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Practice Guideline

European Resuscitation Council Guidelines 2025 Special Circumstances in Resuscitation

Carsten Lott[a,*,1], *Vlasios Karageorgos*[b,c,1], *Cristian Abelairas-Gomez*[d,e], *Annette Alfonzo*[f], *Joost Bierens*[g,h], *Steve Cantellow*[i], *Guillaume Debaty*[j], *Sharon Einav*[k], *Matthias Fischer*[l], *Violeta González-Salvado*[m,n], *Robert Greif*[o], *Bibiana Metelmann*[p], *Camilla Metelmann*[p,q], *Tim Meyer*[r], *Peter Paal*[s], *David Peran*[t], *Andrea Scapigliati*[u,v], *Anastasia Spartinou*[b,w], *Karl Thies*[x], *Anatolij Truhlar*[y,z,aa], *Charles D. Deakin*[ab]

Abstract

These European Resuscitation Council (ERC) Guidelines 2025 on Special Circumstances in Resuscitation are based on the 2025 Consensus on 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 Paediatric Life Support cover the special circumstances in children.

Keywords: Cardiac arrest, Special circumstances, Adults, Special causes, Special settings, Special patient populations

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.

Abbreviations: ABC, Airway-Breathing-Circulation, ACE-I, Angiotensin converting enzyme inhibitors, AED, Automated external defibrillator, AFE, Amniotic fluid embolism, ALS, Advanced life support, BLS, Basic life support, CAB, Circulation - Airway - Breathing, CAD, Coronary artery disease, CKD, Chronic kidney disease, COPD, Chronic obstructive pulmonary disease, CPR, Cardiopulmonary resuscitation, CoSTR, Consensus on 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, $ETCO_2$, End tidal carbon dioxide, HCP, Healthcare professionals, HD, Haemodialysis, ICU, Intensive Care Unit, IHCA, In-hospital cardiac arrest, ILCOR, International Liaison Committee on Resuscitation, 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, SCD, Sudden cardiac death, STEMI, ST-elevation myocardial infarction, TCA, Traumatic cardiac arrest, TOE, Transoesophageal echocardiography, VA-ECMO, Veno-arterial extracorporeal membrane oxygenation, VF, Ventricular fibrillation, VT, Ventricular tachycardia, WHO, World Health Organization

* Corresponding author.
E-mail address: lott@uni-mainz.de (C. Lott).

[1] Both share the first authorship.

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3. Special patient populations, characterised by pre-existing conditions or comorbidities that require different modified treatment strategies.

These ERC Guidelines 2025 on Special Circumstances in Resuscitation are based on the 2025 annual Consensus on Science with Treatment Recommendations (CoSTR) of the International Liaison Committee on Resuscitation (ILCOR).[1] 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 Table 1. The broader methodology used for guideline development is presented in the Executive Summary.[2] These Guidelines were posted for public comments between 15 May and 30 May 2025. A total of 54 individuals submitted 66 comments, leading to 9 changes in the final version. Subsequently, the feedback was reviewed by the writing group, and the Guidelines was thereafter updated where relevant. The Guidelines were presented to, and approved by, the ERC Board and the ERC General Assembly in June 2025.

For the purpose of these Guidelines, 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 these ERC Guidelines 2025 on Special Circumstances in Resuscitation 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 (see Fig. 1). Table 1 showes changes in the ERC Resuscitation Guidelines Special Circumstances from 2021 to 2025.

Table 1 – This table provides a synopsis of the major changes in the ERC Guidelines 2025 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	• There is no evidence that supports the routine use of either steroids or antihistamines	• Antihistamine administration should not delay the administration of adrenaline. • The ERC recommends against the routine use of corticosteroids.
Resuscitation in hyper/ hypokalaemia and other electrolyte disorders	• Protect the heart: Give 10 ml calcium chloride 10 % IV by rapid bolus injection. Consider repeating dose if cardiac arrest is refractory or prolonged.	• No evidence for or against use of IV calcium in hyperkalaemia. • ECG – record pre and post IV calcium. • Recommendation against the routine use of sodium bicarbonate in the treatment of hyperkalaemia in non-cardiac arrest cases. • Hyperkalaemia algorithm revised – emphasis on initiation of potassium-lowering treatments. 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, when core temperature cannot be measured. • 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 of arrested hypothermic patients, e.g. unwitnessed cardiac arrest, asystole as presenting arrhythmia. • New avalanche rescue algorithm. • BLS avalanche rescue algorithm for cases with insufficient personnel on site.

Table 1 (continued)

	Summary of changes	
	ERC guidelines 2021	**ERC guidelines 2025**
Pulmonary embolism		• 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, since defibrillation and the time elapsed from ROSC may influence the results • High-certainty evidence from new RCTs discourages routine emergent/early coronary angiography in stable patients without ST-elevation or equivalent on ECG. Immediate angiography is recommended in case of haemodynamic instability or suspected ongoing ischaemia. • Indications for antiplatelet and anticoagulation treatment in this setting.
Toxic agents		• New update on intoxication with opioids.
Traumatic cardiac arrest	• New algorithm established • Invasive procedures introduced	• Algorithm clarified. • Role of chest compressions specified. • 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 in this setting. • General recommendations were given to consider mechanical CPR, extracorporeal CPR and circulatory support devices in the catheterisation lab.	• Guidance on resuscitation team performance in the catheterisation lab: adequate radiation protective equipment, pre-alerting the surgical team if necessary, clearly defining roles and allowing the interventional cardiologist to focus on corrective procedures rather than leading CPR. • Specifies management of extreme bradycardia/ asystole complicating invasive procedures in the catheterisation lab, consider external or transvenous temporary pacing (included in the management algorithm). • Updated evidence on the role of POCUS and specific indications on the use of transoesophageal echocardiography in this setting. • Updated evidence on the role of mechanical CPR, extracorporeal CPR and circulatory support devices in the catheterisation lab, with specific indications for cardiac arrest in this setting. • New limited evidence on the use of intracoronary adrenaline as an alternative administration route for cardiac arrest in the catheterisation lab, highlighting the need for further research.
Drowning	• First responders not covered	• The introduction of the role of first responders in drowning resuscitation. • Clarifications regarding some topics such as the 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 for perioperative cardiac arrest. • 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 algorithm in Left Ventricular Assist Device patients.

(*continued on next page*)

Table 1 (continued)

	Summary of changes	
	ERC guidelines 2021	**ERC guidelines 2025**
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	• New subchapter Emergency Medical Services includes 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 now expanded by CPR in microgravity.
Asthma & COPD		• New shortened algorithm
Resuscitation in haemodialysis patients	• Focus on haemodialysis unit	• Now focusing on haemodialysis patients rather than on the. • Infographic of resuscitation whilst on dialysis.
Resuscitation in obese patients		• No differences – Standard BLS and ALS recommended.
Resuscitation in patients with pectus excavatum	• Not addressed	• Reduced chest compression depth to 3–4 cm. • 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	• Defines 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. • Recommends delivery within 5 min of collapse if no ROSC. • Use of 4Hs and 4Ts.	• Addendum to the ALS algorithm introducing maternal reversible causes ("4P" – (pre-eclampsia/ eclampsia, puerperal sepsis, placental/ uterine issues, peripartum cardiomyopathy) • adds global mortality statistics and highlights that pregnancy may not be clinically obvious • Suggests manual displacement as preferred, due to practicalities. • De-emphasises 5 min; emphasises immediate preparation for resuscitative hysterotomy. • Practical recommendation to remove fetal monitors to prevent burns. • IV/IO access above diaphragm when possible.

Concise guidelines for clinical practice

General recommendations

- Initiate resuscitation following the standard ALS algorithm in cardiac arrest.
- Always address hypoxia, hypovolaemia, electrolyte disorders, hypothermia, cardiac tamponade, tension pneumothorax, thrombosis, and toxic agents.
- Where appropriate, prioritise treating reversible causes, even if chest compressions are briefly interrupted.

Special causes

Management and prevention of cardiac arrest due to anaphylaxis

- 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 if immediately feasible.
- Immediately inject intramuscular adrenaline 0.5 mg at first suspicion of anaphylaxis and repeat if no improvement occurs within 5 min.
- Give an IV crystalloid fluid bolus early and monitor the response.

Hyper/hypokalaemia and other electrolyte disorders

Hyperkalaemia (Fig. 2)

- Shift potassium into cells
  - o Give 10 units soluble insulin and 25 g glucose IV for treatment of moderate and severe hyperkalaemia. Follow with 10 % glucose infusion at 50 ml/hr for 5 h if pre-treatment blood glucose < 7 mmol/l.
  - o Give nebulised salbutamol (10–20 mg) for moderate and severe hyperkalaemia, as an adjunct to Insulin-glucose therapy.
- Antagonise the effect of hyperkalaemia

SPECIAL CIRCUMSTANCES IN ADULT RESUSCITATION
KEY MESSAGES

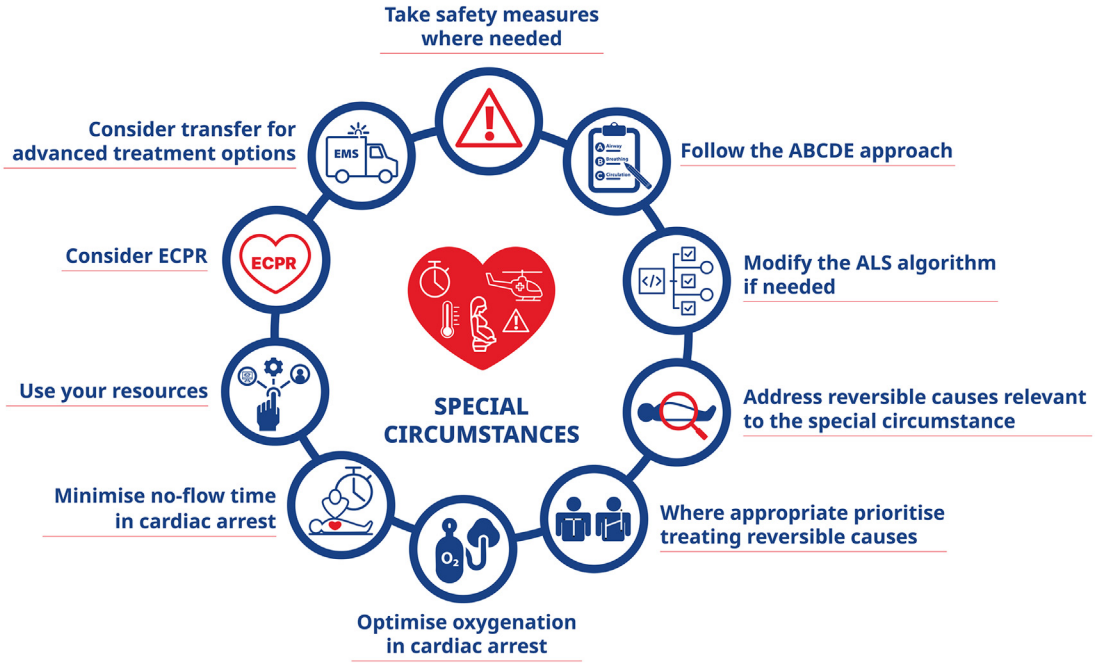

Fig. 1 – Key messages of the ERC Guideline 2025 Special Circumstances in Resuscitation.

- Use IV 10 mL 10 % calcium chloride for patients with severe hyperkalaemia with ECG changes.
- Administer IV 10 mL 10 % calcium chloride and 50 mmol sodium bicarbonate, through separate lines or with flush in between, in the treatment of hyperkalaemic cardiac arrest in all settings of severe hyperkalaemia.

- Remove potassium from the body
  - Give sodium zirconium cyclosilicate 10 g orally.
  - Consider dialysis for patients with refractory severe hyperkalaemia.
- Consider ECPR in accordance with local protocols if initial resuscitation attempt is unsuccessful.

Hypokalaemia

- Treatment is guided by the severity of hypokalaemia and presence of symptoms and/or ECG abnormalities.
- Where appropriate replace potassium and correct magnesium deficit concurrently.
- Give IV 20 mmol potassium chloride over 2–3 min, followed by 10 mmol over 2 min in hypokalaemic cardiac arrest then monitor K^+ level and adjust infusion rate accordingly.

Hyperthermia, malignant hyperthermia and toxin-induced hyperthermia

Hyperthermia

- Measure core temperature to guide treatment.
- Move patient to a cool environment.
- Simple external cooling may involve conductive, convective and evaporative measures (See ERC Guidelines 2025 First Aid).[3]
- 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 application of ice and cold-water immersion (Fig. 3).

Malignant hyperthermia

QR code. 1 – Malignant hyperthermia algorithm.
Treatment of malignant hyperthermia, modified from (Hopkins et al., 2021)[91] and (Kollman-Camaiora et al., 2017).[109]

- Stop triggering agents immediately. This includes turning off and removing vaporiser and changing the ventilator circuit.
- Give IV 2.5 mg/kg dantrolene as soon as possible.
- Start active cooling.
- Give 100 % oxygen and aim for normocapnia using hyperventilation.
- Change the ventilator. If the ventilator cannot be changed, change charcoal filters

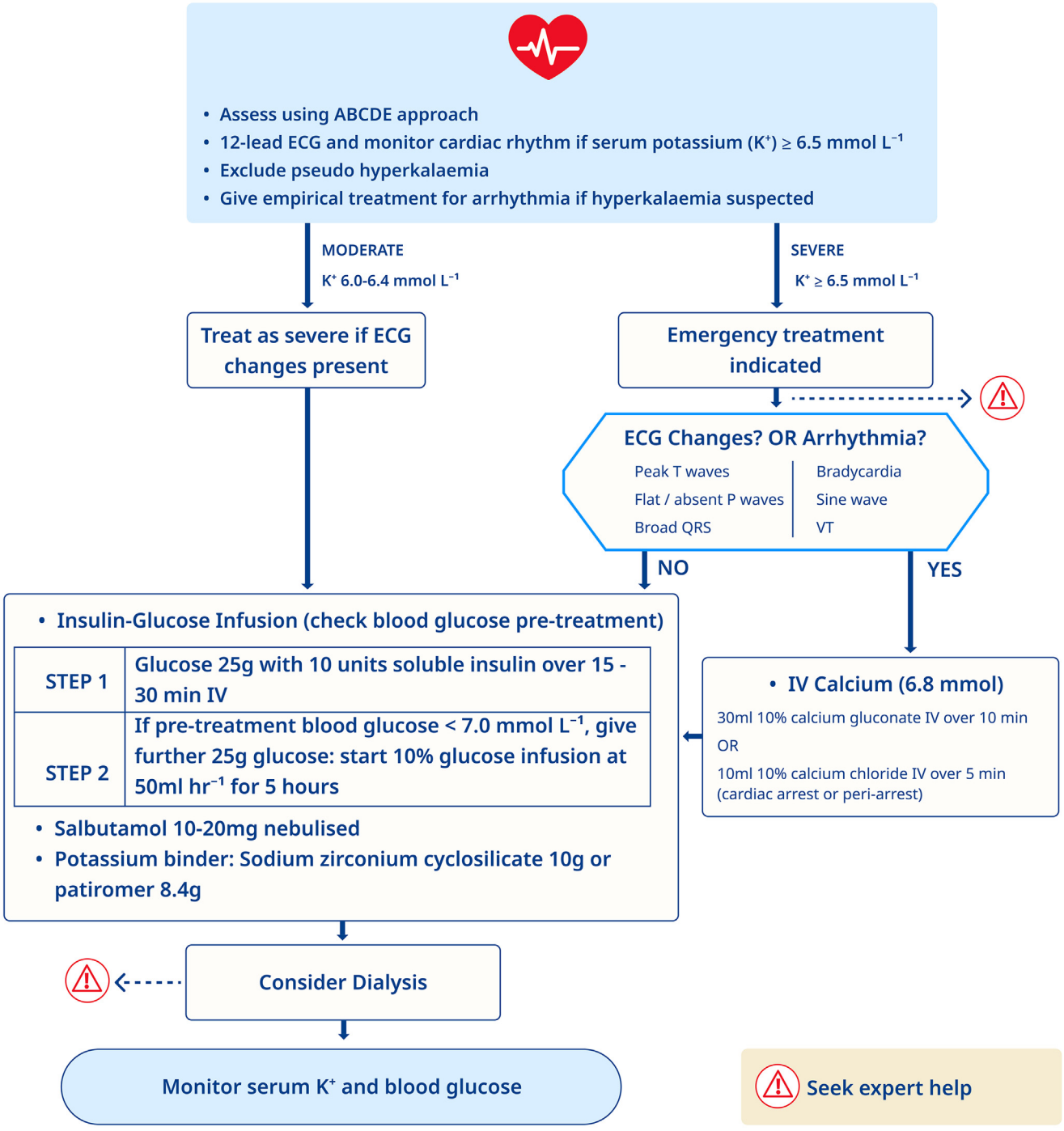

Fig. 2 – Treatment algorithm of hyperkalaemia in adults.
ABCDE – airway breathing circulation disability exposure, ECG – electrocardiography, IV – intravenous, VT – ventricular tachycardia, K – potassium.

- Contact a malignant hyperthermia centre for advice and follow-up.

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.

Accidental hypothermia and avalanche rescue

Accidental hypothermia

- Check vital signs for up to one minute in an unconscious hypothermic patient (Fig. 5).
- Measure core temperature with a low reading thermometer to diagnose accidental hypothermia.
- Use the Swiss Staging System if core temperature cannot be measured.

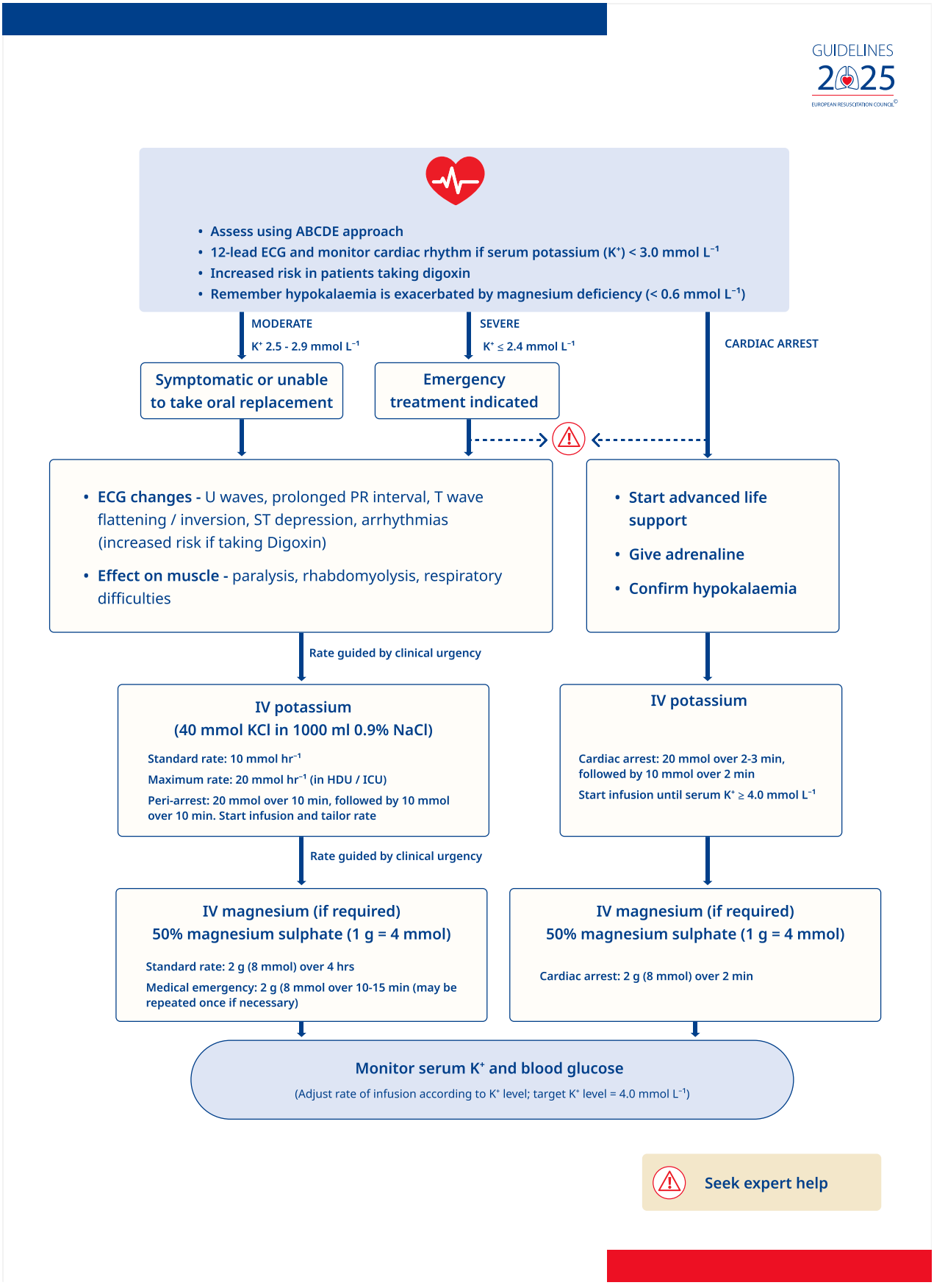

Fig. 3 – Treatment algorithm of hypokalaemia in adults.
ABCDE – airway breathing circulation disability exposure, ECG – electrocardiography, IV – intravenous.

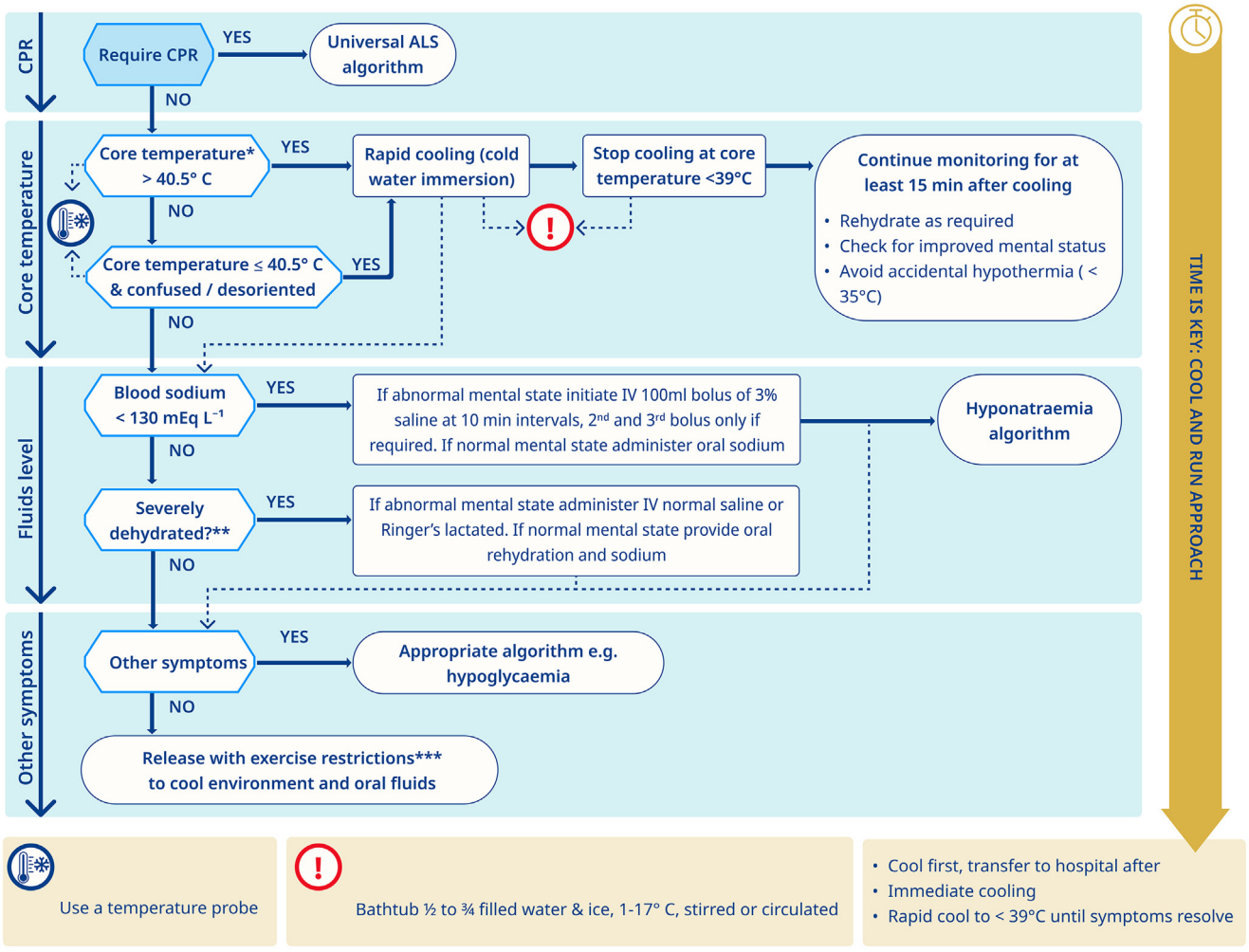

Fig. 4 – Emergency treatment of hyperthermia.
CPR – cardiopulmonary resuscitation, ALS – advanced life support. Footnote: *Core temperature should be measured, e.g. tympanic, rectal, or oesophageal. ** Dry mouth, thirsty, hypotensive. ***Return to activity considerations following a diagnosis of exertional heat illness and exertional head stroke have been published recently elsewhere. [96,99.]

- Transfer hypothermic patients with risk factors for imminent cardiac arrest and those in cardiac arrest directly to an extracorporeal life support (ECPR) centre for rewarming.
- Delay CPR or use intermittent CPR in hypothermic cardiac arrest patients with a core temperature below 28 °C when immediate or continuous CPR is not feasible.
- 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 IV 1 mg adrenaline once to facilitate ROSC unless planning imminent initiation of ECPR. 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 when 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 extracorporeal membrane oxygenation (VA-ECMO).
- Initiate non-extracorporeal life support rewarming if an ECPR centre cannot be reached within a reasonable time (e.g. 6 h).

Avalanche rescue

- Base initiation of CPR in cardiac arrest on core temperature, burial time, and airway patency (Fig. 6).
- Consider proceeding according to the AvaLife algorithm, in multi-burial avalanche accidents with BLS providers only and insufficient numbers of rescuers.

Thrombosis

Pulmonary embolism

- 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 ventricular strain).

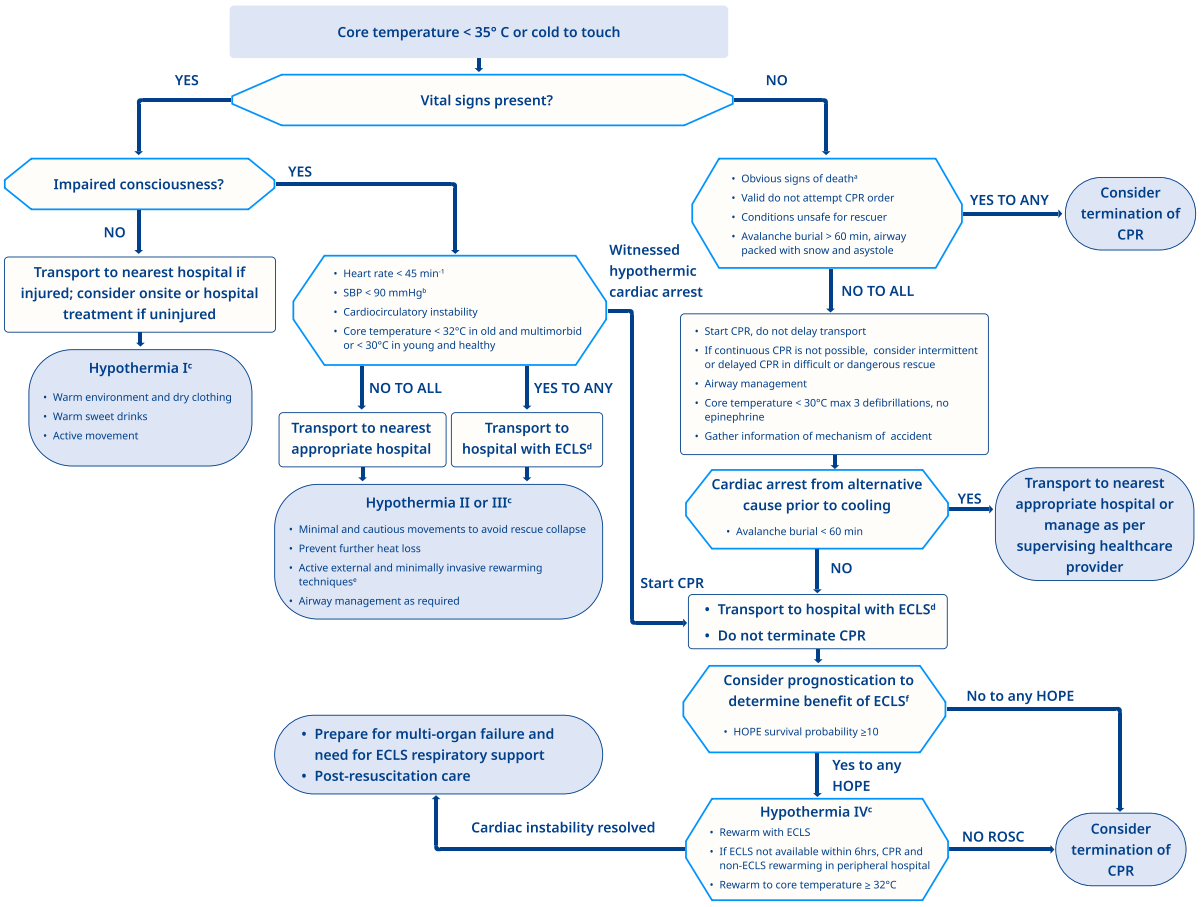

Fig. 5 – Emergency treatment of accidental hypothermia.

[a] Decapitation; truncal transection; whole body decomposed or whole body frozen solid (chest wall not compressible). [b] Systolic blood pressure < 90 mmHg is a reasonable prehospital estimate of cardiocirculatory instability but for in-hospital decisions, the minimum sufficient circulation for a deeply hypothermic patient (e.g., <28 °C) has not been defined. [c] Swiss staging of accidental hypothermia. [d] Direct transport to an ECLS centre is recommended in an arrested hypothermic patient. In remote areas, transport decisions should balance the risk of increased transport time with the potential benefit of treatment in an extra corporal life support centre. Transfer should be commenced if such a centre can be reached within 6 h. [e] Warm environment, chemical, electrical, or forced air heating packs or blankets, and warm IV fluids (38–42 °C). In case of cardiac instability refractory to medical management, consider rewarming with extra corporal life support. [f] If the decision is made to stop at an intermediate hospital to measure serum potassium, a hospital enroute to an extra corporal life support centre should be chosen. The HOPE score [156,158,162] should not be used in children, consider expert consultation instead. ECLS extracorporeal life support, ROSC return of spontaneous circulation, SBP systolic blood pressure.

- 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.
- Consider surgical embolectomy or catheter-directed treatment as alternative to rescue fibrinolytic therapy in rapidly deteriorating patients.

Cardiac arrest due to pulmonary embolism.

- 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 is implemented.

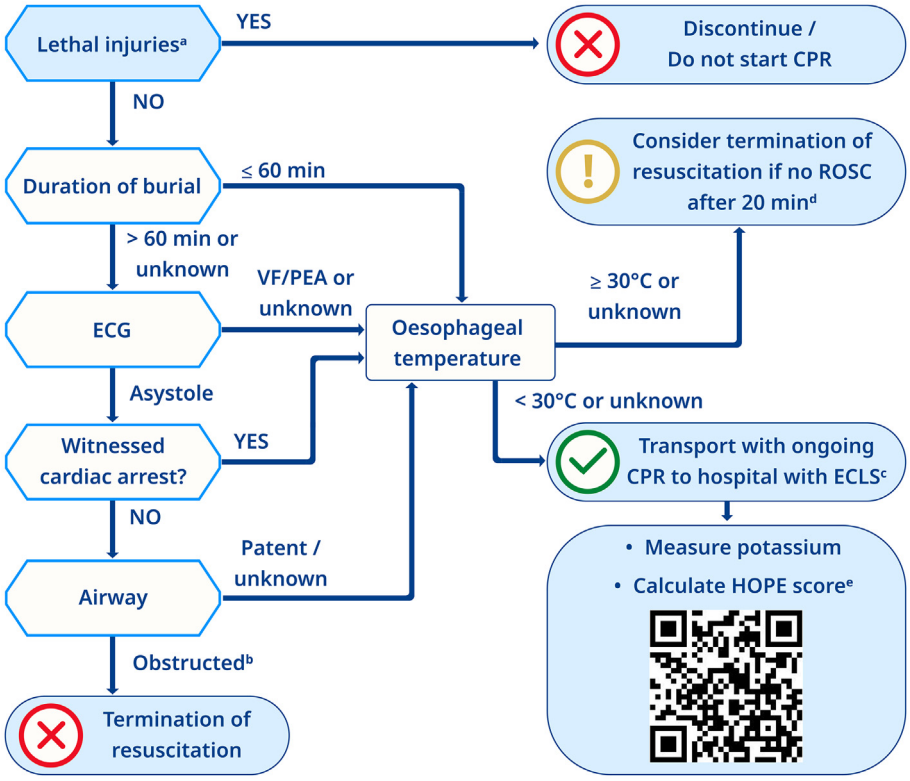

Fig. 6 – Decision-making algorithm for advanced management of critically buried avalanche victims in cardiac arrest.[188]

CPR – cardiopulmonary resuscitation, ROSC – return of spontaneous circulation, ECLS – extracorporeal life support, HOPE – Hypothermia Outcome Prediction after ECLS rewarming for hypothermic arrested patients, ECG – electrocardiography. [a] Decapitation, truncal transection, whole body decomposed. [b] An obstructed or blocked airway requires that both the nose and mouth be completely filled with compact snow or debris. [c] With a deeply hypothermic patient (<28 °C), if rescue is too dangerous consider delayed CPR and if transport is difficult consider intermittent CPR. [d] If core temperature measurement is not available, hypothermic CA may be considered, at the rescuer's discretion, despite a burial duration of < 60 min in a victim with a patent airway and no vital signs when there is a possibility if very rapid cooling (e.g. burial during ascent, low body mass index or small persons, minimally dressed, sweating before burial). [e] In-hospital prognostication of successful rewarming in an avalanche victim should include estimation of the survival probability using the HOPE score. If any doubt exists whether the avalanche victim may have asphyxiated despite critical burial, the HOPE score should be calculated using the NON-ASPHYXIA option. This will reduce the risk of under-treatment. If the HOPE score cannot be determined, the combination of potassium < 7 mmol L^{-1} and a temperature < 30 °C may be used instead to help indicate ECLS rewarming.

- Set-up a multidisciplinary team for making decisions on management of high-risk pulmonary embolism depending on local resources.

Coronary 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:
  - o Perform immediate coronary angiography (and PCI if required) within 120 min of diagnosis.
  - o Consider fibrinolysis in pre-hospital and non-PCI-capable settings if greater delay is expected.
- In patients with sustained ROSC and no ST-elevation on ECG:
  - o Consider immediate coronary angiography (and PCI if required) if the patient is haemodynamically unstable or shows signs of ongoing ischaemia.
  - o 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.
  - o Assess for non-coronary-causes if the clinical context suggests an alternative aetiology of the arrest.
  - o Unless on-going resuscitation is considered futile, transfer patients without sustained ROSC with ongoing CPR, to a PCI centre for consideration for angiography or ECPR depending on available resources and team expertise.

Toxic agents

- Ensure your personal safety (Fig. 7), 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.

Traumatic cardiac arrest (TCA)

- Traumatic cardiac arrest is different from cardiac arrest due to medical causes; this is reflected in the treatment algorithm (Fig. 8).
- 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.

Special settings

Cardiac arrest in the catheterisation laboratory

- Promote adequate training of the staff in technical skills and ALS, 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.

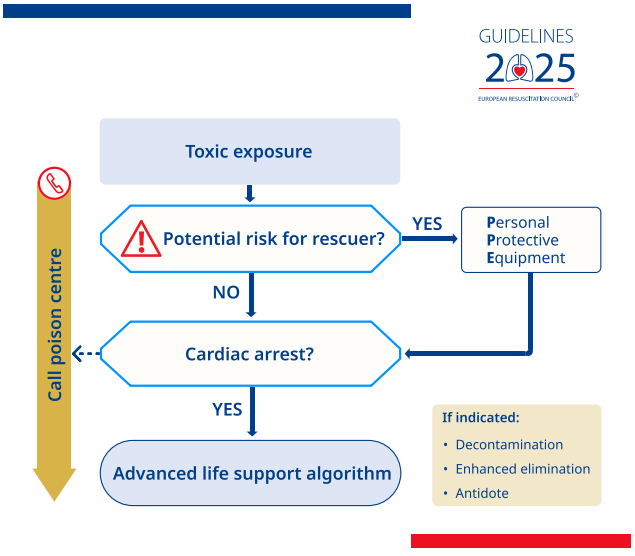

Fig. 7 – Management of toxic exposure.

Drowning

- Rescuers 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.
- Start with 5 ventilations 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.
- Follow the recommendations for hypothermia.

Cardiac arrest in the operating room (OR)

- Prevent and mitigate the risk of cardiac arrest through preoperative screening and identification of high-risk patients, clear communication with surgeons 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 compression 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 ALS courses.

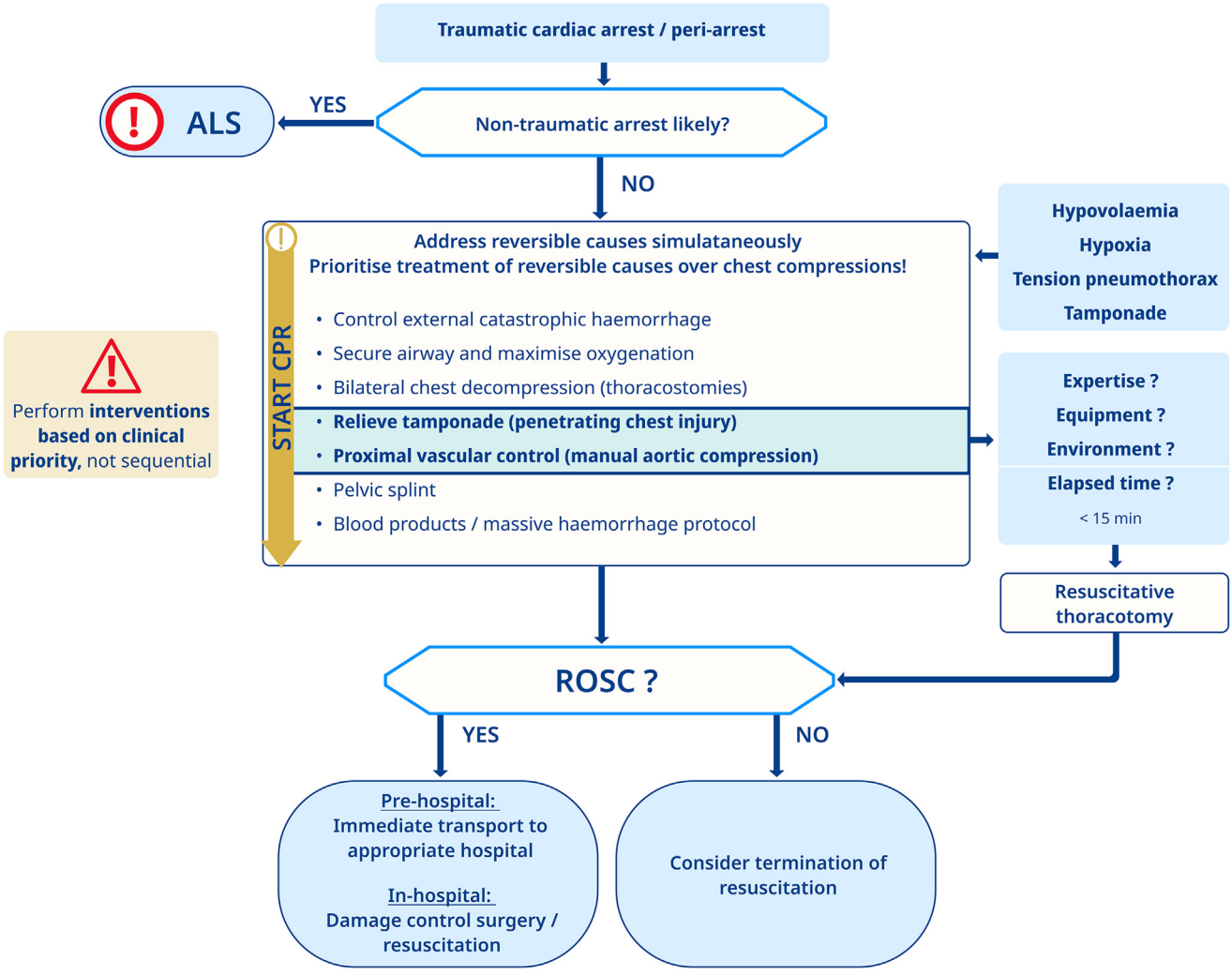

Fig. 8 – Traumatic cardiac arrest algorithm.
ROSC – return of spontaneous circulation, ALS – advanced life support. This algorithm should be used to guide the management of traumatic cardiac arrest (TCA) and peri-arrest trauma patients with impending circulatory collapse. In contrast to the sequential ALS algorithms, it is a framework for context-sensitive decisions on clinically prioritised interventions. The aim is to treat the most urgent, reversible cause based on individualised treatment (e.g. patients with a cardiac tamponade need urgent thoracotomy; patients with pelvic fracture in haemorrhagic shock but with a stable airway may benefit more from immediate application of a pelvic binder and rapid transfer without airway intervention).

Local anaesthetic systemic toxicity

- Stop the local anaesthetic if possible.
- Hyperventilate the patient to increase plasma pH if metabolic acidosis is present.
- Give a lower adrenaline dose ($\leq$1 μg 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 0.25 mL kg^{-1} min^{-1} but do not exceed a maximum cumulative dose of 12 mL kg^{-1} IV 20 % lipid emulsion.
- If ROSC has not been achieved at 5 min, double the rate of lipid infusion and give a maximum of two additional lipid boluses at 5-minute intervals until ROSC has been achieved.
- Consider prolonged resuscitation (>1 h) and ECPR
- Treat seizures by administering benzodiazepines

Cardiac surgery

- 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 min regardless of the patient's location.

- Provide open cardiac compressions once the chest is reopened.
- Reduce IV adrenaline dose (0.05–0.1 mg)
- Consider ECPR for prolonged resuscitation or minimally invasive cases where reopening may be delayed

Left ventricular assist device (LVAD) patients

- Immediately activate specialised teams for unconscious LVAD patients.
- Start CPR while simultaneously attempting to restore device function if multiple rescuers are available.
- Consider delaying CPR for up to 2 min to attempt device restoration if a single rescuer is present.
- Troubleshoot device issues as a priority, following relevant protocols.

Cardiac arrest in sports

- 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 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.

Emergency medical services

- 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.

Inflight cardiac arrest and microgravity resuscitation

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.

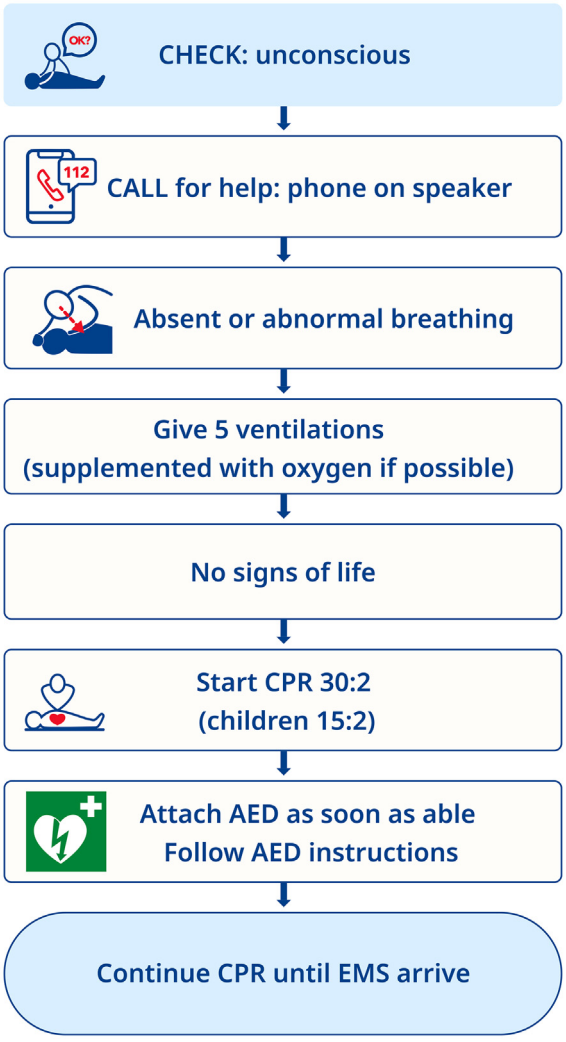

Fig. 9 – CPR after drowning for rescuers with a duty to respond.
CPR – cardiopulmonary resuscitation, EMS – emergency medical services, AED – automatic external defibrillator.

- 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.

Microgravity resuscitation

- Airway management, defibrillation and IV/IO access are similar to terrestrial ALS, but only once the patient is secured.
- Consider mechanical CPR
- 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.

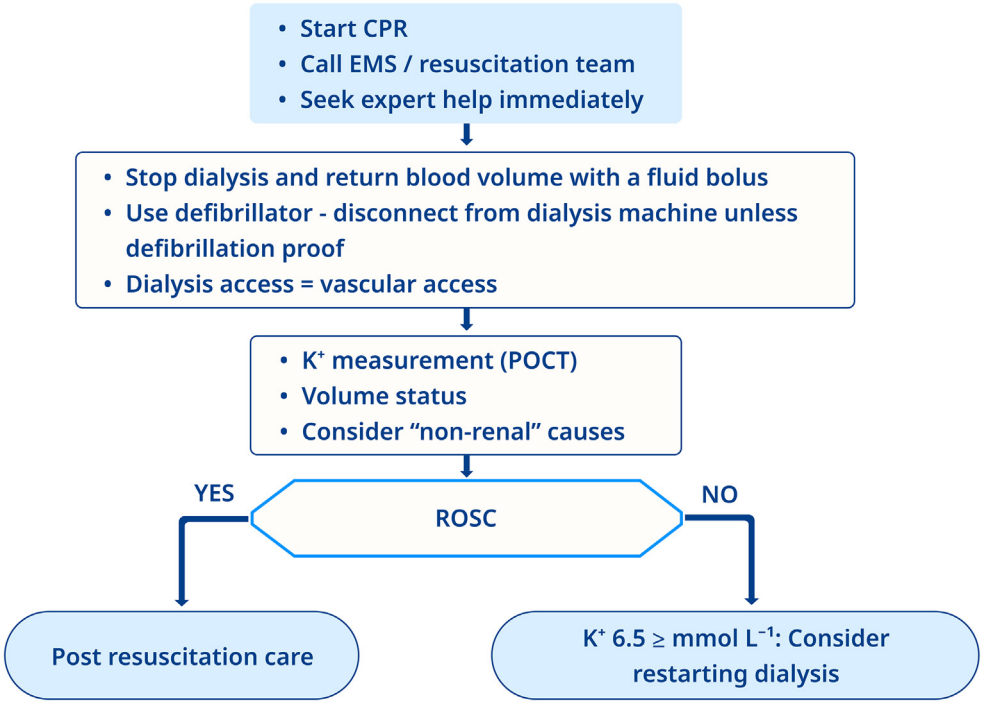

Fig. 10 – Cardiac arrest during dialysis.
Abbreviations: CPR – cardiopulmonary resuscitation; EMS – emergency medical services; K+ – potassium; HD – haemodialysis; ROSC – return of spontaneous circulation; POCT – point of care testing.

Cruise ship

- Use all healthcare resources immediately (personal, equipment).
- Activate helicopter emergency medical service 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.

Special patient groups

Asthma and chronic obstructive pulmonary disease

- 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 ECPR in accordance with local protocols if initial resuscitation efforts are unsuccessful.

Cardiac arrest in haemodialysis patients

- 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.

Resuscitation in obese patients

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

Resuscitation in patients with pectus excavatum

- Consider reduced chest compression depth of 3–4 cm.
- 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.

Cardiac arrest in pregnancy

- Consider pregnancy in any collapsed woman of childbearing age.
- Pregnant and peripartum women can deteriorate to cardiac arrest anywhere and medical facilities and services must be prepared for such events.

- Obstetric-specific early warning systems enable early recognition of deteriorating pregnant patients.
- Relieve aortocaval compression as early as possible and maintain it throughout resuscitation. Manual left uterine displacement in maternal cardiac arrest is suggested due to practicalities.
- Systematically address the 4Hs and 4Ts and seek pregnancy-specific causes of cardiac arrest, the 4Ps: Pre-eclampsia and eclampsia, Puerperal sepsis, Placental and uterine complications and Peripartum cardiomyopathy.
- Resuscitative hysterotomy is a time-sensitive intervention. Preparation to perform the procedure should be undertaken early.
- Resuscitative hysterotomy should be performed as soon as possible at the site of cardiac arrest by a skilled team.
- Post-resuscitation care in pregnant and postpartum women requires a multidisciplinary approach.

The evidence informing the guidelines

Special causes

Management and prevention of cardiac arrest due to anaphylaxis

There is no 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 persons per year in Europe.[4] The most common triggers of anaphylactic reactions are food, medications and hymenoptera venom.[5–7]

This guidance on anaphylaxis is based on the most recent ILCOR CoSTR,[8] guidelines and updates from the World Allergy Organization Anaphylaxis Committee,[9] European Academy of Allergy and Clinical Immunology,[10] North American Practice Parameter,[11] Australasian Society of Clinical Immunology and Allergy,[12] the findings from the UK National Audit Project (NAP 7) of perioperative anaphylaxis.[13] Most of these recommendations are based on observational data, good practice statements and expert consensus.

Recognition of anaphylaxis

According to the World Allergy Organization Anaphylaxis Committee, anaphylaxis is likely when at least one of the following criteria is fulfilled[9]:

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.

The initial approach if suspecting an anaphylactic reaction

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.[14–16] 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 are present without circulation problems.

Give adrenaline

Administer IM adrenaline as first-line drug for the management of anaphylaxis.[17] Initial IM adrenaline both aims provide haemodynamic support as well as acting to inhibit mast cell degranulation. The preferred site is the lateral mid-thigh,[18] standard dose for adults is 0.5 mg.[10] This should be repeated after 3–5 min, according to clinical response.[19] 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 $\mu g\ kg^{-1}\ min^{-1}$ titrated according to response can be administered by appropriately trained personnel when patients are fully monitored.[20]

In a patient with upper airway obstruction or bronchospasm, consider adding nebulised adrenaline.[10,21] However, systemic absorption of inhaled adrenaline is minimal and should not delay concurrent IM adrenaline administration.[22]

Secure vascular access – give intravenous fluids

Anaphylaxis may result in severe hypovolaemia because of peripheral vasodilation and increased vascular permeability (distributive shock).[23,24]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.

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 %.[25]

Other medications to support the circulation

In the case of no response to adrenaline, consider administration of a second vasoactive drug, such as noradrenaline or vasopressin.[10,21]

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.[26]

Corticosteroid administration to prevent protracted symptoms or biphasic reactions has been based on low-certainty evidence.[27,28] 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.[29,30] Based on experts' opinion, the ERC does not recommend the routine use of corticosteroids in the management of anaphylactic reactions.

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.[13]

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 hospitals where this can be implemented.[8]

Hyper/hypokalaemia and other electrolyte disorders

Electrolyte abnormalities are a recognised cause of arrhythmias and cardiac arrest, 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.[31]

Hyperkalaemia has also been subject to a recent ILCOR review (2025).[32]

Hyperkalaemia – Non-cardiac arrest

Hyperkalaemia occurs in 1–10 % of hospitalised patients.[33–35] 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.[36] Dialysis patients are also more prone to severe hyperkalaemia than non-dialysis patients (45.8 % vs 10.3 %) during hospitalisation.[35] In-hospital mortality is significantly higher in patients with hyperkalaemia (18.1 %) compared to those with hypokalaemia (5 %) or normokalaemia (3.9 %).[33] In-hospital mortality is 3.9-fold higher in patients with a serum $K^+ > 6.5$ mmol L^{-1} when compared with normokalaemic patients.[37]

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.[38–40] There is now a role for potassium binders (sodium zirconium cyclosilicate and patiromer) to provide cardio-renal protective therapy.[41,42]

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 mmol L^{-1}), 'moderate' (K^+ 6.0–6.4 mmol L^{-1}), 'severe' ($K^+ \geq 6.5$ mmol L^{-1}), or 'extreme' ($K^+ \geq 9.0$ mmol L^{-1}).

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

Diagnosis of hyperkalaemia

Consider hyperkalaemia in all at risk patients (e.g. renal failure, heart failure, diabetes mellitus, chronic liver disease) with an arrhythmia or cardiac arrest. 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.[43–45]

ECG changes may reflect the severity and rate of rise of serum K^+,[46,47] 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.[46] These ECG signs of hyperkalaemia may develop progressively and include (Fig. 11a):

- 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 > 0.2 s);
- Flattened or absent P waves;
- Widened QRS (>0.12 s);
- 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 h of the presenting ECG.[46] Therefore, delays in treatment may have serious consequences.

Emergency treatment of hyperkalaemia

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 (Fig. 2). 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.[32,48,49]

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}.[32,50] Studies have suggested a possible dose-dependent effect of insulin[51–55] and a potential correlation between the severity of hyperkalaemia and the degree of K^+-lowering with 10 units insulin.[56] These findings are particularly relevant in the resuscitation setting.

Hypoglycaemia remains a major iatrogenic risk with a reported incidence of up to 28 %.[53,57–62] Reducing the dose of insulin (5 units) did not significantly lower the risk of hypoglycaemia (blood

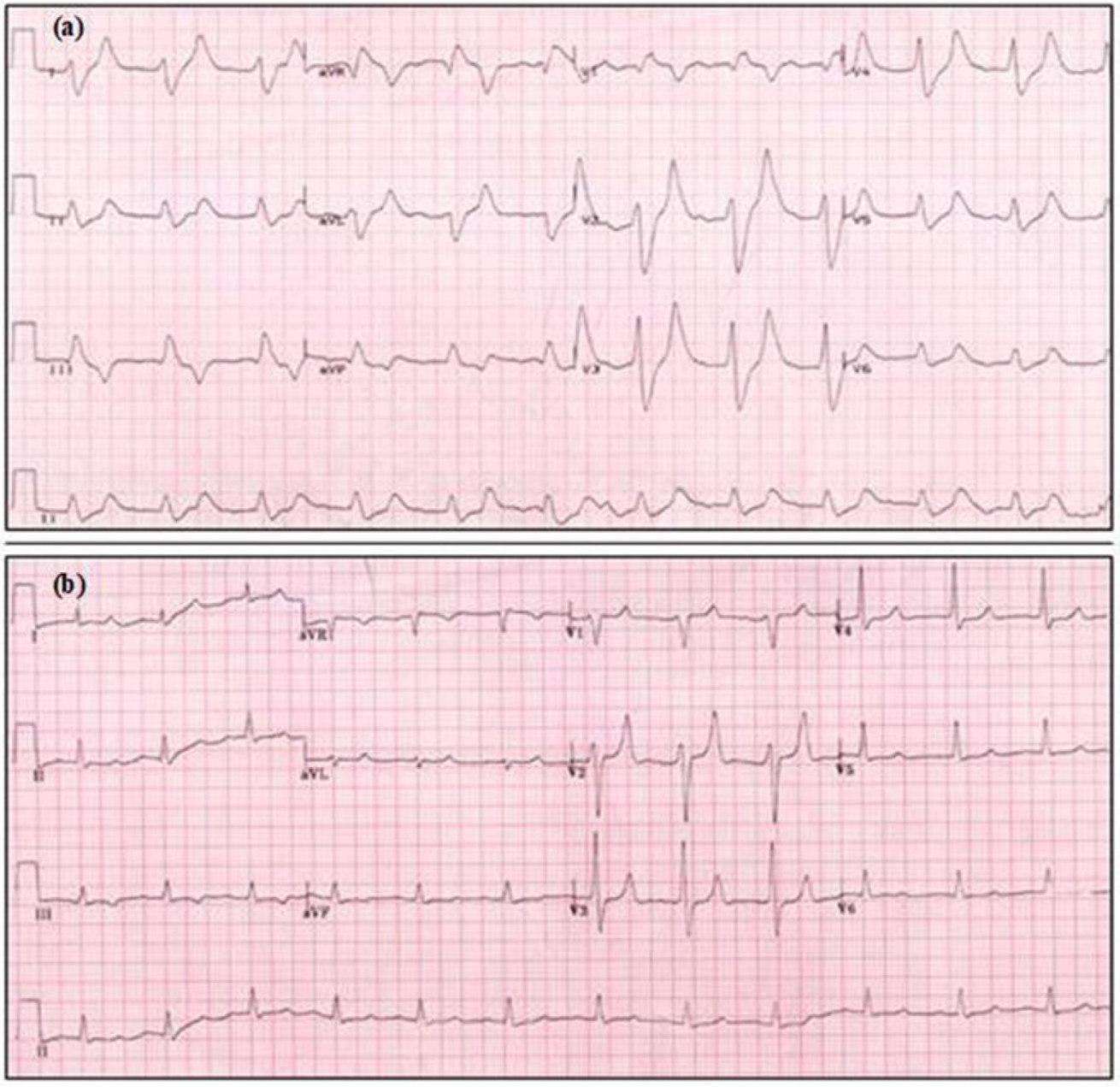

Fig. 11 – Effect of IV calcium on ECG in hyperkalaemia.
ECG on admission (a) and following pre-emptive administration of 20 ml 10 % calcium gluconate IV (b) in a patient who presented with a flaccid paralysis. Response to IV calcium shown with narrowing of QRS complex. Blood gas analyser was unavailable and formal lab result later

glucose < 4 mmol L^{-1}).[51,53,60,61] A scoping review (n= > 15,000) found that the most consistent risk factor for hypoglycaemia is a low pre-treatment blood glucose value.[63] A threshold of < 7 mmol L^{-1} blood glucose has been consistently identifies patients at increased risk of iatrogenic hypoglycaemia.[55,64–67] In one study, administration of 50 g glucose over 4 h resulted in hypoglycaemia in 6.1 % of patients.[59] Based on these collective findings and on evidence that this strategy is effective,[68] a modified protocol which is recommended in the ERC algorithm (Fig. 2) has been developed in order to reduce iatrogenic hypoglycaemia.[50]

Salbutamol. Salbutamol is a beta-2 adrenoceptor agonist and promotes the intracellular shift of K^+. A *meta*-analysis including seven studies administering 10–20 mg inhaled salbutamol demonstrated a reduction in serum K^+ by 0.9 mmol L^{-1} within 120 min.[32] 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.[69,70] On this basis, monotherapy has not been advised. Studies combining insulin-glucose and beta-2 agonists were more effective than either treatment alone.[69,70] The ILCOR review noted a K^+-reduction of 1.2 mmol L^{-1} with combined therapy and supported the use of both drugs.[32] Thus, the ERC recommends simultaneous administration of insulin-glucose and beta-2-agonists for reducing potassium values. Insulin-glucose and salbutamol are effective for 4–6 h, after which rebound hyperkalaemia may occur.

Intravenous calcium salts (calcium chloride or gluconate). 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.[71] 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 (Fig. 11).

The rationale for the 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.[72] In pre-eclampsia, IV calcium ameliorates limb weakness, respiratory depression and cardiac effects induced by iatrogenic hypermagnesaemia.[73]

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.[32]

The Cochrane reviews included no studies evaluating the effect of IV calcium, but suggested that withholding IV calcium in patients with severe hyperkalaemia or ECG changes or both is not advised given potentially life-threatening consequences.[48] 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).

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 min.[32] The Cochrane review also concluded that there is no evidence to support the use of sodium bicarbonate in hyperkalaemia.[48,49] Therefore, the ERC recommends against routine use of sodium bicarbonate in the treatment of hyperkalaemia in non-cardiac arrest cases.

Potassium binders. Novel potassium binders were not included in the ILCOR review, but sodium zirconium cyclosilicate (SZC) and Patiromer are well tolerated and have a role in both acute and chronic hyperkalaemia. SZC has a more rapid onset of action (within 1 h) than Patiromer.[74] Within 48 h, SZC lowers serum K^+ by 1.1 mmol/l and shows greater efficacy with higher potassium levels (1.5 mmol/l K^+-reduction in patients with a serum $K^+ > 6.0$ mmol/l).

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.

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.

Resuscitation of hyperkalaemic cardiac arrest

Hyperkalaemia is the most common metabolic disturbance associated with cardiac arrest and is potentially reversible, with a reported IHCA incidence of 1 %[75] to 12 %,[76] with PEA as the most common initial arrest rhythm.[77] 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.[32]

A study of hyperkalaemic IHCA ($n = 109$; mean serum K^+ 7.8 mmol L^{-1}) used IV calcium and sodium bicarbonate [76] in a very heterogeneous population and achieved 36.7 % ROSC, but only 12.8 % survived > 24 h 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}.[50] All dialysis modalities (with and without ECMO) have been used during CPR, demonstrating that it is technically feasible.[78–81] The mean duration of dialysis to achieve ROSC was 45.4 min (range 15–95 min) and the mean reduction in serum K^+ was 3.2 mmol L^{-1} which would be difficult to achieve with medications alone, particularly during cardiac arrest.[50] 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.[50]

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}).[82] 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 with hyperkalaemic cardiac arrest in all settings.

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].[83] It is usually caused by excessive K^+ loss, transmembrane shift of K^+ into cells, or reduced K^+ intake.[84] 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,[85,86] those treated with digoxin[87] or those undergoing primary percutaneous coronary intervention for ST-elevation myocardial infarction.[88] In acute heart failure, hypokalaemia may be associated with increased short and long-term all-cause mortality after hospital discharge.[89]

Treatment of hypokalaemia

Treatment is guided by the severity of hypokalaemia and presence of symptoms and/or ECG abnormalities, as illustrated in Fig. 3. 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 $L^{-1}K^+$.[90] Correction of any concomitant hypomagnesaemia is essential. Concomitant magnesium deficiency is common in patients with hypokalaemia. Repletion of magnesium will facilitate more rapid correction of hypokalaemia.[91] Seek expert advice for potassium replacement in patients with severe renal impairment (see Fig. 3).

Calcium and magnesium disorders

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

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.[92,93]

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 Medications	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 1 mg kg^{-1} IV Hydrocortisone 200–300 mg IV Pamidronate 30–90 mg 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 (10–30 mL) or 10 % Calcium chloride (5–10 mL) IV repeated if necessary Saline diuresis – 150 mL 0.9 % saline/ hr IV and Furosemide 1 mg/ 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 2 g (8 mmol) IV over 15 min Torsades de pointes: 50 % magnesium sulphate 2 g (8 mmol) IV over 1–2 min Seizures: 50 % magnesium sulphate 2 g (8 mmol) IV over 10 min

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.[94,95] 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.[96] 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.[94–101]

Hyperthermia

Environmental-associated hyperthermia (core temperature > 38 °C) can be avoided by acclimatisation, adequate hydration and avoidance of physical activity in hot weather.[92,98,102] The major risk factor is dehydration, which risks progression to heat syncope, heat exhaustion, heat stroke, and finally multiple organ dysfunction and cardiac arrest. Treatment of hyperthermia see Fig. 4 and Table 3. Core temperature should be measured centrally (e.g. tympanic, rectal, oesophageal) to guide treatment.[103]

Malignant hyperthermia

Prevention of malignant hyperthermia is key; it is a genetic illness that if untreated results in death.[97,108,109] Drugs such as 3,4-methyle nedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines may also cause a condition similar to MH and the use of dantrolene may also be therapeutic in these cases.[110] Always consider malignant hyperthermia, with unexplained, unexpected increases in $ETCO_2$, heart rate or temperature. Further information on malignant hyperthermia management (See QR code. 1).[94,111]

Briefly, stop exposure, provide high flow oxygen, give dantrolene until $ETCO_2$ < 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 (see Fig. 5).

It is essential to contact an expert malignant hyperthermia centre for ongoing advice.[97–112] Dantrolene should be stored centrally where anaesthesia is provided, and algorithms for MH management should be readily available (see QR code 1).

Toxin-Induced hyperthermia

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 (See Table 4).

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.[150] 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 extracorporeal 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,[151–153] but also included other relevant articles.[150,154–161]

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.[154,157]

Measuring the core temperature with a low reading thermometer is the gold standard for diagnosis of hypothermia[157]:

- tympanic in spontaneously breathing patients and
- oesophageal in patients having a tracheal tube or supraglottic airway in situ.[159]

If the core temperature cannot be measured, temperature should be assessed according to the level of consciousness with the Revised Swiss Staging System.[156] (Table 5 and Fig. 12).

- In the Revised Swiss System, "Alert" corresponds to a GCS score of 15; "Verbal" corresponds to a GCS score of 9–14, including confused patients; "Painful" and "Unconscious" correspond to a GCS score < 9. While shivering is not used as a stage-defining sign in the Revised Swiss System, its presence usually means that the temperature is > 30 °C, a temperature at which hypothermic cardiac arrest is unlikely to occur.
- No apparent respiration, no apparent palpable carotid or femoral pulse, no measurable blood pressure – check for signs of life (pulse and, especially, respiration) for up to 1 min.
- The transition of colours between stages represents the overlap of patients within groups. The estimated risk of cardiac arrest is based on accidental hypothermia being the only cause of the clinical findings (Fig. 12). If other conditions impair consciousness, such as asphyxia, intoxication, high altitude cerebral oedema or trauma, the Revised Swiss System may falsely predict a higher risk of cardiac arrest due to hypothermia. Caution should be taken if a patient remains "alert" or "verbal" showing signs of haemodynamic or respiratory instability such as bradycardia, bradypnea, or hypotension because this may suggest transition to a stage with higher risk of cardiac arrest.

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

Table 3 – Treatment of hyperthermia.

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 $Na^+ \leq 130$ 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 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. [92]Mortality is approximately 10 %, and when combined with hypotension approaches 33 %.[98] The outcome worsens if the core temperature is sustained at > 40.5 °C.	Rapidly cool the patient to < 39 °C, preferably < 38.5–38.0 °C as quickly as possible. [98] Prioritise active cooling methods over passive cooling – a cooling rate $\geq$ 0.155 °C min^{-1} is recommended[104]. 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 [104]. 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.[98,639] 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).[105] 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.[105] 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.[105] IV isotonic or hypertonic fluids should be administered (if $Na^+ \leq$130 mmol L^{-1}, for example up to 3 × 100 mL 3 % NaCl at 10 min intervals).[106] 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.[92,107] There may be a requirement for advanced cooling techniques including external or internal devices used for targeted temperature management. There are no specific medications lowering core temperature.

Treatment is progressive in accordance with the degree of hyperthermia.

periods (e.g., less than 1 h).[153] 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 with timely consultation with an ECMO centre being essential.[156,161]

Management of cardiac arrest

The lowest published temperature from which successful resuscitation and rewarming has been achieved is currently 11.8°C[155] for accidental hypothermia and 4.0 °C for induced hypothermia.[162] 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 $\geq$ 28 °C is possible, but unlikely.[151] Some patients may still have minimal vital signs at a core temperature < 24 °C.[163]

Check for signs of life for one minute – not only by clinical examination but also using ECG and ultrasound.[164]

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.[158,160,165]

The prognosis of patients with primary hypothermic cardiac arrest can be excellent,[157] while in cases of secondary hypothermia outcome is more influenced by co-morbidity.[166] Hypothermic patients

Table 4 – Toxins inducing hyperthermia.

Category	Substance	Mechanism	Symptoms	Diagnostic testing	Specific Management
Antipsychotics[113–117]	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[118–122]	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[123–137]	MDMA (Ecstasy), LSD	Central catecholamine release and reuptake inhibition in the CNS, hypermetabolic condition with skeletal muscle stimulation, tachycardia and vasoconstriction	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, agitation, mydriasis Autonomic dysfunction	Urine or blood screening for amphetamines	Ammonium chloride for excretion (urine acidifier). 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[138–142]	Atropine, scopolamine, plant alkaloids (belladonna, brugmansia, amanita)	Blockade of muscarinic receptors. Anticholinergic hyperthermic 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 within 1st hour (time window may be extended as anticholineregics reduce gastrointestinal motility)
Sympathomimetics[143–145]	Ephedrine, pseudoephedrine	Sympathetic system activation Increased metabolic rate and peripheral vasoconstriction	Hyperthermia, tachycardia, hypertension, arrythmias, muscle twitching, nausea, mydriasis, urinary retention	Urine screening, serum toxicology testing for pseudoephedrine/ ephedrine	Benzodiazepines against agitation
Salicylates[146,147]	Aspirin, methyl salicylate	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[148,149]	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

CNS denotes central nervous system, LSD denotes lysergic acid diethylamide, MAOI – Monoaminoxidase inhibitor, MDMA – 3,4-Methylenedioxymethamphetamine, SNRI – Serotonin and norepinephrine reuptake inhibitor, SSRI – Selective serotonin reuptake inhibitor, TCA – Tricyclic antidepressant.

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 functioned well.[151,152,166] 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.[167] 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.[152]

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. Efforts should also focus on maintaining normoxia during resuscitation.

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.[168] 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.

Medication metabolism is slowed, leading to potentially toxic plasma concentrations, therefore, medications should be administered cautiously. The evidence for the efficacy of medication in severe hypothermia is limited and based mainly on animal studies. In severe hypothermic cardiac arrest, the effectiveness of amiodarone is reduced.[169] Adrenaline may be effective in increasing coronary perfusion pressure, but not survival.[170,171] Adrenaline

Table 5 – Swiss staging of accidental hypothermia.[157]

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**

* Consciousness may be impaired by comorbid illness (e.g. trauma, central nervous system pathology, toxic ingestion) or medications (e.g. sedatives, muscle relaxants, opioids) independent of core temperature.

** The risk of cardiac arrest increases < 30 °C, older and sicker patients. Alternative causes should be considered. Some patients still have vital signs < 24 °C.

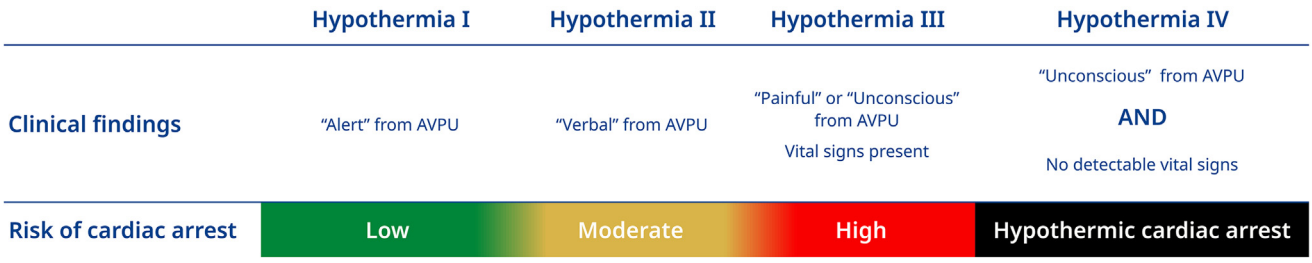

Fig. 12 – Revised Swiss System for staging of accidental hypothermia.[156]
AVPU – alert verbal pain unresponsive.

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 even at lower core temperature in an attempt to achieve ROSC.[172] 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 (Fig. 13).[173]

Establish extracorporeal life support only in cardiac arrest or deteriorating patients (e.g. decreasing blood pressure, increasing acidosis). Primary ECLS may be considered in patients with $ETCO_2$ < 10 mmHg (1.3 kPa) or a systolic blood pressure ≤ 60 mmHg.[174] An unwitnessed cardiac arrest with asystole as first rhythm is not a contraindication for ECLS rewarming.[152] Rewarming should preferably be performed with VA-ECMO rather than cardiopulmonary bypass.[175,176] If ECLS is not available within 6 h, non-extracorporeal life support rewarming may be used.[177,178]

In-hospital prognostication of successful rewarming should be based on the HOPE score.[158,160] 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.[179,180] In hypothermic arrested patients, criteria for ECLS rewarming should not be based on guidelines for normothermic cardiac arrest.[181] This may result in potentially life-saving treatment being withheld in patients with a potential for good neurological outcome.[176]

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.

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.[182] Most avalanche victims die from asphyxia, fewer from trauma or hypothermia. In cases of unwitnessed cardiac arrest presenting in asystole, avalanche victims have a poor chance of survival, even if guidelines are followed.[183–185]

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 dur-

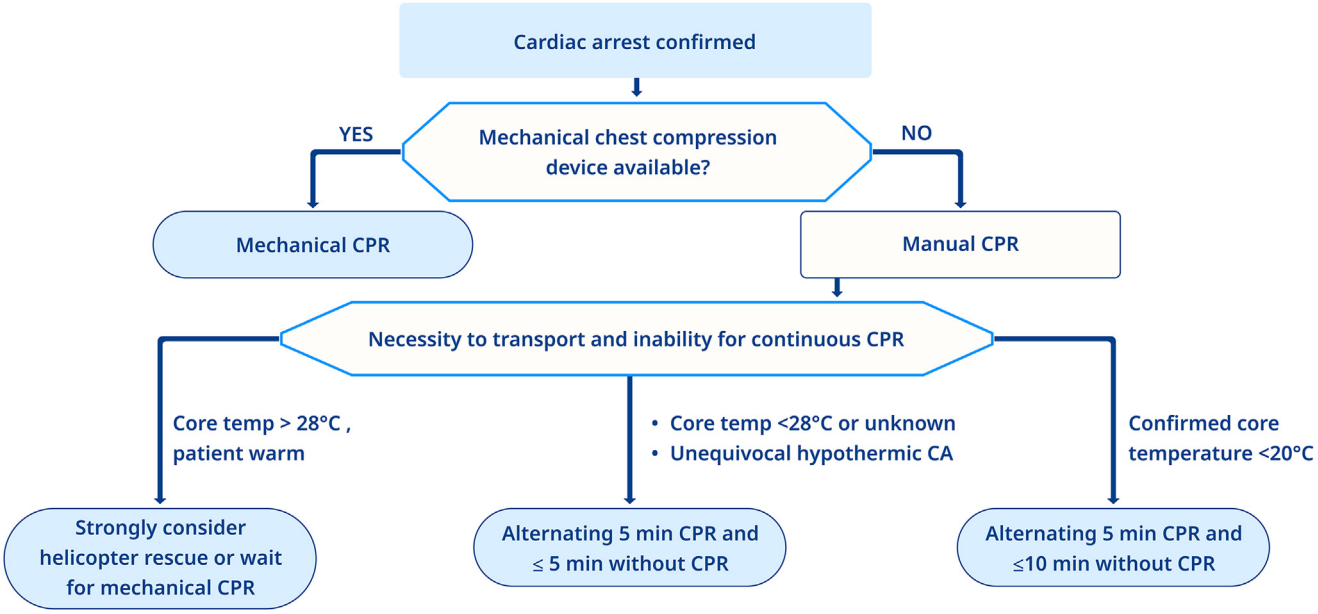

Fig. 13 – Decision making in hypothermic cardiac arrest.
CPR – cardiopulmonary resuscitation. Delayed and intermittent CPR in hypothermic patients when continuous CPR is not possible during dangerous or difficult rescue.[170]

ing daylight hours, the presence of an air pocket (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.[182–184,186,187] The International Commission for Mountain Emergency Medicine performed a scoping review and published guidelines for the management of victims buried in avalanches (Fig. 14).[188]

The AvaLife algorithm should be used in multiple burial scenarios with too few rescuers on site. In this triage situation, AvaLife enables identification of the i) buried subjects who should be excavated first (cut-off < 1.5 m of burial depth) and ii) who should be resuscitated and for how long.[189] AvaLife has been specifically developed for BLS provider.

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.[190–192]

Thrombosis

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).[193] The reported incidence of cardiac arrest caused by PE is 2–7 % of all OHCAs,[194,195] and 5–6 % of all in-hospital cardiac arrests,[75,196] but it is likely to be underestimated. Overall survival is low, even if invasive treatment or ECPR is used.[195,197,198] 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.[199] The 2019 ILCOR summary statement reviewed the use ECPR for cardiac arrest in adults,[200] 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.[201] Additional evidence was identified from the European Society of Cardiology (ESC) guideline on pulmonary embolism.[193]

These ESC Guidelines define 'confirmed pulmonary embolism' as a probability of pulmonary embolism high enough to indicate the need for specific treatment.[193] 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.[197] Constant low $ETCO_2$ readings (below 1.7 kPa (13 mmHg)) while performing high-quality chest compressions may support a diagnosis of pulmonary embolism, although it is a non-specific sign.[202,203] 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 ventricular 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 diseases.[204]

Initial treatment (Fig. 15)

All patients with a 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

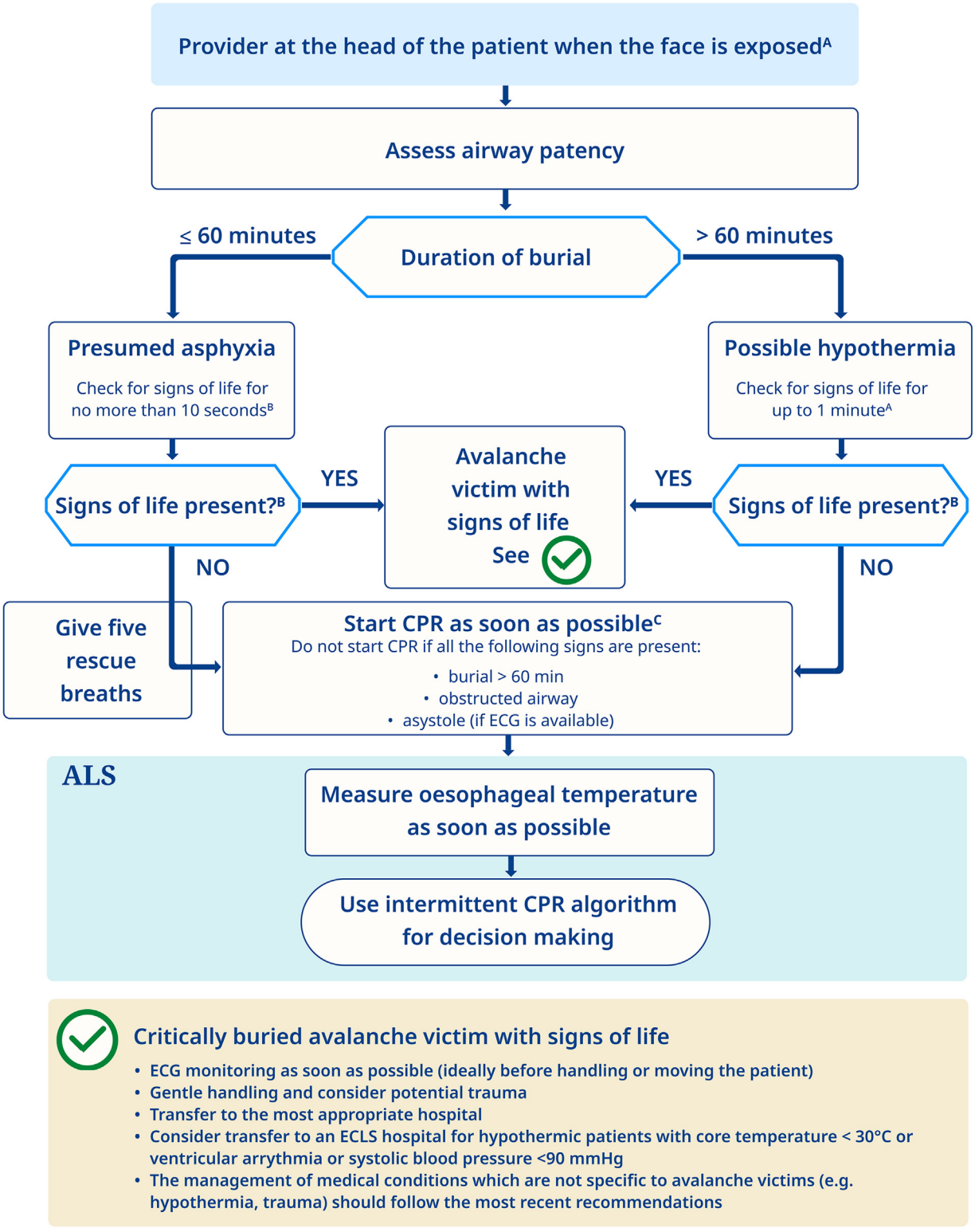

Fig. 14 – Initial management of critically buried avalanche victim.[188]

CPR – cardiopulmonary resuscitation, ECG – electrocardiography, ECLS – extracorporeal life support. Figure legend. [A] Assess for lethal injuries: decapitation; truncal transection; whole body decomposed. If present, do not start CPR. [B] Signs of life include any of the following: A, V or P from AVPU (alert, responsive to verbal stimuli, responsive to pain, unresponsive) or Glasgow Coma Scale > 3, any visible movement, respirations, or a palpable carotid or femoral pulse (for experienced ALS providers). [C] Standard compression / ventilation rates. Drug dose and defibrillation depending on core temperature or, if not available, burial duration. If ventricular fibrillation persists after three shocks, delay further attempts until the core temperature is > 30 °C.

hypoxaemia will not be possible without simultaneous pulmonary reperfusion.[193]

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 ($\leq$ 500 mL over 15–30 min) may increase cardiac output, and the use of vasopressors and/or inotropes is frequently beneficial.[193]

IV anticoagulation should be initiated while awaiting the results of diagnostic tests. Unfractionated heparin is recommended for patients with shock and hypotension, and for whom primary reperfusion is considered. Thrombolytic treatment of acute pulmonary embolism restores pulmonary perfusion more rapidly than anticoagulation with heparin alone.[206]

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.[201] 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 IV – max. 100 mg).[197,207,208] When thrombolytic drugs have been administered, continue CPR for at least 60–90 min.[195,209,210] Based on expert opinion, thrombolysis or surgical embolectomy should be considered for pregnant women with high-risk pulmonary embolism and PE causing cardiac arrest.[193]

Table 6 – Non-specific patient characteristics observed with higher incidence in OHCA caused by pulmonary embolism compared with other causes[193,198,205].

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
Female biological sex
Lower age
EMS witnessed cardiac arrest
PEA as first observed rhythm
Low $ETCO_2$ readings during high quality 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)

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.[211–213] Treatment decisions should be made by a highly experienced interdisciplinary team, involving a thoracic surgeon or interventional cardiologist.[193,214]

Extracorporeal CPR. Some observational studies suggest the use of ECPR if cardiac arrest is associated with pulmonary embolism[198,215] ECPR maintains circulation and gas exchange. Time to ECPR correlates with neurological outcome.[216] 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.[201] It is recommended that adequate ECPR flow is established within 60 min of the onset of cardiac arrest. [217] Favourable neurological outcomes in patients with pulmonary embolism undergoing ECPR is inferior to outcomes related to other aetiology.[198,218] Despite the extremely high mortality rate of patients with cardiac arrest refractory to standard ALS, ECPR is also an option to increase the pool of organ donors.[198]

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.[219,220] While CAD prevalence increases with age, it is also the most common cause of cardiac death in adults aged 35 to 50 years.[221,222]

A recent systematic review and *meta*-analysis reported significant CAD in 75 % of OHCA cases,[223] 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.[224,225]

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.[226]

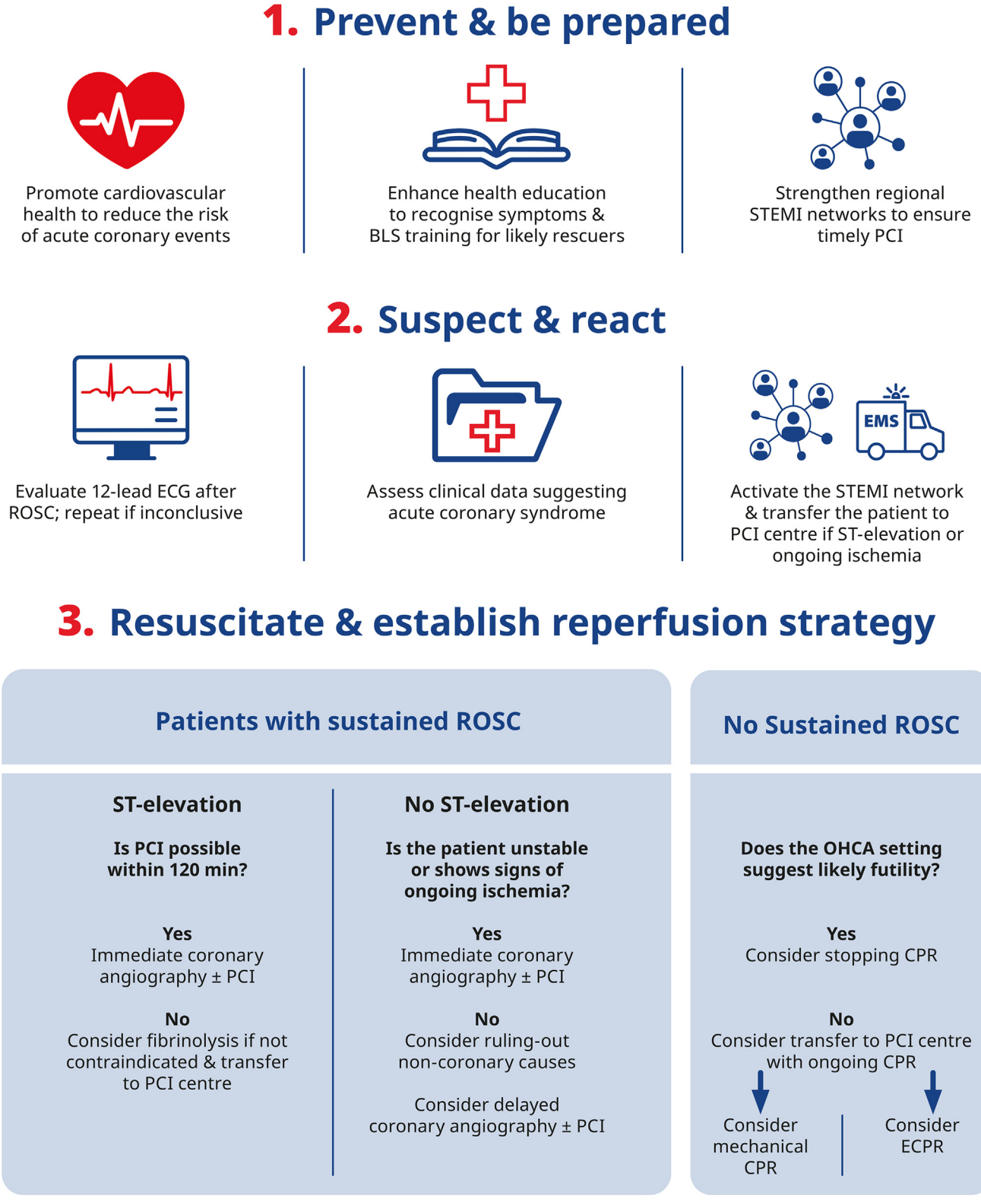

Fig. 15 – Coronary thrombosis algorithm.

BLS – basic life support, STEMI – ST elevation myocardial infarct, PCI – percutaneous coronary intervention, ECG – electrocardiography, OHCA – out-of-hospital cardiac arrest, ROSC – return of spontaneous circulationg.

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.[201]

Along with initiatives raising public awareness, healthcare systems should establish regional STEMI networks to ensure equal and timely access to percutaneous coronary intervention (PCI).[225] A recent systematic review and *meta*-analysis found that such networks may reduce STEMI case-fatality by 35 % and long-term mortality by 27 %.[227] Integration of the emergency transport system was a critical factor for success.

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, with a higher percentage of false-positive for STEMI if the ECG is performed $\leq$ 8 min from ROSC.[228] ST elevation remains the most sensitive and specific sign of coronary artery occlusion; however, its absence does not completely exclude the condition.[229] 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.[225] Additionally, non-cardiac conditions, such as subarachnoid haemorrhage, can also cause ST-changes,[230] 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—[231] is recommended. Once clinical suspicion is established, the STEMI network should be promptly activated to ensure early transfer to a centre with PCI capability.

Resuscitate according to ALS algorithm & establish reperfusion strategy

Patients with ROSC and ST-elevation. OHCA resuscitation should follow the standard ALS protocol (see ALS chapter). Conscious patients with persistent ST-elevation (or equivalent) after ROSC should undergo immediate coronary angiography and PCI within 120 min of diagnosis, as per standard STEMI management. If significant delays are expected, pre-hospital fibrinolysis may be considered unless CPR was prolonged or traumatic or there are other contraindications, followed by immediate transfer to PCI centre. [225] Comatose patients with ST-elevation after ROSC have significantly worse prognosis, and no RCTs have directly compared early versus delayed angiography in this group. However, since early angiography is the standard of care for STEMI and no evidence suggests otherwise, both the ILCOR [232] and the European Society of Cardiology[225] recommend the same approach for these patients.

Pharmacological therapy should align with STEMI protocols for non-arrest patients.[225] In patients targeted for a primary PCI strategy, aspirin is recommended and early administration of a P2Y12 inhibitor (ticagrelor or prasugrel over clopidogrel, or IV cangrelor if oral intake is not possible) may be considered. Unfractioned heparin (IV) is the preferred anticoagulant, with enoxaparin (SC) or bivalirudin (IV) as alternatives.

Patients with ROSC and no ST-elevation. While previous observational studies suggested a potential benefit of early coronary angiography in resuscitated patients without ST-elevation on ECG,[233,234] recent RCTs have not confirmed this. A 2021 ILCOR systematic review and *meta*-analysis[235] compared routine early (2–6 h) versus delayed (within 24 h) coronary angiography in unconscious adult patients with ROSC after OHCA. This included data from the Direct or Subacute Coronary angiography in Out-of-hospital Cardiac Arrest-DISCO trial ($n = 79$),[236] Coronary Angiography After Cardiac Arrest Trial-COACT ($n = 552$)[237] and Randomized Pilot Clinical Trial of Early Coronary Angiography-PEARL ($n = 99$)[238] – the latter being underpowered. No survival or neurological benefit was found for early versus delayed angiography in patients without ST-elevation. In 2022, an updated ILCOR CoSTR[232] added the results of the Angiography after Out-of-Hospital Cardiac Arrest without ST–Segment Elevation-TOMAHAWK ($n = 554$) trial,[239] which also found no advantage of a routine early approach. Accordingly, the Task Force deemed both early and delayed angiography reasonable in patients without ST-elevation after ROSC (weak recommendation, low-certainty evidence).[232] Two latter RCTs, Emergency vs Delayed Coronary Angiogram in Survivors of Out-of-Hospital Cardiac Arrest-EMERGE ($n = 279$)[240] and Coronary angiography in Out-of-hospital cardiac arrest Patients without ST-segment Elevation-COUPE ($n = 66$)[241] have reinforced these findings, despite variations in study protocols and outcome definitions, as well as limited statistical power.

Importantly, patients with shock, electrical instability, or signs of ongoing ischaemia were excluded or allowed to cross over to the emergency arm. Thus, current evidence does not support routine early over delayed angiography in stable patients without ST-elevation on the post-ROSC ECG. However, individual decisions should consider the patient's haemodynamic status and signs of ongoing ischaemia. Also, ruling out non-coronary causes is recommended if the clinical context suggests a potential alternative aetiology for the arrest.

Pharmacological treatment should generally follow the recommendations for non-ST-elevation acute coronary syndrome if suspected.[225] Aspirin is recommended, but routine early use of P2Y12 inhibitors is discouraged if early angiography is planned. Unfractioned heparin (IV) and fondaparinux (SC) are the preferred anticoagulants for the early and delayed angiography strategies, respectively. However, if there are concerns about other non-coronary causes for the arrest, withholding antiplatelet and anticoagulant therapies until further evaluation is reasonable.

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.[242] 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.[8,243] 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.[244,245] A multicentre

RCT found no difference in 30-day survival with favourable neurological outcomes.[246] 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.[243]

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 (See Fig. 7). The therapeutic strategy of intoxication consists of the three pillars:

- Decontamination
- Enhanced elimination
- Administration of antidotes

Helpful online databases on poison centres and toxins can be found in the QR codes 2 and 3). 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.[99] These updated recommendations are based on recent scientific evidence, systematic reviews, and expert consensus. A separate subchapter addresses local anaesthetic systemic toxicity.

QR code. 2 – World directory of poisons centres – provided by the World Health Organization.

QR code. 3 – PubChem – world's largest collection of freely accessible chemical information – provided by the National Library of Medicine.

Intoxication with opioids

An ILCOR systematic review found heterogenous results on the benefit of opioid-specific advanced life support-level therapies for cardiac arrest.[247] Existing evidence is not sufficient to recommend administration of an opioid-antagonist (e.g. naloxone) for cardiac arrest caused by opioid poisoning.

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.[248]

ECPR

ECPR seems to be associated with increased survival in intoxicated patients in refractory cardiogenic shock or cardiac arrest.[249]

Traumatic cardiac arrest

Traumatic cardiac arrest is associated with a very high mortality and good neurological outcome is reported in less than 50 % of those who survive. 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 nine systematic reviews and evidence updates and consensus from the expert group.[250–258]

In Europe, traumatic cardiac arrest accounts for 4 % of all cardiac arrests occurring in the pre-hospital setting.[259] Registry data for survival range from 0 %[260] to 37 %.[261] A recent systematic review[255] 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.[253]

Factors relevant for prognostication are listed in Table 7.

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.[271] However, neurologically intact survivors initially presenting in this state have been reported. [272] 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.

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.[273]

A considerable proportion of trauma-related deaths can be attributed to management errors. In an urban German population, the

Table 7 – Prognostication in traumatic cardiac arrest.[254,262–270.]

–	+
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

prevalence of potentially preventable prehospital deaths from trauma was reported as 15.1 %,[274] whereas in an urban U.S. population, this figure was higher at 29 %.[275] In Australia, the rate of potentially preventable trauma deaths was found to be 20 %.[276] 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.[277]

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 %).[278] The key pathophysiological mechanisms leading to traumatic cardiac arrest are listed in Table 8:

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.[266]
- 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.

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 (pseudo-PEA), chest compressions may even reduce the remaining cardiac output by further decreasing venous return.[279–281] Therefore, chest compressions must not delay immediate treatment of reversible causes.

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

Table 8 – Major causes of traumatic cardiac arrest and their pathophysiology.

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 (contributing factor)	Ischaemia from prolonged hypoxia & hypoperfusion	Cardiac dysfunction, reduced contractility
Neurogenic shock (contributing factor)	Loss of sympathetic tone	Aggravates other shock states, worsening hypotension

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.[282]

Providers without the training to manage reversible causes should follow the standard ALS algorithm and prioritise rapid transport under ALS or BLS, depending on system capability. Those with the necessary expertise should make case-by-case decisions based on available resources, likely reversible causes, and regional guidelines.

The role of inotropes and vasopressors

Although adrenaline may increase the rates of ROSC with medical cardiac arrest,[283] the role of inotropes and vasopressors in traumatic cardiac arrest (TCA) is unclear.[284] They may serve as a temporary bridge until reversible causes are addressed. Their use may also be considered in late-stage shock with cardiac dysfunction or mixed shock states such as trauma with a neurogenic component. The primary aim in traumatic cardiac arrest is to identify and treat any reversible cause which should take preference over inotrope/vasopressor therapy.

Pre-hospital care

The TCA algorithm (Fig. 8) applies not only to patients in traumatic cardiac arrest but also to *peri*-arrest trauma patients who present with signs of impending circulatory collapse. In both scenarios, the response must be immediate, targeted, and adapted to the capabilities of the prehospital system. The choice between a "stay and treat" strategy versus "scoop and run" or "treat-as-you-go" depends less on geography and more on the available skill set, equipment, and system infrastructure. In highly trained systems with extended capabilities, life-saving interventions may be feasible on scene. In others, immediate transfer may be the best chance for survival, provided that basic haemorrhage control and oxygenation are ensured during transport. Regardless of the system configuration, prolonged prehospital time must be avoided.[273] Early pre-alerting of the Major Trauma Centre (MTC) is essential. In cases of ongoing haemorrhage or severe physiological compromise, the patient should be declared "Code Red" (or equivalent), enabling the hospital to activate the massive transfusion protocol, mobilise the trauma team, and ensure immediate access to surgical care upon arrival.

The recently published practice guidelines of the Royal College of Surgeons of Edinburgh on traumatic cardiac arrest provide a comprehensive overview of prehospital management.[285]

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.[286] Fig. 8 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, radiologists, intensivists and nursing staff, is essential to ensure that each critical intervention is initiated without delay, according to the patient's immediate needs.

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.[273] Compressible external haemorrhage can be treated with elevation (of a bleeding limb), direct or indirect pressure, pressure dressings, tourniquets and topical haemostatic agents.[273,287] 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.[288]

Immediate aortic occlusion is recommended as a last resort measure in patients with exsanguinating and uncontrollable infradiaphragmatic 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.[289] Both are highly specialised interventions, appropriate expertise and equipment. A recent UK REBOA study[290] in traumatic shock revealed a possible increase in mortality. The ERC does not recommend the use of REBOA outside of clinical trials.

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.[291,292] Recent evidence suggests additional benefit from whole blood transfusion.[258,293]

Hypoxia. In traumatic cardiac arrest, hypoxaemia can be caused by airway obstruction, traumatic asphyxia or impact brain apnoea.[294] Impact brain apnoea is an underestimated cause of morbidity and mortality in trauma, but not necessarily associated with an unsurvivable brain injury.[295] 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.[296] 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.[297]

Controlled ventilation in circulatory compromised patients is associated with major risks because of the increase in intrathoracic pressure[298,299] that may lead to further decreased cardiac output caused by:

- Impeded 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.[265,300,301]

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.[302] 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.[278] 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 with tracheal deviation and jugular venous distention).[302] 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 available.[303–306]

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.[307,308] 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.[309]

While resuscitative thoracotomy is often performed for the relief of cardiac tamponade, it is not exclusively indicated for this condition. Resuscitative thoracotomy is also applicable for direct haemorrhage control, aortic cross-clamping, and open cardiac compressions.[282]

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.[251] 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 min 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 min

If any of the four criteria is not met, resuscitative thoracotomy is futile and exposes the team to unnecessary risks.[310] A retrospective cohort study found that prehospital resuscitative thoracotomy for traumatic cardiac arrest was associated with 3.8 % of 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 min of traumatic cardiac arrest for cardiac tamponade and 5 min after exsanguination-induced cardiac arrest.[311]

Post resuscitation care after traumatic cardiac arrest

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. 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.

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 min, as the risk of irreversible organ damage then outweighs its benefits.[312–314]

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

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 or whole blood), tranexamic acid, and point-of-care coagulation monitoring to guide targeted haemostatic therapy.

Special settings

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 among others in this setting include severe ischaemia, tamponade, arrythmias, vascular perforation/dissection, or anaphylaxis.

A recent ILCOR scoping review[315] estimated the overall incidence of cardiac arrest in the catheterisation lab as 0.2 % to 0.5 % across all procedures.[316,317] For PCI, the incidence is slightly higher (0.8–2 %),[318–320] and for ST-elevation myocardial infarction higher still (STEMI – 1.9 % to 5.5 %), primarily with an initial rhythm of ventricular fibrillation.[321–325]

Overall initial survival rates range from 67 % to 77 % at the event time[316,317,326] and between 38 % to 56 % at hospital discharge.[317,326] For PCI and STEMI, survival to discharge is higher (82–100 %).[321,322,325]

Prevent and be prepared

Adequate Education is essential to ensuring the best cardiac arrest outcomes 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 (See Fig. 16).

Studies support periodic simulation emergency drills to enhance technical and non-technical skills in the catheterisation lab.[327] Training in non-technical skills has the potential to minimize errors in this setting.[328] 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 has resulted in fewer complications.[329]

Detect and react

Patient's monitored vital parameters including ECG must be checked regularly 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 images and precision.[330]

Cardiac arrest in the catheterisation lab should prompt an immediate call for help and activation of the resuscitation team.[316] 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 also be alerted. Importantly, as highlighted by the Joint British Societies' guideline on management of cardiac arrest in the catheterisation lab,[331] 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.

Resuscitate and treat possible causes

Cardiac arrest in the catheterisation lab should be generally managed according to the ERC Guidelines 2025 ALS,[164] 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.[331]

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.[332] 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.

Mechanical CPR

An ILCOR systematic review evaluated the impact of mechanical chest compression devices compared with manual compression on cardiac arrest outcomes,[242] and found evidence against the routine use of automated mechanical chest compressions to replace manual compressions for in-hospital cardiac arrest. 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[315] 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.[242] Importantly, if used,

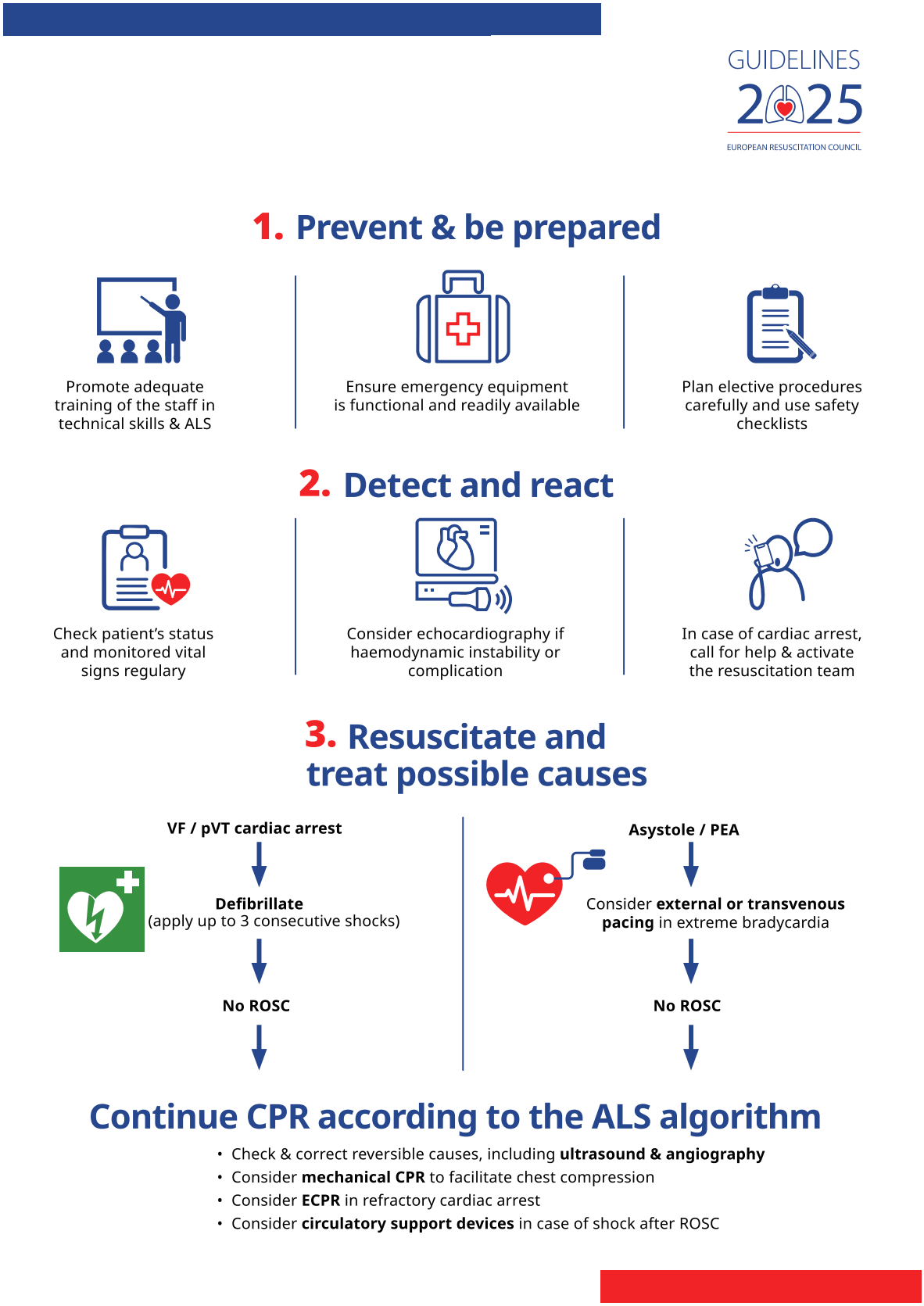

Fig. 16 – Treatment of cardiac arrest in catheterisation laboratory.
ALS – advanced life support, VF – ventricular fibrillation, pVT – pulseless ventricular tachycardia, ROSC – return of spontaneous circulation, PEA – pulseless electrical activity, POCUS –point of care ultrasound.

pauses during device placement should be minimized, and correct positioning ensured to avoid visceral injuries.

Extracorporeal CPR

The use of veno-arterial extracorporeal membrane oxygenation (VA-ECMO) to support refractory cardiac arrest patients (ECPR) was recently updated by ILCOR.[243] 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[315] 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.[333] 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. ECPR facilitates performing diagnostic or therapeutic procedures, including coronary angiography/angioplasty, pulmonary embolectomy or cardiac surgery.

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.[334] 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.

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.[334] 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.

Drowning

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[335]. This decline might be attributed to legislation and regulations (e.g., pool fencing and mandatory lifejacket use),[336] as well as effective preventive measures (e.g., day-care centres for children and water safety training).[337] 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.[338–340] Most fatal drownings never reach the health system because rescue or retrieval often occurs too late for medical intervention.[341]

Recommendations are based on a scoping review in 2021 and a systematic review in 2023.[342,343] 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.[344]

Drowning is defined as 'the process of experiencing respiratory impairment from submersion or immersion in liquid'.[345] Immersion is a situation in which the person stays in a liquid with the airway above the liquid 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 the drowning mechanism to become fatal.[346,347] Submersion time is the most robust and independent prognostic factor.[348] The Adult BLS CoSTR 2020 recommends submersion time as the sole indicator guiding prognosis and decisions regarding clinical management and search and rescue operations.[349] Less than 5–10 min of submersion is associated with a better prognosis.[348–350]

The extent of lung damage depends on the amount and degree of pollution of the aspirated water.[346] Salinity is of no proven predictive value.[348,349,351]

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.[352] In drowning, low water temperature may exert a protective effect by reducing brain metabolism.[346,348,349] However, this phenomenon is exceedingly rare and is most likely in small individuals who cool quickly in water below 6 °C.[353] Submersion longer than 60 min with good neurological outcomes has been reported,[354] particularly in children immersed in cold water, preceding the submersion.[354,355] These reports suggest that if water temperature is above 6 °C, survival or successful resuscitation becomes extremely unlikely after about 30 min of submersion, whereas in water at or below 6 °C, this threshold may extend to around 90 min.[354]

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.[356] 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 flotation 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.[357] Rescue material and flotation devices also reduce the rescue time.[357]

Physical fatigue is a limiting factor when CPR is required, especially after water rescue.[358,359] If possible, a person not involved in the rescue should perform CPR.[357] Spinal injuries are uncommon after drowning.[360] 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.[361] 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.

Cardiac arrest caused by drowning

Evidence showed better prognosis when drowned persons are ventilated when initiating CPR.[362–364] For this reason, in-water ventilation and on-board resuscitation are gaining acceptance within the lifesaving community, as these techniques are feasible with training.[342,362,365–367]. In practice, some circumstances do not allow this. For example, due to lack of training, absence of flotation equipment or the conditions of the water. Then, it is more practical to wait until the victim is onshore.

Onshore, first responders and EMS should follow the ABC approach.[362,365] CPR should start with 5 ventilations. If the person remains unconscious without normal breathing, continue with standard CPR (Fig. 9). Starting CPR with compression is only advised if the rescuer is unwilling to provide ventilations.[368]

For simplicity, untrained bystanders should start with chest compressions whilst trained first aid providers should consider ventilations, since the time to ventilation is critical in restoring cardiac arrest in drowned victims. In isolated respiratory arrest, ventilations can prevent cardiac arrest (see Fig. 10).

In drowning, an initial shockable rhythm is present in less than 10 % of cases because of the cardiac response to hypoxia. Although victims removed from the water might be in cardiac arrest due to cardiac cause, for example because of arrhythmias caused by autonomic conflict[352] or the presence of cardiovascular disease,[369] CPR should not be delayed by AED application.

Oxygen should be provided early because drowning is a respiratory event. However, a recent study showed that it is difficult to provide evidence of the impact of oxygen on prognosis.[370] Airway management should be based on the competencies and training of the first responder or EMS.[365] Tracheal intubation compared with supraglottic airway devices was associated with higher ROSC, but not associated with survival or favourable neurological outcome at one month.[371]

Impact of hypothermia

Most drowned victims are hypothermic (core body temperature < 35 °C), which affects diagnosis, treatment and prognosis.[346,348,372,373] In general, guidelines for accidental hypothermia can also be applied to hypothermic drowned victims, including the one-minute vital sign check, accurately measuring core temperature, the alternative treatment regarding defibrillation, use of medication when body temperature is below 30 °C, and access to extracorporeal life support rewarming.[372] Hypothermia reflects a longer submersion time, resulting in poorer neurological outcome and survival. However, when drowning occurs in cold water, generally considered below 6 °C, and notably after a period of immersion, associated hypothermia may exert a neuroprotective effect.[374]

The Adult BLS CoSTR 2020 suggests not using water temperature when making prognostic decisions[349] and no specific guidelines exist to indicate precisely when to move from rescue to recovery—a stance that also acknowledges the exclusion of rare favourable outcomes in icy water. This is partly due to the wide variety of drowning scenarios regarding submersion 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.[374]

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,[375–377] with a higher incidence in low-resource settings,[378] in older-frailer patients, and in newborns and infants.[379,380] Overall survival rate is > 50 %.[381] Strong predictors of intraoperative cardiac arrest are higher scores in the American Society of Anesthesiologists (ASA) physical status, sepsis, urgent or emergency case, complexity of the case, anaesthetic technique and age.[381] The leading causes are complications during cardiac surgery, major haemorrhage, bradyarrhythmias and septic shock.[378,381]

Specifics of intraoperative cardiac arrest and its treatment

Mostly, gradual physiological deterioration leads to intraoperative cardiac arrest.[382] 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 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 50 mmHg despite interventions to treat the underlying cause.[375,383,384] Sudden fall in $ETCO_2$ in such cases is a strong indicator for cardiac arrest. 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.[385–387] For pre-arrest patients (SBP < 50 mmHg), 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 1 mg of intravenous adrenaline should be given.[375,388]

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, pre-

pare resuscitation drugs and fluids, establish advanced airway management (if not already undertaken) and use a mechanical ventilator delivering 100 % oxygen as soon as possible.[389] Current data suggest that mechanical ventilation yields a similar PaO_2 to that of manual ventilation with a self-inflating bag.[390–393] 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.[389]

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.[394,395] Consider simultaneous left lateral decubitus and head-down positioning in cases of massive air embolism[396–398] 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 compressions are effective only if the circulating volume is replaced simultaneously and haemorrhage control (e.g. surgery, endoscopy, endovascular techniques) is initiated immediately.[389]
- 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 the potential benefit.[389,399,400]
- 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 these guidelines.

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.[8,401,402] 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.[8,403]

There is no evidence to support the use of hypothermic temperature control after adult intraoperative cardiac arrest.[404] Therefore, post-arrest temperature control should follow local post-resuscitation protocols.

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 min.[405,406] 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.[407,408] Seizure management involves administering benzodiazepines in incremental doses (e.g. IV lorazepam 0.1 mg kg^{-1} IV, midazolam 0.05–0.1 mg kg^{-1} IV), 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 (especially propofol). 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,[131,408–410] but without documented harm.[411] A 20 % lipid emulsion (at least 4 bags of 250 mL) should be available if large doses of local anaesthetics are used (e.g. operating rooms, labour wards, emergency department).[409,412] During cardiac arrest, after an initial 20 % lipid emulsion IV bolus (1.5 mL/kg IV over 1 min), an infusion should be started (0.25 mL kg^{-1} min IV). If ROSC is not achieved in 5 min, the infusion rate should be doubled with two additional boluses at 5-minute intervals.[409,412] A maximum cumulative dose of 12 mL kg^{-1} IV 20 % lipid emulsion is recommended to avoid fat overload.[411] It is suggested that adrenaline IV be given at a lower dose ($\leq$1 μg kg^{-1}) in local anaesthetic intoxication because its arrhythmogenic and acidotic effects may impede sustained ROSC – this has been shown in animal studies.[409,413–416]

QR code. 4 – Cardiac arrest due to local anaesthetic toxicity management.

Further information on cardiac arrest due to local anaesthetic toxicity can be found at the QR code. 4.

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.[417–421] 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 Cardio-Thoracic Surgery,[422,423] the Society of Thoracic Surgeons expert consensus document for the resuscitation of patients who arrest after cardiac surgery,[424] the British Societies

Guidelines,[425] and the ILCOR CoSTR for the Management of Implantable Left Ventricle Assist Devices recipients.[426]

Ensure adequate initial and refresher training of staff in resuscitation technical skills, ALS and teamwork through simulated cardiac surgery scenarios, including training to perform an emergency resternotomy. Teamwork, 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.[427]

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.[423,424]

Safety checklists reduce complications and mortality in non-cardiac surgery and should be implemented for the management of cardiac arrest in these settings.[428]

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).[429–431] Echocardiography should be performed in case of haemodynamic instability; consider transoesophageal echocardiography for more precise diagnosis.[432,433] Continuous ECG monitoring enables early identification of arrhythmias; supraventricular tachycardias are the most frequent in this setting.[434]

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 $ETCO_2$.[423,424]

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.[423,424].

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 (Fig. 17).

In patients with VF/pVT, defibrillation of up to three stacked shocks are prioritised within a maximum time of 60 s.[435,436] If these fail, immediate resternotomy and internal defibrillation are advised.[436] In case of asystole or extreme bradycardia, attempt epicardial pacing (DDD mode at 80 to 100 beats/min 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.[423,424]

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, aiming to reach a systolic blood pressure > 60 mmHg; failure to attain this value despite adequate

Table 9 – Common causes of patient deterioration after cardiac surgery and management.

• Haemorrhage • 'Medical' bleeding: post-operative coagulopathy • 'Surgical' bleeding: operative trauma	• Correct hypothermia and hypotension, 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

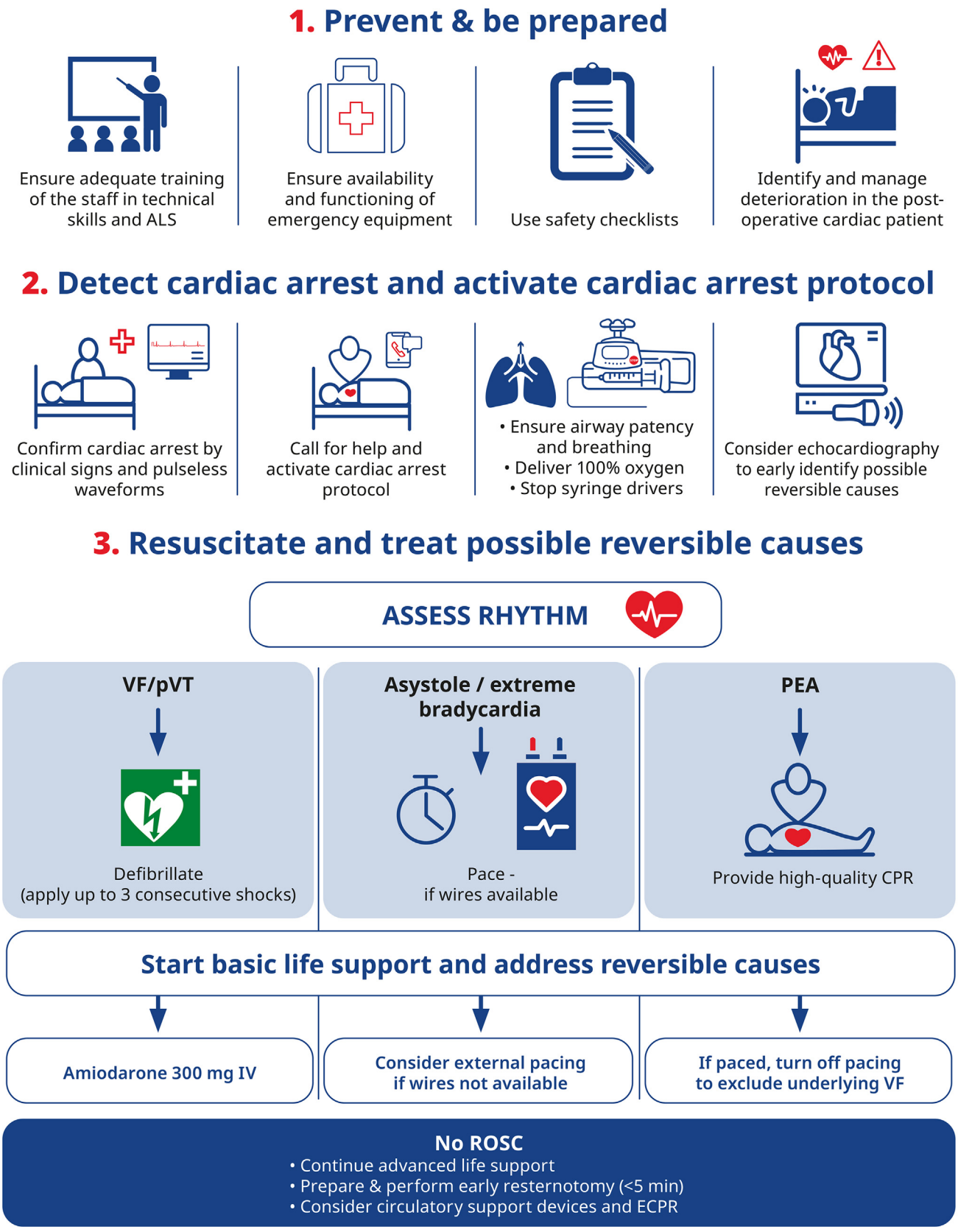

Fig. 17 – Post-cardiac surgery cardiac arrest algorithm.
ALS – advanced life support, VF – ventricular fibrillation, pVT – pulseless ventricular tachycardia, ROSC – return of spontaneous circulation, PEA – pulseless electrical activity, IV – intravenous.

performance may indicate tamponade or severe haemorrhage, requiring emergency resternotomy.[423,424] Compared with external compressions, internal cardiac compression provides better coronary and systemic perfusion pressure, which may justify chest reopening.[437,438] 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 to 100 % and remove positive end-expiratory pressure. If a tension pneumothorax is suspected, emergency decompression is necessary.[423,424]

Medications during resuscitation. As a general principle, stop all infusions other than those needed for resuscitation. Amiodarone (300 mg) or lidocaine (100 mg) may be administered intravenously after three failed shocks to treat VF/ pVT.[424,439]. Injecting adrenaline (1 mg) shortly after cardiac surgery is controversial. The European Association of Cardio-Thoracic 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[423,424,439], although lower doses (50–100 mcg boluses) may be considered in *peri*-arrest situations based on expert consensus.[375,388,424,429,437]

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 (TOE) 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,

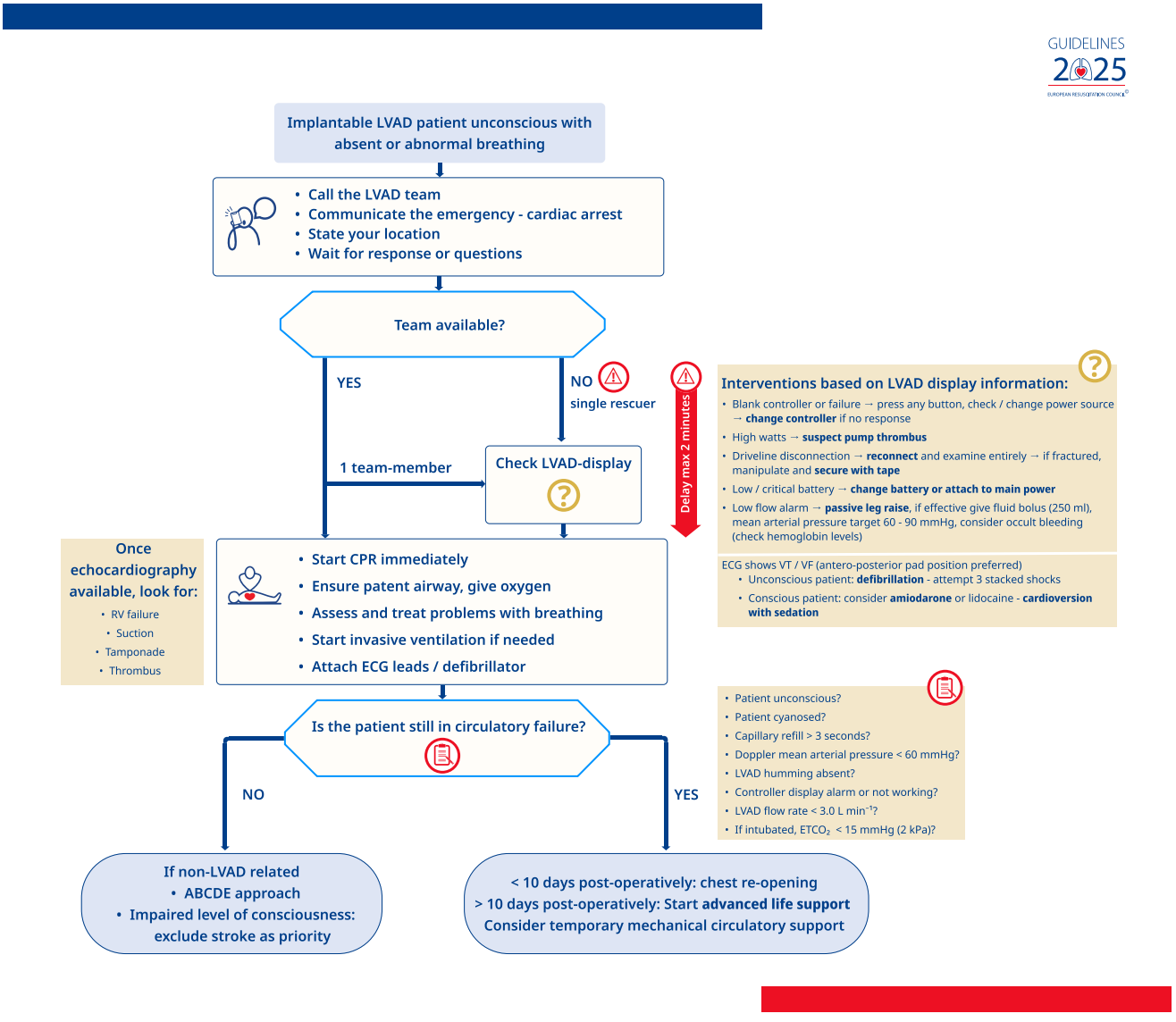

Fig. 18 – Left ventricular assist device ALS algorithm.
LVAD – left ventricular assist device, ALS – advanced life support, VT – ventricular tachycardia, VF – ventricular fibrillation, ECG – electrocardiography.

dynamic left ventricular outflow tract obstruction and aortic dissection can generally be identified using TOE.[432,433,440–447] TOE 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.[448,449]

Early resternotomy

Refractory cardiac arrest requires resternotomy within 5 min, in order to perform open cardiac compressions compressions, release any tamponade and correct underlying causes. This is a safe procedure in the ICU,[450] leading to higher survival rates, especially if performed with minimal delay and in the presence of surgically repairable problem on reopening.[451] Resternotomy should be attempted as part of the resuscitation protocol of cardiac patients until at least day 10 post-operatively.[424]

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 improving coronary and brain perfusion if coordinated with cardiac compression (1:1 ratio, with maximal amplification).[452] 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 if cardiac compressions 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.[424] However, there is limited data addressing this specific scenario, since most studies relate to cardiogenic shock or paediatric populations.[453]

Left ventricular assist devices. 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 the absence of responsiveness and breathing. When performed, conventional CPR is recommended. The risk of device dislodgement during CPR appears minimal.[425,426]

The ERC recommendations are based on the ILCOR 2025 CoSTR and the British Societies guidelines 2025[425,426] on the management of emergencies in implantable LVAD recipients in transplant centres (Fig. 18).

- Immediate activation of specialised teams for unconscious LVAD patients.
- Start CPR while simultaneously attempting to restore the device if multiple rescuers are available. Consider delaying CPR for up to 2 min 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 the 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. 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 the 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 & 4 T.

Cardiac arrest in sports

The absolute risk of experiencing of cardiac arrest during physical exercise is small.[454] The incidence of cardiac death associated with sport or exercise in the general population is reported between 0.46 and 6.8 per 100,000 person-years.[455–458] Large population-based studies of cardiac arrest in athletes from the USA and Europe indicate regional differences in the underlying causes of death.[459,460] About a third survived; most cases occurred during non-elite competitive or recreational sports, and bystander CPR was performed in 75 %. In patients aged $\leq$ 35 years, premature coronary artery disease and sudden arrhythmic death syndrome predominated, followed by myocarditis. In athletes aged $\geq$ 35 years, coronary artery disease predominated.[461] The likelihood of sport-related cardiac arrest is highest for males aged between 40 and 60 and with pre-existing cardiomyopathy. In 22 % of cases no pathology was found and survival was better with early resuscitation, use of AED, and presence of professional staff.[462] Highest incidences are reported for football players during competition and for running and gym exercise during non-competition.[463]

Cardiac arrest during sport or exercise requires rapid recognition and effective treatment if the affected individual is to survive. Improved survival of cardiac arrest during sport is attributed to witnessed events, prompt resuscitation and availability of an AED.[455,464] This evidence supports the importance of planning, adhering and implementing standard BLS in event-prone sport events.

For those athletes who have a shockable cardiac rhythm during cardiac arrest, a minimum of three shocks should be delivered on the field of play if the shockable rhythm persists.

The medical team should consider moving the athlete in cardiac arrest to a designated rendezvous point, in the safest and most effi-

cient way. If that is not possible, CPR should be continued on the field.[465] These plans should be rehearsed regularly.

Unlike in professional sports, recreational athletes, supporters and fans may be less aware of the risk of 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.[466] Once an athlete is successfully resuscitated, thorough cardiological assessment and follow-up are vital before considering a return to sport.[467]

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,[467] but the AHA and American College of Cardiology conclude that there is insufficient evidence to support this as a screening measure.[468,469] 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 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.[470]

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 diseases, the presence of the signs or symptoms suggestive of cardiovascular disease and the desired or anticipated exercise intensity.[471] In this context, screening approaches should be adapted to the specific characteristics and risk profile of the target population.[467,470]

Commotio cordis

Commotio cordis is a rare but potentially fatal cause of 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.[472,473] It has historically been cited as a leading cause of cardiac arrest in young athletes, particularly in sports involving high-velocity projectiles such as baseball, lacrosse, and hockey[474,475]. In football, commotio cordis has been reported in 9 % of cardiac arrests in players $\leq$ 35 years old, with 79 % of cases caused by a ball impact to the chest.[462]

Survival rates are approximately 66 % in 2008–2023.[474] Witnessed CA, early recognition, immediate CPR initiation, and rapid defibrillation with an AED remain the most important factors for survival[474–477] Survival rates are 40 % when resuscitation occurs within 3 min, but drop to just 5 % when delayed beyond this window.[475,476]

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.[478,479]

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.[480] However, validity of data should be assessed before use.[481,482] Feedback systems have a positive effect on the quality of care and medical personnel are willing to receive feedback.[483,484] 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.[485]

Resuscitation during transport

CPR quality is reduced if performed during transportation, affecting correct hand position, chest compressions rate and depth, pauses, and overall CPR quality.[201,486] 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.

Arterial line in the prehospital setting

Invasive blood pressure measurement in OHCA patients in a HEMS situation is feasible,[383,487–491] and can help prehospital teams to guide resuscitation and post-resuscitation care,[383,487,489,492] specifically in haemodynamically unstable patients.[493,494]

Resuscitation by two-member ALS crews

Teams larger than two ALS members improve CPR quality and efficiency in rhythm recognition, adrenaline administration, and intubation.[495] No significant no-flow fraction differences were found between two-, three-, and four-member teams.[495] Two-paramedic teams were slower and more error-prone than paramedic-emergency medical technician teams.[495] The evidence is not robust enough to support formal recommendations for education, protocols, or equipment regarding ALS provided by two HCPs. However, studies suggest that pre-filled syringes and automation-assisted protocols might improve team performance.[495]

Inflight cardiac arrest and microgravity resuscitation

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.[496,497] A *meta*-analysis reported an incidence 0.09 cardiac arrest events per million passengers.[498] Not all airlines are equipped with an AED and ALS equipment.[499–501]

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. If ROSC is unlikely (pre-existing conditions, absence of an AED), the expected time for diver-

sion and landing would be too long to improve the prognosis reasonably.

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.

Recommendations exist from the German Society of Aerospace Medicine and the European Society of Aerospace Medicine Space Medicine Group.[502] However, a recent simulation study[503] using underwater methods to approximate microgravity found that all commonly recommended CPR techniques[504] (including the Reverse-Bear Hug, Schmitz Hinkelbein, Cologne, and Evetts-Russomano) resulted in rates below 5 % of effective chest compressions. In the absence of data from real space environment, it is not possible to endorse any particular CPR method.

The ERC key recommendations are summarised here

- Airway management, defibrillation and IV/IO access are similar to terrestrial ALS, but only once the patient is secured.
- Consider mechanical CPR
- 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.

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 general population, because access to healthcare facilities is more complicated, on-board resources are limited, and transfers can be prolonged.[505] 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.[506] Qualified medical air transportation is an option to cover long distances to medical facilities.

Special patient groups

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[507] and the Global Initiative for Asthma Strategy 2021,[508] while for chronic obstructive lung disease (COPD) recommendations are based on those of the Global initiative for chronic obstructive lung disease.[509] A recent ILCOR Evidence Update[8] 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.

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/45 mmHg and arterial pH $\leq$ 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.[510,511] In selected unconscious cases with severe hypoxemia, veno-venous ECMO may be an option to prevent hypoxic cardiac arrest.[512,513]

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.[514–519] Cardiac arrest in obstructive lung disease is usually associated with a non-shockable rhythm and poor survival rates.[520,521]

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.[522,523] 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.[524–530] If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest while temporarily disconnecting the tracheal tube may relieve air trapping.[524,527,529] 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).[528] 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.[531]

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 insen-

sible losses. Give fluids cautiously to prevent potential adverse effects.[532]

ECPR has been used successfully in patients with life threatening asthma.[513,533] Consistent with the ERC Guidelines 2025 ALS, ECPR may be considered if conventional therapies fail and there is immediate access to this treatment.

Cardiac arrest in haemodialysis patients

Chronic kidney disease is a global health problem affecting 1 in 10 people worldwide[534] and the WHO projects it will become the 5th most prevalent chronic condition by 2040.[535]

Risk factors for cardiac arrest in dialysis patients are summarised in Table 10. 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.[536–540] The risk period extends over the 12 h 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.[541,542] Clinically significant arrhythmias are also more common on dialysis compared with non-dialysis days.[543]

Out-of-hospital cardiac arrest in haemodialysis patients

Cardiac arrest accounts for 47.1 % of deaths in HD patients, occurring 20 times more frequently in dialysis patients compared with the general population.[544–546] 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.[537] 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.[547]

Table 11 was reproduced with permission from UK Kidney Association Hyperkalaemia Guideline in adults.[50]

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.[546] Following cardiac arrest, 24 % of haemodialysis patients survived to hospital discharge and 15 % still were still alive at 1 year following cardiac arrest.[537] The poor outcome in one report may reflect the low rate of bystander CPR and AED use.[548,549]

Table 10 – Risk factors for cardiac arrest in haemodialysis 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 h 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^{-1})
5. History of heart disease
6. QT-prolonging medications
7. Non-compliance with diet and / or dialysis regimen

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.[537–539,550] Multiple studies in HD patients demonstrate ROSC in 44–72 %[551–553] and survival to hospital discharge ranges from 22-31 %.[551–555] Duration of CPR and older age were predictors of a poor outcome.[555] The 'Get With The Guidelines-Resuscitation' registry shows comparable neurological outcomes in HD patients compared with non-dialysis patients (17 % vs 16 %, $p = 0.07$).[551] These findings suggest that outcomes for HD patients are no worse than other patients dispelling the perception of futility.[556] Nevertheless, a large national registry of IHCA found that dialysis patients had lower overall resuscitation quality compared with case-matched non-dialysis patients.[551] Shortfalls in resuscitation practice in dialysis patients include a delay in initiation of CPR[546] and failure to achieve timely first defibrillation for a shockable rhythm.[536,546,551]

Modifications to cardiopulmonary resuscitation in haemodialysis patients

Start resuscitation by following the standard ALS algorithm. Assign a trained dialysis nurse to operate the HD machine (Fig. 10). 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.[557] Most are equipped with AEDs,[546,558] but manual defibrillators may also be used by trained staff. Dialysis clinics are predominantly nurse-led units. 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.[537,546] 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

Table 11 – Cardiac arrest during dialysis in outpatient clinics.
Reproduced with permission from UK Kidney Association Hyperkalaemia Guideline in adults.

Study	N=	Before HD	During HD	After HD
Karnik 2001[538]	400	7 %	81 %	12 %
La France 2006[539]	38	8 %	78 %	14 %
Davis 2008[537]	110	10 %	70 %	20 %

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.

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.[538,559,560] 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).[561] 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).[551] 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 cardiac death, but this is only achievable with home haemodialysis.

Resuscitation in obese patients

Obesity is defined by WHO as a body mass index exceeding 30 kg m^{-2}.[562] Obesity can increase cardiovascular morbidity and mortality directly and indirectly.[563] Given the global increase in obesity, an ILCOR scoping review assessed the evidence for cardiac arrest treatment and outcomes in obese patients.[564] 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.[564] 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.[565,566] 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.[567]

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.[568] 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.[569,570]

Surgically placed corrective "Nuss bars" complicate CPR, as they require significantly increased force to deliver proper chest compression depth.[571] Rescuer fatigue and organ damage (liver lacerations specifically) are to be considered when mechanical chest compressions are delivered.[572] Defibrillation in the presence of a Nuss bar may divert electrical current flow through the low resistance metallic bar when defibrillation pads are placed anterolaterally.[573]

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 3–4 cm, 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.[574]

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.[575]

These Guidelines has been informed by an ILCOR scoping review[334] and international guidelines.[575–583] Most guidance is based on observational data, expert opinion and physiological principles. This update introduces a new process for management of maternal cardiac arrest along with key figures and tables to support clinical application.

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.[584] While there are limited data on precise causes of maternal cardiac arrest, MBRRACE-UK reports causes of maternal mortality from 2020 to 2022 to be 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.[575] 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.[585]

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 resuscitative hysterotomy.[586] 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.[587]

Prevention of maternal cardiac arrest

Maternal cardiac arrest is usually preventable, though in certain causes, such as amniotic fluid embolism, arrest may occur before intervention.[588] Approach any deteriorating pregnant or peripartum patient following the ABCDE approach. There is evidence to suggest that implementing an obstetric-specific early warning system enables early recognition of deteriorating pregnant patients; however, uptake across Europe is low.[589–593] Early involvement of an obstetrician, along with simultaneous activation of maternal and neonatal resuscitation teams, is essential.

Aortocaval compression

Aortocaval compression (i.e., compression of the inferior vena cava and the aorta by the gravid uterus) usually manifests by the 20th week of gestation. This may significantly reduce venous return and subsequently cardiac output in the supine position.[594,595] In healthy pregnant patients with preserved intrinsic compensatory mechanisms, the effects of aortocaval compression may be absent or minimal.[596] 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 (Fig. 19), though supporting data are derived from non-cardiac arrest and simulation studies. ILCOR conducted a scoping review on this topic and found insufficient evidence to recommend either method over the other during CPR in pregnant patients.[8] 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.[597–599] However, wedges are rarely available, and simulation and animal studies have questioned the effectiveness of chest compressions in the left lateral position.[600,601]

Accordingly, the ERC suggests performing manual left uterine displacement in maternal cardiac arrest. It can be achieved by placing one or both hands below the uterus, on the patient's right side, and pushing upwards and to the left, or if standing on the patient's left, reaching across to cup the uterus from below and lifting upward and leftward (Fig. 19).[602,603]

Definitive relief of aortocaval compression may be achieved only by resuscitative hysterotomy (sometimes known as "perimortem Caesarean section").

Resuscitative hysterotomy

Resuscitative hysterotomy should be performed by a skilled team at the site of cardiac arrest to achieve ROSC by relieving aortocaval compression.[604] Previous guidelines recommended starting the procedure at 4 min and completing uterine evacuation by 5 min.[605] The maternal cardiac arrest supplement to the ALS algorithm (Table 12) shifts the focus to preparing for resuscitative hysterotomy, highlighting the time-sensitive nature of the intervention.

- The ILCOR review found insufficient evidence to support a specific time for initiating resuscitative hysterotomy.
- Time-sensitive resuscitative hysterotomy depends on clearly designated team competences, rapid system activation, and equipment readiness, all requiring training and rehearsal.[606,607]
- If arrest results from hypotension despite optimal resuscitation, it is suggested that resuscitative hysterotomy be performed as soon as possible.
- The procedure should not be considered futile beyond 5 min, however, the benefit declines steadily the longer emptying the uterus is delayed.[586,608]
- In exceptional cases, ECPR may be initiated immediately, with uterine evacuation postponed due to anticoagulation-related bleeding risks.[609,610]
- In the prehospital setting, the procedure requires adequate access to the patient and a trained clinician, otherwise prioritise time-critical transport to hospital

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.

Extracorporeal life support

A retrospective analysis of peripartum patients requiring ECPR from the International Registry of Extracorporeal Life Support Organization, identified 280 patients,[611] with 70 % survival. Survival rates were higher when extracorporeal membranous oxygenation (ECMO) was initiated prior to cardiac arrest.

If available, early ECMO can be considered by the multidisciplinary team in pregnant patients with signs of impending circulatory collapse.

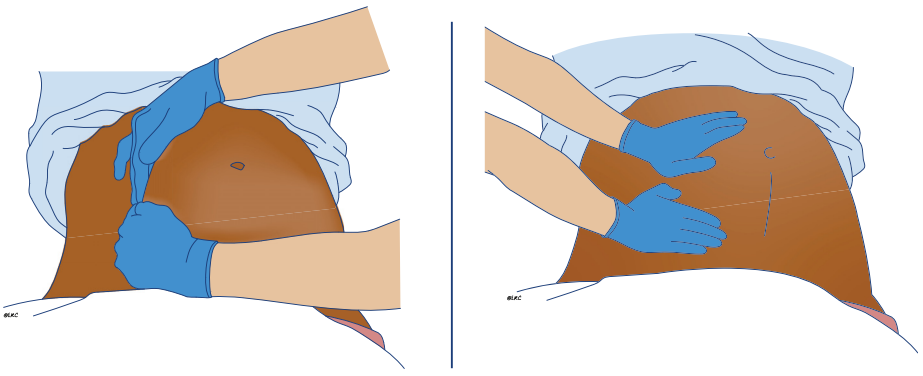

Fig. 19 – Manual left uterine displacement manoeuvre.

Table 12 – Modifications to advanced life support in obstetric patients.

ALS during pregnancy	Rationale
Detection of cardiac arrest: unconscious + abnormal breathing = suspected cardiac arrest	*No change*
Call for help – "maternal arrest team"	Consider pregnancy in any collapsed woman of childbearing age. Call maternal cardiac arrest team (including an obstetrician and neonatologist)
Manual (left) uterine displacement throughout	Relieve aortocaval compression to improve cardiac output as early as possible and maintain it throughout resuscitation. Establish manual left uterine displacement when two or more team members are available—one performing manual left uterine displacement, the other performing CPR. (Fig. 19)
Chest compression quality parameters and ventilation-compression-ratio	*No change*
Airway management	Aspiration and failed intubation risk are both increased. Use a stepwise approach (bag-mask, tracheal tube or supraglottic airway if tracheal intubation fails), according to rescuer skills. Try to achieve a ramped position. Intubation should be performed by an experienced person. Equipment considerations: - Short-handled laryngoscope for large breasts - Video laryngoscope as standard for intubation - Smaller tracheal tube with guidance
Defibrillation – Shock energy	*No change*
Pad position	No change: ensure defibrillation pads are placed beneath, not over, enlarged breast tissue
Fetal monitoring	Remove internal & external fetal monitors before defibrillation
Identify common and reversible causes	See Table 13 on 4Hs, 4Ts and 4Ps
Medications	
Vascular access early	Consider IV/IO access above the diaphragm if possible .
Dosage and time of adrenaline, amiodarone and lidocaine	*No change*
Calcium chloride	10 ml IV calcium chloride 10 %: for Mg overdose, low calcium or hyperkalaemia
Magnesium	2 g IV: for polymorphic VT 4 g IV: for eclampsia
Tranexamic acid	1 g IV: for haemorrhage
Resuscitative Hysterotomy (Perimortem Caesarean section)	In patients > 20 weeks' gestation or fundus above umbilicus. Resuscitative hysterotomy is a time-sensitive intervention. Preparation to perform should be undertaken early. Resuscitative hysterotomy should be performed as soon as possible at the site of cardiac arrest by a skilled team.
Post-resuscitation care	Stabilise mother to stabilise fetus and prepare for major obstetric haemorrhage.

Relieve aortocaval compression. Aortocaval compression should be relieved as early as possible and maintained throughout resuscitation (Fig. 19).

Chest compressions. Currently, there is no strong evidence to support modifying hand position for chest compressions during pregnancy. 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.[612] 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.[613]

With regard to mechanical chest compression devices, there is insufficient evidence to make a recommendation for or against their use, though in certain scenarios, practical alternatives are unavailable – for example, during extrication or transport in the prehospital setting.[614]

Airway management. Pregnancy increases the risk of aspiration of gastric contents and difficult intubation.[615–617] Manage the airway as directed by the ERC Guidelines 2025 ALS (bag-mask, tracheal tube or supraglottic airway if tracheal intubation fails) 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.[617]

Defibrillation. When using anterolateral positioning, ensure defibrillation pads are placed beneath, not over, enlarged breast tissue. Defibrillation energy levels are the same as those for non-pregnant adults.[618] To avoid burns, external fetal monitoring should be removed before defibrillation.

Medications. Medications used in the resuscitation of pregnant women are the same as for the general adult population. While there is limited evidence, intravenous or intraosseous access should be established above the level of the diaphragm whenever possible, based on physiological considerations.[619]

Reversible causes. The 4Hs and 4Ts are important considerations in all cardiac arrests, including during pregnancy, and may prompt pregnancy-specific diagnoses such as hypovolaemia from a ruptured ectopic pregnancy or pulmonary embolism, the latter being more common in pregnant and postpartum women (Table 13). However, there are also pregnancy-specific causes of cardiac arrest, which are now introduced as the 4Ps:

Pre-eclampsia and eclampsia: Follow established guidelines for management.[620] Initiate immediate treatment for severe hypertension, defined as $\geq$ 160/110 mmHg. In severe cases give 4 g IV magnesium sulphate over 5 to 15 min to treat and prevent eclampsia, followed by 1 g/h over 24 h.[620] Consider pulmonary oedema and treat accordingly. Magnesium toxicity from treatment overdose may cause cardiac arrest (see Table 14).

Puerperal sepsis: In suspected puerperal sepsis, take blood cultures, give early broad-spectrum antibiotics, and consider obstetric sources such as chorioamnionitis, retained products, endometritis, or surgical site infection that may require intervention.

Placental and uterine complications: Examples include placental abruption, placenta praevia or accreta, uterine atony, rupture, inversion, and amniotic fluid embolism. Seek clinical signs of overt or occult haemorrhage (vaginal or intra-abdominal). Examine the abdomen for peritonism and free fluid (use ultrasound if available), assess for hypovolaemia, check haemoglobin concentration and assess clotting function. Request blood products, alert the obstetric team, and prepare for urgent surgical intervention. The management of amniotic fluid embolism is supportive: maintain oxygenation, treat haemorrhage with blood products, correct disseminated intravascular coagulation (DIC), and immediately involve obstetric, anaesthetic and critical care teams.

Peripartum cardiomyopathy: Treat as acute heart failure. Consider intravenous diuretics, vasodilators (e.g. nitrates), and inotropes if indicated. Avoid fluid overload. Seek early echocardiography and cardiology input.

Table 13 – Potentially reversible causes of cardiac arrest in obstetric patients: 4Hs,4Ts,4Ps.

Potentially reversible causes in pregnancy	Obstetric-specific considerations
Hypoxia	- high risk of aspiration - high risk of difficult airway - reduced lung capacity - higher oxygen demand
Hypovolaemia	- aortocaval compression - haemorrhage (e.g., ectopic pregnancy and other pregnancy-related causes below, DIC) - distributive (e.g., high regional block, anaphylaxis)
Hypo-/hyperkalaemia, other electrolyte disorders	- also consider magnesium levels
Hypo-/hyperthermia	
Thrombosis (coronary and pulmonary)	- high risk for pulmonary embolism - myocardial infarction - coronary and aortic dissection - (amniotic fluid embolism)
Tamponade (cardiac)	
Tension pneumothorax	- nitrous oxide in pre-existing pneumothorax
Toxic agents (poisoning)	- local anaesthetic and other perioperative drugs
Pre-eclampsia and eclampsia	- Follow guidelines - Consider pulmonary oedema - Magnesium overdose may cause cardiac arrest
Puerperal sepsis	- take blood cultures and give early broad-spectrum antibiotics - Chorioamnionitis, retained products, endometritis, - Surgical site infection
Placental and uterine complications	- Placental abruption, placenta praevia, placenta accreta, uterine atony, uterine rupture, uterine inversion, amniotic fluid embolism)
Peripartum cardiomyopathy	- Treat as acute heart failure. - Early echocardiography - Consult cardiology experts

Table 14 – Suggested pre-prepared equipment to perform Resuscitative Hysterotomy.We suggest as a minimum a scalpel and 3 cord clamps to be available in all resuscitation carts.

For staff	- Sterile gloves (non-latex) - Gowns - Masks
For mother	- Skin preparation solution - 3 scalpels (1: incision, 2: umbilical cord, 3: backup) - Dissection scissors - Laparotomy sponges - 4 haemostat clamps - Retractor - Gauze (ideally haemostatic)
For baby	- 3 cord clamps - Hat, 4 towels, heated incubator - Equipment to resuscitate the neonate

Cardiovascular disease

Percutaneous coronary intervention is the preferred reperfusion strategy for STEMI in pregnancy.[582] 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.[621] Point-of-care ultrasound is invaluable in cases where it is unclear if the cause of arrest is due to concealed bleeding or thromboembolism.[575]

Haemorrhage

This is a leading cause of maternal morbidity and mortality.[622] 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.[623]

Management includes:

- Immediate involvement of obstetric, anaesthetic, and critical care teams who should follow existing guidelines for the management of major obstetric haemorrhage.[623,624]
- Activation of the major obstetric haemorrhage protocol, with prompt blood transfusion and correction of coagulopathy.
- Administration of tranexamic acid 1 g IV in cases of suspected bleeding.[625–628]

Maternal considerations for post resuscitation care

Post resuscitation care in pregnancy aligns with standard adult management, with some important additional considerations. Hypothermic temperature control has been reported in isolated cases during early pregnancy, with fetal heart rate monitoring in place. There was no evidence of harm and maternal and neonatal outcomes were favourable.[629,630] 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 amniotic fluid embolism.

Maternal critical illness requires a multidisciplinary approach making it essential to involve all relevant specialties, including obstetrics, anaesthesia, critical care and neonatology.[631]

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.

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 time-sensitive resuscitative hysterotomy and neonatal resuscitation.[593]

Pregnant and peripartum women can deteriorate to cardiac arrest anywhere and medical facilities and services must be prepared for such events. There should be[632–638]:

- Plans and equipment in place for resuscitation of both the pregnant woman and newborn (see Table 14).
- Early involvement of obstetric, anaesthetic, critical care, and neonatal teams.
- Regular multidisciplinary training in obstetric emergencies, using simulated resuscitation scenarios.
- Review and debriefing following clinical events to support learning and team performance

Collaborator

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Disclosures

Declarations of competing interests for all ERC Guidelines authors are displayed in a COI table which can be found online at https://doi.org/10.1016/j.resuscitation.2025.110753.

Author details

[a]*Department of Anesthesiology, University Medical Centre, Johannes-Gutenberg University, Mainz, Germany* [b]*Cardiopulmonary Resuscitation Lab, School of Medicine, University of Crete, Heraklion, Crete, Greece* [c]*Department of Anesthesiology, Onassis Hospital, Athens, Greece* [d]*Faculty of Education Sciences and CLINURSID Research Group, Universidade de Santiago de Compostela, Santiago de Compostela, Spain* [e]*Simulation and Intensive Care Unit of Santiago (SICRUS) Research Group, Health Research Institute of Santiago, University Hospital of Santiago de Compostela-CHUS, Santiago de Compostela, Spain* [f]*Departments of Renal and Internal Medicine, Victoria Hospital, Kirkcaldy, Fife, UK* [g]*Extreme Environments Laboratory, School of Sport, Health and Exercise Science, University of Portsmouth, UK* [h]*Maatschappij tot Redding van Drenkelingen, Amsterdam, the Netherlands* [i]*Nottingham University Hospitals NHS Trust, Nottingham, UK*[j]*Univ. Grenoble Alpes, Emergency Department*

and Mobile Intensive Care Unit, CNRS, UMR 5525, TIMC, 38000 Grenoble, France [k]Maccabi Healthcare Services and Hebrew University, Faculty of Medicine, Israel [l]University Medical Center Schleswig-Holstein, Institute for Emergency and Rescue Medicine, Kiel, Germany [m]Cardiology Department, University Clinical Hospital of Santiago de Compostela, Spain [n]Institute of Health Research of Santiago de Compostela (IDIS), Biomedical Research Networking Centres on Cardiovascular Disease (CIBER-CV), A Coruna, Spain [o]Faculty of Medicine, University of Bern, Switzerland[p]Department of Anaesthesia, Intensive Care Medicine, Emergency Medicine and Pain Medicine, University Medicine Greifswald, Germany[q]Department of Anesthesiology and Intensive Care Medicine, University Hospital Ulm, Germany [r]Institute of Sports and Preventive Medicine, Saarland University, Saarbrücken, Germany [s]Department of Anaesthesiology and Intensive Care Medicine, St. John of God Hospital, Paracelsus Medical University, Salzburg, Austria[t]Department of Anaesthesia and Intensive Care Medicine, Charles University, Third Faculty of Medicine and FNKV University Hospital in Prague, Czech Republic [u]Department of Cardiovascular Sciences, Fondazione Policlinico Universitario A. Gemelli, IRCCS, Italy [v]Institute of Anesthesiology and Intensive Care, Catholic University of the Sacred Heart, Italy[w]Department of Emergency Medicine, University Hospital of Heraklion, Crete, Greece [x]Bielefeld University, Medical School and University Medical Center OWL, EvKB, Department of Anesthesiology, Intensive Care, Emergency Medicine, Transfusion Medicine and Pain Therapy, Bielefeld, Germany [y]Emergency Medical Services of the Hradec Králové Region, Hradec Králové, Czech Republic [z]Department of Anaesthesiology and Intensive Care Medicine, Charles University in Prague, University Hospital Hradec Králové, Czech Republic [aa]Department of Anaesthesiology and Intensive Care Medicine, Charles University, Faculty of Medicine in Hradec Králové, Czech Republic [ab]Departement of Anaesthetics, University Hospital Southampton, Southampton SO168AD, UK

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