOMES
To understand:

- **the advantages and disadvantages of tracheal intubation during cardiopulmonary resuscitation**
- **some methods for confirming correct placement of a tracheal tube**
- **the role of cricothyroidotomy**
- **the role of automatic ventilators in the peri-arrest period**

TRACHEAL INTUBATION

was perceived as the optimal method of providing and maintaining a clear and secure airway during resuscitation. Increasing evidence supports the use of alternative airway management methods, especially the use of SGAs for rescuers not sufficiently competent in tracheal intubation. Only properly trained personnel with a high level of skill and competence should perform tracheal intubation. Rescuers must weigh the risks and benefits of intubation against the need to provide effective chest compressions. The intubation attempt may require some interruption of chest compressions but, once an advanced airway is in place, ventilation will not require interruption of chest compressions. The intubation attempt should interrupt chest compressions for less than 5 seconds. If intubation is not achievable within these constraints, recommence bag-mask ventilation or place a SGA.

Personnel competent in advanced airway management should be able to undertake laryngoscopy without stopping chest compressions; a brief pause in chest compressions will be required only as the tube is passed through the vocal cords. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until ROSC. After intubation, tube placement must be confirmed and the tube secured adequately.

Confirmation of correct tracheal tube placement

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine use of techniques to confirm correct placement of the tracheal tube reduce this risk and are strongly recommended.

Waveform end-tidal CO_2 detectors (capnography) is the gold standard in almost all settings, including out-of-hospital, emergency department, and in-hospital locations where tracheal intubation is performed. It is the most sensitive and specific way to confirm and continuously monitor the position of a tracheal tube and supplements the

clinical assessment (auscultation and visualisation of tube through cords). Waveform capnography cannot discriminate between tracheal and bronchial tube placement - careful auscultation is essential. Furthermore, waveform capnography may be a sensitive indicator proper chest compression and of ROSC (especially useful in PEA).

CRICOTHYROIDOTOMY

Occasionally, ventilation with a bag-mask, or tracheal intubation, or other airway device will not enable ventilation of the lungs of a patient. That requires an emergency surgical access to the trachea from the anterior neck below the level of the obstruction. Surgical cricothyroidotomy is nowadays the gold standard for this emergency neck rescue, applying a "scalpel-bougie-tube" technique to enable ventilation of the patient's lungs.

BASIC MECHANICAL VENTILATION

Very few studies address the specifics of ventilation during advanced life support. Studies indicate that healthcare personnel ventilate excessive during cardiac arrest and hyperventilation should be avoided.

Various small portable automatic ventilators are available to be used during resuscitation. Initial suggested settings are the delivery of a tidal volume of 6 ml kg^{-1} ideal body weight at 10 breaths min^{-1}. In the presence of a spontaneous circulation, the correct setting will be determined by checking the patient's arterial blood gas values. Avoid chest compression attempts during the inspiratory phase by synchronising chest compressions with the ventilator. That might be of less importance after the placement of a tracheal tube.

KEY LEARNING POINTS

- **When undertaken by someone with appropriate skills and experience, tracheal intubation is an effective airway management technique during cardiopulmonary resuscitation.**
- **In unskilled hands, prolonged chest compression interruptions, the high risk of failure (e.g. unrecognised oesophageal intubation) make tracheal intubation attempts potentially harmful.**
- **Automatic ventilators may be a useful adjunct during cardiopulmonary resuscitation. Their safe use requires appropriate training.**

Details of the clinical assessment of correct tube placement, oesophageal detection device, other carbon dioxide (CO_2) detector devices (disposable colorimetric end-tidal carbon dioxide ($ETCO_2$) detectors, non-waveform electronic digital $ETCO_2$ devices), the perceived advantages and disadvantages of tracheal intubation, and a detailed description of a surgical cricothyroidotomy procedure in cardiac arrest are described in the airway chapter in CoSy.

CHAPTER 9
DEFIBRILLATION, CARDIOVERSION AND PACING

LEARNING OUTCOMES

To understand:

- **the mechanism of defibrillation**
- **the factors affecting defibrillation success**
- **the importance of minimizing interruptions in chest compressions during defibrillation**
- **how to deliver a shock safely using either a manual or automated external defibrillator (AED)**
- **the indications for cardiac pacing in the peri-arrest setting**
- **how to apply non-invasive, transcutaneous electrical pacing**
- **the problems associated with temporary transvenous pacing and how to correct them**
- **how to manage patients with implanted permanent pacemakers and cardioverter defibrillators in the setting of cardiac arrest and in the peri-arrest setting**

INTRODUCTION

Defibrillation is a crucial link in the chain of survival and one of the few interventions that has been shown to improve outcome from cardiac arrest. The probability of successful defibrillation declines rapidly with time; early defibrillation is critical and the first goal of the ALS team. Every minute that passes before defibrillation increases mortality by 10-12%.

Continuous, high-quality chest compressions with minimal interruptions are the other goal of the ALS team. Every 5-second increase in pre-shock pause halves the chance of successful defibrillation.

In addition to defibrillation, cardiac pacing can be appropriate in some cardiac arrest or peri-arrest settings. The ALS provider does not need to have a detailed technical knowledge of permanent cardiac pacemakers or implanted cardioverter defibrillators (ICDs) but needs to recognize when they are present and how they influence cardiac arrest management.

FACTORS AFFECTING DEFIBRILLATION SUCCESS

Defibrillation is the passage of an electrical current of sufficient magnitude across the myocardium to depolarize a critical mass of cardiac muscle simultaneously, enabling a normal rhythm to take over. The delivered current is influenced by transthoracic impedance and electrode position; as little as 4% reaches the myocardium.

Transthoracic impedance

Current flow is inversely proportional to transthoracic impedance. Transthoracic impedance is influenced by electrode-to-skin contact, electrode size and phase of ventilation. The impedance may increase from 70 to 150 Ohm with poor technique.

Electrode-to-skin contact

Patients with a very hairy chest may have increased impedance and may suffer burns from poor contact. If a razor is immediately available, excessive hair can be quickly removed. Transdermal drug patches must also be removed before applying the electrodes.

Electrode size

The optimal electrode size is unknown; the recommendation is that the area's sum should be a minimum of 150 cm^2. Self-adhesive pads are recommended.

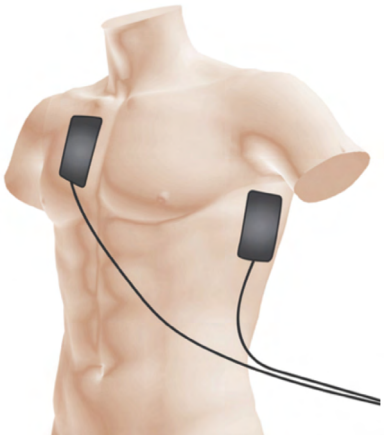
Respiratory phase

Transthoracic impedance is minimal at end-expiration. Positive end-expiratory pressure (PEEP) increases impedance and should be minimized during defibrillation.

Electrode position

The fibrillating part of the heart should lie directly between the electrodes. For patients in VF/pVT, the standard position is one electrode in the suprasternal area below the right clavicle and one electrode in the mid-axillary line on the left, approximately level with electrode V6. Bilateral or antero-posterior are other possible pad positions. In case of implantable medical devices (e.g., pacemaker or ICD), place the pad at least 8 cm away from the device or use an alternative position.

Figure 9.1 Standard position of defibrillator pads



CPR or defibrillation first?

A specific period of CPR before the first rhythm analysis is not recommended. Defibrillation must be performed as soon as possible.

Strategies for minimizing the pre-shock pause

The pre-shock pause should be less than 5 seconds. Continue chest compressions during charging and communicate effectively as a leader. The safety check before the moment of defibrillation should be rapid but efficient. Minimize post-shock pause by immediately resuming chest compressions after defibrillation.

Shock sequence

Immediately after giving a single shock, without reassessing pulse or rhythm, resume CPR. If VF/pVT should persist, additional perfusion of the myocardium is needed. Even if an organized rhythm should return, it takes time for a pulse to become palpable. Chest compressions during a perfusing rhythm do not increase the chance of VF recurrence.

Witnessed, monitored VF/pVT with immediate access to defibrillation

If a patient has a monitored and witnessed cardiac arrest with VF/pVT and the defibrillator is directly available in the immediate vicinity (such as the cardiac catheter laboratory, the coronary care unit, or possibly the operating room), a three-stacked shock sequence can be used.

- 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 and escalate energy levels.
- Start chest compressions and continue CPR for two minutes if the third shock is unsuccessful. Count these three defibrillations for amiodarone administrations. However, for adrenaline timing, count the resuscitation cycles, not the administered shocks.

Shock energy and waveforms

The recommendations for energy levels are based on consensus. For biphasic defibrillators the initial shock should be at least 150J. Use 360J when using monophasic defibrillators. If the first shock is not successful and the defibrillator is capable of delivering shocks of higher energy, it is reasonable to increase the energy for subsequent shocks.

Importance of uninterrupted chest compressions

The importance of early, uninterrupted chest compressions is emphasized throughout this manual. They should only be interrupted for rhythm checks and shock delivery and resumed as soon as a shock has been delivered. With two persons present, the person operating the defibrillator applies the electrodes and charges during compressions. The entire process of pausing compressions, standing clear and immediately resuming chest compressions should be achieved in < 5 seconds.

SAFETY

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

Safe use of oxygen during defibrillation

An oxygen-enriched atmosphere may cause fire and burns to the patient when there is a spark. Use self-adhesive pads to minimize this risk. In addition:

- Take off any oxygen mask or nasal cannula and place them at least 1 meter away from the patient's chest.
- Leave ventilation bags connected to the tracheal tube or supraglottic airway, ensuring there is no residual PEEP.
- If the patient is connected to a ventilator, leave the ventilator tubing connected.

AUTOMATED EXTERNAL DEFIBRILLATORS

AED's are safe and effective when used by laypeople with minimal or no training. Defibrillation many minutes before professional help arrives is possible. Providers should continue CPR while attaching an AED and follow the voice prompts when they are spoken. Standard AED's are suitable for use in children older than eight years.

Automated rhythm analysis

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

In-hospital use of AEDs

AED's should be considered for the hospital setting to facilitate defibrillation within 3 minutes of collapse. Healthcare providers with a duty to perform CPR should be trained, equipped, and authorized to do defibrillation.

Public access defibrillation (PAD) programs

An organized and practised response with rescuers trained and equipped to recognize emergencies, activate the EMS system, provide CPR, and use the AED will improve survival from out-of-hospital cardiac arrest. Public access defibrillation programs are most likely to improve cardiac arrest survival if established in locations where witnessed cardiac arrest is expected to occur (such as airports, casinos and sports facilities).

Sequence for the use of an AED or shock-advisory defibrillator

1. Make sure the victim, any bystanders, and you 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 both things yourself.
3. Start CPR according to the guidelines.
4. As soon as the AED arrives:
   - Switch on the AED and attach the electrode pads. If more than one rescuer is present, continue CPR while this is done.
   - Follow the voice/visual directions.
   - Ensure that nobody touches the victim whilst the AED is analyzing the rhythm.

5A. If a shock **IS** indicated:

- Ensure that nobody touches the victim.
- Push the shock button as directed.
- Continue as directed by the voice/visual prompts.

5B. If **NO** shock is indicated:

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

6. Continue to follow the AED prompts until:

- Qualified help (e.g., ambulance or resuscitation team) arrives and takes over.
- The victim starts to breathe normally.
- You become exhausted.

Notes

- The AED's carrying case must contain some strong scissors for cutting through clothing and a disposable razor for shaving excessive chest hair to obtain good electrode contact.
- According to the guidelines, if ALS providers are using the AED, they should implement other ALS interventions (advanced airway, ventilation, IV access, drug delivery, etc.).

MANUAL DEFIBRILLATION

Manual defibrillators allow for rapid shock without having to wait for automated rhythm analysis. The operator, however, has to be skilled in ECG rhythm recognition. Manual defibrillators often have additional functions, such as the ability to deliver synchronized shocks and external pacing.

Sequence for the use of a manual defibrillator

This sequence is an integral part of the ALS treatment algorithm in chapter 4.

Using handheld defibrillator paddles

As handheld paddles are still in use in some countries, the following recommendations apply for their usage:

1. Confirm VF. If in doubt, use a printout rhythm strip.
2. Hand over to an assigned team member for defibrillation.
3. Select the appropriate energy on the defibrillator (maximum energy for monophasic devices), leave the paddles in the defibrillator and press the charge button.

10a. Apply conductive gel to the patient's chest.

10b. 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 if not using a closed system. Ensure that the rescuer giving the compressions is the only person touching the patient.

11a. Once the defibrillator is charged, tell the rescuer doing the chest compressions to" Stop"; when clear, give the shock.

11b. Move one of the charged paddles to the patient's chest.

11c. Once safely positioned and kept in place, move the second paddle to the patient's chest.

11d. Deliver shock and return both paddles to the defibrillator.

12. Use the "Start" command without reassessing the rhythm or feeling for a pulse. Restart CPR using a ratio of 30:2, starting with chest compressions. The interruption for shock delivery should not exceed 3 seconds.

13. The command goes back to the team leader.

SYNCHRONIZED CARDIOVERSION

Carry out cardioversion under general anaesthesia or analgesia/conscious sedation, administered by a healthcare professional competent in the technique being used.

For electrical cardioversion of atrial or ventricular tachyarrhythmias, the shock must be synchronized with the R wave of the electrocardiogram. Avoiding the relative refractory period in the T wave minimizes the risk of inducing VF. Electrodes are applied in the same way as attempted defibrillation. Most manual defibrillators incorporate a switch that enables shock triggering by R wave detection. The operator must anticipate a slight delay between pressing the buttons and the discharge of the shock. The same safety precautions must be met as for attempted defibrillation. If a second shock is needed, reactivate the synchronisation mode if necessary.

For a broad-complex tachycardia, start with 120–150 J biphasic shock (200 J monophasic) and increase in increments if this fails. Lower-energy shocks will often terminate atrial flutter and regular narrow-complex tachycardia: start with 70–120 J biphasic (100 J monophasic).

For atrial fibrillation an initial synchronised shock at maximum defibrillator output rather than an escalating approach is a reasonable strategy.

For atrial fibrillation and flutter use anteroposterior defibrillator pad positions when it is practicable to do so.

INTERNAL DEFIBRILLATION

Internal defibrillation using paddles applied directly across the ventricles requires considerably less energy than used for external defibrillation. For biphasic shocks, use 10-20J. Do not exceed 50J using this method.

CARDIAC PACING AND PACEMAKERS

Non-invasive transcutaneous pacing

Non-invasive transcutaneous pacing can be established very quickly, requires a minimum of training and can be initiated by nurses, paramedics and physicians. Patient discomfort is a significant disadvantage, and IV analgesia and sedation may be required in conscious patients.

- Avoid any unnecessary delay in starting pacing and pay careful attention to technique.
- Using scissors or a razor, quickly remove excess chest hair from the skin where the electrode pad is applied.
- Make sure that the skin is dry.
- Attach ECG monitoring electrodes and leads if necessary - these are needed with some transcutaneous pacing devices.
- Position the electrode pads in the conventional right pectoral-apical positions if possible, paying attention to the sufficiently lateral position of the apical pad. If this position is prevented (e.g., by chest trauma, pacemaker, or ICD implant), anterior-posterior (A-P) positions can be used.
- If you are using a pacing device that is not capable of defibrillation, use the anterior-posterior placement for the pacing electrodes so that defibrillator pads can still be used in the 'conventional' right pectoral and apical positions if cardiac arrest occurs.
- Different transcutaneous pacing devices have different properties. For example, some require the operator to increase the current delivered with each pacing stimulus until electrical capture is achieved. Others use a constant current that cannot be adjusted and longer pulse duration (duration of the pacing stimulus) than other devices. Make sure that you are familiar with the operation of the device that you are using.
- Most transcutaneous pacing devices offer pacing in demand mode; the pacemaker will be inhibited if it detects a spontaneous QRS complex. However, if there is a lot of movement artefact on the ECG this may inhibit the pacemaker. Avoid movement artefact as far as possible. If artefact still appears to be inhibiting the pacemaker, switch to fixed-rate pacing mode.

- Select an appropriate pacing rate, usually in the range of 60–90 min^{-1} for adults. In some circumstances (for example complete AV-block with an idioventricular rhythm at 50 min^{-1}), a slower pacing rate of 40 or even 30 min^{-1} may be appropriate to deliver pacing only during sudden ventricular standstill or more extreme bradycardia.
- If the pacing device has an adjustable energy output, set this at its lowest value and turn on the pacemaker. Gradually increase the output while observing the patient and the ECG. As the current is increased, a pacing spike will appear on the ECG. Increase the current until each pacing spike is followed immediately by a QRS complex, indicating electrical capture (typically with a current of 50-100 mA using a device with adjustable output).
- Check that a T wave follows the apparent QRS complex. Continued failure to achieve electrical capture may indicate non-viable myocardium, but other conditions (e.g., hyperkalemia) may prevent successful pacing.
- Check that the electrical capture has a palpable pulse; the absence of a pulse with electrical capture constitutes PEA.

Although there is no hazard from transcutaneous pacing to other people in contact with the patient, there is no benefit in trying transcutaneous pacing during chest compressions. When transcutaneous pacing produces an adequate pulse, seek expert help immediately to decide about a transvenous pacing system.

Implanted permanent pacing systems

Problems with implanted permanent pacing systems are rare. Occasional fracture of a lead may occur (spontaneous or following trauma). When assessing the patient using the ABCDE-approach, check during "E" for the presence of an implanted device and try to determine if it is a pacemaker or an ICD.

ICD's function as demand pacemakers for bradyarrhythmia, sometimes provide biventricular pacing for heart failure and deliver defibrillation if required. Implant position is similar to pacemakers. ICD's sometimes misdiagnose arrhythmias, delivering inappropriate shocks. Holding a magnet on the skin over the device temporarily disables shocks.

If the patient has a cardiac pacemaker or implantable cardioverter-defibrillator (ICD), electrodes must be placed at least 8 cm away from the pacemaker unit. Alternative pad positions such as antero-posterior or bilateral are also possible. To avoid the risk of shock to rescuers due to unannounced ICD discharges, wear gloves and avoid skin-to-skin contact.

KEY LEARNING POINTS

- **For the patient in VF/pVT, early defibrillation is the only effective means of restoring a spontaneous circulation.**
- **When using a defibrillator, minimize interruptions in chest compressions**
- **Non-invasive pacing can be delivered by any ALS provider and is the immediate treatment for bradyarrhythmia that is a potential risk to the patient who does not respond to initial drug treatment.**
- **Non-invasive pacing is a temporary measure to be used until either a stable and effective spontaneous rhythm returns, or a competent person stablishes transvenous pacing.**
- **Special precautions are necessary during resuscitation attempts in patients with implanted pacemakers and ICDs.**

CHAPTER 10
SUPPORTING TECHNIQUES AND SKILLS

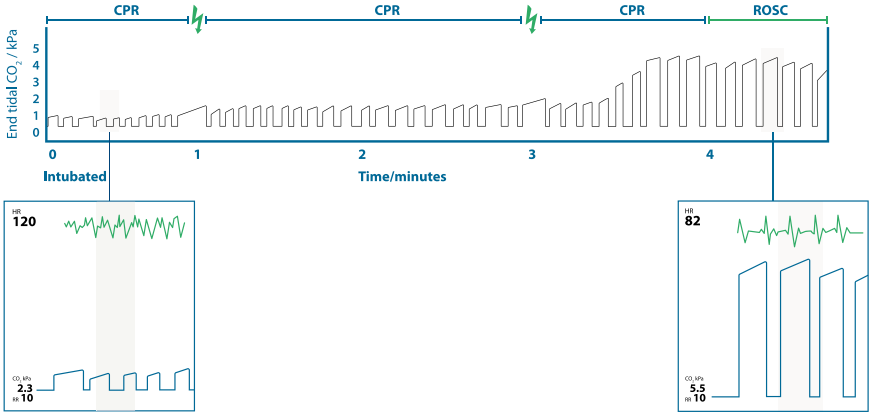
LEARNING OUTCOMES
To understand:

- **How to use capnography during CPR**
- **How to interpret arterial blood gases in the peri-arrest situation**
- **How to use ultrasound in the peri-arrest situation**
- **Indications for mechanical CPR devices**
- **Indications for eCPR**

CAPNOGRAPHY

The concentration of CO_2 can be measured in expired air and is expressed as either percentage by volume or as a partial pressure, both of which are very similar numerically. The concentration varies throughout expiration, being maximal at the end and it is this value, the end-tidal CO_2 ($ETCO_2$) that is most useful. Figure 10.1 shows CO_2 curves during resuscitation starting with low quality chest compression, increase in CO_2 indicates good quality chest compressions with immediate sustained increase at ROSC.

Figure 10.1 Waveform capnography



During a cardiac arrest, blood flow to the lungs ceases and despite continued production, if ventilation is maintained, $ETCO_2$ falls to zero. Once chest compressions are started, blood flow to the lungs will be partially restored and if the patient is ventilated, the end-tidal CO_2 will increase, proportionately to the cardiac output generated.

Information gained from monitoring $ETCO_2$ during cardiopulmonary resuscitation

- **Tube placement**
  Capnography has a high sensitivity and specificity for confirming placement of a tracheal tube in the airway.
- **Quality of CPR**
  The more efficient the chest compression, the greater the cardiac output which delivers more CO_2 to the lungs from where it is exhaled thus generating a higher end-tidal concentration. High-quality chest compressions will result in typical $ETCO_2$ values of 2.0-2.5 kPa.
- **Return of Spontaneous Circulation (ROSC)**
  An increase in $ETCO_2$ during CPR may indicate that ROSC has occurred. However, chest compression should not be interrupted based on this sign alone.
- **Prognostication**
  Although high and increasing $ETCO_2$ values are associated with increased rates of ROSC and survival after CPR, do not use a low $ETCO_2$ value alone to decide if a resuscitation attempt should be stopped.

BLOOD GASES

Interpreting the analysis of an arterial blood sample to determine a patient's acid-base status and respiratory gas exchange is a key component in the management of any patient in the peri-arrest situation. There are 4 key pieces of information in the results of an arterial blood sample.

- **PaO_2**
  The concentration of oxygen in inspired air is 21% - representing a partial pressure of 21 kPa (158 mmHg). In a healthy individual breathing air, the PaO_2 is normally higher than 11 kPa (83 mmHg) i.e. about 10 kPa (75 mmHg) lower than the inspired partial pressure. This can be used as a rule of thumb to estimate the PaO_2 for any given inspired concentration, in that it should be numerically about 10 less than the inspired concentration (%). For example, 40% inspired oxygen should result in a PaO_2 of approximately 30 kPa (225 mmHg). With increasing lung injury, the gap between inspired concentration and PaO_2 increases. This is important to recognise because for someone breathing 50% oxygen, a PaO_2 of 13 kPa (98 mmHg) is not 'normal'.

- **pH**
  The acidity or alkalinity of the blood is determined by the concentration of hydrogen ions [H^+]; the greater the concentration, the more acid the blood. The pH scale is a logarithmic scale expressing the hydrogen ion concentration between 1 and 14. The pH of a normal arterial blood sample lies between 7.35 and 7.45.

- **$PaCO_2$**
  Carbon dioxide (CO_2) is an important waste product of metabolism. Under normal circumstances, it is transported in the blood to the lungs where it is excreted during expiration. The normal $PaCO_2$ is 5.3 kPa (40 mmHg) with a range of 4.7-6.0 kPa (35 – 45 mmHg). If the metabolic production of CO_2 is constant, the only factor that affects the amount in the blood is the rate at which it is removed by alveolar ventilation. A decrease in alveolar ventilation will reduce excretion of CO_2 causing an increase in $PaCO_2$ and the production of more hydrogen ions. If the pH decreases below 7.35 an acidaemia has been produced. As the primary cause of the acidaemia is a problem with the respiratory system, we call this process a respiratory acidosis. Conversely, an increase in alveolar ventilation that removes CO_2 faster than it is generated reduces $PaCO_2$, reducing the concentration of hydrogen ions. As a result, the pH will increase and if it exceeds 7.45 an alkalaemia has been produced. Again, the primary cause is the respiratory system and we call this process a respiratory alkalosis.

- **Base excess**
  For clarity, only changes in base excess are discussed; however, bicarbonate will also change numerically in the same direction. The base excess is a measure of the amount of excess acid or base in the blood as a result of a metabolic derangement. It is calculated as the amount of strong acid or base that would have to be added to a blood sample with an abnormal pH to restore it to normal (pH 7.4). A patient with a base excess of 8 mmol l^{-1} would require 8 mmol l^{-1} of strong acid to return their pH to normal. Conversely, a patient with a base deficit of 8 mmol l^{-1} will require the addition of 8 mmol l-1 of strong base to normalise their pH.

    - A base excess more negative than - 2 mmol l^{-1} indicates a metabolic acidosis.
    - A base excess greater than + 2 mmol l^{-1} indicates a metabolic alkalosis.

Interpretation of the result of blood gas analysis is achieved best by following strictly five steps.

- **Step 1 – how is the patient?**
  This will often provide useful clues to help with interpretation of the results. For example, one might reasonably predict that analysis of arterial blood shortly after successful resuscitation would show signs of a respiratory acidosis caused by a period of inadequate ventilation and a metabolic acidosis due to the period of anaerobic metabolism during the arrest producing lactic acid. Consequently, we would expect the patient to have a very low pH with changes in both $PaCO_2$ and base excess.

- **Step 2- is the patient hypoxaemic?**
  The PaO_2 while breathing room air should be 10.0-13.0 kPa (75-98 mmHg). However, if the patient is receiving supplemental oxygen, the PaO_2 must be interpreted in light of the inspired oxygen concentration. As discussed above, the inspired partial pressure of oxygen can be regarded as the numerical equivalent of the inspired concentration (%). If there is a difference of greater than 10 kPa between the two values, there is a defect in oxygenation, proportional to the magnitude of the difference.
- **Step 3 - is the patient acidaemic (pH < 7.35) or alkalaemic (pH> 7.45)?**
  If the pH is within or very close to the normal range then this suggests normality or a chronic condition with full compensation. In principle, the body never overcompensates and this should enable the primary problem to be determined.
- **Step 4 - what has happened to the $PaCO_2$? Is the abnormality wholly or partially due to a defect in the respiratory system?**
  - If the $PaCO_2$ is increased in acidaemia there is a respiratory acidosis that may be accounting for all or part of the derangement. There could also be a metabolic component.
  - If the the $PaCO_2$ is reduced in alkalaemia there is a respiratory alkalosis, but this is an unusual isolated finding in a patient breathing spontaneously, with a normal respiratory rate. It is seen more often in patients who are being mechanically ventilated with excessively high rates and/or tidal volumes. As a result, $PaCO_2$ decreases, there is a reduction in H+ and an alkalosis develops.
- **Step 5 - what has happened to the base excess? Is the abnormality wholly or partially due to a defect in the metabolic system?**
  - Is the base excess reduced (more negative than -2 mmol l^{-1}) in acidaemia there is a metabolic acidosis accounting for all or part of the derangement. There could be a respiratory component if the $PaCO_2$ is also increased, a situation commonly seen after a cardiac arrest.
  - Is the base excess increased (> + 2 mmol l^{-1}) in alkalaemia there is a metabolic alkalosis accounting for all or part of the derangement. There could be a respiratory component if the $PaCO_2$ is also decreased, but this would be very unusual.

ULTRASOUND

Point-of-care ultrasound (POCUS) imaging is already commonly used in emergency care settings. Its use during CPR is also increasing.

- Only skilled operators should use intra-arrest POCUS.
- POCUS must not cause additional or prolonged interruptions in chest compressions.

- POCUS may be useful to diagnose treatable causes of cardiac arrest such as cardiac tamponade and pneumothorax.
- Pulmonary embolism may be difficult to detect using ultrasound as the right ventricle will dilate during cardiac arrest irrespectively of the cause of arrest.

MECHANICAL CHEST COMPRESSIONS DEVICES

The routine use of automated mechanical chest compression devices is not recommended. They are a reasonable alternative when sustained high-quality manual chest compressions are impractical or compromise provider safety. Examples include transporting to hospital in an ambulance or helicopter, during percutaneous coronary intervention, diagnostic imaging such as a CT scan, as a bridge to establishing extra-corporeal CPR or maintaining circulation prior to organ retrieval when resuscitation is unsuccessful. Mechanical devices should be used only in settings where teams are trained in their deployment to avoid long pauses in chest compressions.

ECPR

Extracorporeal CPR (eCPR) is defined by the ELSO (Extracorporeal Life Support Organization) as the application of rapid-deployment veno-arterial extracorporeal membrane oxygenation (VA-ECMO) to provide circulatory support in patients in whom conventional CPR is unsuccessful in achieving sustained ROSC. The use of eCPR has increased for both IHCA and OHCA in recent years. eCPR may be considered as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it can be implemented. Commonly used inclusion criteria are:

- Witnessed cardiac arrest with bystander CPR.
- Time to establishing eCPR is less than 60 minutes from starting CPR.
- Younger patients (e.g. less than 65 to 70 years) and no major comorbidities precluding a return to independent life.
- Known or suspected treatable underlying cause of cardiac arrest.

Establishing an eCPR programme requires a whole system approach (in- and out-of hospital) and considerable resources to implement effectively,

KEY LEARNING POINTS

- **Capnography is essential during CPR**
- **Arterial blood gases add essential information in the peri-arrest situation**
- **If used by skilled personnel in an appropriate environment there is a number of technical adjuncts available to support CPR**

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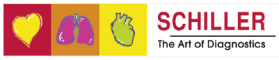

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