[h1] European Resuscitation Council Guidelines 2025: Special Circumstances in Resuscitation

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[h1] Abstract

This European Resuscitation Council (ERC) guideline on Special Circumstances in Resuscitation is based on the 2025 Consensus of Science with Treatment Recommendations (CoSTR) of the International Liaison Committee on Resuscitation (ILCOR), reviews by the expert writing group and relevant peer-reviewed literature. The guideline chapter provides guidance for laypeople and healthcare professionals on the modifications required to basic and advanced life support in adults for the prevention and treatment of cardiac arrest for in-hospital and out-of-hospital cardiac arrest. The ERC Guidelines 2025 on Paediatric Life Support cover the special circumstances in children. REF

[h1] Keywords

Cardiac arrest, special circumstances, adults, special causes, special settings, special patient populations

[h1] Abbreviations

ABC: Airway-breaths-compressions
ACE-I: Angiotensin converting enzyme inhibitors
AED: Automated external defibrillator
ALS: Advanced life support
BLS: Basic life support
CAB: Compressions-airway-breaths
CAD: Coronary artery disease
CKD: Chronic kidney disease
COPD: Chronic obstructive pulmonary disease
CPR: Cardiopulmonary resuscitation
CoSTR: Consensus or Science with Treatment Recommendations
DVT: Deep venous thrombosis
ECG: Electrocardiogram
ECMO: Extracorporeal membrane oxygenation
ECPR: Extracorporeal cardiopulmonary resuscitation
ED: Emergency department
EMS: Emergency medical services
ERC: European Resuscitation Council
ETCO2: End tidal carbon dioxide
HCP: Healthcare professionals
HD: Haemodialysis
ICU: Intensive Care Unit
IHCA: In-hospital cardiac arrest
ILCOR: International Liaison Committee on Resuscitation
ILS: International Lifesaving Federation
IM: Intramuscular
IV: Intravenous
LVAD: Left Ventricular Assist Device
MRA: Mineralocorticoid receptor antagonist
NIV: Non-invasive ventilation
OHCA: Out-of-hospital cardiac arrest
OR: Operating room
PAD: Public access defibrillator
PCI: Percutaneous coronary intervention
PE: Pulmonary embolism
PEA: Pulseless electrical activity
POCUS: Point of care ultrasonography
PPE: Personal protective equipment
RCT: Randomised controlled trial
REBOA: Resuscitative endovascular balloon occlusion of the aorta
ROSC: Return of spontaneous circulation
SCA: Sudden cardiac arrest
STEMI: ST-elevation myocardial infarction
TCA: Traumatic cardiac arrest
TOE: Transoesophageal echocardiography
VA-ECMO: Veno-arterial extra-corporeal membrane oxygenation
VF: Ventricular fibrillation
VT: Ventricular tachycardia
WHO: World Health Organization

[h1] Introduction

Cardiac arrest, regardless of its aetiology, requires immediate intervention, involving rapid recognition, emergency request for assistance, and high-quality cardiopulmonary resuscitation (CPR), with minimal interruptions. Effective management involves the prompt identification and treatment of reversible causes, often summarised in the mnemonic '4Hs and 4Ts'. However, standard life support protocols are not universally applicable, as special circumstances need modifications to these guidelines. The ERC categorise these special circumstances into three parts:

1. Special causes leading to specific interventions.
2. Special settings where unique site factors or aetiologies require tailored approaches.
3. Special patient populations, characterised by pre-existing conditions or comorbidities that require different modified treatment strategies.

This ERC Guideline on Special Circumstances in Resuscitation is based on the 2025 annual Consensus of Science with Treatment Recommendations (CoSTR) of the International Liaison Committee on Resuscitation (ILCOR). Many topics addressed in these Special Circumstances guidelines were not part of the ILCOR review. Thus, several recommendations are based on consensus of the ERC Guidelines 2025 Special Circumstances in Resuscitation writing group based on additional systematic or scoping reviews, or selected original articles. Specialists in the field of these topics formed this expert group which evaluated the available literature and presented an overview to this ERC Guideline 2025 Writing Group, which was discussed in several meetings to reach consensus on the recommendations. These were subsequently approved by all Special Circumstances in Resuscitation writing group members and the ERC Guidelines 2025 Steering Committee. The broader methodology used for guideline development is presented in the Executive Summary. This Guideline was posted for public comments in May/June 2025. A total of [INSERT NUMBER] individuals from [INSERT COUNTRIES] submitted [INSERT NUMBER] comments, leading to [INSERT CHANGES] in the final version. Subsequently, the feedback was reviewed by the writing group, and the Guideline was thereafter updated where relevant. The Guideline was presented to, and approved by, the ERC Board and the ERC General Assembly on xy June 2025.

For the purpose of this guideline, the term CPR relates to the specific technical skills of cardiopulmonary resuscitation (e.g. performance metrics of chest compression and ventilation), whilst resuscitation is used as a generic term covering the broader range of skills and interventions. The term bystander is used to describe rescuers who happen to be at the

scene to provide help, and the term first responder is used for those who have additional training and are alerted to attend the scene of a cardiac arrest. Healthcare Professionals (HCP) are defined as those who work in any healthcare sector (prehospital or in-hospital). Laypeople are persons not working in the healthcare sector. Basic Life Support (BLS) is defined as initiating the chain of survival, early high-quality chest compression, effective ventilation, and the early use of an AED. Any form of resuscitation education beyond BLS is described generically as advanced life support (neonatal, paediatric and adult life support). Where the term 'ALS' is used, this refers specifically to the ERC adult Advanced Life Support course.

The writing group of this 2025 ERC Guideline on Special Circumstances considered the recently introduced ERC approach to diversity, equality, equity, and inclusion and applied it whenever possible, recognising that this is a field for improvement in the development of evidence-informed guidelines. The ERC aims to advance resuscitation practices and improve patient care on a global scale.

[h1] Key points

- Take safety measures where needed
- Follow the ABCDE approach in critically ill patients
- Minimise no-flow time in cardiac arrest
- Optimise oxygenation in cardiac arrest
- Follow the ALS algorithm in cardiac arrest
- Use your resources
- Address reversible causes relevant to the special circumstance
- Where appropriate, prioritise treating reversible causes
- Briefly interrupt chest compressions if needed
- Modify the ALS algorithm if needed
- Consider transfer for advanced treatment options
- Consider ECPR in specific circumstances

[h1] Summary of key changes or new evidence

Table 1. This table provides a synopsis of the major changes in the ERC resuscitation guidelines 2025 on Special Circumstances in Resuscitation. Guidance from the 2021 ERC Guidelines for Resuscitation that is not listed here is still valid and applicable.

	Summary of changes	
	ERC guidelines 2021	**ERC guidelines 2025**
Anaphylaxis	• We suggest repeating the IM adrenaline dose if there is no improvement in the patient's condition after about 5-min	• Repeat IM after 3-5 minutes, according to clinical response.
Resuscitation in hyper/hypokalaemia and other electrolyte disorders	• Introduction of management algorithm	• No evidence for or against use of IV calcium in hyperkalaemia. • ECG – record pre and post IV calcium. • Hyperkalaemia algorithm revised. • Guidance in cardiac arrest added.
Hyperthermia, malignant hyperthermia and toxin induced hyperthermia		• Figure for treatment of malignant hyperthermia added. • Section on toxin-induced hyperthermia with table added.
Accidental hypothermia and avalanche rescue	• Scoring systems established	• Revised Swiss Staging for hypothermia. • Heart rate <45/min as new high-risk criterion for hypothermia-induced cardiac arrest. • Criteria for qualification for extracorporeal life support do not apply for rewarming

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		of arrested hypothermic patients. • New avalanche rescue algorithm. • BLS avalanche rescue algorithm for cases with insufficient personnel on site.
Pulmonary embolism	• overview	• Specific drug and dosage schemes added.
Coronary thrombosis	• Individualised approach concerning reperfusion strategy in NSTEMI patients was recommended. Brief evaluation to rule out non-coronary causes and performing urgent coronary angiography in case of suspected myocardial ischaemia were suggested.	• Assess 12-lead electrocardiogram after ROSC for ischaemic features and consider repeating if findings are inconclusive • Routine emergent/early coronary angiography in stable patients without ST-elevation or equivalent on ECG discouraged. • Immediate angiography recommended in haemodynamic instability or suspected ongoing ischaemia. • Indications for antiplatelet and anticoagulation treatment in this setting.
Toxic agents	• Extensive subchapter	• New update on intoxication with opioids.
Traumatic cardiac arrest	• New algorithm established	• Algorithm clarified. • Role of chest compressions specified.

	• Invasive procedures introduced	• REBOA de-emphasised
Cardiac arrest in the catheterisation laboratory	• Mentioned but not specifically included in the management algorithm. • Point of care ultrasonography (POCUS) may be considered to identify reversible causes of cardiac arrest. • General recommendations were given to consider mechanical CPR, extracorporeal CPR and circulatory support devices.	• Guidance on resuscitation team performance in the catheterisation lab. • Specifies management of extreme bradycardia/ asystole complicating invasive procedures, consider external or transvenous temporary pacing. • Updated role of POCUS and specific indications on transoesophageal echocardiography. • Update mechanical CPR, extracorporeal CPR and circulatory support devices, with specific indications for cardiac arrest. • Limited evidence on intracoronary adrenaline.
Drowning and water rescue	• First responders not covered	• Introduction of first responders in drowning resuscitation. • Clarifications on role of bystanders during rescue, spinal stabilisation, and the emphasis on ventilation by bystanders and first responders.

Cardiac arrest in the operating room	• Less detailed	• Sudden decrease in systolic pressure <50 mmHg despite interventions is criterion to initiate chest compressions. • Lower initial IV adrenaline dose and increment dosing. • Early ECPR for patients with delayed ROSC. • Open chest compressions as an option solely for trained healthcare professionals. • Emphasis on human factors development.
Local anaesthetic systemic toxicity	• No treatment algorithm	• New detailed algorithm.
Resuscitation in cardiac surgery and Left Ventricular Assist Device patients	• LVAD patients not covered	• Revised algorithm for cardiac arrest after cardiac surgery. • Addition of cardiac arrest in Left Ventricular Assist Device patients' algorithm.
Cardiac arrest in sports	• Short overview	• More robust and comprehensive data on incidence and survival. • Characterisation of causes by age group (±35 years). • Awareness about risk in recreational and non-elite athletes. • Specific guidance for high-attendance or televised events.

Resuscitation during transport	• Limited to team approach in EMS	• Initial approach of two-member ALS crews, HEMS and air ambulances and the use of arterial line in the prehospital setting. • Inflight cardiac arrest is expanded by CPR in microgravity guidelines.
Asthma & COPD		• New shortened algorithm
Resuscitation in haemodialysis patients	• Focus on haemodialysis unit	• Focus on haemodialysis patients. • Infographic of resuscitation whilst on dialysis.
Resuscitation in obese patients		• Standard BLS and ALS recommended.
Resuscitation in patients with pectus excavatum	• Not addressed	• Reduced chest compression depth of 3-4cm. • Increased force is required to deliver effective chest compressions in case of Nuss bar correction. • Use of anteroposterior pad placement for defibrillation.
Resuscitation during Pregnancy	• Definition of maternal cardiac arrest as occurring during pregnancy and up to 6 weeks post-partum. • Recommends manual left uterine displacement; left lateral tilt discussed with caveats.	• Definition retained. • Manual displacement preferred in non-theatre settings. • Prioritisation of immediate preparation for resuscitative hysterotomy, de-emphasising 5 minutes time limit.

	• Recommends delivery within 5 minutes of collapse if no ROSC. • Use of 4Hs and 4Ts. • Advice for regular training, team coordination.	• Addition of 4Ps (pre-eclampsia/eclampsia, puerperal sepsis, placental/uterine issues, peripartum. cardiomyopathy). • Remove fetal monitors to prevent burns during defibrillation. • Recommendation for IV/IO access above diaphragm when possible. • Preparation for major obstetric haemorrhage. • In-situ simulation, audits, and table of equipment for resuscitative hysterotomy. • Introduction of a maternal cardiac arrest algorithm

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[h1] Concise guidelines for clinical practice

[h1] General recommendation

- Initiate resuscitation following the standard ALS algorithm in cardiac arrest.
- Address reversible causes relevant to the special circumstance.
- Where appropriate, prioritise treating reversible causes, even if chest compressions are briefly interrupted.

[h1] Special causes

[h2] Management and prevention of cardiac arrest due to anaphylaxis

- Address Hypoxia, Hypovolaemia, (toxic agents).
- Prompt recognition of anaphylaxis is crucial.
- Recognise anaphylaxis by the presence of airway, breathing, or circulation problems with or without skin and mucosal changes.
- Remove or stop the trigger.
- Immediately inject intramuscular adrenaline 0.5 mg at first suspicion of anaphylaxis and repeat if no improvement occurs within 5 minutes.
- Give an IV crystalloid fluid bolus early and monitor the response.

[h2] Hyper/hypokalaemia and other electrolyte disorders

- Address Hyperkalaemia, Hypokalaemia.

[h3] Hyperkalaemia

Insert Figure E1 (XII)

- Shift potassium into cells
  - Give 10 units soluble insulin and 25g glucose IV for treatment of moderate and severe hyperkalaemia.
  - Give nebulised salbutamol (10-20mg) for moderate and severe hyperkalaemia, as an adjunct to Insulin-glucose therapy.
- Antagonise the effect of hyperkalaemia
  - Use IV calcium 30 mL 10% calcium gluconate for patients with severe hyperkalaemia with ECG changes.
  - Administer IV calcium 10 mL 10% calcium chloride and 50mmol sodium bicarbonate, through separate lines or with flush in between, in the treatment of hyperkalaemic cardiac arrest in all settings.
- Remove potassium from the body
  - Consider dialysis for patients with refractory severe hyperkalaemia.

- Consider ECPR in accordance with local protocols if initial resuscitation attempt is unsuccessful.

[h3] Hypokalaemia

Insert Figure E2 (XV)

- Where appropriate replace potassium and correct magnesium deficit concurrently.

[h2] Hyperthermia, malignant hyperthermia and toxin induced hyperthermia

- Address Hypoxia, Hyperkalaemia.

[h3] Hyperthermia

- Measure core temperature to guide treatment.
- Move patient to a cool environment.
- Simple external cooling may involve conductive, convective and evaporative measures (See section First Aid).
- With heat syncope and heat exhaustion quick removal to a cool place, simple external cooling and provision of fluids are sufficient
- With heat stroke prioritise active cooling methods that achieve the most rapid cooling rate such as ice and cold-water immersion.

[h3] Malignant hyperthermia

- Stop triggering agents immediately. (Includes turning off and removing vaporiser and changing the ventilator circuit).
- Give 2,5 mg/kg dantrolene IV.
- Start active cooling.
- Give 100% oxygen and aim for normocapnia using hyperventilation.
- Change ventilator. If ventilator cannot be changed, change charcoal filters
- Contact a malignant hyperthermia centre for advice and follow-up.

[h3] Toxin-induced hyperthermia

- Minimise exposure and absorption of the toxin.
- Use active cooling techniques. Antipyretics have no benefit as central thermoregulatory mechanisms are affected by toxins.

[h2] Accidental hypothermia and avalanche rescue

[h3] Accidental hypothermia

Insert Figure Hypo1 here

- Address Hypoxia, Hyperkalaemia, Hypothermia.
- Check vital signs for up to one minute in an unconscious hypothermic patient.

- Measure core temperature with a low reading thermometer to diagnose accidental hypothermia.
- Use the Swiss Staging System if core temperature cannot be measured.
- Transfer hypothermic patients with risk factors for imminent cardiac arrest and those in cardiac arrest directly to an extracorporeal life support centre for rewarming.
- Delay CPR in hypothermic arrested patients <28°C when CPR on site is not feasible; use intermittent CPR when continuous CPR is not possible.
- Delay further defibrillation attempts if ventricular fibrillation (VF) persists after three shocks, until core temperature is >30°C.
- Below 30°C adrenaline will accumulate and may have more detrimental than beneficial effects. Give 1mg adrenaline once to facilitate ROSC unless planning imminent initiation of extra-corporeal life support. Increase administration intervals for adrenaline to 6-10 min if the core temperature is 30-35°C.
- Consider use of a mechanical CPR device if transport is prolonged, or there are difficulties with the terrain.
- Base in-hospital prognostication of successful rewarming on the Hypothermia Outcome Prediction after Extracorporeal life support (HOPE) score.
- Rewarm hypothermic arrested patients with veno-arterial extra-corporeal membrane oxygenation (VA-ECMO).
- Initiate non-extracorporeal life support rewarming if an extracorporeal life support centre cannot be reached within a reasonable time (e.g. 6 hours).

[h3] Avalanche rescue

- Base initiation of CPR in cardiac arrest on core temperature, burial time, and airway patency as described in figures AVA 1 and 2.
- Consider proceeding according to the AvaLife algorithm, in multi-burial avalanche accidents with BLS providers only and insufficient numbers of rescuers.

[h2] Thrombosis

[h3] Pulmonary embolism

- Address Hypoxia, Hypovolaemia, Thrombosis.
- Consider pulmonary embolism in all patients with sudden onset of progressive dyspnoea and absence of known heart or pulmonary disease.
- Obtain 12-lead ECG (exclude acute coronary syndrome, look for right ventricle strain).
- Identify haemodynamic instability and high-risk pulmonary embolism.
- 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.
- Set-up a multidisciplinary team for making decisions on management of high-risk pulmonary embolism depending on local resources.
- Consider surgical embolectomy or catheter-directed treatment as alternative to rescue thrombolytic therapy in rapidly deteriorating patients.
- Low $ETCO_2$ values (<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.
- Use fibrinolytic drugs for cardiac arrest when pulmonary embolism is the suspected cause of cardiac arrest.
- Use fibrinolytic drugs or surgical embolectomy or percutaneous mechanical thrombectomy for cardiac arrest when pulmonary embolism 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.

[h3] Coronary thrombosis

Insert Figure coronary thrombosis

- Address Hypoxia, Cardiac tamponade, Thrombosis.
- Enhance health education to recognise symptoms and minimise delays in seeking medical care
- Promote BLS training for likely rescuers of high-risk groups.
- Strengthen regional networks to ensure timely percutaneous coronary intervention (PCI).
- Transfer the patient to a centre with PCI capability and activate existing STEMI networks in case of ST-elevation or suspected ongoing ischaemia.
- In patients with sustained ROSC and ST-elevation on ECG:
  - perform immediate coronary angiography (and PCI if required) within 120 min of diagnosis.
  - consider pre-hospital fibrinolysis if greater delay is expected and CPR was not prolonged or traumatic, then transfer immediately to PCI centre.
- In patients with sustained ROSC and no ST-elevation on ECG:

- Consider immediate coronary angiography (and PCI if required) if the patient is haemodynamically unstable or shows signs of ongoing ischaemia.
- In stable patients without ischaemic signs, emergent cardiac catheterisation laboratory evaluation should not be systematic and can be delayed if there is no estimated high probability of acute coronary occlusion.
- Assess for non-coronary-causes if the clinical context suggests an alternative aetiology.
- Unless on-going resuscitation is considered futile, transfer patients without sustained ROSC with ongoing CPR, to a PCI centre for consideration for angiography

[h2] Toxic agents

Insert figure toxic agents 1

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Toxic agents.
- Ensure your personal safety, (Fig. XX) as direct skin contact (e.g., mouth to mouth ventilation) might transmit toxic agents.
- Assess all patients in cardiac arrest for potential intoxication.
- Reduce absorption, consider using specific treatment measures as antidotes, decontamination and enhanced elimination.
- Administer antidotes, where available, as soon as possible.
- Be prepared to continue resuscitation for a prolonged period of time, as the toxin concentration may fall as it is metabolised or excreted during extended resuscitation measures.
- Consult regional or national poison centres for information on treatment of the poisoned patient.

[h2] Traumatic cardiac arrest (TCA)

Insert figure trauma 1

- Address Hypoxia, Hypovolaemia, Hypothermia, Cardiac Tamponade, Tension pneumothorax.
- Traumatic cardiac arrest is different from cardiac arrest due to medical causes; this is reflected in the treatment algorithm (Figure Trauma1).
- The response to traumatic cardiac arrest is time critical and success depends on a well-established chain of survival, including focused pre-hospital and specialised trauma centre care.

- Early and aggressive management of reversible causes (e.g. haemorrhage control, airway management, chest decompression) is essential for survival.
- Ultrasound aids in identifying the cause of cardiac arrest and guides resuscitative interventions.

[h1] Special Settings

[h2] Cardiac arrest in the catheterisation laboratory

Insert cath lab algorithm 2025

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Hypothermia, Cardiac tamponade, Tension pneumothorax, Thrombosis, Toxic agents.
- Promote adequate training of the staff in technical skills and advanced life support, and consider periodic emergency drills.
- Ensure emergency equipment is readily available and functional.
- Plan elective procedures carefully to minimise potential complications and promote the use of safety checklists.
- Consider echocardiography in case of haemodynamic instability or suspected complication.
- Resuscitate according to the ALS algorithm, BUT modify:
  - Apply 3 consecutive shocks in case of shockable rhythm
  - Consider external or transvenous pacing for extreme bradycardia
- Consider, in selected cases depending on clinical context, team expertise and availability:
  - Mechanical CPR, if manual compression is not feasible or safe for the provider.
  - Extracorporeal CPR in selected patients with refractory cardiac arrest, especially if it allows for critical procedures to correct reversible causes.
  - Circulatory support devices, for selected patients in cardiogenic shock after achieving ROSC.

[h2] Drowning and water rescue

Insert figure Algorithm

- Address Hypoxia, Electrolyte disorders, Toxic agents.
- Bystanders and first responders should prioritise their safety and use the safest rescue technique.
- Bystanders should call for professional help and use rescue techniques with which they feel confident, based on their competencies.

- First responders should use rescue material and flotation devices they are trained to use.
- Spine immobilisation in water should not delay removing the victim from the water when resuscitation is required.
- Follow the recommendations for hypothermia
- Start with 5 rescue breaths using 100% inspired oxygen when available, continue with standard CPR protocol.
- Airway and ventilation equipment can be used when the provider is trained appropriately.
- Increase ventilation pressure gradually when a high inspiration pressure is needed, in order to avoid stomach inflation.
- Consider scaling up to ECPR if initial resuscitation is unsuccessful, in accordance with local protocols.

[h2] Cardiac arrest in the operating room (OR)

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Hypothermia, Cardiac tamponade, Tension pneumothorax, Thrombosis, Toxic agents.
- Prevent and mitigate the risk of cardiac arrest through pre-operative screening and identification of high-risk patients, clear surgical communication about potential critical procedures, advanced monitoring and continuous presence of an anaesthesiologist during patient instability.
- Start chest compressions if the systolic blood pressure suddenly decreases below 50 mmHg, in association with fall in $ETCO_2$, despite appropriate interventions.
- Inform the surgeon and the operating room team of the cardiac arrest.
- Initiate high-quality chest compressions and adjust the height of the operating table to improve work efficiency.
- Ensure the airway is secure, review the $ETCO_2$ tracing, and deliver effective ventilation, administering 100% oxygen. Exclude unrecognised oesophageal intubation.
- Use ultrasound to guide resuscitation addressing the reversible causes.
- Exclude tension pneumothorax.
- Consider early ECPR as therapy for selected patients when conventional CPR is failing.
- Trained healthcare professionals may consider open chest cardiac compressions in specific cases as an alternative, if ECPR is unavailable

- Human factors are crucial to improve survival of intraoperative cardiac arrest – ensure familiarity with equipment, assign strategies and roles during surgical team time outs and include perioperative cardiac arrest in multidisciplinary and interprofessional team training, in-situ simulation, and advanced life support courses

[h2] Local anaesthetic systemic toxicity

- Address Hypoxia, Toxic agents
- Stop local anaesthetic if possible.
- Hyperventilate the patient to increase plasma pH if metabolic acidosis is present.
- Give a lower adrenaline dose (≤ 1µg kg^{-1} instead of 1 mg IV bolus)
- Give an initial IV bolus of 20% lipid emulsion at 1.5 $mL.kg^{-1}$over 1 min, followed by an infusion at 15 mL kg^{-1} h^{-1}.
- If ROSC has not been achieved at 5 minutes, double the rate of lipid infusion and give a maximum of two additional lipid boluses at 5-minute intervals until ROSC has been achieved.
- Do not exceed a maximum cumulative dose of 12 mL kg^{-1} IV 20% lipid emulsion.
- Consider prolonged resuscitation (>1 hour) and ECPR
- Treat seizures by administering benzodiazepines

[h2] Cardiac Surgery

Insert Figure CS1 near here (2025 rebranded algorithm)

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Hypothermia, Cardiac tamponade, Tension pneumothorax, Thrombosis, Toxic agents
- Confirm cardiac arrest by clinical signs and pulseless pressure waveforms.
- Consider ultrasound to identify reversible causes.
- Provide up to 3 consecutive shocks in VF/pVT
- Use epicardial pacing at maximum output in asystole or extreme bradycardia
- Perform re-sternotomy up to 10 days post-surgery within 5 minutes regardless of the patient's location.
- Provide internal cardiac compressions once the chest is reopened.
- Reduce adrenaline dose (0.05-0.1 mg)
- Consider ECPR for prolonged resuscitation or minimally invasive cases where reopening may be delayed

[h3] Left ventricular assist device (LVAD) patients

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Cardiac tamponade, Tension pneumothorax, Thrombosis.
- Immediately activate specialised teams for unresponsive LVAD patients.
- Start CPR while simultaneously attempting to restore device function if multiple rescuers are available.
- Consider delaying CPR for up to 2 minutes to attempt device restoration if a single rescuer is present.
- Troubleshoot device issues as a priority, following relevant protocols.

[h2] Cardiac arrest in sports

- Address electrolyte disorders, cardiac tamponade, tension pneumothorax, thrombosis.
- Screening as primary prevention plays an important role, but remains controversial.
- All sports and exercise facilities should undertake a risk assessment which considers the likelihood and consequence of sudden cardiac arrest and put in place mitigation strategies to reduce the risk.
- Gain immediate and safe access to the field of play.
- Awareness programs in sport events have proven to be feasible to raise awareness amongst target groups not yet involved with cardiac arrest.

[h2] Emergency medical services

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Hypothermia, Cardiac tamponade, Tension pneumothorax, Thrombosis, Toxic agents.
- Healthcare professionals should provide resuscitation at the scene rather than undertake ambulance transport with ongoing resuscitation, unless there is an appropriate indication to justify transport (bridging to in-hospital treatment).
- Consider mechanical CPR for transport with ongoing resuscitation.
- Consider obtaining invasive arterial blood pressure to guide resuscitation and post-resuscitation care already in the prehospital setting if it is feasible.
- EMS systems should use registry and data provided from equipment data (e. g. defibrillators) for debriefing and continuous quality improvement.

[h2] Inflight cardiac arrest and microgravity resuscitation

[h3] Inflight cardiac arrest

- Healthcare 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).
- Overhead-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 planned route leads over an area where no airport can be reached for a longer period of time 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) there is no medical need for flight diversion - follow airline policy.

[h3] Microgravity resuscitation

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Hypothermia, Cardiac tamponade, Tension pneumothorax, Thrombosis, Toxic agents.
- Perform standard resuscitation in a way that victim and rescuer are fixed.
- The crew member with the highest medical qualification on scene decides on termination of resuscitation.

[h2] Cruise ship

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Hypothermia, Cardiac tamponade, Tension pneumothorax, Thrombosis, Toxic agents.
- Use all healthcare resources immediately (personal, equipment).
- Activate HEMS if close to the coastline.
- Consider early telemedicine support.
- Have all equipment needed for ALS available on board.
- In case of insufficient number of health care professionals to treat CA, call for further medical staff via an on-board announcement.

[h1] Special Patient Groups

[h2] Asthma and chronic obstructive pulmonary disease

Insert Figure asthma algorithm 2025

- Address Hypoxia, Tension pneumothorax.
- Treat life threatening hypoxia with 100% oxygen.

- Check for evidence of (tension) pneumothorax.
- Provide endotracheal intubation (because of high inflation pressures).
- Consider manual decompression and disconnection from ventilator to manage dynamic hyperinflation.
- Consider E-CPR in accordance with local protocols if initial resuscitation efforts are unsuccessful.

[h2] Cardiac arrest in haemodialysis patients

Insert Figure special consideration in…

- Address Hypovolaemia, Electrolyte disorders, Hypothermia.
- Assign a trained dialysis nurse or technician to operate the dialysis machine.
- Stop dialysis and return the patient's blood volume with a fluid bolus.
- Disconnect from dialysis machine (unless defibrillation-proof) and beware of wet surfaces.
- Leave dialysis access open and use for drug administration.
- Dialysis may be required in the early post resuscitation period.

[h2] Resuscitation in obese patients

- Obese patients should receive standard resuscitation treatment – no deviation from standard BLS and ALS is needed.

[h2] Resuscitation in patients with pectus excavatum

- Address Hypoxia, Hypovolaemia, Electrolyte disorders, Hypothermia, Cardiac tamponade, Tension pneumothorax, Thrombosis, Toxic agents.
- Consider reduced chest compression depth of 3-4cm
- In the case of a Nuss bar correction, substantially increased force is required to deliver effective chest compressions
- Consider early implementation of ECPR if chest compressions are ineffective.
- Use anteroposterior pad placement for defibrillation using standard energies.

[h2] Cardiac arrest in pregnancy

Insert figure 1 Maternal ALS Algorithms….

- Address Hypovolaemia, Thrombosis.
- Provide Immediate manually displacement of the uterus laterally (left uterine

displacement) or tilting the patient leftward by at least 15–30 degrees to relieve aortocaval compression.

- Consider early perimortem Caesarean delivery (resuscitative hysterotomy) ideally within 5 minutes.

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[h1] The evidence informing the guidelines

[h1] Special Causes

[h2] Management and prevention of cardiac arrest due to anaphylaxis

There is not a universally accepted definition of anaphylaxis. It is an acute systemic hypersensitivity reaction, usually rapid in onset and may be fatal if not appropriately managed. Diagnosis of anaphylaxis is clinical.

The estimated incidence of anaphylaxis is 1.5-7.9 per 100.000 person per year in Europe.[1] The most common triggers of anaphylactic reactions are food, drugs and hymenoptera venom. [2-4] This guidance on anaphylaxis is based on the most recent ILCOR CoSTR[5], guidelines and updates from the World Allergy Organization Anaphylaxis Committee[6], European Academy of Allergy and Clinical Immunology,[7] North American Practice Parameter[8], Australasian Society of Clinical Immunology and Allergy,[9] the findings from the UK National Audit Project (NAP 7) of perioperative anaphylaxis.[10] Most of these recommendations are based on observational data, good practice statements and expert consensus.

[h3] Recognition of anaphylaxis

According to the World Allergy Organization Anaphylaxis Committee, anaphylaxis is likely when at least one of the following criteria is fulfilled[6]:

1. *Acute onset within minutes to several hours, with simultaneous involvement of the skin, mucosal tissue, or both (e.g. generalised hives, pruritus or flushing, swollen lips, tongue or uvula) and one of the following:*
   a. *Respiratory compromise (e.g., dyspnoea, wheeze/bronchospasm, stridor, reduced peak expiratory flow, hypoxemia)*
   b. *Reduced blood pressure or associated symptoms of end-organ dysfunction (e.g., hypotonia, syncope, incontinence)*
   c. *Severe gastrointestinal symptoms (e.g., severe crampy abdominal pain, repetitive vomiting), especially after exposure to non-food allergens*
2. *Acute onset of hypotension or bronchospasm or laryngeal involvement after exposure to a known or highly probable allergen for that patient (minutes to several hours), even in the absence of typical skin involvement.*

Anaphylaxis can occur without any skin involvement or with skin involvement remote to the site of exposure, and an anaphylactic reaction may initially present only with respiratory or cardiovascular involvement. During general anaesthesia or mechanical ventilation in emergency or intensive care settings, anaphylaxis may clinically manifest through suddenly

increased ventilation pressures and prolonged expiration, combined with a decrease in blood pressure, with or without skin and mucosal changes.

Early recognition of symptoms suggesting anaphylactic reaction, should elicit immediate reaction.

[h3] The initial approach if suspecting an anaphylactic reaction is:

Stop or remove the trigger if possible - stop or remove any potential or probable trigger. Do not delay treatment if removing the trigger is not feasible.

Ensure the patient is lying - do not sit or stand the patient up - fatal anaphylaxis has been associated with upright posture or patient sitting/ standing up.[11-13]. Position patients according to their symptoms and specifically consider:

- Supine position with raised legs, to improve venous return
- Sitting position with legs stretched if airway or breathing symptoms without circulation problem.

[h3] Give adrenaline

Administer IM adrenaline as first-line drug for the management of anaphylaxis. [14]

The preferred site is the lateral mid-thigh [15] standard dose for adults is 0.5 mg.[7] This should be repeated after 3-5 minutes, according to clinical response.[16] Auto-injectors or intranasal adrenaline applicators can be used for early self-administration or injection by a rescuer. If symptoms fail to resolve or the patient is in a monitored area e.g. intensive care unit (ICU), operating room (OR), emergency department (ED)), IV administration of adrenaline, starting at a dose of 0.1 micrograms kg^{-1} min^{-1} titrated according to response can be administered by appropriately trained personnel when patients are fully monitored.[17]

In a patient with upper airway obstruction or bronchospasm, consider adding nebulized adrenaline.[7, 18] However, systemic absorption of inhaled adrenaline is minimal and should not delay concurrent IM adrenaline administration.[19]

[h3] Secure vascular access - give intravenous fluids

Anaphylaxis may result in severe hypovolaemia because of peripheral vasodilation and increased vascular permeability (distributive shock).[20, 21]Fluid resuscitation is essential, in addition to the vasoconstrictive effect of adrenaline. Immediately after the first adrenaline dose, ensure adequate vascular access (e.g. wide bore IV catheter, intraosseous access).

Administer crystalloid solution in bolus doses of 10-20 mL kg^{-1} and repeat according to the patient's response. Large volumes of fluids might be required to restore haemodynamic stability.

[h3] Give oxygen

Provide 15 L min^{-1} 100% oxygen to every patient with an anaphylactic reaction and titrate to an oxygen saturation targeting 94-98%.[22]

[h3] Other drugs to support the circulation

In the case of no response to adrenaline, consider administration of a second vasoactive drug, such as noradrenaline or vasopressin.[7, 18]

[h3] Role of steroids and antihistamines in the management of anaphylaxis

H1- antihistamines antagonise the pro-inflammatory action of histamine, which can relieve the cutaneous symptoms of histamine release. However, their onset of action is slow, and they should not delay the administration of adrenaline.

Caution: Rapid IV administration of first generation H1-antihistamines might cause or exaggerate hypotension. [23]

Corticosteroid administration to prevent protracted symptoms or biphasic reactions has been based on low-certainty evidence.[24, 25] Recent studies indicate increased risk for negative outcomes (second dose of adrenaline, hospitalisation or ICU admission) in patients treated with corticosteroids in the pre-hospital setting. [26, 27]Based on experts' opinion, the ERC recommends not routinely using corticosteroids in the management of anaphylactic reactions.

[h3] Considerations for cardiac arrest in anaphylaxis

In a case-series of perioperative cardiac arrest, 94% of patients with anaphylactic cardiac arrest had initial pulseless electrical activity. [10]

Expert consensus is to follow the standard ALS protocol, including IV adrenaline administration and correction of relevant reversible causes, in particular hypovolaemia and hypoxia. Consider ECPR in cases of refractory cardiac arrest, specifically in hospital when this can be implemented.[5]

[h2] Hyper/hypokalaemia and other electrolyte disorders

Electrolyte abnormalities are a recognised cause of arrhythmias and cardiac arrest, but potassium disorders (hyperkalaemia and hypokalaemia) are most commonly seen in clinical practice and a U-shaped association between serum K^+ and mortality has been shown. [28]

Hyperkalaemia has also been subject to a recent ILCOR review (2025). [29]

[h3] Hyperkalaemia – non-cardiac arrest

Hyperkalaemia occurs in 1 – 10% of hospitalised patients.[30-32] In patients with chronic kidney disease (CKD), hyperkalaemia results in a significantly higher risk of in-hospital major adverse cardiovascular events and arrhythmias compared to patients without hyperkalaemia. [33] Dialysis patients are also more prone to severe hyperkalaemia than non-dialysis patients (45.8% vs 10.3%) during hospitalisation.[32] In-hospital mortality is significantly higher in patients with hyperkalaemia (18.1%) compared to those with hypokalaemia (5%) or normokalaemia (3.9%).[30] Similarly, in-hospital mortality is 3.9-fold higher in patients with a serum $K^+ > 6.5$ mmol L^{-1} when compared with normokalaemic patients.[34]

Renal and cardiac disease often co-exist and these patients are at high risk of hyperkalaemia exacerbated by drug therapy (e.g ACE-inhibitors, angiotensin II antagonists and mineralocorticoid receptor antagonists). However, down-titration or stopping these drugs in response to hyperkalaemia is associated with worse patient outcomes. [35-37]There is now a role for potassium binders (sodium zirconium cyclosilicate and patiromer) to provide cardio-renal protective therapy.[38, 39]

Hyperkalaemia is evident with a serum potassium (K^+) concentration greater than 5.5 mmol/l, although hyperkalaemia is a continuum, and its severity guides treatment.

Hyperkalaemia is classified as 'mild' (K^+ 5.5 – 5.9 5 mmol L^{-1}), 'moderate' (K^+ 6.0 – 6.4 5 mmol L^{-1}), 'severe' ($K^+ \geq 6.5$ 5 mmol L^{-1}), or 'extreme' ($K^+ \geq 9.0$ 5 mmol L^{-1}).

The risk of hyperkalaemia increases with multiple simultaneous risk factors (e.g. concomitant use of ACE-I and/or MRA in the presence of CKD).

[h3] Diagnosis of hyperkalaemia

Consider hyperkalaemia in all at risk patients with an arrhythmia or cardiac arrest (e.g. renal failure, heart failure, diabetes mellitus, chronic liver disease). Limb weakness, flaccid paralysis or paraesthesia may be indicators of severe hyperkalaemia.

Confirm hyperkalaemia using point-of-care testing, as formal laboratory samples will take time.[40-42]

ECG changes may reflect the severity and rate of rise of serum K^+,[43, 44] but it may be normal even in severe hyperkalaemia. When the diagnosis of hyperkalaemia can be established based on the ECG, treatment can be initiated before the laboratory result is available.[43]

These ECG signs of hyperkalaemia may develop progressively and include (Figure XIVa):

- Tall, peaked (tented) T waves (i.e. T wave larger than R wave in more than one lead);
- First degree heart block (prolonged PR interval > 0.2s);
- Flattened or absent P waves;

- Widened QRS (>0.12s);
- Sine wave;
- Ventricular tachycardia;
- Bradycardia;
- Cardiac arrest (PEA, VF/VT, asystole).

In patients with severe hyperkalaemia, arrhythmias or cardiac arrest have been shown to occur in 15% of patients within 6 hours of the presenting ECG.[43] Therefore, delays in treatment may have serious consequences.

[h3] Emergency treatment of hyperkalaemia

Insert Figure E3 (XII)

Treatment is guided by the severity of hyperkalaemia and the presence of ECG changes. Avoid delay in initiating potassium-lowering treatments (i.e. insulin-glucose, salbutamol and sodium zirconium cyclosilicate). Follow a systematic approach as outlined in the hyperkalaemia treatment algorithm (Figure XI). The treatment of mild hyperkalaemia is not within the scope of this guideline. Systematic reviews of the pharmacological treatment of hyperkalaemia have been conducted both by Cochrane and ILCOR. [29, 45, 46]

[H4] Insulin and glucose

This is the most effective and reliable K^+-lowering therapy which works by shifting K^+ into cells. The conventional dose of soluble insulin (10 units) has been shown to reduce serum K^+ by 0.7 – 1.4 mmol L^{-1}.[29, 47] Studies have suggested a possible dose-dependent effect of insulin [48-52] and a potential correlation between severity of hyperkalaemia and degree of K^+-lowering with 10 units insulin.[53] These findings are particularly relevant in the resuscitation setting. Hypoglycaemia remains a major iatrogenic risk with a reported incidence of up to 28%.[50, 54-59] Reducing the dose of insulin (5 units) did not significantly lower the risk of hypoglycaemia (blood glucose < 4 mmol L^{-1}). [48, 50, 57, 58]A scoping review (n= > 15,000) found that the most consistent risk factor for hypoglycaemia is a low pre-treatment blood glucose value.[60] A threshold of < 7 mmol L^{-1}blood glucose has been consistently identifies patients at increased risk of iatrogenic hypoglycaemia.[52, 61-64] In one study, administration of 50 g glucose over 4 hours resulted in hypoglycaemia in 6.1 % of patients. [56] Based on these collective findings, a modified protocol which is recommended in the ERC algorithm (figureXIII) has been

developed in order to reduce iatrogenic hypoglycaemia,[47] based on evidence that this strategy is effective.[65]

[H4] Salbutamol

Salbutamol is a beta-2 adrenoceptor agonist and promotes the intracellular shift of K^+. A meta-analysis including seven studies administering 10-20mg inhaled salbutamol demonstrated a reduction in serum K^+ by 0.9 mmol L^{-1} within 120 minutes.[29] Early studies suggested that salbutamol may be less effective in patients receiving non-selective beta-blockers and in up to 40% of patients with end-stage kidney disease.[66, 67] On this basis, monotherapy has not been advised. Studies combining insulin-glucose and beta2-agonists were more effective than either treatment alone.[66, 67] The ILCOR review noted a K^+-reduction of 1.2 mmol L^{-1} with combined therapy and supported use of both drugs.[29] Thus the ERC recommends simultaneous administration of insulin-glucose and beta2-agonists for reducing potassium values. Insulin-glucose and salbutamol are effective for 4-6 hours, after which rebound hyperkalaemia may occur.

[H4] Intravenous calcium salts (calcium chloride or gluconate)

Insert Figure E4 (XI)

Intravenous calcium has been used for decades for the treatment of hyperkalaemia, particularly in the presence of ECG changes, despite lacking clinical evidence. A recent randomised trial suggested harm from the routine use of IV calcium during OHCA, however patients with hyperkalaemia were excluded.[68] In resuscitation practice, the use of IV calcium has been restricted to the treatment of electrolyte disorders (i.e. hyperkalaemia, hypocalcaemia, hypermagnesaemia) and calcium channel blocker overdose. In clinical practice, the improvement in ECG changes after IV calcium may be seen even before K^+-lowering drugs have taken effect (Figure XIVb).

The rationale for use of IV calcium in hyperkalaemia and hypermagnesaemia is '*antagonism*' of its effect on cardiac and skeletal muscle (i.e. membrane stabilisation), but efficacy may be influenced by the dose and rate of administration. An alternative mechanism has been proposed in an animal study where IV calcium was shown to restore '*conduction*' resulting in improvement of hyperkalaemia-induced ECG changes.[69] In pre-eclampsia, IV calcium ameliorates limb weakness, respiratory depression and cardiac effects induced by iatrogenic hypermagnesaemia.[70]

The recent ILCOR systematic review included only one non-cardiac arrest study of IV calcium in adults and found no evidence to support a clinical effect of IV calcium in hyperkalaemia, although considered the study to have a critical risk of bias.[29]

The Cochrane reviews included no studies evaluating the effect of IV calcium, but suggested withholding IV calcium in patients with severe hyperkalaemia or ECG changes.[45] There is currently insufficient evidence for or against the use of IV calcium in the treatment of hyperkalaemia, therefore the ERC continues to recommend the administration of IV calcium to patients most at risk of arrhythmias (i.e. severe hyperkalaemia with ECG changes present).

[H4] Sodium bicarbonate

The ILCOR review included five studies in adults using variable doses of bicarbonate (50-390 mmol) and found a K^+-lowering of only 0.1 mmol L^{-1} within 60 minutes.[29] The Cochrane review also concluded that there is no evidence to support the use of sodium bicarbonate in hyperkalaemia.[45, 46] Therefore, the ERC recommends against routine use of sodium bicarbonate in treatment of hyperkalaemia in non-cardiac arrest cases.

[h3] Indications for dialysis

Dialysis is the most definitive treatment for hyperkalaemia, and the main indications with hyperkalaemia are:

- Severe life-threatening hyperkalaemia with or without ECG changes or arrhythmia;
- Hyperkalaemia resistant to medical treatment;
- End-stage kidney disease;
- Oliguric acute kidney injury (urine output < 400 mL/day);
- Marked tissue breakdown (e.g. rhabdomyolysis).

Following dialysis, rebound hyperkalaemia may also occur.

[h3] Pre-hospital setting and hyperkalaemia

The management and identification of patients with hyperkalaemia in the pre-hospital setting can be challenging, because of the limited diagnostic options.

[h3] Resuscitation of hyperkalaemic cardiac arrest

Insert Figure E5 (XIV)

Hyperkalaemia is the most common metabolic disturbance associated with cardiac arrest and is potentially reversible, with a reported IHCA incidence of 1% [71] to 12% [72], with PEA as the commonest initial arrest rhythm.[73] The ILCOR review included only one study of hyperkalaemia in cardiac arrest (high risk of bias due to resuscitation time). This review describes decreased rates of ROSC with calcium, sodium bicarbonate or both.[29]

A study of hyperkalaemic IHCA (n = 109; mean serum K^+ 7.8 mmol L^{-1}) used IV calcium and sodium bicarbonate [72] in a very heterogeneous population and achieved 36.7% ROSC, but only 12.8% survived > 24 hours and 3.7% survived to hospital discharge. Given the poor outcome in patients with extreme hyperkalaemia, the authors proposed that dialysis may be an option.

Dialysis is the definitive treatment for hyperkalaemia, but is rarely used in cardiac arrest. A review of case reports of dialysis during CPR demonstrate successful outcomes in patients with a serum K^+ ranging between 8.2 – 10.2 mmol L^{-1}.[47] All dialysis modalities (with and without ECMO) have been used during CPR, demonstrating that it is technically feasible.[74-77] The mean duration of dialysis to achieve ROSC was 45.4 minutes (range 15-95 minutes) and the mean reduction in serum K^+ was 3.2 mmol L^{-1}which would be difficult to achieve with drugs alone, particularly during cardiac arrest.[47] The severity of hyperkalaemia appears to be a good indicator of the likelihood of achieving and sustaining ROSC. A protocol for initiation of dialysis during CPR has been reported.[47]

A study assessing the outcome of OHCA following anti-hyperkalaemic medical treatment (n=465; mean K^+ 6.9 mmol L^{-1}, IQR 5.7-8.4 mmol L^{-1}) found no effect on ROSC, despite achieving K^+-lowering (mean K^+ 5.4 mmol L^{-1}, IQR 4.4-6.8 mmol L^{-1}).[78] There is currently insufficient evidence for or against the use of IV calcium in the treatment of hyperkalaemia, therefore the ERC continues to recommend the administration of IV calcium to patients hyperkalaemic cardiac arrest in all settings.

[h2] Hypokalaemia

Hypokalaemia is defined as a serum K^+ < 3.5 mmol L^{-1} (mild (K^+ 3.0 – 3.4 mmol L^{-1}), moderate (K^+ 2.5 – 2.9 mmol L^{-1}) or severe (K^+ < 2.5 mmol L^{-1}or symptomatic). [79] It is usually caused by excessive K^+ loss, transcellular shift of K^+ into cells, or reduced K^+ intake.[80] Hypokalaemia is associated with higher in-hospital mortality and an increased risk of ventricular arrhythmias. Risks are increased in patients with pre-existing heart disease,[81, 82] those treated with digoxin[83] or those undergoing primary percutaneous coronary intervention for ST-elevation

myocardial infarction.[84] In acute heart failure, hypokalaemia may be associated with increased short and long-term all-cause mortality after hospital discharge.[85]

[h3] Treatment of hypokalaemia

Treatment is guided by the severity of hypokalaemia and presence of symptoms and/or ECG abnormalities, as illustrated in Figure XIII. Slow replacement of potassium is preferable, but in an emergency, rapid IV replacement, preferable via central route is required. The target level is 4 mmol $L^{-1}K^{+}$.[86] Correction of any concomitant hypomagnesaemia is also essential. Seek expert advice for potassium replacement in patients with severe renal impairment. Concomitant magnesium deficiency is common in patients with hypokalaemia. Repletion of magnesium will facilitate more rapid correction of hypokalaemia.[87]

[h2] Calcium and magnesium disorders

The recognition and treatment of calcium and magnesium disorders is summarised in Table 2.

Table 2: Calcium and magnesium disorders with associated clinical presentation, ECG manifestations and recommended treatment.

Disorder	Causes	Presentation	ECG	Treatment
Hypercalcaemia Calcium > 2.6 mmol L^{-1}	Primary or tertiary hyperparathyroidism Malignancy Sarcoidosis Drugs	Confusion Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Short QT interval Prolonged QRS interval Flat T waves AV block Cardiac arrest	Guided by underlying cause Fluid replacement IV Furosemide 1mg kg^{-1} IV Hydrocortisone 200-300mg IV Pamidronate 30-90mg IV
Hypocalcaemia Calcium < 2.1 mmol L^{-1}	Chronic kidney disease Acute pancreatitis Calcium channel blocker overdose Toxic shock syndrome Rhabdomyolysis Tumour lysis syndrome Massive blood transfusion	Paraesthesia Tetany Seizures AV-block Cardiac arrest	Prolonged QT interval T wave inversion Heart block Cardiac arrest	Bolus: 10-20 mL 10% Calcium gluconate over 5-10 min Infusion: 100 mL 10% Calcium gluconate in 1000 mL 0.9% Saline or 5% Glucose at 50 mL h^{-1} IVI (monitor Ca^{2+} level and adjust rate) 50% Magnesium sulphate 4-8 mmol IV (if necessary)
Hypermagnesaemia Magnesium > 1.1 mmol L^{-1}	Renal failure Iatrogenic	Confusion Flaccid paralysis Respiratory depression Hypotension AV-block Cardiac arrest	Prolonged PR and QT intervals T wave peaking AV block Cardiac arrest	Consider treatment when magnesium > 1.75 mmol L^{-1}: 10% Calcium gluconate (10-30mL) or 10% Calcium chloride (5-10 mL) IV repeated if necessary

				Saline diuresis – 150 mL 0.9% saline/ hr IV and Furosemide 1mg/kg IV Haemodialysis – when renal function impaired or severe symptoms, but risk of causing hypocalcaemia. Ventilatory support if necessary
Hypomagnesaemia Magnesium < 0.6 mmol L^{-1}	GI loss Polyuria Starvation Alcoholism Malabsorption	Tremor Ataxia Nystagmus Seizures Arrhythmias – torsade de pointes Cardiac arrest	Prolonged PR and QT intervals ST-segment depression T-wave inversion Flattened P waves Increased QRS duration Torsades de pointes	Severe or symptomatic: 50% magnesium sulphate 2g (8 mmol) IV over 15 min Torsades de pointes: 50% magnesium sulphate 2g (8 mmol) IV over 1-2 min Seizures: 50% magnesium sulphate 2g (8 mmol) IV over 10 min

[h2] Hyperthermia and malignant hyperthermia

Hyperthermia results from a body temperature above normal (core temperature 36.5-37.5°C) because of failed thermoregulation. Extremes of age and multimorbidity are specific risk factors.[88, 89]

Malignant hyperthermia is a rare pharmacogenetic disorder of skeletal muscle calcium homeostasis characterised by muscle contracture and life-threatening hypermetabolic crisis following exposure of genetically predisposed individuals to halogenated anaesthetics,

succinylcholine, or neuroleptics.[90, 91] Rarely, malignant hyperthermia can be triggered non-pharmacologically. The most common clinical signs and symptoms include hypercapnia (34%), sinus tachycardia (25%), hyperthermia (20%), masseter spasm (11%), generalised muscle rigidity (3%), acidosis, hyperkalaemia and death if left untreated.[92] This section is based on two systematic reviews, five non-systematic reviews, and two scoping reviews the latter being most recently performed on June 15 2024.[90-97]

[h3] Hyperthermia

Environmental-associated hyperthermia (core temperature >38°C) can be avoided by acclimatisation, adequate hydration and avoidance of physical activity in hot weather.[88, 94, 98] The major risk factor is dehydration, which risks progression to heat syncope, heat exhaustion, heat stroke, and finally multiple organ dysfunction and cardiac arrest (Table 3). Core temperature should be measured centrally (e.g. tympanic, rectal, oesophageal) to guide treatment.[99]

Table 3

Degree of hyperthermia	Symptoms	Treatment
Mild - Heat syncope	Transient loss of consciousness and fast return to normal neurologic baseline.	Remove patient to a cool environment, passive cooling, resting and administration of oral isotonic or hypertonic fluids (the latter only if Na^+ ≤130 mmol L^{-1}).
Moderate - Heat exhaustion	Intense thirst, weakness, discomfort, anxiety, dizziness, syncope. Caused by mild to moderate hyperthermia (>40°C) due to exposure to high environmental heat or excessive exercise.	Additionally, lie patient flat and administer isotonic IV fluids. Simple external cooling measures are usually not required but may involve conductive (e.g. cold floor, ice sheets; commercial ice packs to hands, feet and cheeks), convective (cold water immersion, cold shower) and evaporative measures (spraying cold water, fanning bare skin).
Severe - Heat stroke	Triad of severe hyperthermia (core temperature >40°C), neurological symptoms and	Rapidly cool the patient to <39°C, preferably <38.5-38.0°C as quickly as possible. [94]. Prioritise active cooling methods over passive cooling - a cooling rate

	recent passive environmental exposure (classic or passive heat stroke) or excessive exercise (exertional heat stroke). Symptoms include central nervous system dysregulation (e.g. altered mental state, seizure, coma), tachycardia, tachypnoea and arterial hypotension. [88]Mortality is approximately 10%, and when combined with hypotension approaches 33%.[94] The outcome worsens if the core temperature is sustained at >40.5°C.	≥0.16°C min^{-1} is recommended (Figure X) [100]. It is important to account for the time between symptom onset and the provision of care when selecting a cooling method, reaching the target temperature within 30 min of onset of heat stroke should be the goal [100]. For exertional heatstroke, a cooling rate faster than 0.10°C min^{-1} is safe and desirable. Ice or cold-water immersion (from neck down) or full body conductive cooling should be used, cooling rates of 0.2-0.35°C min^{-1} can be achieved. [94, 101]. Cold water immersion should be continued until the symptoms have resolved or for a reasonable amount of time, e.g. 15 min, because benefit outweighs risk (weak recommendation, very low certainty evidence). [102] if cold water immersion is not available, a combination of simple cooling techniques may be used, including conductive, convective and evaporative measures, although there are no comparative studies to guide the best option (Figure X). [102] A systematic review concluded that water immersion (1-26°C water) lowers body temperature faster and more effectively compared to passive cooling (low to very low certainty of evidence). Misting and fanning cooling techniques are marginally faster than passive cooling, and cold showers (20.8°C) cool faster than passive cooling. [102] IV isotonic or hypertonic fluids should be administered at room temperature (if Na^+ ≤130 mmol L^{-1}, for example up to 3 x 100 mL 3% NaCl at 10 minute intervals). [103] Additional electrolyte replacement with isotonic fluids

		should be considered and substantial amounts of fluids may be required. Follow the ABCDE approach in any patient with deteriorating vital signs. Critically ill patients will require aggressive and extended treatment in an intensive care unit.[88, 104] There may be a requirement for advanced cooling techniques including external or internal devices used for targeted temperature management. There are no specific drugs lowering core temperature.

Insert Figure 1 here (revised hyperthermia algorithm)

Insert NEW Figure X near here. New Figure X legend. Ability to reach target final core temperature (°C) after 30 min based on the cooling modality and starting temperature. Green reflects a final core temperature <39°C (preferred), yellow reflects a final core temperature 39-40°C (borderline), and red reflects a final core temperature >40°C (inadequate). [100]

[h3] Malignant hyperthermia

Insert Figure 2 here (MH algorithm)

Prevention of malignant hyperthermia is key; it is a genetic illness that if untreated results in death.[93, 105, 106] Drugs such as 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines may also cause a condition similar to MH and the use of dantrolene may also be therapeutic in these cases.[107] Always consider malignant hyperthermia, with unexplained, unexpected increases in $ETCO_2$, heart rate or temperature. [90]Follow guidance according to Figure 2. [90, 108]

Briefly, stop exposure, provide high flow oxygen, give 2,5 mg/kg dantrolene IV until $ETCO_2$ $ETCO_2$ <45 mmHg (6 kPa) with normal minute ventilation and core temperature <38.5°C, cool the patient. If cardiac arrest occurs, follow the universal ALS algorithm and continue to cool the patient. Apply the same cooling techniques as for post-resuscitation care targeted temperature management.

It is essential to contact an expert malignant hyperthermia centre for ongoing advice.[93] [109] Dantrolene should be stored centrally where anaesthesia is provided, and algorithms for MH management should be readily available.

[h3] Toxin-Induced hyperthermia

Table 4

Category	Substance	Mechanism	Symptoms	Diagnostic testing	Specific Management
Antipsychotics [110-114]	Risperidone, Arapiprazole, Haloperidol, Olanzapine, Quetiapine, Clozapine, Blonanserine	Dopamine antagonism /acute withdrawal of dopamine agonists →neuroleptic malignant syndrome (NMS)	Hyperthermia, muscle rigidity, tremor, autonomic dysfunction, altered mental status	Blood testing for antipsychotics	Benzodiazepines against agitation. Bromocriptine for hypodopaminergic state. Dantrolene as muscle relaxant
Antidepressants [115-119]	Lithium, MAOI, SSRI, SNRI, TCA	Serotonin syndrome	Hyperthermia, flushing, shivering, akathisia, agitation, mydriasis, autonomic dysfunction	Blood testing for antidepressants, serum lithium level	Benzodiazepines against agitation. Consider Chloropromazine. Consider sodium bicarbonate for TCA
Recreative/Party Drugs [120-134]	MDMA (Ecstasy), LSD	Central catecholamine release and reuptake inhibition in the CNS, hypermetabolic condition with skeletal muscle stimulation	Hyperthermia, euphoria, hallucinations, agitation, shivering, mydriasis, nausea	Urine screening (MDMA, LSD)	Benzodiazepines for agitation. Dantrolene for hyperthermia. Consider carvedilol for MDMA
	Methamphetamine		Hyperthermia, hallucinations, tremor,	Urine or blood screening for amphetamines	Ammonium chloride for excretion (urine acidifier).

		, tachycardia and vasoconstriction	agitation, mydriasis Autonomic dysfunction		Activated charcoal to reduce absorption
	Cocaine	Nonselective dopamine, serotonin, noradrenaline reuptake inhibitor. CNS stimulation, sympathetic system activation. Direct sodium channel blockade. Coronary vasoconstriction	Hyperthermia, hallucinations, tremors, agitation, mydriasis, autonomic dysfunction, arrythmias, chest pain	Urine and blood screening	Consider sodium bicarbonate for broad complex tachycardia or cardiac arrest, avoid β-blockers
Anticholinergics [135-139]	Atropine, scopolamine, plant alkaloids (belladonna, brugmansia, amanita)	Blockade of muscarinic receptors. Anticholinergic hypertermic syndrome	Hyperthermia. tachycardia, sweating Inhibiton, dry skin & mucus membranes, flushing, mydriasis, altered mental status	Urine and serum screening	Benzodiazepines and central acting cholinesterase inhibitors against agitation. Activated charcoal within1st hour (time window may be extended as anticholinergics reduce gastrointestinal motility)
Sympathomimetics [140-142]	Ephedrine, pseudoephedrine	Sympathetic system activation Increased metabolic rate and peripheral	Hyperthermia, tachycardia, hypertension, arrythmias, muscle	Urine screening, serum toxicology testing for pseudoephedrine/ ephedrine	Benzodiazepines against agitation

		vasoconstriction	twitching, nausea, mydriasis, urinary retention		
Salicylates [143, 144]	Aspirin, methyl salicylate	Uncoupling of oxidative phosphorylation leading to increased heat production. Uncoupling of oxidative phosphorylation leading to increased heat production	Mild hyperthermia, nausea, tachypnea, confusion, tinnitus	Serum salicylate levels	Urine alkalization with $NaHCO_3$. Prevent hypoglycemia. Consider dialysis for drug excretion and cooling
Miscellaneous [145, 146]	Dinitrophenol (DNP)	Uncoupling of oxidative phosphorylation leading to increased heat production	Hyperthermia, sweating, flushing, agitation, tachycardia & tachypnea, fatigue	Serum toxicology testing for DNP	Consider dantrolene

Intoxication may present with hyperthermia as an early sign. Risk is higher in people taking psychotropic drugs (for treatment or recreation), sympathomimetics, anticholinergics, salicylates, weight-loss drugs or following ingestion of wild mushrooms.

Carry out an initial ABCDE approach after applying appropriate personal protective equipment (PPE) as in all cases of unknown toxic agents. Specific symptoms and the patient's history may help to identify the suspected substance, even before toxicology results are available. For most poison-induced hyperthermia cases, prompt symptomatic treatment is key. Active cooling and targeted strategies for specific symptoms: benzodiazepines to reduce agitation and tremor, dantrolene for muscle contraction and rigidity, and measures to reduce toxin concentrations.

[h2] Accidental hypothermia and avalanche rescue

Accidental hypothermia is defined as a decrease in core temperature <35°C, during which vital signs and consciousness fade and finally disappear.[147] However, vital signs may be minimal but still be present with temperatures <24°C.

In hypothermic patients with spontaneous circulation, key interventions are insulation, and hospital triage followed by transfer and rewarming. In hypothermic patients with cardiac arrest, key interventions are continuous CPR and extra corporeal life support rewarming. These measures may result in good neurological outcome even with prolonged no-flow or low-flow (i.e. CPR) times, if hypothermia (e.g. <30°C) ensued before cardiac arrest. A scoping review up to February 13th 2025 found three systematic reviews for this recommendation,[148-150] but also included other relevant articles.[147, 151-158]

[h3] Prevention from cardiac arrest

Primary accidental hypothermia is induced by exposure to cold, while secondary accidental hypothermia is triggered by illness and other external causes. Primary hypothermia is common in outdoor activities (mainly athletes and lost persons) and urban environments (e.g. homeless and intoxicated persons), while the incidence of secondary hypothermia is increasing among old and multimorbid persons in the indoor environment.[151, 154]

Measuring the core temperature with a low reading thermometer is the gold standard for diagnosis of hypothermia[154]: (Table Hypo 1)

- tympanic in spontaneously breathing patients and
- oesophageal in patients have a tracheal tube or supraglottic airway in situ. [156]

If the core temperature cannot be measured, temperature should be assessed according to the level of consciousness with the revised Swiss Staging System.[153] (Figure Hypo 2).

Insert Figure hypo 2

Hypothermic patients should be protected from the cold environment through minimal exposure and insulation and be transferred as fast as possible to the next appropriate hospital for rewarming. While not harmful, active rewarming is impractical during brief transport periods (e.g., less than 1 hour).[150] Hypothermic patients with prehospital cardiac instability (i.e. heart rate <45/min, systolic blood pressure <90 mmHg, ventricular arrhythmia, core temperature <30°C) should be rewarmed in-hospital using minimally invasive techniques.[153, 158] Insert Table 5

Table 5.

Stage	Clinical Findings	Core temperature (°C) (if available)
Hypothermia I (mild)	Conscious*	35-32°C
Hypothermia II (moderate)	Impaired consciousness*	<32-28°C
Hypothermia III (severe)	Unconscious*; vital signs present	<28°C
Hypothermia IV (severe)	Apparent death; Vital signs absent	Variable**

[h3] Management of cardiac arrest

Insert Figure Hypo1

The lowest published temperature from which successful resuscitation and rewarming has been achieved is currently 11.8°C[152] for accidental hypothermia and 4.0°C for induced hypothermia.[159] A systematic review reported only five patients (28-75 years of age) who had arrested at a body core temperature >28°C, suggesting that cardiac arrest caused by primary hypothermia at ≥28°C is possible, but unlikely.[148] Some may still have minimal vital signs at a core temperature <24°C.[160]

Check for signs of life for one minute - not only by clinical examination but also using ECG and ultrasound. (REFER TO ERC 2025 ALS SECTION)

In hypothermic cardiac arrest, outcome relevant information should be collected to estimate the survival probability from hypothermic cardiac arrest with the HOPE (Hypothermia Outcome Prediction after ECLS rewarming for hypothermic arrested patients) -score [155, 157, 161]

The prognosis of patients with primary hypothermic cardiac arrest can be very good,[154] while in cases of secondary hypothermia outcome is more influenced by co-morbidity.[162]

Hypothermic patients in witnessed and unwitnessed cardiac arrest have good chances of neurological recovery if hypothermia developed before hypoxia and cardiac arrest, and if the chain of survival is functioning well.[148, 149, 162] Hypothermia diminishes the oxygen demand of the body (6-7% per 1°C cooling) and thereby protects the most oxygen dependent organs of the body (brain and heart) against hypoxic damage.[163] A systematic review of witnessed hypothermic cardiac arrest patients (n=214) reported a survival to hospital-discharge rate of

73%, with 89% of survivors having a favourable neurologic outcome. A systematic review of unwitnessed hypothermic cardiac arrest patients (n=221) reported a survival rate of 27%, with 83% of survivors having a favourable neurological outcome. Of note, the first rhythm was asystole in 48% of these survivors.[149]

Hypothermic cardiac arrest patients should receive continuous CPR until circulation has been re-established. Chest compression and ventilation rate should follow the standard ALS algorithm as for normothermic patients.

Hypothermic cardiac arrest is often refractory to defibrillation and adrenaline. Defibrillation attempts have been successful in patients with a core temperature >24°C, however, ROSC tends to be unstable with lower temperature.[164] The hypothermic heart may be unresponsive to cardioactive drugs, attempted electrical pacing and defibrillation. If ventricular fibrillation or pulseless ventricular tachycardia persist after three shocks, it is reasonable to delay further attempts until the core temperature is >30°C.

Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drug given. The evidence for the efficacy of drugs in severe hypothermia is limited and based mainly on animal studies. In severe hypothermic cardiac arrest, the effectiveness of amiodarone is reduced.[165] Adrenaline may be effective in increasing coronary perfusion pressure, but not survival.[166] [167] Adrenaline may induce myocardial injury and impaired neurologic recovery; it is reasonable to withhold adrenaline and other CPR drugs until the patient has been warmed to a core temperature ≥30°C. If access to extracorporeal life support is delayed, since shorter CPR duration might be associated with better outcome, it may be reasonable to administer 1 mg of adrenaline in an attempt to achieve ROSC.[168] Once 30°C has been reached, the intervals between drug doses should be doubled when compared with those during normothermia (i.e. adrenaline every 6–10 min). Once normothermia is achieved (≥35°C), use standard drug protocols.

If possible, transfer arrested hypothermic patients (or those at risk of arrest), directly to a hospital able to provide extracorporeal life support. If prolonged transport is required or the terrain is difficult, mechanical CPR is suggested. In hypothermic arrested patients with a body temperature <28°C delayed CPR may be used when CPR is too dangerous, and intermittent CPR can be used when continuous CPR is not possible, for example because of technically difficult rescue (Figure 3).[169]

Establish extracorporeal life support only in cardiac arrest or deteriorating patients (e.g. decreasing blood pressure, increasing acidosis). Primary extracorporeal life support

rewarming may be considered in patients with ETCO2 <10mmHg or a systolic blood pressure ≤60mmHg.[170] An unwitnessed cardiac arrest with asystole as first rhythm is not a contraindication for extracorporeal life support rewarming.[149] Rewarming should preferably be performed with VA-ECMO rather than cardiopulmonary bypass.[171, 172] If extracorporeal life support is not available within 6 hours, non-extracorporeal life support rewarming may be used.[173, 174]

Insert Figure Hypo 3 near here

In-hospital prognostication of successful rewarming should be based on the HOPE score (Table Hypo 2).[155, 157] The 5A score is a screening tool for predicting in-hospital mortality among elderly patients with accidental hypothermia with or without cardiac arrest which may also guide treatment options,[175, 176] In hypothermic arrested patients, criteria for extra corporal life support rewarming should not be based on guidelines for normothermic cardiac arrest (Figure 4). This may result in potentially life-saving treatment being withheld in patients with a potential for good neurological outcome.[172]

Insert Figure Hypo 4 here

[177]

Emergency medical services and hospitals should install structured protocols to improve prehospital triage, transport and treatment, as well as in-hospital management of hypothermic patients.

[h3] Avalanche rescue

The chances of surviving avalanche burial is steadily improving, due to collaborative efforts to improve avalanche search and rescue, and subsequent medical interventions.[178] Most avalanche victims die from asphyxia, fewer from trauma or hypothermia. In cases of an unwitnessed cardiac arrest presenting in asystole, avalanche victims have a poor chance of survival, even if guidelines are followed.[179-181]

Several factors improve the likelihood of good outcome from avalanche burial. These include non-critical burial (i.e. head and chest out of the snow), superficial burial, burial for a short time, burial during daylight hours, the presence of an air pocket is present (i.e. open airways in addition to any space in the snow in front of nose and mouth), retrieval before cardiac arrest, witnessed cardiac arrest and ROSC in the first few minutes of CPR.[178-180, 182, 183] The International Commission for Mountain Emergency Medicine performed a scoping review

and published guidelines for the management of victims buried in avalanches (Figures AVA 1 and 2).[184]

Insert Figures AVA 1 and 2 near here

The AvaLife algorithm should be used in multiple burial scenarios with too few rescuers on site. In this triage situation, AvaLife enables dentification of the i) buried subjects who should be excavated first (cut-off <1.5m of burial depth) and ii) who should be resuscitated and for how long.[185] AvaLife has been specifically developed for BLS providers.

The quality of CPR can be compromised by several factors, including the confined space of the burial site, chest compressions on snow, hypobaric hypoxia on mountains leading to faster exhaustion of rescuers, and long and difficult extrication and transport of victims. Mechanical chest compression devices can be helpful in technically difficult and prolonged rescues.[186-188]

[h2] Thrombosis

[h2] Pulmonary embolism

Cardiac arrest from acute pulmonary embolism (PE) is the most serious clinical presentation of venous thromboembolism, in most cases originating from a deep venous thrombosis (DVT).[189] The reported incidence of cardiac arrest caused by PE is 2–7% of all OHCAs,[190, 191] and 5–6% of all in-hospital cardiac arrests,[71, 192] but it is likely to be underestimated. Overall survival is low, even if invasive treatment or ECPR is used.[191, 193, 194] Specific treatments for cardiac arrest resulting from PE include administration of fibrinolytics, surgical embolectomy and percutaneous mechanical thrombectomy.

The 2020 ILCOR systematic review explored the influence of specific treatments (e.g. fibrinolytics, or any other) yielding favourable outcomes.[195] The 2019 ILCOR summary statement reviewed the use ECPR for cardiac arrest in adults,[196] while the 2022 ILCOR summary statement updated evidence on sensitivity and specificity of POCUS for specific pathophysiological states, including PE, and reviewed four small additional observational studies on cardiac arrest from PE which were published since the previous review. No additional evidence was found to change the management.[197] Additional evidence was identified from the European Society of Cardiology (ESC) guideline on pulmonary embolism.[189]

These ESC Guidelines define 'confirmed pulmonary embolism' as a probability of pulmonary embolism high enough to indicate the need for specific treatment.[189] Clinical history, assessment, capnography, and echocardiography (if available) can assist in the diagnosis of acute pulmonary embolism during CPR. Cardiac arrest commonly presents as PEA. [193] Constant low ETCO2 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. [198, 199] If a 12-lead ECG can be obtained before cardiac arrest, changes indicative of right ventricular strain may be helpful for decision making.

Common symptoms preceding cardiac arrest are described in Table 6

Acute pulmonary embolism can cause right ventricle pressure overload and dysfunction, POCUS might be helpful for detection, (Table 6). but no individual echocardiographic parameter provides fast and reliable information on right ventricle size or function. Signs of right ventricular overload or dysfunction may also be caused by other cardiac or pulmonary disease.[200]

Table 6.

Non-specific patient characteristics observed with higher incidence in OHCA caused by pulmonary embolism compared with other causes.

Symptoms preceding cardiac arrest
Sudden onset of dyspnoea
Pleuritic or substernal chest pain
Cough
Haemoptysis
Syncope (otherwise unexplained and/or repeated collapses)
Signs of DVT (unilateral low extremity swelling)
Signs of right ventricular strain on 12-lead ECG (if obtained before cardiac arrest) • Inversion of T waves in leads V1–V4 • QR pattern in V1 • S1 Q3 T3 pattern (i.e. a prominent S wave in lead I, a Q wave and inverted T wave in lead III) • Incomplete or complete right bundle-branch block
Assessment during resuscitation
EMS witnessed cardiac arrest

First observed rhythm PEA
Low ETCO2 readings during CPR (<1,7 kPa/13 mmHg)
Presence of right heart dilation with poor filling of the left heart, D sign (straightening of interventricular septum) and/or intracardiac thrombi (intra-arrest POCUS)

[h3] Initial treatment

All patients with sudden onset of progressive dyspnoea, especially in patients without pre-existing cardiac or pulmonary disease are suspicious for pulmonary embolism. Hypoxaemia is usually reversed with administration of oxygen, while in some patients, correction of hypoxaemia will not be possible without simultaneous pulmonary reperfusion.[189]

High-risk pulmonary embolism is suspected with shock or persistent arterial hypotension and is immediately life-threatening. Leave hypotensive patients lying flat if breathing allows, to prevent further progression of hypotension and cardiac arrest. Acute right ventricular failure is the leading cause of death in patients with high-risk pulmonary embolism. Aggressive volume expansion is of no benefit and may even worsen right ventricular function. However, cautious volume loading (≤ 500 mL over 15–30 min) may increase cardiac output, and the use of vasopressors and/or inotropes is frequently beneficial.[189]

IV anticoagulation should be initiated while awaiting the results of diagnostic tests. Unfractionated heparin is recommended for patients with shock and hypotension, and in whom primary reperfusion is considered. Thrombolytic treatment of acute pulmonary embolism restores pulmonary perfusion more rapidly than anticoagulation with heparin alone.[201]

[h3] Modifications to ALS

Fibrinolysis

If pulmonary embolism is the suspected cause of cardiac arrest, fibrinolytic drugs should be administered, based on evidence from an ILCOR CoSTR.[197] There is insufficient evidence to recommend optimal drug and dosing strategy for fibrinolysis during CPR. ROSC and survival were observed after recombinant tissue type plasminogen activator (alteplase, bolus 50 mg IV with or without additional 50 mg after 30 min, or 0.6–1.0 mg kg^{-1} IV - max. 100 mg).[193, 202, 203] When thrombolytic drugs have been administered, continue CPR for at least 60–90 min.[191, 204, 205] Based on expert opinion, thrombolysis or surgical embolectomy should be

considered for pregnant women with high-risk pulmonary embolism[189] and PE causing cardiac arrest.

Surgical embolectomy or percutaneous mechanical thrombectomy

Successful surgical embolectomy and percutaneous mechanical thrombectomy have been reported in cardiac arrest patients and are recommended if PE is the known cause of cardiac arrest, and the specialised skills are available.[206-208] Treatment decisions should be made by a highly experienced interdisciplinary team, involving a thoracic surgeon or interventional cardiologist.[189, 209]

Extracorporeal CPR

Some observational studies suggest the use of ECPR if cardiac arrest is associated with pulmonary embolism[194, 210] ECPR maintains circulation and gas exchange. Time to ECPR correlates with neurological outcome.[211] 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[197]. It is recommended that adequate ECPR flow is established within 60 min of onset of cardiac arrest. [212] Favourable neurological outcomes in patients with pulmonary embolism undergoing ECPR is inferior to outcomes related to other aetiology.[194, 213] Despite the extremely high mortality rate of patients with cardiac arrest with refractory standard ALS, ECPR is also an option to increase the pool of organ donors.[194]

[h2] Coronary thrombosis

Coronary artery disease (CAD) remains the leading cause of OHCA in adults, either due to ventricular arrhythmias triggered by acute myocardial ischaemia, or those arising from the fibrotic arrhythmogenic substrate in patients with previous myocardial infarction.[214, 215] While CAD prevalence increases with age, it is also the most common cause of sudden cardiac death in adults aged 35 to 50 years.[216, 217]

A recent systematic review and meta-analysis reported significant CAD in 75% of OHCA cases, [218]ranging from 88% in patients with initial shockable rhythm and ST-elevation on ECG, to 54% in non-shockable cases without ST-elevation. Culprit lesions were identified in nearly 60%, more often in patients with shockable rhythm and ST-elevation, while acute coronary occlusion was found in around 40%, with a higher prevalence in patients with ST-elevation. Among those with refractory cardiac arrest, 75% had significant CAD and 70% had a culprit lesion, often involving the left main coronary artery.

Prevention strategies for CAD as a cause of OHCA should comprise promoting healthy lifestyles in asymptomatic individuals, as well as addressing modifiable risk factors of atherosclerosis in patients with known disease, most effectively through cardiac rehabilitation.[219, 220]

Health education should target time reduction from symptom onset (i.e. chest pain) to seeking medical help, in order to enable early diagnosis and treatment. Behaviour change techniques (action planning, information about health consequences, signs and symptoms and instruction on what to do) might be helpful for this particular purpose.[221]

An updated 2022 ILCOR CoSTR concluded that BLS training consistently improved BLS skills, knowledge, and increased confidence to perform CPR. Thus, BLS training for likely bystanders of high-risk populations is recommended and should be actively promoted by healthcare professionals.[197]

Along with initiatives raising public awareness, healthcare systems should establish regional STEMI networks to ensure equal and timely access to percutaneous coronary intervention (PCI).[220] A recent systematic review and meta-analysis found that such networks may reduce STEMI case-fatality by 35% and long-term mortality by 27%.[222] Integration of the emergency transport system was a critical factor for success.

[h4] Detect features suggesting coronary thrombosis and activate STEMI network

A 12-lead electrocardiogram (ECG) should be obtained and evaluated after ROSC to identify possible ischaemic features. Additional ECGs may help decision-making, as defibrillation and the time elapsed from ROSC to ECG acquisition may affect findings.[223] ST elevation remains the most sensitive and specific sign of coronary artery occlusion; however, its absence does not completely exclude the condition.[224] Other ECG patterns —such as bundle branch block or diffuse ST-depression with concurrent ST-segment elevation in aVR and/or V1— may suggest coronary occlusion and should be considered if the clinical context is compatible.[220] Additionally, non-cardiac conditions, such as subarachnoid haemorrhage, can also cause ST-changes,[225] highlighting the importance of clinical correlation.

A comprehensive approach integrating ECG findings and clinical information suggesting a possible coronary cause —such as a history of CAD, chest pain before arrest, or an initial shockable rhythm—[226] is recommended. Once clinical suspicion is established, the STEMI network should be promptly activated to ensure early transfer to a centre with PCI capability.

[h4] Patients without sustained ROSC

Mechanical CPR and, ultimately, ECPR might be the only therapeutic option for refractory OHCA. The first has not proved consistent superiority over manual CPR, but it may facilitate delivering high-quality chest compression during transportation or while performing coronary angiography.[233] Studies addressing ECPR in this setting have reported conflicting results. An updated ILCOR systematic review compared ECPR with manual or mechanical CPR for refractory OHCA, assessing survival and neurological outcomes.[5, 234] This included two single-centre RCTs: the ARREST (n=30) trial was terminated early because of superiority in the ECPR arm for primary outcome (survival to hospital discharge). Interpretation of included studies was difficulted due to different study designs and inconsistent findings.[235, 236] A multicentre RCT found no difference in 30-day survival with favourable neurological outcomes.[237] Given these inconclusive findings but considering the potential risk-benefit balance in this poor-prognosis scenario, ECPR may be considered as a rescue therapy for selected OHCA patients when conventional CPR fails to achieve ROSC, provided it can be implemented.[234]

[h2] Toxic agents

Intoxication is one of the eight reversible causes of cardiac arrest. Thus, all patients with cardiac arrest should be screened for signs of intoxication, especially in suspicious cases, unexpected cardiac arrests or in cases with more than one casualty. If intoxication is likely, resuscitation teams should avoid contamination by first donning appropriate personal protective equipment (PPE). Direct skin contact or mouth to mouth ventilation might transmit toxic agents and should therefore be avoided. The therapeutic strategy of intoxication consists of the three pillars:

- Decontamination
- Enhanced elimination
- Administration of antidotes

Figure YY provides helpful online databases on poison centres and toxins. The patient's temperature should be measured because hypo- or hyperthermia may occur during drug overdose, as mentioned earlier. The 2021 ERC guidelines provide a detailed overview about intoxication.[95] These updated recommendations are based on recent scientific evidence, systematic reviews, and expert consensus. A separate subchapter addresses local anaesthetic systemic toxicity.

[h3] Intoxication with opioids

An ILCOR systematic review found heterogenous results on the benefit of opioid-specific advanced life support-level therapies for cardiac arrest.[238] Existing evidence is not sufficient to recommend administration of an opioid-antagonist (e.g. naloxone) for cardiac arrest caused by opioid poisoning. Administration of an opioid antagonist is warranted, if it is unclear whether a patient with suspected opioid poisoning is actually in cardiac arrest.

[h3] Intoxication with cardiac glycoside

A narrative review on treatment of patients with haemodynamic instability caused by cardiac glycoside poisoning found an improved haemodynamic status and survival after administration of digoxin immune-Fab fragments. Favourable outcomes were seen in patients receiving magnesium, cardioversion, or cardiac pacing.[239]

[h3] ECPR

ECPR seems to be associated with increased survival in intoxicated patients in refractory cardiogenic shock or cardiac arrest.[240]

[h2] Traumatic cardiac arrest

Traumatic cardiac arrest is associated with a very high mortality and good neurological outcome is reported in less than 50% of the survivors. The response to traumatic cardiac arrest is time-critical and success depends on a well-established chain of survival, including advanced prehospital and specialised trauma centre care. Immediate resuscitative efforts in traumatic cardiac arrest focus on simultaneous treatment of reversible causes, which takes priority over chest compressions. Evidence is based on eight systematic reviews and evidence updates and on consensus from the expert group.[241-248]

In Europe, traumatic cardiac arrest accounts for 4% of all cardiac arrests occurring in the pre-hospital setting.[249] Registry data for survival range from 0%[250] to 37%.[251] A recent systematic review[246] of traumatic cardiac arrest identified 36 studies, and a total of 51,722 patients, reporting an overall mortality rate of 96.2%, with 43.5% of survivors achieving a favourable neurological outcome. The presence of a physician at the prehospital scene was associated with improved outcomes (6.1% vs 2.4% survival and 57.0% vs 38.0% favourable neurological outcome, with or without physician respectively). Another systematic review confirmed these findings.[244]

Factors relevant for prognostication are listed in Table 7.

Table 7.

Prognostication	-	+

	Age	Emergency surgery
	Female	Major trauma centre care
	Increased ISS	Shockable ECG rhythm
	Head injury	Reactive pupils
	Shock on admission	Respiratory activity
	Need for blood transfusion	Spontaneous eye or extremity movements
	CPR in the emergency department	Organised ECG rhythm
	Children	Shorter CPR duration
		Reduced prehospital time
		Penetrating chest injury
		Witnessed arrest
		Cardiac motion in ultrasound

[h3] Withholding resuscitation

The American College of Surgeons and the National Association of EMS Physicians recommend withholding resuscitation in situations where death is inevitable or established and in trauma patients presenting with apnoea, pulselessness and without organised ECG activity.[252] However, neurologically intact survivors initially presenting in this state have been reported. [253] The ERC based on expert consensus recommends the following approach: Consider withholding resuscitation in traumatic cardiac arrest in any of the following conditions:

- no signs of life within the preceding 15 min.
- massive trauma incompatible with survival (e.g. decapitation, extensive cardiac destruction, massive head injury with loss of brain tissue).

We suggest termination of resuscitative efforts if there is:

- no ROSC within 20 min after reversible causes have been addressed.
- no detectable ultrasonographic cardiac activity in PEA 20 min after reversible causes have been addressed.

[h3] Preventable Deaths

For a diagnosis of TCA, there must be plausible signs of traumatic injury or, at least, a mechanism of injury consistent with trauma. In the absence of an identifiable mechanism or visible signs of injury, the standard ALS algorithm should be followed instead. The use of ultrasound supports findings in this context, aiding in differentiating peri-arrest from TCA, identifying reversible causes, and guiding resuscitative efforts accordingly.[254]

A considerable proportion of trauma-related deaths can be attributed to management errors. In an urban German population, the prevalence of potentially preventable prehospital deaths from trauma was reported as 15.1%,[255] whereas in an urban U.S. population, this figure was higher at 29%.[256] In Australia, the rate of potentially preventable trauma deaths was found to be 20%.[257] Throughout these studies, exsanguination was identified as a leading cause of preventable death. Taking these data into account, the ERC recommends participation in an accredited trauma management training program for those involved in the care of trauma patients. Ideally such a training format reflects the standard team approach provided in the European systems. [258]

The reversible causes associated with TCA are uncontrolled haemorrhage (48%); tension pneumothorax (13%); asphyxia (13%); and cardiac tamponade (10%). The presenting rhythms found in traumatic cardiac arrest are usually PEA or asystole, depending on the time interval between circulatory arrest and the first ECG recording; PEA (66%); asystole (30%); VF (4%).[259] The key pathophysiological mechanisms leading to traumatic cardiac arrest are listed in table 8:

Table 8.

Cause	Mechanism	Impact on Circulation
Hypovolaemic shock	Severe blood loss → inadequate preload & cardiac output	Decrease in right ventricular filling→ hypotension, decrease in cardiac output → tissue hypoperfusion including coronary perfusion, → tissue hypoxia → metabolic acidosis, shock, ECG typically PEA, followed by asystole
Tension pneumothorax	Increased intrathoracic pressure → impaired venous return	
Cardiac tamponade	Pericardial blood accumulation → restricted ventricular filling	

Hypoxia/asphyxia	Airway compromise, lung injury, or Brain Impact Apnoea	Progressive hypoxaemia → bradycardia → PEA → asystole
Metabolic acidosis	Ischaemia from prolonged hypoxia & hypoperfusion	Cardiac dysfunction, reduced contractility
Neurogenic shock	Loss of sympathetic tone (contributing factor)	Aggravates other shock states, worsening hypotension

As these mechanisms progress beyond a critical threshold, irreversible circulatory failure ensues:

- Progressive bradycardia and hypotension (caused by severe hypoxia and acidosis)
- PEA with minimal cardiac activity on ultrasound (Pseudo-PEA) refers to a state where the heart exhibits weak contractile activity on ultrasound but fails to generate an effective pulse or circulation.[260]
- PEA without with no cardiac activity detectable on ultrasound. The mortality of this condition approaches 100%.
- Asystole → The final stage of cardiac arrest, with no electrical or mechanical activity.

[h3] Effectiveness of chest compressions

In cases of cardiac arrest due to hypovolaemia, cardiac tamponade, or tension pneumothorax, the effectiveness of chest compressions is uncertain. Their impact likely depends on ventricular filling, which is compromised in these conditions, and in peri-arrest states, chest compressions may even reduce the remaining cardiac output by further decreasing venous return. [261-263] Therefore, chest compressions must not delay immediate treatment of reversible causes. Although the effectiveness of chest compressions in traumatic cardiac arrest is limited and context-dependent, the administration of IV adrenaline remains beneficial and is supported by the ERC.[264]

Following resuscitative thoracotomy, open cardiac compressions might be beneficial. Patients sustaining traumatic cardiac arrest within the first six hours of hospital admission may benefit from open cardiac compression instead of closed chest compressions; in one retrospective trial, open cardiac compressions in traumatic cardiac arrest were associated

with a higher long-term survival both in penetrating and blunt trauma. However, the study does not address the management of reversible causes.[265]

Providers who are not trained to manage reversible causes should adhere to the standard ALS algorithm until additional support arrives. Those with the necessary expertise should make case-by-case decisions and respective treatment based on available resources, suspected reversible causes, and regional guidelines.

[h3] Pre-hospital care

Shorter prehospital times are linked to higher survival rates in cases of major trauma and traumatic cardiac arrest.[254] The recently published practice guidelines of the Royal College of Surgeons of Edinburgh on traumatic cardiac arrest provide a comprehensive overview of prehospital management.[266] Depending on the expertise of the prehospital team and the available resources, either the full traumatic cardiac arrest ERC algorithm (fig TCA) can be implemented, or standard ALS may be applied in cases where advanced trauma resuscitation is not feasible.

[h3] In-hospital care

Successful treatment of traumatic cardiac arrest requires a highly coordinated, team-based approach, where all life-saving interventions are carried out in parallel rather than sequentially, based on the clinical priorities dictated by the patient's presentation. The focus is on rapid identification and treatment of all potentially reversible causes to maximise the chances of survival. A prospective observational study in patients with exsanguinating injuries and systolic blood pressure <90 mmHg showed lower 30-day mortality (17.5% vs. 72.0%, p<0.001) when prioritising circulation (CAB approach) over tracheal intubation (ABC approach), thus enabling for resuscitative bleeding control before ventilation.[267] Figure Trauma 1 illustrates the traumatic cardiac (peri-)arrest algorithm, which is based on the universal ALS algorithm but integrates trauma-specific modifications to accommodate the unique challenges of traumatic cardiac arrest management. A multidisciplinary team approach, including trauma surgeons, emergency physicians, anaesthetists, radiologist, intensivists and nursing staff, is essential to ensure that each critical intervention is initiated without delay, according to the patient's immediate needs.

[h3] Treatment of the reversible causes in traumatic cardiac arrest

Hypovolaemia

The treatment of severe hypovolaemia has several components. The main principle is to achieve immediate haemostasis. Temporary haemorrhage control can be lifesaving.[254] Compressible external haemorrhage can be treated with elevation (of a bleeding limb), direct or indirect pressure, pressure dressings, tourniquets and topical haemostatic agents.[254, 268] Non-compressible haemorrhage is more difficult to address and splints (pelvic splint), blood products, IV fluids and tranexamic acid can be used while transferring the patient for surgical haemorrhage control.[269]

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 and cross-clamping (or manual compression) of the descending aorta or resuscitative endovascular balloon occlusion of the aorta (REBOA). There is no evidence for one technique being superior to the other. [270] Both are highly specialised interventions, requiring careful patient selection and appropriate expertise and equipment. A recent UK REBOA study[271] in traumatic shock revealed a possible increase in mortality.

In hypovolaemic traumatic cardiac arrest, immediate restoration of the circulating blood volume with blood products is mandatory. Prehospital transfusion of fresh plasma and packed red blood cells may improve survival, especially in cases with prolonged prehospital time.[272, 273] If whole blood transfusion offers any advantages over component therapy is unclear.[274, 275]

Hypoxia

In traumatic cardiac arrest, hypoxaemia can be caused by airway obstruction, traumatic asphyxia or impact brain apnoea.[276] Impact brain apnoea is an underestimated cause of morbidity and mortality in trauma, but not necessarily associated with an un-survivable brain injury.[277] Data from the National Trauma Audit and Research Network database in UK reported a 15% survival rate with 90% favourable outcome among traumatic cardiac arrest patients with brain impact apnoea.[278] Brain impact apnoea may aggravate the course of traumatic brain injury and can lead to asphyxiation if left untreated. Effective airway management and ventilation with oxygen can prevent and reverse hypoxic cardiac arrest. In a recent national Dutch study, 52% of traumatic cardiac arrest were attributed to traumatic brain injury related apnoea.[279]

Controlled ventilation in circulatory compromised patients is associated with major risks because of the increase in intrathoracic pressure[280, 281] that may lead to further decreased cardiac output caused by:

- impeding venous return to the heart, particularly in severe hypovolaemia.
- reduced diastolic filling, particularly in cardiac tamponade
- conversion of pneumothorax into a tension pneumothorax
- increase in blood loss from venous bleeding sites

Minimising intrathoracic pressure using low tidal volumes and minimal PEEP may help optimise cardiac preload. Ventilation should be monitored with capnography and adjusted to achieve normocapnia.[282-284]

Tension pneumothorax

Tension pneumothorax is a reversible cause of cardiac arrest and must be excluded in traumatic cardiac arrest. It may lead to cardiac arrest by obstructing venous return through mediastinal shift and impairing effective gas exchange. Positive pressure ventilation can convert a simple pneumothorax into a tension pneumothorax.[285] The prevalence of tension pneumothorax is approximately 0.5% in all major trauma patients treated in the prehospital setting and 13% of those develop traumatic cardiac arrest.[259] Diagnosis of tension pneumothorax in a patient with cardiac arrest or haemodynamic instability must be based on clinical examination or POCUS. The symptoms include haemodynamic compromise (hypotension or cardiac arrest) in conjunction with signs suggestive of a pneumothorax (preceding respiratory distress, hypoxia, absent unilateral breath sounds on auscultation, subcutaneous emphysema and mediastinal shift (tracheal deviation and jugular venous distention)).[285] The presentation of a tension pneumothorax is not always classical, but when it is suspected in the presence of cardiac arrest or severe hypotension, chest decompression by open thoracostomy should be carried out immediately. To decompress the chest in traumatic cardiac arrest, perform bilateral thoracostomies in the 4th intercostal space at the midaxillary line, allowing extension to a clamshell thoracotomy if required. Needle thoracocentesis should be attempted if the provider is not competent to perform a thoracostomy, or if the necessary surgical instruments are not immediately.[286-289]

Cardiac tamponade and resuscitative thoracotomy

Cardiac tamponade is a frequent cause of cardiac arrest in penetrating chest trauma. Those with the clinical ability, competence, and equipment to perform it, can restore circulation with immediate resuscitative thoracotomy via a clamshell or left anterolateral incision.[290, 291]

A systematic review on needle pericardiocentesis in traumatic cardiac tamponade concluded that there remains a limited role for this method in non-trauma centres where definitive surgical management is not immediately available.[292]

While resuscitative thoracotomy is often performed for the relief of cardiac tamponade, it is not exclusively indicated for this condition. Resuscitative thoracotomy is also indicated for direct haemorrhage control, aortic cross-clamping, and open cardiac compressions, which is associated with better outcomes compared with closed chest compressions in the resuscitation of patients with traumatic cardiac arrest.[265]

A recent systematic review and meta-analysis examined resuscitative thoracotomy in civilian thoracic trauma, analysing 49 studies across both pre-hospital and emergency department settings.[242] The study found that timing is critical, as performing pre-hospital resuscitative thoracotomy more than five minutes after arrival at the scene was associated with increased neurological complications, while a delay of more than ten minutes from the initial encounter to resuscitative thoracotomy was linked to higher mortality rates. Patients with an Injury Severity Score of 25 or higher and those without signs of life also had poorer outcomes. Overall, mortality was higher in pre-hospital resuscitative thoracotomy (93.5%) compared to ED-resuscitative thoracotomy (81.8%) ($P = 0.02$). Among emergency department-resuscitative thoracotomy cases, blunt trauma patients had significantly higher mortality (92.8%) compared with penetrating trauma patients (78.7%) ($P < 0.001$). In summary, emergency resuscitative thoracotomy is a high-risk intervention, with better survival rates in the hospital, and timely intervention within 5–10 minutes is crucial for improving outcomes. Blunt trauma patients have poorer survival prospects compared with those with penetrating injuries.

The prerequisites for a successful resuscitative thoracotomy can be summarized as the 'four E rule':

- Expertise: teams that perform resuscitative thoracotomy 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 resuscitative thoracotomy and to deal with the intrathoracic findings is mandatory.
- Environment: ideally resuscitative thoracotomy should be carried out in an operating room. Resuscitative thoracotomy 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 resuscitative thoracotomy should not be longer than 15 minutes

If any of the four criteria is not met, resuscitative thoracotomy is futile and exposes the team to unnecessary risks.[293] A retrospective cohort study found for prehospital resuscitative thoracotomy for traumatic cardiac arrest was associated with 3.8% patients surviving with favourable neurological outcome, all presenting with cardiac tamponade following penetrating trauma, and none of the patients with exsanguination or other pathology surviving with favourable neurological outcome. There were no survivors beyond 15 minutes of traumatic cardiac arrest for cardiac tamponade and 5 minutes after exsanguination-induce cardiac arrest.[294]

[h3] Post resuscitation care after traumatic cardiac arrest

As in severe trauma management, early whole-body CT scanning can help identify major injuries and guide initial management following ROSC by detecting life-threatening conditions such as ongoing haemorrhage, pneumothorax, or cardiac tamponade, which may require immediate intervention. As with all aetiologies of cardiac arrest, optimising cardiac output is essential for restoring oxygenation and meeting the metabolic demands of post-arrest physiology. This includes fluid resuscitation and vasopressor support as needed to maintain adequate tissue perfusion and oxygen delivery.

The principle of damage control resuscitation is advocated for uncontrolled haemorrhage in trauma. Damage control resuscitation integrates permissive hypotension, haemostatic resuscitation, and damage control surgery. Evidence supports a conservative IV fluid approach, maintaining blood pressure at a level no more than is necessary to sustain a radial pulse, until surgical haemostasis is achieved. However, caution is needed in traumatic brain injury, where raised intracranial pressure may necessitate a higher blood pressure to achieve sufficient cerebral perfusion pressure. The duration of hypotensive resuscitation should not exceed 60 minutes, as the risk of irreversible organ damage then outweighs its benefits.[295-297]

Temperature management is crucial, as hypothermia exacerbates coagulopathy, acidosis, and haemodynamic instability (the 'lethal triad'). Active warming strategies, including fluid warming, external warming devices, and maintaining an optimal resuscitation environment, should be implemented early to prevent further deterioration.

Coagulation management is a key component of resuscitation, as trauma-induced coagulopathy can worsen haemorrhage and organ dysfunction. Strategies include early

administration of blood products (e.g., plasma, platelets, fibrinogen), tranexamic acid, and point-of-care coagulation monitoring to guide targeted haemostatic therapy.

[h1] Special Settings

[h2] Cardiac arrest in the catheterisation laboratory

The occurrence and outcomes of cardiac arrest in the cardiac catheterisation laboratory ('cath lab') vary depending on the setting, patient profile and type of intervention performed. Cases range from routine elective procedures to the emergent treatment of critically ill patients with cardiac ischaemia, shock, or life-threatening arrhythmias. Additionally, structural heart interventions, often targeting elderly and high-risk patients, are becoming increasingly prevalent and complex. Causes of cardiac arrest in this setting include severe ischaemia, tamponade, vascular perforation/dissection, or anaphylaxis, among others.

A recent ILCOR scoping review[298] estimated the overall incidence of cardiac arrest in the catheterisation lab as 0.2% to 0.5% across all procedures.[299, 300] For PCI, the incidence is slightly higher (0.8% - 2%),[301-303] and for ST-elevation myocardial infarction higher still (STEMI - 1.9% to 5.5%), primarily with an initial rhythm of ventricular fibrillation.[304-308]

Overall initial survival rates range from 67% to 77% at the event time[299, 300, 309] and between 38% to 56% at hospital discharge.[300, 309] For PCI and STEMI, survival to discharge is higher (82% - 100%).[304, 305, 308]

[h3] Prevent and be prepared

Adequate training is essential to ensuring the best outcomes of cardiac arrest in the catheterisation lab. Staff should be trained in advanced life support and prepared to quickly recognise and manage procedure-specific risks and complications, including pacing, pericardiocentesis, initiation of ventricular assist devices, or ECPR. Emergency equipment should be functional and readily available.

Studies support periodic simulation emergency drills to enhance technical and non-technical skills in the catheterisation lab.[310] Training in non-technical skills has the potential to minimize errors in this setting.[311] Careful risk-benefit assessment and planning for elective procedures are advised to minimise complications. The use of safety checklists has been reported as effective in reducing human errors and resulted in fewer complications.[312]

[h3] Detect and react

Patient's monitored vital signs and ECG must be regularly checked to detect early complications. Echocardiography should be readily accessible in case of haemodynamic instability or suspected complication (e.g. cardiac tamponade). Transoesophageal echocardiography (TOE), often used for monitoring structural heart interventions in the catheterisation lab, may facilitate early detection of complications with higher quality image and precision.[313]

Cardiac arrest in the catheterisation lab should prompt an immediate call for help and activation of the resuscitation team.[299] While catheterisation lab staff must promptly initiate CPR, additional support is needed to sustain CPR while addressing reversible causes. Depending on the suspected underlying complication or cause of arrest, the cardiac surgery team should be also alerted. Importantly, as highlighted by the Joint British Societies' guideline on management of cardiac arrest in the catheterisation lab,[314] all emergency team members should don lead aprons before entering, since fluoroscopy may often be necessary. Ensure that an adequate supply of lead aprons, protection for eyes and thyroid is readily available in advance.

[h3] Resuscitate and treat possible causes

Cardiac arrest in the catheterisation lab should be generally managed according to the ALS protocol (see ALS chapter), with some modifications. In the presence of monitored VF/pVT, immediate defibrillation with up to three stacked shocks if a shockable rhythm persists is recommended before starting chest compressions. This approach aligns with the recommendations for witnessed arrests in monitored patients with a shockable rhythm when a defibrillator is readily available. In case of PEA/ asystole, CPR should be initiated and adrenaline injected. However, extreme bradycardia may be treated with external or transvenous temporary pacing, especially if it arises from atrio-ventricular block complicating certain procedures, such as transaortic valve implant, tricuspid valve interventions or catheter ablation. During cardiac arrest, invasive procedures to correct reversible causes –such as PCI, pericardiocentesis, or mechanical support device initiation– might be needed during on-going CPR or immediately after ROSC.[314]

[h3] Point of care ultrasound (POCUS)

ILCOR reviewed the diagnostic accuracy of POCUS in detecting reversible causes of cardiac arrest in different scenarios, concluding that POCUS evaluation may be considered during cardiac arrest in the catheterisation lab if performed by experienced personnel without interrupting CPR, especially when a specific reversible cause is clinically suspected.[315] In patients already undergoing TOE at the time of cardiac arrest, maintaining the TOE probe in place could be considered if the airway is secured, since TOE may offer additional feedback and assist in guiding ECPR or mechanical support device placement if performed by a skilled operator, without interfering with chest compression. However, the use of these tools should not compromise adherence to the ALS protocol.

[h3] Mechanical CPR.

An ILCOR systematic review evaluated the impact of mechanical chest compression devices compared with manual compression on cardiac arrest outcomes,[233] and found evidence against the routine use of automated mechanical chest compressions to replace manual compressions for in-hospital cardiac. However, mechanical CPR was considered a reasonable alternative when high-quality manual compressions may compromise the safety of the provider or interfere with critical procedures, such as those performed in the catheterisation lab. Additionally, the ILCOR scoping review on cardiac arrest in the catheterisation lab[298] described outcomes of cardiac arrest in the catheterisation lab following the use of mechanical CPR, mainly during PCI or cannulation for mechanical circulatory support or extracorporeal CPR. The mixed cohorts included in the remaining studies and the inconsistent outcome reporting made interpretation difficult, so the overall results in this setting are uncertain. Until new evidence is produced, mechanical CPR may be used in the catheterisation lab following the ILCOR CoSTR considerations discussed above.[233] Importantly, if used, pauses during device placement should be minimized, and correct positioning ensured to avoid visceral injuries.

[h3] Extracorporeal CPR

The use of veno-arterial extracorporeal membrane oxygenation (VA-ECMO) to support refractory cardiac arrest patients (ECPR) was recently updated by ILCOR.[234] In the in-hospital setting, ECPR was considered as a rescue therapy for selected patients with refractory cardiac arrest if implementation is feasible. A recent ILCOR scoping review [298] covered the use of ECPR in the catheterisation lab. Most evidence derived from the Extracorporeal Life Support Organization registry with 39% survival to hospital discharge.[316] Despite its potential

advantages in sustaining resuscitation while addressing reversible causes, evidence is limited due to study heterogeneity and high risk of bias. Nevertheless, considering the risk-benefit ratio, ECPR may be considered for selected cardiac arrest patients in the catheterisation lab based on clinical circumstances, available resources and experience. Of note, extracorporeal CPR facilitated performing diagnostic or therapeutic procedures, including coronary angiography/angioplasty, pulmonary embolectomy or cardiac surgery.

[h3] Mechanical circulatory support

A variety of temporary devices can be used in the catheterisation lab to assist cardiac pumping function, offering different levels of haemodynamic support for severe heart failure or during high-risk procedures. Common examples include the intra-aortic balloon pump, Impella®, TandemHeart® or VA-ECMO.

The use of mechanical circulatory support devices for cardiogenic shock after ROSC in any setting was addressed by ILCOR.[317] This showed no survival benefit at different follow-up points when comparing early routine device use with standard care. However, most evidence derived from cardiogenic shock patients rather than those specifically resuscitated from cardiac arrest. In summary, mechanical circulatory support in patients with cardiogenic shock after ROSC in the catheterisation lab may be considered in selected cases. Individualised decisions should consider the clinical picture, device availability and team expertise. When used, close monitoring for complications is recommended to ensure prompt management.

[h3] Intracoronary adrenaline. The recent ILCOR scoping review on cardiac arrest in the catheterisation lab included a comparison of intracoronary adrenaline with either peripheral intravenous or central venous administration.[317] Adrenaline via central intravenous and intracoronary routes was associated with higher rates of ROSC, survival to hospital discharge, and survival with good neurological outcomes compared with peripheral intravenous administration. However, intracoronary administration was associated with an increased risk of stent thrombosis. Despite these promising findings, current evidence is insufficient to recommend routine intracoronary administration of adrenaline during cardiac arrest in the catheterisation lab.

[h2] Drowning and water rescue

Once the World Health Organization recognised accidental drowning as a serious public health problem—especially in low-resource settings—annual global deaths from drowning decreased from 370,000 in 2000 to 300,000 in 2021[318]. This decline might be attributed to legislation and regulations (e.g., pool fencing and mandatory lifejacket use),[319], as well as effective preventive measures (e.g., day-care centres for children and water safety training).[320] Fatal drownings are associated with several risk factors: 1) sex: males account for up to six times more drownings than females; 2) age: although in Europe drowning is the fourth leading cause of death in children aged 5–14 years, those older than 50 years of age have the highest incidence; 3) location: inland waterways represent a significant risk location; 4) circumstances such as alcohol consumption and migrant status increase the likelihood of drowning. The WHO statistics do not include fatalities from drowning caused by suicide (the most frequent cause of drowning death in several European countries), transport accidents, or natural disasters such as floods.[321-323] Most fatal drownings never reach the health system because rescue or retrieval often occurs too late for medical intervention.[324]

Recommendations are based on a scoping review in 2021 and a systematic review in 2023. [325, 326] There are no RCTs. Most recommendations are good practice statements or expert opinions based on indirect evidence and are in line with the position statements from the medical commission of the International Lifesaving Federation (ILS).[327]

Drowning is defined as 'the process of experiencing respiratory impairment from submersion or immersion in liquid'.[328] Immersion is a situation in which the person stays in a liquid with the airway above surface, while submersion refers to the state in which the airway remains submerged. When respiratory impairment progresses during submersion, the heart slows down and finally stops due to prolonged hypoxia, and only resuscitation can prevent fatal drowning.[329, 330] Submersion time is the most robust and independent prognostic factor[331]. The Adult BLS CoSTR 2020 recommends submersion time as the sole indicator guiding prognosis and decisions regarding clinical management and search and rescue operations.[332] Less than 5-10 min of submersion is associated with a better prognosis.[331-333]

The extent of lung damage depends on the amount and degree of pollution of the aspirated water.[329] Salinity is of no proven predictive value.[331, 332, 334]

The autonomic conflict, caused by simultaneous stimulation of the sympathetic and parasympathetic autonomic nervous system may explain the genesis of cardiac arrhythmias in circumstances with cold water immersion and breath-holding.[335] In drowning, low water

temperature may exert a protective effect by reducing brain metabolism.[329, 331, 332] However, this phenomenon is exceedingly rare and is most likely in small individuals who cool quickly in water below 6°C.[336] Submersion longer than 60 min with good neurological outcomes have been reported,[337] particularly in children immersed in cold water, preceding the submersion.[337, 338] These reports suggest that if water temperature is above 6°C, survival or successful resuscitation becomes extremely unlikely after about 30 minutes of submersion, whereas in water at or below 6°C, this threshold may extend to around 90 minutes.[337]

[h3] Water rescue

Water rescue is needed to prevent drowning, to interrupt drowning, and to provide immediate life-saving intervention. Many untrained people have died while attempting a rescue.[339] Rescuers should never deliberately place themselves in danger. For this reason, it is strongly recommended that bystanders, especially those who are unable to swim, do not enter deep water. Instead, they should rely on indirect rescue methods, such as using public rescue equipment, throwing any available floatation device or reaching out with a long object, while seeking professional assistance. First responders with rescue training have the competencies to select and apply the appropriate rescue technique, rescue material and flotation devices where possible.[340] Rescue material and flotation devices also reduce the rescue time.[340]

Physical fatigue is a limiting factor when CPR is required, especially after water rescue.[341, 342] If possible, a person not involved in the rescue should perform CPR.[340] Spinal injuries are exceptional after drowning.[343] Spinal stabilisation should not delay assessment of vital signs or resuscitation. If resuscitation is not needed and clear signs of a cervical spine injury are present, it is recommended that at least three people carry out spinal motion restriction during extrication from water, preferably with at least one of them specifically trained in the procedure.[344] If the required people are unavailable, the extrication should not be postponed any further. In every situation of a spinal injury in water, emergency medical services should be alerted immediately.

[h3] Cardiac arrest caused by drowning

To reduce hypoxia, in-water ventilation and on-board resuscitation are gaining acceptance within the lifesaving community, as these techniques are feasible with training. [325, 345-348]. The prognosis is better when drowned persons are ventilated when initiating CPR,[346, 349, 350],

1654 therefore, first responders and EMS should follow the ABC approach. [345, 346] CPR should start
1655 with 5 ventilations. If the person remains unconscious without normal breathing, continue
1656 with standard CPR (Figure X). Starting CPR with compression is only advised if the rescuer is
1657 unwilling to provide rescue breaths.[351]
1658 For simplicity, laypeople should start with chest compressions whilst trained first aid
1659 providers should consider rescue breaths, since the time to ventilation is critical in restoring
1660 cardiac arrest in drowned victims. In isolated respiratory arrest, rescue breaths can prevent
1661 cardiac arrest.
1662 In drowning, an initial shockable rhythm is present in less than 10% of cases because of the
1663 cardiac response to hypoxia. Although victims removed from the water might be in cardiac
1664 arrest following arrhythmias caused by autonomic conflict[335], CPR should not be delayed by
1665 AED application. Although, oxygen should be provided early because drowning is a
1666 respiratory event, a recent study did not observe improved oxygen saturation or survival
1667 after oxygen administration by lifeguards.[352] Airway management should be based on the
1668 competencies and training of the first responder or EMS. [345] Tracheal intubation compared
1669 with supraglottic airway devices was associated with higher ROSC, but not associated with
1670 survival or favourable neurological outcome at one month.[353]
1671
1672 **[h3] Impact of hypothermia**
1673 Most drowned victims are hypothermic (core body temperature < 35°C), which affects
1674 diagnosis, treatment and prognosis.[329, 331, 354, 355] In general, guidelines for accidental
1675 hypothermia can also be applied to hypothermic drowned victims, including the one-minute
1676 vital sign check, accurately measuring core temperature, the alternative treatment regarding
1677 defibrillation, use of medication when body temperature is below 30°C, and access to
1678 extracorporeal life support rewarming.[354] Hypothermia reflects a longer submersion time,
1679 resulting in poorer neurological outcome and survival. However, when drowning occurs in
1680 cold water, generally considered below 6°C, and notably after a period of immersion,
1681 associated hypothermia may exert a neuroprotective effect.[356]
1682 The Adult BLS CoSTR 2020 suggests not using water temperature when making prognostic
1683 decisions[332] and no specific guidelines exist to indicate precisely when to move from rescue
1684 to recovery—a stance that also acknowledges the exclusion of rare favourable outcomes in
1685 icy water. This is partly due to the wide variety of drowning scenarios regarding submersion
1686 time, water temperature, available resources, or environmental hazards. Some agencies or

countries have local or national protocols. In the absence of definitive guidance, it is crucial that those on scene reach a consensus on the point at which further medical intervention is deemed futile.[356]

[h2] Cardiac arrest in the operating room (OR)

Cardiac arrest in the operating room is a rare event with an incidence of about 3/10,000 anaesthetics[357-359], with a higher incidence in low resource settings[360], in older-frailer patients, and in newborns and infants.[361, 362] Overall survival rate is >50%.[363] Strong predictors of intraoperative cardiac arrest are higher American Society of Anesthesiologists (ASA) physical status, sepsis, urgent or emergency case, complexity of the case, anaesthetic technique and age.[363] The leading causes are complications during cardiac surgery, major haemorrhage, bradyarrhythmias and septic shock.[360, 363]

[h3] Specifics of intraoperative cardiac arrest and its treatment

Mostly, gradual physiological deterioration leads to intraoperative cardiac arrest.[364] Cardiac arrest treatment in the OR follows the general ALS algorithm. However, several modifications intended to identify reversible causes are required. Key interventions include immediately calling for help, informing the surgical and anaesthesia team, and ensuring the presence of a sufficiently skilled people.

High-risk surgical patients are often monitored with invasive blood pressure pre-arrest. Recent proposals suggest starting chest compression if systolic blood pressure stays below 50mmHg despite interventions to treat the underlying cause. [357, 365, 366] Adjust the position and the height of the operating table or trolley to optimise delivery of high-quality chest compressions.

Low initial end-tidal capnography values ($ETCO_2$ <2.7 kPa or 20 mmHg) are linked to inadequate chest compression quality, indicating the need for improvement and possibly suggesting rescuer fatigue.[367-369] For pre-arrest patients, initial incremental boluses of 50–100 µg adrenaline intravenously have been proposed, rather than the standard 1 mg bolus. In pre-arrest patients, higher adrenaline doses may induce severe hypertension or tachyarrhythmias. If a low-dose adrenaline bolus fails, the standard 1mg of intravenous adrenaline should be given.[357, 370]

In circumstances where the probability of cardiac arrest is high, a defibrillator should be readily available in standby mode and self-adhesive defibrillation electrodes should be

applied before induction of anaesthesia. Safe defibrillation should be performed immediately in case of a shockable rhythm. Ensure adequate venous access, prepare resuscitation drugs and fluids, establish advanced airway management (if not already undertaken) and use a mechanical ventilator delivering 100% oxygen as soon as possible.[371] Current data suggest that mechanical ventilation yields a similar PaO_2 to that of manual ventilation with a self-inflating bag.[372-375] If there is a qualified sonographer able to perform ultrasound (transthoracic/ transoesophageal) with minimum interruptions to chest compressions, they should be summoned to aid diagnosis.[371]

Chest compression is optimally performed in the supine position, but in case of a cardiac arrest in prone position with an advanced airway in place, follow the ILCOR Good Practice Statement to initiate CPR in prone position.[376, 377] Consider simultaneous left lateral decubitus and head-down positioning in cases of massive air embolism[378-380] if it does not affect chest compression quality.

Identification of reversible causes should be prioritised and treated appropriately:

- If arrest is caused by significant blood loss, chest compression is effective only if the circulating volume is replaced simultaneously and haemorrhage control (e.g. surgery, endoscopy, endovascular techniques) is initiated immediately.[371]
- In cases of severe subdiaphragmatic exsanguination, resuscitative endovascular balloon occlusion of the aorta (REBOA) may be considered in an attempt to slow intravascular volume depletion. However, there is limited evidence on improved survival and further studies are required to elucidate potential benefit.[371, 381, 382]
- ECPR should be considered in cases where conventional CPR fails or when prolonged resuscitation is required.
- Open cardiac compressions should be performed only by trained healthcare professionals in cases of intraoperative cardiac arrest or as part of resuscitative thoracotomy for trauma patients.
- If arrest occurs during laparoscopic or robotic surgery, consider releasing any pneumoperitoneum and deflate the abdomen to enhance venous return during CPR; during thoracoscopy, stop CO_2 insufflation and rule out contralateral pneumothorax.
- For other reversible causes consult the specific subchapters of this guideline.

Human factors in intraoperative cardiac arrest

As in every resuscitation event, a designated team leader should direct and coordinate the resuscitation team and their assistants, focusing on high-quality chest compressions and ventilations, minimising no-flow times, addressing reversible causes simultaneously, and preventing focus on low-priority distracting tasks. Surgery must be stopped unless it addresses a reversible cause of cardiac arrest. There may be a need to cover the surgical field to enable access to the patient and to perform resuscitation tasks.

Successful management of intraoperative cardiac arrest requires not only individual technical skills and a well-organised team response, but also an institutional safety culture embedded in everyday practice through continuous education, training and multidisciplinary cooperation.[5, 383, 384] Institutional protocols for responding to potential arrest situations (e.g. massive transfusion protocols) and checklists will help to optimise the response to cardiac arrest in the operating room environment.[5, 385]

There is no evidence to support the use of hypothermic temperature control after adult intraoperative cardiac arrest (ref -post-resus GL2025 chapter). Therefore, post-arrest temperature control should follow local post-resuscitation protocols.

[h3] Local Anaesthetic Systemic Toxicity

Cardiac arrest is a rare complication of local anaesthetic overdose, which is often caused by inadvertent intravascular injection. Direct action of local anaesthetics on the sodium channels of cardiac myocytes causes cardiovascular collapse, usually within 1–5 min, but onset was reported from 30 sec to 60 minutes.[386, 387] Early symptoms are peri-oral numbness, metallic taste, dizziness, tinnitus and blurred vision followed by substantial hypotension, dysrhythmias, and generalised seizures. The emergency diagnosis is often based on exclusion of other causes.[388, 389] Seizure management involves administering benzodiazepines in incremental doses (e.g. IV lorazepam 0.1 mg kg^{-1}, midazolam 0.05-0.1 mg kg^{-1}), followed by stepwise doses of propofol or pentothal (up to induction doses) if needed. Attention should be given to securing the airway and maintaining ventilation while preventing cardiovascular collapse during sedative drug administration. Intravenous lipid therapy is recommended as rescue therapy to treat cardiovascular collapse and cardiac arrest although its use is based on very low-certainty evidence,[128, 389-391] but without documented harm.[392] A 20% lipid emulsion (4x250 mL) should be available if large doses of local anaesthetics are used (e.g. operating rooms, labour wards, emergency department).[390, 393]During cardiac arrest, after an initial 20% lipid emulsion IV bolus (1.5 mL kg^{-1} over 1 min), an infusion should be started (15

mL kg^{-1} h^{-1}). If ROSC is not achieved in 5 min, the infusion rate should be doubled with two additional boluses at 5-minute intervals.[390, 393] A maximum cumulative dose of 12 mL kg^{-1} IV 20% lipid emulsion is recommended to avoid fat overload.[392] It is suggested that adrenaline IV be given at a lower dose (≤ 1µg kg^{-1}) in local anaesthetic intoxication because its arrhythmogenic and acidotic effects may impede sustained ROSC – this has been shown in animal studies.[390, 394-397]

[h3] Cardiac surgery

The incidence of cardiac arrest following cardiac surgery is 2-5%, with higher survival rates (around 50%) that are higher than other causes.[398-402] This is largely due to early detection of, and the high incidence of, reversible causes. Common causes of cardiac arrest in this setting include arrhythmias causing VF, accounting for up to 50% of cases, followed by cardiac tamponade and major bleeding, which often presents as PEA.

Evidence-based recommendations for the management of cardiac arrest following cardiac surgery derive from the European Association for CardioThoracic Surgery[403, 404], the Society of Thoracic Surgeons expert consensus document for the resuscitation of patients who arrest after cardiac surgery,[405] , the British Societies Guidelines, [406, 407] and the ILCOR CoSTR for the Management of Implantable Left Ventricle Assist Devices recipients.[406, 407]

Ensure adequate initial and refresher training of staff in resuscitation technical skills, ALS and team working through simulated cardiac surgery scenarios, including training to perform an emergency resternotomy. Team work, including human factors such as situational awareness and communications skills, should be included in the training and acquired through practice in simulated scenarios. Roles should be previously allocated to staff in every setting to ensure effective coordination of resuscitation efforts.[408]

Emergency equipment should include small resternotomy sets containing only the essential elements to open the chest, be standardised, adequately marked, readily available where patients with recently performed interventions are recovering and periodically checked. [404, 405]

Safety checklists reduce complications and mortality in non-cardiac surgery and should be implemented for the management of cardiac arrest in these settings. [409]

Detect cardiac arrest and activate cardiac arrest protocol

Early signs of deterioration can be identified in the monitored post-operative patient after careful examination. Hypotension is a common sign of several post-operative complications (Table 9).[410-412]. Echocardiography should be performed in case of haemodynamic instability; consider transoesophageal echocardiography for more precise diagnosis.[413, 414] Continuous ECG monitoring enables early identification of arrhythmias; supraventricular tachycardias are the most frequent in this setting [415]

Table 9.

• Haemorrhage • 'Medical' bleeding: post-operative coagulopathy • 'Surgical' bleeding: operative trauma	• Correct hypothermia and hypertension, avoid haemodilution • Consider blood product transfusion and use of haemostatic agents, guided by haematological tests • Check chest drains to identify active bleeding and perform echocardiography to exclude cardiac tamponade; consider early re-operation if suspected
• Low cardiac output state • Inadequate preload • Excessive afterload • Decreased ventricular contractility • Diastolic dysfunction	• Perform echocardiography to assess ventricular function • Ensure adequate ventricular filling • Correct systemic vasoconstriction • Maintain atrioventricular coordination • Correct metabolic disturbances and hypocalcaemia • Consider inotropic or mechanical circulatory support
Graft or valve failure	• Check for ECG abnormalities • Perform echocardiography • Consider percutaneous intervention or re-operation
Arrhythmias	• Correct electrolytic disturbances • Consider antiarrhythmic, electrical cardioversion or pacing
Vasodilation • Rewarming • Analgesics / sedatives • Sepsis • Anaphylaxis • Adrenal insufficiency • Vasoplegic syndrome	• Correct specific underlying causes • Consider haemodynamic-guided IV fluid therapy • Consider vasopressor support

Cardiac arrest can be detected by checking ECG rhythm, clinical examination and review of vital signs, including pressure waveforms (arterial, central venous and pulmonary artery pressures, and pulse oximetry) and ETCO2. [404, 405]

[h3] Resuscitate and treat possible causes in cardiac surgery

Key modifications to the standard ALS algorithm include immediate correction of reversible causes and if this is not successful, emergent resternotomy.[404, 405]

Resuscitate and treat possible causes:

- Start resuscitation according to ALS but with modification:
    - VF/ pVT : Apply up to 3 consecutive shocks (< 1 min).
    - Asystole/ extreme bradycardia: Apply early pacing – maximum output (< 1 min).
    - PEA: Correct potentially reversible causes. If paced rhythm, transiently turn off pacing to exclude VF.

➔ No ROSC:

- Initiate chest compression and ventilation.
- Consider POCUS/ TOE.
- Perform early resternotomy (< 5 min).
- Consider circulatory support devices and ECPR (Figure CS1).

In patients with VF/pVT, defibrillation of up to three stacked shocks are prioritised within a maximum time of 60 seconds.[416, 417] If these fail, immediate resternotomy and internal defibrillation are advised.[417] In case of asystole or extreme bradycardia, attempt epicardial pacing (DDD mode at 80 to 100 beats.min^{-1} and at maximum output voltages) or transcutaneous pacing should be attempted for one minute before initiating chest compressions. PEA should trigger immediate external chest compressions, searching for reversible causes and preparing for early resternotomy. In the presence of a pulseless stimulated rhythm, pause the pacing to exclude underlying VF and, if indicated, perform defibrillation.[404, 405].

If ROSC is not achieved following defibrillation or pacing, or in case of PEA, initiate compressions and ventilations should be initiated while preparing for emergency resternotomy. Perform external compressions at 100-120 beats min^{-1}, aiming to reach a

systolic blood pressure > 60 mmHg; failure to attain this value despite adequate performance may indicate tamponade or severe haemorrhage, requiring emergency resternotomy.[404, 405] Compared with external compressions, internal cardiac compression provides better coronary and systemic perfusion pressure, which may justify chest reopening.[418, 419] Airway management in this setting follows the usual indications for ALS. In mechanically ventilated patients check the position and patency of the tracheal tube, increase inspiratory oxygen increased to 100% and remove positive end-expiratory pressure. If a tension pneumothorax is suspected, emergency decompression is necessary.[404, 405]

Drugs during resuscitation

As a general principle, stop all infusions other than needed for resuscitation. Amiodarone (300 mg) or lidocaine (100 mg) may be administered intravenously after three failed shocks to treat VF/ pVT.[405, 420]. Injecting adrenaline (1 mg) shortly after cardiac surgery is controversial. The European Association of CardioThoracic Surgery and Society of Thoracic Surgeons discourage the routine use of adrenaline based on the concern that intense hypertension induced by adrenaline may cause bleeding or disruption of surgical anastomoses after ROSC[404, 405, 420], although lower doses (50-100 mcg boluses) may be considered in peri-arrest situations based on expert consensus.[357, 370, 405, 410, 418]

POCUS and Transoesophageal echocardiography.

In a peri-arrest patient following cardiac surgery, consider transthoracic POCUS, although it may give limited views of the posterior cardiac chambers and any surrounding posterior cardiac tamponade. Transoesophageal echocardiography is a preferable alternative, giving clearer 360° views to include posterior cardiac structures, and enables uninterrupted chest compressions. The presence of cardiac tamponade, intrathoracic haemorrhage, pleural effusions, hypovolemia, dynamic left ventricular outflow tract obstruction and aortic dissection can generally be identified using TOE.[413, 414, 421-428] Transoesophageal echocardiography can evaluate the effectiveness of chest compressions by real-time imaging of the emptying and filling of compressed cardiac chambers, potentially leading to repositioning of hands for chest compressions.[429, 430]

[h3] Early resternotomy

Refractory cardiac arrest requires resternotomy within 5 minutes, in order to perform internal heart compression, release any tamponade and correct underlying causes. This is a

safe procedure in the ICU,[431] leading to higher survival rates, especially if performed with minimal delay and in the presence of surgically repairable problem on reopening.[432] Resternotomy should be attempted as part of the resuscitation protocol of cardiac patients until at least day 10 post-operatively.[405]

[h3] Circulatory support devices

Intra-aortic balloon pump

In patients supported by intra-aortic balloon pump who present in cardiac arrest, the device may contribute to improve coronary and brain perfusion if coordinated with cardiac compression (1:1 ratio, with maximal amplification).[433] The ECG trigger of the balloon is not reliable during resuscitation and should be switched to pressure trigger mode, or to internal mode at 100 beats min^{-1} if massage is interrupted for a significant interval.

Extracorporeal CPR

ECPR may be considered if resternotomy fails to revert cardiac arrest or as an initial approach, alternative to resternotomy, for patients undergoing minimally invasive cardiac surgery or those who arrest >10 days after initial sternotomy.[405] However, there is limited data addressing this specific scenario, since most studies relate to cardiogenic shock or paediatric populations.[434]

Insert modified LVAD algorithm

Mechanical circulatory support devices especially left ventricular assist devices (LVAD) are being increasingly used as a bridge to heart transplantation or recovery in advanced heart failure patients. Patients with these devices often lack traditional clinical signs such as a pulse, even when haemodynamically stable and therefore assessment of the circulatory state using palpation, non-invasive blood pressure measurement and pulse oximetry may be difficult. Recognition of cardiac arrest is based on absence of responsiveness and breathing. When performed, conventional CPR is recommended. The risk of device dislodgement during CPR appears minimal.[406, 407]

The ERC recommendations are based on the ILCOR 2025 CoSTR and British Societies guidelines 2025[406, 407] on the management of emergencies in implantable LVAD recipients in transplant centres, the algorithm from which is reproduced below.

- Immediate activation of specialised teams for unresponsive LVAD patients.

- Start CPR while simultaneously attempting to restore device if multiple rescuers are available. Consider delaying CPR for up to 2 minutes to attempt device restoration if a single rescuer is present.
- Troubleshoot device issues as a priority, following relevant protocols:
  - Low Flow Alarms: Check for volume responsiveness with leg raise technique. If alarm resolves and hypovolaemia is confirmed, proceed with fluid resuscitation. Check haemoglobin values as soon as possible to evaluate the context of hypovolaemia haemorrhage or dehydration. In cases of low flow resulting from high blood pressure, manage afterload with antihypertensives.
  - Pump Thrombosis: Administer anticoagulation or consider mechanical circulatory support like ECMO.
  - Electrical Failure: Ensure connection to a power source and integrity of powerlines. Replace the batteries or the controller.
  - Arrhythmias: Defibrillation or cardioversion should be performed if required, ensuring adequate sedation if the patient is still conscious. Remember a functioning LVAD may provide sufficient brain perfusion even in fibrillating hearts.
- Simultaneously (if second responder available) address Airway, Breathing, Cardiac (Monitor/Defibrillator)
- Determine the adequacy of circulation. Evaluate the patient for MAP >60 mmHg, absence of cyanosis, and presence of an audible LVAD hum. Echocardiography and invasive monitoring may be required for precise assessment. In cases of inadequate circulation, consider temporary mechanical support or corrective surgery. If device
  - Is working well and patient not in circulatory failure: Perform a complete ABCDE assessment
  - Is still not working or patient is in circulatory failure: Start CPR and
    -consider chest reopening if <10 days from implantation
    - deliver standard ALS, addressing 4H & 4T

[h2] Cardiac arrest in sports

The absolute risk of experiencing of sudden cardiac arrest during physical exercise is small.[435] The incidence of sudden cardiac death associated with sport or exercise in the general population is reported between 0.46 and 6.8 per 100,000 person-years.[436-439] Large population-based studies of sudden cardiac arrest in athletes from the USA and Europe

indicate regional differences in the underlying causes of death.[440, 441] About a third survived; most cases occurred during non-elite competitive or recreational sports, and bystander CPR was performed in 75%. In patients aged ≤35 years, premature coronary artery disease and sudden arrhythmic death syndrome predominated, followed by myocarditis. In athletes aged ≥ 35 years, coronary artery disease predominated.[442] The likelihood of sport-related sudden cardiac arrest is highest for males aged between 40 and 60 and with pre-existing cardiomyopathy. Interestingly in 22% of cases no pathology was found, and survival was better with early resuscitation, use of AED, and presence of professional staff.[443] Highest incidences are reported for football players during competition and for running and gym exercise during non-competition.[444]

Sudden cardiac arrest during sport or exercise requires rapid recognition and effective treatment if the affected individual is to survive. Improved survival of sudden cardiac arrest during sport is attributed to witnessed events, prompt resuscitation and availability of an AED.[436, 445] This evidence supports the importance of planning, adhering and implementing standard BLS in event-prone sport events.

The medical team should consider moving the athlete in cardiac arrest to a designated rendezvous point, in the safest and most efficient way. If that is not possible, CPR should be continued on the field.[446] These plans should be rehearsed regularly.

Unlike in professional sports, recreational athletes, supporters and fans may be less aware of the risk of sudden cardiac arrest, highlighting the importance of awareness programs. Initiatives such as the ERC–UEFA cooperation in 2024 during the European championships demonstrate how strategic collaborations between sports and health organisations can raise awareness and improve outcomes.[447] Once an athlete is successfully resuscitated, thorough cardiological assessment and follow-up are vital before considering a return to sport.[448]

[h3] Prevention

There is no consensus between the major organisations on preventative measures. The International Olympic Committee recommend cardiac screening for athletes. The European Society of Cardiology recommends 12-lead ECG as a screening tool for all young athletes,[448] but the AHA and American College of Cardiology conclude that there is insufficient evidence to support this as a screening measure.[449, 450] Processes applied often follow economic views rather than a health care perspective.Primary prevention includes a targeted history and a physical examination, and the addition of a 12-lead ECG to at least prevent some

unnecessary deaths, but this may still overlook several important conditions associated with sudden cardiac arrest including high-risk anomalous coronary origins, aortopathies and adrenergically-mediated arrhythmias. Beyond an ECG, there are insufficient data to support additional routine testing.[451]

For older participants in sports and exercise, a medical evaluation of their individual risk should include the current level of physical activity, their known cardiovascular, metabolic, or renal disease, the presence of the signs or symptoms suggestive of cardiovascular disease and the desired or anticipated exercise intensity.[452] In this context, screening approaches should be adapted to the specific characteristics and risk profile of the target population.[448, 451]

[h3] Commotio cordis

Commotio cordis is a rare but potentially fatal cause of sudden cardiac arrest, triggered by a blunt, non-penetrating impact to the precordium, typically occurring within a 20-millisecond window during the rising phase of the T wave, inducing VF.[453, 454] It has historically been cited as a leading cause of sudden cardiac arrest in young athletes, particularly in sports involving high-velocity projectiles such as baseball, lacrosse, and hockey[455, 456]. In football, commotio cordis has been reported in 9% of sudden cardiac arrests in players ≤35 years old, with 79% of cases caused by a ball impact to the chest.[443]

Survival rates are approximately 66% in 2008-2023.[455] Early recognition, immediate CPR initiation, and rapid defibrillation with an AED remain the most important factors for survival[455-458] Survival rates are 40% when resuscitation occurs within 3 minutes, but drop to just 5% when delayed beyond this window.[456, 457]

Protective gear, such as chest protectors in lacrosse has been introduced in attempt to prevent commotio cordis, but recent studies suggest that they do not reduce the risk of VF in critical impacts.[459, 460]

[h2] Emergency medical services (EMS) and transportation

EMS play a crucial role in the chain of survival by providing timely, high-quality care to patients experiencing life-threatening emergencies, including cardiac arrest. This chapter provides evidence-based recommendations for resuscitation practices in the context of EMS (including specialised medical transport services) and transportation.

EMS collect valuable data that can support continuous quality improvement and provide feedback to healthcare providers involved in CPR.[461] However, validity of data should be

assessed before use.[462, 463] Feedback systems have a positive effect on the quality of care and medical personnel are willing to receive feedback.[464, 465] A recent systematic review showed improvements in documentation, protocol adherence, and small effects on cardiac arrest performance, clinical decision-making, ambulance times, and survival rates.[466]

[h3] Resuscitation during transport

CPR quality is reduced if performed during transportation, affecting correct hand position, chest compression rate and depth, pauses, and overall CPR quality.[197, 467] Only a few indications justify ongoing resuscitation during transport (advanced interventions not available in the prehospital setting as outlined in this chapter). Whenever possible, mechanical CPR should be implemented to mitigate the risk to both patient and rescuer.

[h3] Arterial line in the prehospital setting

Invasive blood pressure measurement in OHCA patients in a HEMS situation is feasible,[365, 468-472] and can help prehospital teams to guide resuscitation and post-resuscitation care,[365, 468, 470, 473] specifically in haemodynamically unstable patients.[474, 475]

[h3] Resuscitation by two-member ALS crews

Teams larger than two ALS members improve CPR quality and efficiency in rhythm recognition, adrenaline administration, and intubation. [476] No significant no-flow fraction differences were found between two-, three-, and four-member teams. [476] Two-paramedic teams were slower and more error-prone than paramedic-emergency medical technician teams. [476] The evidence is not robust enough to support formal recommendations for training, protocols, or equipment regarding ALS provided by two HCPs. However, studies suggest that pre-filled syringes and automation-assisted protocols might improve team performance.[476]

[h2] Inflight cardiac arrest and microgravity resuscitation

[h3] Inflight cardiac arrest

Although air travel is safe in general, physiological changes during air travel, passenger demographics, pre-existing medical conditions and the number of passengers aboard larger aircraft and long distance flights raise the probability of in-flight emergencies.[477, 478] A meta-

analysis reported an incidence 0.09 cardiac arrest events per million passengers.[479] Not all airlines are equipped with an AED and ALS equipment.[480-482]

Medical professional help should be sought via 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 (e.g. galley). Overhead-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.

[h5] Microgravity resuscitation

CPR is challenging even for trained professionals on earth, and spaceflight adds further complications due to microgravity, confined space, and limited resources. With the rise of commercial missions by private companies, clear guidelines are essential for managing medical emergencies in both long-duration and short-term space missions.

The recommendations for microgravity CPR are based on guidelines from the German Society of Aerospace Medicine and the European Society of Aerospace Medicine Space Medicine Group.[483] . Alternative techniques of CPR including hypo- and microgravity are also summarised in a recent scoping review.[484]

Fig. 1 – The Evetts-Russomano technique (Image: MedizinFoto Uniklinik Koeln).[484]

Fig. 2 – The Reverse Bear Hug technique (Image: MedizinFoto Uniklinik Koeln).[484]

Fig. 3 – The Handstand technique (Image: MedizinFoto Uniklinik Koeln).[484]

Key recommendations are summarised here, for more details see the original guideline.[483]

- The use the Evetts-Russomano method for initial CPR should be used. If chest compressions are inadequate, rescuers should switch to the Reverse-Bear-Hug method.
- Applying chest compressions using the handstand-method if space and provider height allow, once the patient is secured on the Crew Medical Restraint System.
- Airway management, defibrillation and IV/IO access are similar to terrestrial ALS, but only once the patient is secured.

- Consult telemedicine support during cardiac arrest in low earth orbit if feasible and manpower allows.
- The crewmember with the highest medical qualification should decide on termination of resuscitation, consulting telemedicine support.

[h3] Cruise ships

There is limited evidence about treatment of OHCA on cruise ships. The ERC guidelines recommend adhering to standard BLS and ALS protocols. Outcome from cardiac arrest on cruise ships is expected to be worse, compared with the overall population, because access to healthcare facilities is more complicated, on-board resources are limited, and transfers can be prolonged.[485] A medical first-responder team should be available 24/7. All equipment necessary for ALS should be available onboard and readily accessible. An AED should be onboard and requested immediately. Where there are too few numbers of crew health care professionals, an onboard announcement should be made to call for further medical professional help. In most cruise ships telemedicine is available, and it should be used as early as possible.[486] Qualified medical air transportation is an option to cover long distances to medical facilities.

[h1] Special Patient Groups

[h2] Asthma and chronic obstructive pulmonary disease

Evidence based recommendations for the management of acute life-threatening asthma are based on the British Thoracic Society, Scottish Intercollegiate Guidelines Network [487] and the Global Initiative for Asthma Strategy 2021,[488], while for chronic obstructive lung disease (COPD) recommendations are based on those of the Global initiative for chronic obstructive lung disease. [489] A recent ILCOR Evidence Update [5] did not reveal any new data on management of cardiac arrest IN asthma patients beyond the 2021 ERC guidelines. Patients with an exacerbation of obstructive lung disease (asthma/ COPD) are at high risk for cardiac arrest. Further steps in the treatment of acute severe asthma are summarized in Figure1. -> new short algorithm

For COPD, the Global initiative for chronic obstructive lung disease guidelines recommend that supplemental oxygen is titrated to achieve a target saturation of 88-92%, with frequent monitoring of blood gases to ensure adequate oxygenation without carbon dioxide retention. Pharmacological therapy comprises inhaled short-acting beta-2 agonists with or

without short-acting anticholinergics (with repeated dosing as required), systemic corticosteroids and, in case a bacterial infection is suspected, antibiotics. Non-invasive ventilation is recommended in the presence of respiratory acidosis ($PaCO_2$ < 6 kPa (35 mmHg) and arterial pH ≤ 7.35); severe dyspnoea with clinical signs of fatigue or increased work of breathing. Escalation to invasive ventilation may be necessary if NIV fails, if the patient cannot tolerate NIV, or there are factors such as agitation, reduced consciousness, high aspiration risk, cardiovascular instability, or life-threatening hypoxia. However, be alert to the higher risk of life-threatening hypotension after emergency intubation and mechanical ventilation in patients with raised arterial CO_2 and obstructive lung disease.[490, 491] In selected unresponsive cases with severe hypoxemia, Veno-Venous ECMO may be an option to prevent hypoxic cardiac arrest.[492, 493]

[h3] Treatment of cardiac arrest caused by obstructive lung disease

Cardiac arrest in patients with obstructive lung disease may be caused by hypoxia, hypovolaemia, toxic agents (arrhythmias caused by stimulant drugs e.g. beta-adrenergic agonists, aminophylline), electrolyte disturbance (hypokalaemia), tension pneumothorax and/ or the effects of gas trapping leading to reduced venous return and hypotension.[494-499] Cardiac arrest in obstructive lung disease is usually associated with a non-shockable rhythm and poor survival rates. [500, 501]

Because of extremely elevated inflation pressure during severe asthma attacks there is a significant risk of gastric inflation and aspiration with simultaneous hypoventilation of the lungs when attempting to ventilate a severe asthmatic patient with bag valve mask devices. The trachea should be intubated as soon as possible during cardiac arrest caused by asthma, by someone who is trained and competent to do so.

Tension pneumothorax may develop because of high airway pressure, which, if left untreated, may cause cardiac arrest. Check for signs of tension pneumothorax and treat accordingly. [502, 503]. Disconnect from positive pressure ventilation if air-trapping and hyperinflation occurs and apply pressure to manually reduce the hyperinflation. Some case reports have described ROSC in patients with air trapping when the tracheal tube was disconnected from the breathing system.[504-510] If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest while temporarily disconnecting the tracheal tube may relieve air trapping.[504, 507, 509] Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.

Ventilating the lungs with a slower respiratory rate of 8-10 breaths per minute and sufficient tidal volume to cause the chest to rise during CPR should minimise dynamic hyperinflation of the lungs (air trapping).[508]. In mechanically ventilated patients with severe asthma, increasing the expiratory time (achieved by reducing the respiratory rate and changing inspiration to expiration time ratio) provides only moderate gains in terms of reduced gas trapping when a minute volume of less than 10 L min^{-1} is used.[511]

No studies evaluating the use of IV fluids for cardiac arrest caused by obstructive lung disease were identified. Consider giving intravenous fluids to patients with obstructive lung disease, particularly those with an acute exacerbation of asthma, because they be dehydrated as a result of reduced oral intake and/or increased insensible losses. Give fluids cautiously to prevent potential adverse effects.[512]

ECPR has been used successfully in patients with life threatening asthma. [493, 513] Consistent with ALS guidelines, ECPR may be considered if conventional therapies fail and there is immediate access to this treatment.

[h3] Cardiac arrest in haemodialysis patients

Chronic kidney disease is a global health problem affecting 1 in 10 people worldwide[514] and the WHO projects it will become the 5th most prevalent chronic condition by 2040.[515]

Risk factors for cardiac arrest in dialysis patients are summarised in Table XI. Fluid and electrolyte disturbances are common, with the highest risk period being just before the first dialysis session of the week, i.e. on Mondays or Tuesdays.[516-520] The risk period extends over the 12 hours after start of treatment. This suggests that the non-physiological rapid removal of toxins, fluid and electrolytes, most pronounced during the first session of the week, accounts for the high risk period.[521, 522] Clinically significant arrhythmias are also more common on dialysis compared with non-dialysis days.[523]

Risk Factors for Cardiac Arrest in HD Patients

1) Hyperkalaemia
2) Excessive fluid shifts during dialysis
3) The 3-day inter-dialytic interval - 'weekend break'

- In-centre dialysis is delivered 3 days per week (Mon/Wed/Fri or Tue/Thu/Sat)
- highest risk period is just prior to the first session of the week (i.e. Monday or Tuesday) as K^+ level peak and fluid accumulates
- risk extends for 12 hours after initiation of dialysis on the first session of the week after rapid fluid and electrolyte shifts from peak levels

4) Low potassium dialysate fluid (1 mmol/L)
5) History of heart disease
6) QT-prolonging medications
7) Non-compliance with diet and/ or dialysis regimen

Figure x: Risk factors for cardiac arrest in haemodialysis patients

[h3] Out-of-hospital cardiac arrest in haemodialysis patients

Sudden cardiac arrest accounts for 47.1% of deaths in HD patients, occurring 20 times more frequently in dialysis patients compared with the general population.[524-526] Within a dialysis centre, the odds of VF was found to be 5-fold greater in patients during dialysis and 14-fold greater in patients who arrest after dialysis compared with events occurring before dialysis.[517] In a cohort of dialysis patients with a wearable cardioverter defibrillator, VT/VF was the initial rhythm in 78.6%, with most occurring during dialysis.[527]

Study	N=	Before HD	During HD	After HD
Karnik 2001 [518]	400	7%	81%	12%
La France 2006 [519]	38	8%	78%	14%
Davis 2008 [517]	110	10%	70%	20%

Table 10 Timing of cardiac arrest during dialysis in out-patient clinics. Reproduced with permission from UK Kidney Association Hyperkalaemia Guideline in adults.[47]

HD – haemodialysis

A three-fold increase in odds to hospital discharge with favourable neurological outcome has been shown when CPR is initiated by dialysis staff rather than awaiting the arrival of

emergency services.[526] Following cardiac arrest, 24% of haemodialysis patients survived to hospital discharge and 15% still were still alive at 1 year following cardiac arrest.[517] The poor outcome in one report may reflect the low rate of bystander CPR and AED use.[528529]

[h3] In-hospital cardiac arrest in haemodialysis patients

The incidence of in-hospital cardiac arrest (IHCA) in patients receiving haemodialysis (HD) is approximately 20 times higher than in the general population (6.3% vs. 0.3%), with dialysis patients accounting for 17% of all IHCA cases. The incidence of cardiac arrest specifically in dialysis clinics ranges from 3.4 to 7.8 per 100,000 dialysis sessions. Most events are witnessed, with 70–80% occurring during treatment.[517-519530] Multiple studies in HD patients demonstrate ROSC in 44 – 72%[531-533] and survival to hospital discharge ranges from 22-31%.[531-535] Duration of CPR and older age were predictors of a poor outcome.[535] The 'Get With The Guidelines-Resuscitation' registry shows comparable neurological outcomes in HD patients compared with non-dialysis patients (17% vs 16%, p=0.07).[531] These findings suggest that outcomes for HD patients are no worse than other patients dispelling the perception of futility.[536] Nevertheless, a large national registry of IHCA found that dialysis patients had lower overall resuscitation quality compared with case-matched non-dialysis patients.[531] Shortfalls in resuscitation practice in dialysis patients include a delay in initiation of CPR [526] and failure to achieve timely first defibrillation for a shockable rhythm.[516, 526, 531]

[h3] Modifications to cardiopulmonary resuscitation in haemodialysis patients

Figure Y (concise guidelines – resuscitation in dialysis patients).[47]

Start resuscitation by following the standard ALS algorithm. Assign a trained dialysis nurse to operate the HD machine. Stop the HD machine and return blood volume to the patient with a fluid bolus. If the HD machine is not defibrillation-proof disconnect it from the patient in accordance with the International Electrotechnical Committee (IEC) standards. Keep the dialysis access open so that it can be used for drug administration The Kidney Disease Outcomes Initiative guideline on the management of cardiovascular disease in dialysis patients recommends that all dialysis units should have on-site capability for external defibrillation.[537] Most are equipped with AEDs,[526, 538] but manual defibrillators may also be used by trained staff. Dialysis clinics are predominantly nurse-led units and staff training and confidence may influence the rate of nurse-led defibrillation. When dialysis staff apply the AED before arrival of paramedics, there is a greater proportion of shockable primary arrest

rhythms.[517, 526] Given the higher chance of survival with shockable rhythm, avoid delays in defibrillation. All of the reversible causes (4 Hs and 4 Ts) apply to dialysis patients. Electrolyte imbalances and fluid shifts during dialysis are common causes of cardiac arrest. Consider hyperkalaemia if cardiac arrest occurs pre or early in the dialysis session. For management of hyperkalaemic cardiac arrest, refer to Section *Hyperkalaemia*. Consider hypokalaemia if cardiac arrest occurs mid-late or immediately post dialysis.
Dialysis may be required in the early post-resuscitation period, guided by fluid status and serum biochemistry. Patient transfer to an intensive care unit with dialysis facilities is essential.

[h3] Prevention of cardiac arrest in dialysis patients

Avoiding hyperkalaemia and volume overload requires patient adherence and careful dialysis prescription, but may reduce the risk of cardiac arrest.[518, 539, 540] The use of potassium binders on non-dialysis days to facilitate use of a higher dialysate potassium content (3 mmol) can reduce the occurrence of clinically significant arrhythmias (bradycardia, ventricular tachycardia, and/or asystole).[541] The higher frequency of cardiac arrest during dialysis and the equivalent survival in dialysis patients compared with non-dialysis patients may reflect a higher likelihood of a reversible cause (i.e. electrolyte or fluid disturbance).[531] The highest risk period is the 'weekend break.' It is conceivable that removing this inter-dialytic gap with short, frequent (4-5 per week) dialysis sessions may reduce risk of sudden cardiac death, but this is only achievable with home haemodialysis.

[h2] Resuscitation in obese patients

Obesity is defined by WHO as a body mass index exceeding 30 kg m^{-2}.[542] Obesity can increase cardiovascular morbidity and mortality directly and indirectly.[543] Given the global increase in obesity, an ILCOR scoping review assessed the evidence for cardiac arrest treatment and outcomes in obese patients.[544] In adults, the effect of obesity on neurological outcome, survival to hospital discharge, longer term survival (months to years), and ROSC was variable. Few studies reported resuscitation quality indicators, and no studies reported adjustments to resuscitation techniques, or provider outcomes. Current evidence indicates that it is reasonable to use standard resuscitation protocols.

Delivery of effective CPR may be challenging in obese patients.[544] Rescuer fatigue may make it advisable to change the rescuer performing chest compressions more often than every two minutes. The use of mechanical chest compression devices might be considered although body dimensions and slope of the anterior chest wall limit usability of most devices in very obese patients. Obesity is associated with difficult mask ventilation and might pose difficulties with airway management.[545, 546] Experienced providers should secure a patent airway (supraglottic airway devices or tracheal intubation) early to minimise the period of bag-mask ventilation. Although mortality seems to be higher in obese patients than normal-weighted patients receiving ECPR, it should not be withheld in obese patients.[547]

[h2] Resuscitation in patients with pectus excavatum

This chest wall deformation (sometimes associated with congenital heart disease) reduces available space for heart and lungs with direct pressure to these organs and increases the risk of cardiac arrest.[548] The heart is often displaced leftward, which can complicate effective chest compressions. Reports suggest reduced chest compression depth (3-4 cm) to minimise the risk of organ injury in the presence of reduced anteroposterior thoracic diameter . [549, 550] Surgically placed corrective “Nuss bars” complicate CPR, as they require significantly increased force to deliver proper chest compression depth. [551]Rescuer fatigue and organ damage (liver lacerations specifically) are to be considered when mechanical chest compressions are delivered.[552] Defibrillation in the presence of a Nuss bar may divert electrical current flow through the low resistance metallic bar when pads are placed anterolaterally.[553]

Based on very indirect data from case reports, anatomical and simulation studies reporting cardiac arrest in patients with pectus excavatum, the following deviations from the standard algorithm might be considered:

- During CPR, consider a lower chest compression depth of 3-4cm, because the reduced anteroposterior thoracic diameter, limits the effective compression depth achievable and increases the risk of direct trauma to the heart, lungs and great vessels.
- In case of a Nuss bar correction, substantially increased force is required to deliver effective chest compressions because increased chest wall rigidity, potentially accelerating rescuer fatigue.
- Use mechanical chest compressions with caution.

- If chest compressions are ineffective consider early implementation of ECPR to ensure sufficient organ perfusion.
- Use anteroposterior pad placement for defibrillation using standard energies to ensure optimal current flow through the myocardium, despite the presence of the metallic Nuss Bar. [554]

[h2] Cardiac arrest in pregnancy

Maternal cardiac arrest refers to cardiac arrest that occurs at any stage in pregnancy or within six weeks after birth. Maternal mortality remains high, with an estimated 287,000 cases globally in 2020 – equivalent to one death every two minutes. The majority occur in low- and middle-income countries. A significant proportion of maternal cardiac arrests occur outside maternity units. Pregnancy is not always immediately apparent, particularly in its early stages. However, considering pregnancy in reproductive-age patients is essential for identifying reversible causes such as concealed ectopic bleeding and modifiable factors such as aortocaval compression after 20 weeks' gestation.[555]

This guideline has been informed by an ILCOR scoping review [317]and international guidelines.[555-563] Most guidance is based on observational data, expert opinion and physiological principles. This update introduces a new maternal cardiac arrest algorithm, together with key figures and tables to support clinical practice.

US data reveal that 32% of obstetric patients experiencing cardiac arrest have no history of a pre-existing disorder, 36.1% had respiratory insufficiency while 33.3% had hypotension as the most common antecedent conditions. Pulseless electrical activity is the most common rhythm.[564] While there are few data on precise causes of maternal cardiac arrest, reported causes of maternal mortality are thromboembolism (16%), COVID-19 (14%), cardiac disease (13%), mental health conditions (11%), sepsis (9%), epilepsy and stroke (9%), obstetric bleeding (7%), early pregnancy disorders (5%), cancer (3%), and pre-eclampsia (3%). Risk factors included Black and Asian ethnicity, social deprivation, maternal age >35, and obesity.[555] In contrast, global data from low- and middle-income countries identified haemorrhage (27.1%), hypertensive disorders (14%), and sepsis (10.7%) as the leading direct causes of maternal deaths.[565]

The UK Obstetric Surveillance System identified 66 cases of cardiac arrest in pregnancy in a three year period with a survival rate of 58%, poor outcomes linked to OHCA, and delays in perimortem Caesarean delivery.[566] The UK NAP7 survey reported an incidence of

perioperative cardiac arrest in obstetric patients of 7.9 per 100,000 anaesthetic encounters and a survival rate of 82%, with haemorrhage, high neuraxial block, and bradyarrhythmias as the leading causes.[567]

[h3] Prevention of cardiac arrest in the pregnant patient

Maternal cardiac arrest is usually preventable, though in certain causes, such as amniotic fluid embolism, arrest may occur before intervention.[568] Approach any deteriorating pregnant or peripartum patient following the ABCDE format. There is evidence to suggest that implementing an obstetric-specific early warning system enables early recognition of deteriorating pregnant patients.[569-572] Early involvement of an obstetrician, along with simultaneous activation of maternal and neonatal resuscitation teams, is essential.

[h3] Aortocaval compression

Aortocaval compression (i.e., compression of the inferior vena cava and the aorta by the gravid uterus) manifests by the 20th week of gestation. This may significantly reduce venous return and subsequently cardiac output in the supine position.[573, 574] In healthy pregnant patients with preserved intrinsic compensatory mechanisms, the effects of aortocaval compression may be absent or minimal.[575] However, in critically ill or hypotensive patients, aortocaval compression may precipitate cardiac arrest and limit the effectiveness of cardiopulmonary resuscitation.

Aortocaval compression may be relieved by manual left uterine displacement or left lateral tilt, though supporting data are derived from non-cardiac arrest and simulation studies. An ILCOR scoping review on this topic found insufficient evidence to recommend either method over the other during CPR in pregnant patients.[5] Magnetic resonance imaging suggests that a left lateral tilt of approximately 30° is needed to partially relieve inferior vena cava compression. This may be achieved by applying lateral tilt on an operating table or by placing wedges under the right hip for patients on a conventional bed.[576-578] However, wedges are rarely available, and simulation and animal studies have questioned the effectiveness of chest compressions in the left lateral position.[579, 580] Accordingly, the ERC suggest performing manual left uterine displacement in maternal cardiac arrest occurring outside of the operating theatre. It can be achieved by placing one or both hands below the uterus, on the right side of the patient, and pushing upwards and to the left (see Figure 2).[581, 582]

Definitive relief of aortocaval compression may be achieved only by resuscitative hysterotomy.

[h3] Resuscitative Hysterotomy

Resuscitative hysterotomy should be performed at the site of cardiac arrest to achieve ROSC by relieving aortocaval compression.[583] Previous guidelines recommended starting the procedure at 4 minutes and completing uterine evacuation by 5 minutes. [584] The new maternal cardiac arrest algorithm (Figure 1.) shifts the focus to preparing for resuscitative hysterotomy at the earliest opportunity to ensure timely intervention.

- The ILCOR review found insufficient evidence to support a specific time for initiating resuscitative hysterotomy.
- Timely resuscitative hysterotomy depends on clear applied team competences, rapid activation of the systems that needs proper training and rehearsal, and equipment readiness. [585, 586]
- If cardiac arrest persists despite optimal treatment, resuscitative hysterotomy should not be delayed
- Resuscitative hysterotomy might be beneficial, even beyond 5 minutes post cardiac arrest. [566, 587]
- In exceptional cases, ECPR may be initiated immediately, with uterine evacuation postponed due to anticoagulation-related bleeding risks. [588, 589]

While fixed timing for resuscitative hysterotomy is de-emphasised, early intervention remains paramount to maximise the chance of return of spontaneous circulation and reduce the risk of hypoxic brain injury.

[h3] Extracorporeal life support

A retrospective analysis of peripartum patients requiring ECPR from the International Registry of Extracorporeal Life Support Organization, identified 280 patients,[590] with 70% survival. Survival rates were higher when ECPR was initiated prior to cardiac arrest. Therefore, if available, consider starting extracorporeal life support in pregnant patients before the onset of cardiac arrest.

[h3] Modifications to advanced life support in obstetric patients (Table 1., Figure 1).

[h4] Reduce Aortocaval Compression

Reduction of aortocaval compression should be achieved as early as possible and maintained throughout resuscitation.

Insert figure 2 here

[h4] Chest Compressions

A magnetic resonance imaging study of cardiac remodelling in the third trimester of pregnancy showed an increase in left ventricular mass but no cephalad displacement.[591] However, a subsequent transthoracic echocardiography study of left ventricular position during the third trimester demonstrated that it is located approximately 6 cm cranial to the distal tip of the xiphoid process.[592] Currently, there is no strong evidence to support modifying hand position for chest compressions during pregnancy.

With regard to mechanical chest compression devices, there is insufficient evidence to make a recommendation for or against their use.[593]

[h4] Airway Management

Pregnancy increases the risk of aspiration of gastric contents and failed intubation.[594-596] Manage the airway as directed by the ALS guidelines according to skills of the HCP. Early tracheal intubation facilitates oxygenation and ventilation, while protecting against aspiration, but it is considered an expert skill and should be performed in accordance with current obstetric-anaesthesia guidelines.[596]

[h4] Defibrillation

To avoid burns, external fetal monitors should be removed before defibrillation.

Defibrillation energy levels are the same as those for non-pregnant adults.[597]

[h4] Drugs

While there is limited evidence, intravenous or intraosseous access should be established above the level of the diaphragm whenever possible, based on physiological considerations.[598]

[h4] Reversible Causes

The 4Hs and 4Ts are also important considerations in cardiac arrest during pregnancy. They may prompt relevant considerations specific to pregnancy, like hypovolaemia from a bleeding ectopic pregnancy or pulmonary embolism in pregnancy. However, there are also pregnancy-specific causes of cardiac arrest, which are now introduced as the 4Ps, which are:

- Pre-eclampsia and eclampsia,
- Puerperal sepsis,

- Placental and uterine complications,
- Peripartum cardiomyopathy.

The ERC recommends following existing guidance on the management of pre-eclampsia and eclampsia .[599] Table 2 outlines potentially reversible causes of cardiac arrest in obstetric patients, including the 4Hs, 4Ts, and 4Ps.

Haemorrhage - is a leading cause of maternal morbidity and mortality.[600] Life-threatening haemorrhage can occur both antenatally and postnatally. Ensure that a massive/major obstetric haemorrhage protocol is available in all units, and that it is reviewed regularly and rehearsed in collaboration with all team members including transfusion services and portering staff.[601]

ERC recommends prophylactic administration of tranexamic acid, based on meta-analyses showing a significant reduction in estimated blood loss .[602-604] Follow existing guidelines for the management of major obstetric haemorrhage.[601, 605]

Cardiovascular Disease

Percutaneous coronary intervention is the preferred reperfusion strategy for STEMI in pregnancy.[562] Consider thrombolysis if timely PCI is unavailable. A review of 200 cases of thrombolysis for massive pulmonary embolism in pregnancy reported maternal mortality rate of 1% supporting its safe use in pregnancy when clinically indicated.[606] Point-of-care ultrasound is invaluable in cases where it is unclear if the cause of arrest is due to concealed bleeding or thromboembolism and should be used by an experienced HCP.[555]

[h3] Maternal considerations for post resuscitation care

Hypothermic temperature control has been used safely in early pregnancy with fetal heart monitoring, resulting in favourable maternal and fetal outcomes.[607, 608] The continued focus should be to resuscitate the mother. Once the mother is stabilised, fetal resuscitation will also be optimised. This includes treating maternal pathology, optimising maternal physiology, and ensuring necessary investigations and imaging. Preparation for major obstetric haemorrhage in anticipation of ROSC is essential following resuscitative hysterotomy or thrombolysis, as well as for other maternal cardiac arrest causes such as postpartum haemorrhage or amniotic fluid embolism.

Maternal critical illness is a multidisciplinary approach, making it essential to involve all appropriate specialists (obstetricians, anaesthesiologists, critical care specialists).[609]

The psychological impact of adverse maternal outcomes should be acknowledged, with support offered to the patient, their family, and the staff involved in their care.

[h3] Preparation for cardiac arrest in pregnancy

Gaps in preparedness, including equipment availability and multidisciplinary coordination, remain widespread and may hinder advanced life support in pregnancy, including timely resuscitative hysterotomy and neonatal resuscitation.[610]

A pregnant woman can deteriorate to cardiac arrest anywhere and medical facilities and services must be prepared. There should be:[611-616]

- Plans and equipment in place for resuscitation of both the pregnant woman and newborn (see table 3).
- Early involvement of obstetric, anaesthetic, critical care, and neonatal teams.
- Regular multidisciplinary training in obstetric emergencies, using simulated resuscitation scenarios. [611, 613]
- Review and debriefing following clinical events to support learning and team performance

[h1] Conflict of Interest statement

CL is ERC Board Director of Training and Education. VK is YoungERC committee member and SEC-BLS member, CAG is SEC-BLS member.-RG is chair of the ILCOR Task Force EIT, and ERC Board Director of Guidelines and ILCOR.

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