RESUS 8904 No. of Pages 68
ARTICLE IN PRESS
RESUSCITATION XXX (2021) XXX –XXX

Available online at www.sciencedirect.com

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances

Carsten Lott[a,*], Anatolij Truhlář[b,c], Annette Alfonzo[d], Alessandro Barelli[e], Violeta González-Salvado[f], Jochen Hinkelbein[g], Jerry P. Nolan[h,i], Peter Paal[j], Gavin D. Perkins[k,l], Karl-Christian Thies[m], Joyce Yeung[k,l], David A. Zideman[n], Jasmeet Soar[o], the ERC Special Circumstances Writing Group Collaborators[1]

[a] Department of Anesthesiology, University Medical Center, Johannes Gutenberg-University Mainz, Germany
[b] Emergency Medical Services of the Hradec Králové Region, Hradec Králové, Czech Republic
[c] Department of Anaesthesiology and Intensive Care Medicine, Charles University in Prague, University Hospital Hradec Králové, Hradec Králové, Czech Republic
[d] Departments of Renal and Internal Medicine, Victoria Hospital, Kirkcaldy, Fife, UK
[e] Anaesthesiology and Intensive Care, Catholic University School of Medicine, Teaching and Research Unit, Emergency Territorial Agency ARES 118, Rome, Italy
[f] Cardiology Department, University Clinical Hospital of Santiago de Compostela, Institute of Health Research of Santiago de Compostela (IDIS), Biomedical Research Networking Centres on Cardiovascular Disease (CIBER-CV), A Coruña, Spain
[g] Department of Anaesthesiology and Intensive Care Medicine, University Hospital of Cologne, Cologne, Germany
[h] Resuscitation Medicine, University of Warwick, Warwick Medical School, Coventry, CV4 7AL, UK
[i] Anaesthesia and Intensive Care Medicine, Royal United Hospital, Bath, BA1 3NG, UK
[j] Department of Anaesthesiology and Intensive Care Medicine, Hospitallers Brothers Hospital, Paracelsus Medical University, Salzburg, Austria
[k] Warwick Clinical Trials Unit, Warwick Medical School, University of Warwick, Coventry, UK
[l] University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK
[m] Department of Anesthesiology, Critical Care and Emergency Medicine, Bethel Medical Centre, OWL University Hospitals, Bielefeld University, Germany
[n] Thames Valley Air Ambulance, Stokenchurch, UK
[o] Southmead Hospital, North Bristol NHS Trust, Bristol, UK

Abstract

These European Resuscitation Council (ERC) Cardiac Arrest in Special Circumstances guidelines are based on the 2020 International Consensus on Cardiopulmonary Resuscitation Science with Treatment Recommendations. This section provides guidelines on the modifications required to basic and advanced life support for the prevention and treatment of cardiac arrest in special circumstances; specifically special causes (hypoxia, trauma, anaphylaxis, sepsis, hypo/hyperkalaemia and other electrolyte disorders, hypothermia, avalanche, hyperthermia and malignant hyperthermia, pulmonary embolism, coronary thrombosis, cardiac tamponade, tension pneumothorax, toxic agents), special settings (operating room, cardiac surgery, catheter laboratory, dialysis unit, dental clinics, transportation (in-flight, cruise ships), sport, drowning, mass casualty incidents), and special patient groups (asthma and COPD, neurological disease, obesity, pregnancy).

* Corresponding author.
E-mail address: lott@uni-mainz.de (C. Lott).
https://doi.org/10.1016/j.resuscitation.2021.02.011
Available online xxx

Introduction

Irrespective of the cause of cardiac arrest, the most important interventions are universal and according to the chain of survival.[1] These include early recognition and calling for help, management of the deteriorating patient to prevent cardiac arrest, prompt defibrillation and high-quality cardiopulmonary resuscitation (CPR) with minimal interruption of chest compressions, treatment of reversible causes, and post-resuscitation care. In certain conditions, however, basic and advanced life support interventions may require modification. This guideline for resuscitation in special circumstances is divided into three parts: special causes, special settings and special patients. The first part covers treatment of potentially reversible causes of cardiac arrest, for which specific treatment exists, and which must be identified or excluded during advanced life support (ALS). For improving recall during ALS, these are divided into two groups of four, based upon their initial letter – either H or T – and are called the '4Hs and 4Ts': Hypoxia; Hypovolaemia; Hypo-/hyperkalaemia and other electrolyte disorders; Hypo-/hyperthermia; Thrombosis (coronary and pulmonary); Tamponade (cardiac); Tension pneumothorax; Toxic agents (poisoning). The second part covers cardiac arrest in special settings, where universal guidelines have to be modified due to specific locations or location-specific causes of cardiac arrest. The third part is focused on patients with specific conditions, and those with certain long-term comorbidities where a modified approach and different treatment decisions may be necessary.

Many of the chosen topics were not part of the ILCOR reviews. ILCOR has published reviews on pulmonary embolism,[2] extracorporeal CPR (ECPR),[2] drowning[3] and evidence updates on pregnancy[2] and opioid toxicity.[2] Most of the evidence is derived from individual systematic reviews, scoping reviews and evidence updates, recommendations are provided as expert consensus following discussion in the writing group. Whenever an ILCOR systematic review or a GRADE like systematic review informs the recommendation, the level of recommendation is provided.

There are no major changes in the 2021 adult Special Circumstances guidelines. There is greater emphasis on the priorisation of recognition and management for reversible causes in cardiac arrest due to special circumstances. The guidelines reflect the increasing evidence for extracorporeal CPR (eCPR) as management strategy for selected patients with cardiac arrest in settings in which it can be implemented. This ERC guideline follows European and international guidelines for treatment recommendations (electrolyte disorders, sepsis, coronary thrombosis, accidental hypothermia and avalanche rescue). The trauma section has been revised with additional measures for haemorrhage control, the toxic agents section comes with an extensive supplement, focusing on management of specific toxic agents. Prognostication of successful rewarming in hypothermic patients follows more differentiated scoring systems (HOPE score; ICE score). In avalanche rescue priority is given to ventilations as hypoxia is the most likely reason of cardiac arrest. Caused by the increasing number of patients from that special settings, recommendations for cardiac arrest in the catheterisation laboratory and in the dialysis unit have been added.

These guidelines were drafted and agreed by the Resuscitation in Special Circumstances Writing Group members. The methodology used for guideline development is presented in the Executive summary.[4] The guidelines were posted for public comment in October 2020. The feedback was reviewed by the writing group and the guidelines was updated where relevant. The Guideline was presented to and approved by the ERC General Assembly on 10th December 2020.

Key guideline highlights are summarised in Fig. 1.

Concise guidelines for clinical practice

Special causes

Hypoxia

- Follow the standard ALS algorithm when resuscitating patients with asphyxial cardiac arrest.
- Treat the cause of the asphyxia/hypoxaemia as the highest priority because this is a potentially reversible cause of the cardiac arrest.
- Effective ventilation with the highest feasible inspired oxygen is a priority in patients with asphyxial cardiac arrest.

Hypovolaemia

Traumatic cardiac arrest (TCA)

- Resuscitation in TCA should focus on the immediate, simultaneous treatment of reversible causes.
- The response to TCA is time critical and success depends on a well-established chain of survival, including focused pre-hospital and specialised trauma centre care.
- TCA (hypovolemic shock, obstructive shock, neurogenic shock) is different from cardiac arrest due to medical causes; this is reflected in the treatment algorithm (Fig. 2).
- Use ultrasound to identify the underlying cause of cardiac arrest and target resuscitative interventions.
- Treating reversible causes simultaneously takes priority over chest compressions. Chest compression must not delay treatment of reversible causes in TCA.
- Control haemorrhage with external pressure, haemostatic gauze, tourniquets and pelvic binder.
- 'Don't pump an empty heart'.
- Resuscitative thoracotomy (RT) has a role in TCA and traumatic peri-arrest.

Anaphylaxis

- Recognise anaphylaxis by the presence of airway (swelling), breathing (wheeze or persistent coughing), or circulation (hypotension) problems with or without skin and mucosal changes. This can be in the context of a known trigger in a patient with an allergy, or suspected anaphylaxis in a patient with no previous history of allergy.
- Call for help early.
- Remove or stop the trigger if feasible.
- Give intramuscular (IM) adrenaline (0.5 mg (which is 0.5 ml of a 1 mg in 1 ml ampoule of adrenaline)) into the anterolateral thigh as soon as anaphylaxis is suspected. Repeat the IM adrenaline if there is no improvement in the patient's condition after about 5 min.
- Ensure the patient is lying and do not suddenly sit or stand the patient up.
- Use an ABCDE approach and treat problems early (oxygen, fluids, monitoring).
- Give an IV crystalloid fluid bolus early and monitor the response – large volumes of fluids may be needed.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

Fig. 1 – Special circumstances summary infographic.

- Consider IV adrenaline as a bolus (20–50 mcg) or infusion for refractory anaphylaxis or in specialist care settings where the skills are available.
- Consider alternative vasopressors (vasopressin, noradrenaline, metaraminol, phenylephrine) in refractory anaphylaxis.
- Consider IV glucagon in patients taking beta-blockers.
- Start chest compressions and ALS as soon as cardiac arrest is suspected and follow standard guidelines.
- Consider ECLS or ECPR for patients who are peri-arrest or in cardiac arrest as a rescue therapy in those settings where it is feasible.
- Follow existing guidelines for the investigation and follow-up care of patients with suspected anaphylaxis and confirmed anaphylaxis.

Sepsis

Cardiac arrest prevention in sepsis

- Follow the Surviving Sepsis Guidelines Hour-1 bundle for the initial resuscitation of sepsis and septic shock (Fig. 3).

Specifically:

- Measure lactate level.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

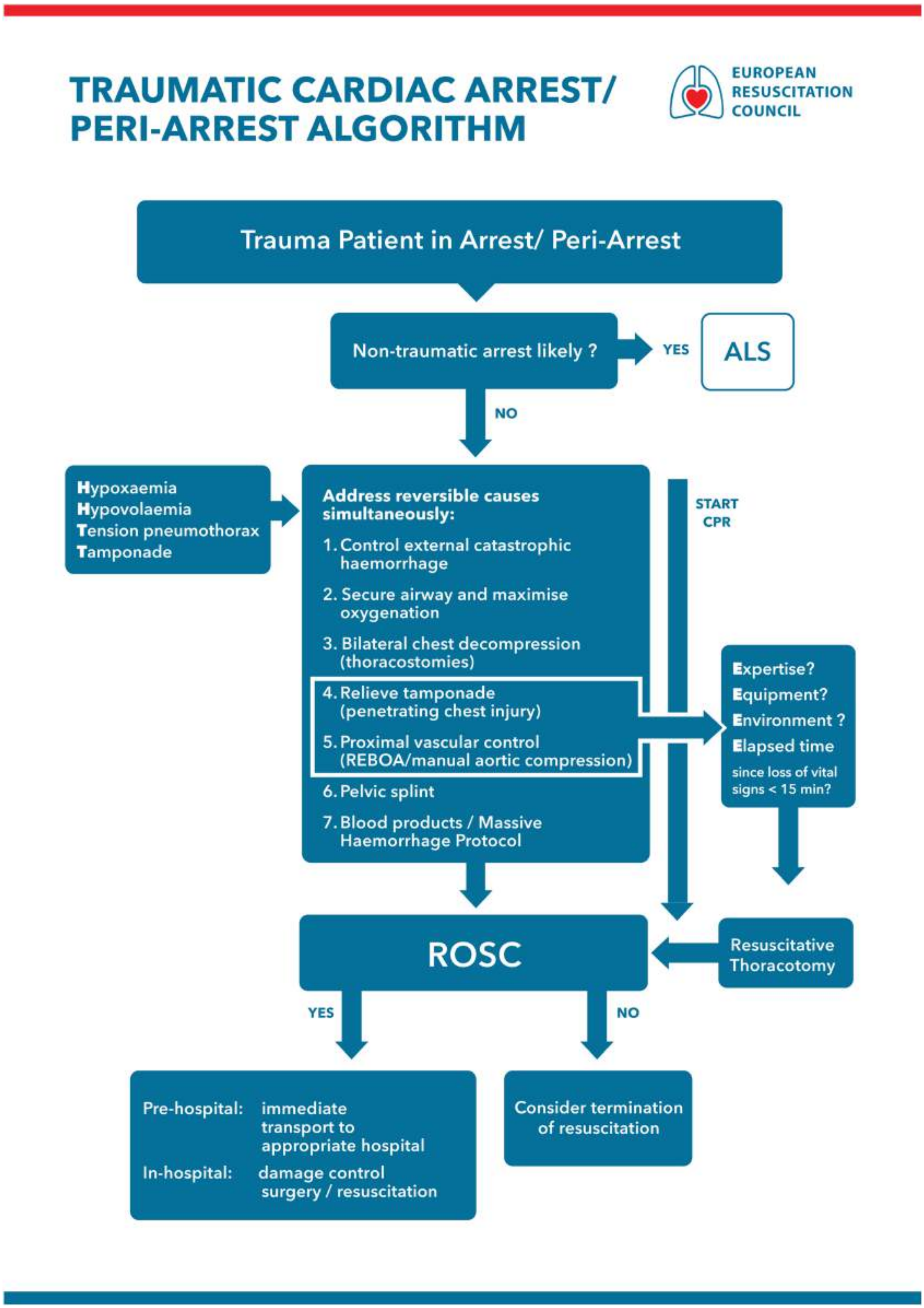


Fig. 2 – Traumatic cardiac arrest algorithm.

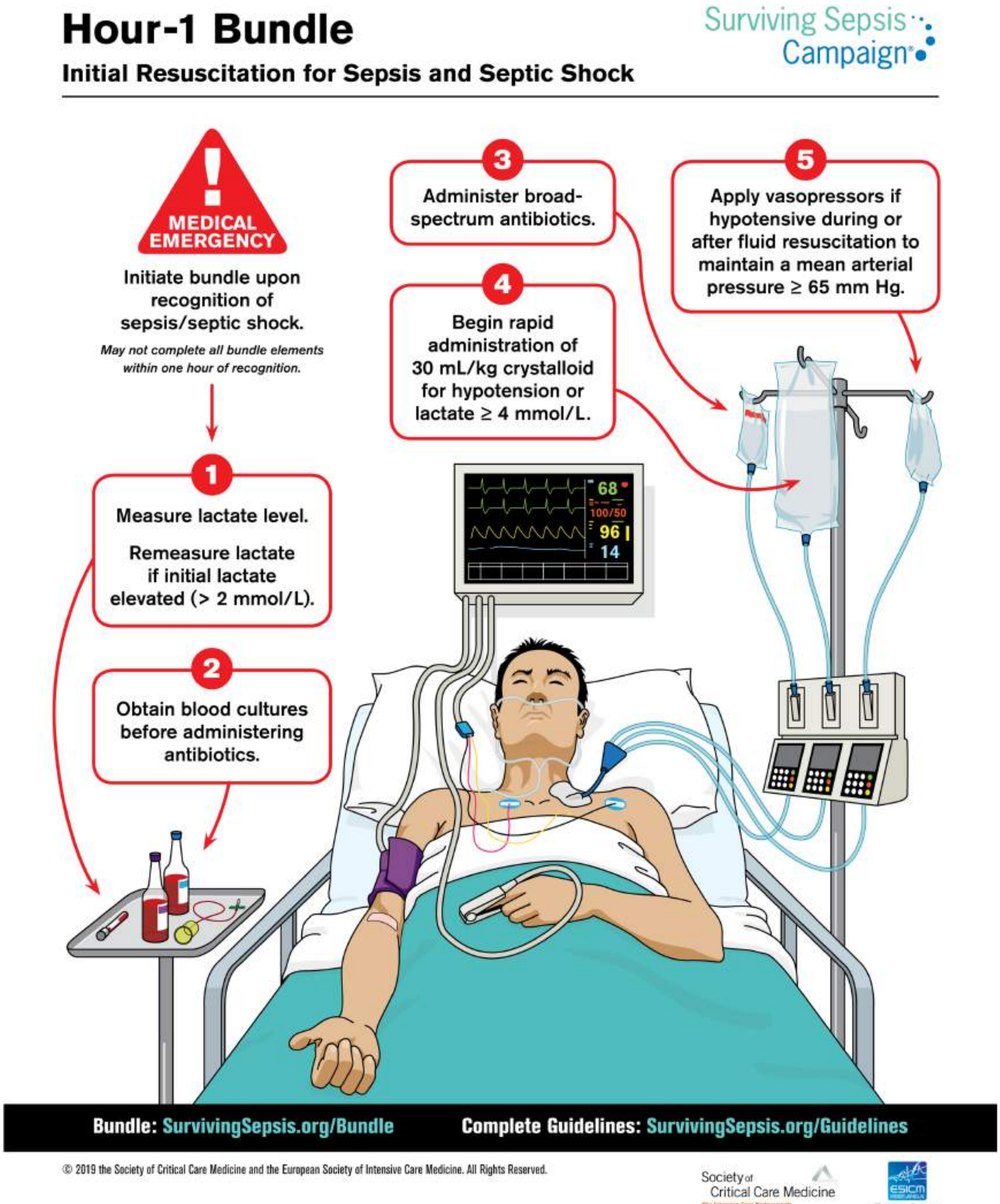


Fig. 3 – Initial Resuscitation for Sepsis and Septic Shock Hour-1 Bundle (published with permission of the Society of Critical Care Medicine, 500 Midway Drive, Mount Prospect, IL 60056-5811 USA, www.sccm.org).

- Obtain blood cultures prior to administration of antibiotics.
- Administer broad-spectrum antibiotics.
- Begin rapid administration of 30 ml kg^{-1} crystalloid for hypotension or a lactate $\geq$4 mmol l^{-1}.
- Apply vasopressors if the patient is hypotensive during or after fluid resuscitation to maintain mean arterial pressure $\geq$65 mmHg.

Cardiac arrest treatment due to sepsis

- Follow standard ALS guidelines including giving the maximal inspired oxygen concentration.
- Intubate the trachea if able to do so safely.
- Intravenous (IV) crystalloid fluid resuscitation with a 500 ml initial bolus. Consider administering further boluses.
- Venepuncture for venous blood gas/lactate/electrolytes.

- Control the source of sepsis, if feasible, and give antibiotics early.

Hypo-/hyperkalaemia and other electrolyte disorders

- Consider hyperkalaemia or hypokalaemia in all patients with an arrhythmia or cardiac arrest.
- Check for hyperkalaemia using point-of-care testing if available.
- The ECG may be the most readily available diagnostic tool.

Treatment of hyperkalaemia

- Protect the heart (Fig. 4).
- Shift potassium into cells.
- Remove potassium from the body.
  - Consider dialysis initiation during CPR for refractory hyperkalaemic cardiac arrest.
  - Consider ECPR.
- Monitor serum potassium and glucose levels.
- Prevent the recurrence of hyperkalaemia.

Patient not in cardiac arrest

Assess patient:

- Use the ABCDE approach and correct any abnormalities, obtain IV access.
- Check serum K^+ level – use blood gas analyser if available and send a sample to the laboratory.
- Perform an ECG – look for signs of hyperkalaemia.
- Cardiac monitoring – if the serum $K^+ \geq 6.5$ mmol/l or if the patient is acutely unwell.

Follow hyperkalaemia algorithm guided by the severity of hyperkalaemia and ECG changes.

Moderate hyperkalaemia (serum K^+ 6.0–6.4 mmol/l)

- Shift K^+ into cells: Give 10 units short-acting insulin and 25 g glucose (250 ml glucose 10%) IV over 15–30 min (onset in 15–30 min; maximal effect 30–60 min; duration of action 4–6 h; monitor blood glucose). Follow up with 10% glucose infusion at 50 ml/h for 5 h in patients with a pre-treatment blood glucose <7 mmol/l.
- Remove K^+ from the body: Consider oral administration of a potassium binder, e.g. Sodium Zirconium Cyclosilicate (SZC), or a cation exchange resin e.g., Patiromer or calcium resonium according to local practice.

Severe hyperkalaemia (serum $K^+ \geq 6.5$ mmol/l) without ECG changes

- Seek expert help early.
- Shift K^+ into cells: Give insulin/glucose infusion (as above).
- Shift K^+ into cells: Give salbutamol 10–20 mg nebulised (onset 15–30 min; duration of action 4–6 h).
- Remove K^+ from the body: Give SZC (onset in 60 min) or Patiromer (onset in 4–7 h) and consider dialysis.

Severe hyperkalaemia (serum $K^+ \geq 6.5$ mmol/l) with toxic ECG changes

- Seek expert help early.
- Protect the heart: Give 10 ml calcium chloride 10% IV over 2–5 min (onset 1–3 min, repeat ECG, further dose if toxic ECG changes persist).
- Shift K^+ into cells: Give insulin/glucose infusion (as above).
- Shift K^+ into cells: Give salbutamol 10–20 mg nebulised (as above).
- Remove K^+ from the body: Give SZC or Patiromer (see above) and consider dialysis at outset or if refractory to medical treatment.

Patient in cardiac arrest

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

Treatment of hypokalaemia

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

Hypothermia

Accidental hypothermia

- Assess core temperature with a low reading thermometer, tympanic in spontaneously breathing, oesophageal in patients with a tracheal tube or a supraglottic device with an oesophageal channel in place (Fig. 5).
- Check for the presence of vital signs for up to one minute.
- Prehospital insulation, triage, fast transfer to a hospital and rewarming are key interventions.
- Hypothermic patients with risk factors for imminent cardiac arrest (i.e., core temperature <30 °C, ventricular arrhythmia, systolic blood pressure <90 mmHg) and those in cardiac arrest should ideally be directly transferred to an extracorporeal life support (ECLS) centre for rewarming.
- Hypothermic cardiac arrest patients should receive continuous CPR during transfer.
- Chest compression and ventilation rate should not be different to CPR in normothermic patients.
- If ventricular fibrillation (VF) persists after three shocks, delay further attempts until the core temperature is >30 °C.
- Withhold adrenaline if the core temperature is <30 °C.
- Increase administration intervals for adrenaline to 6–10 min if the core temperature is >30 °C.
- If prolonged transport is required or the terrain is difficult, use of a mechanical CPR device is recommended.
- In hypothermic arrested patients <28 °C delayed CPR may be used when CPR on site is too dangerous or not feasible, intermittent CPR can be used when continuous CPR is not possible (Fig. 6).

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

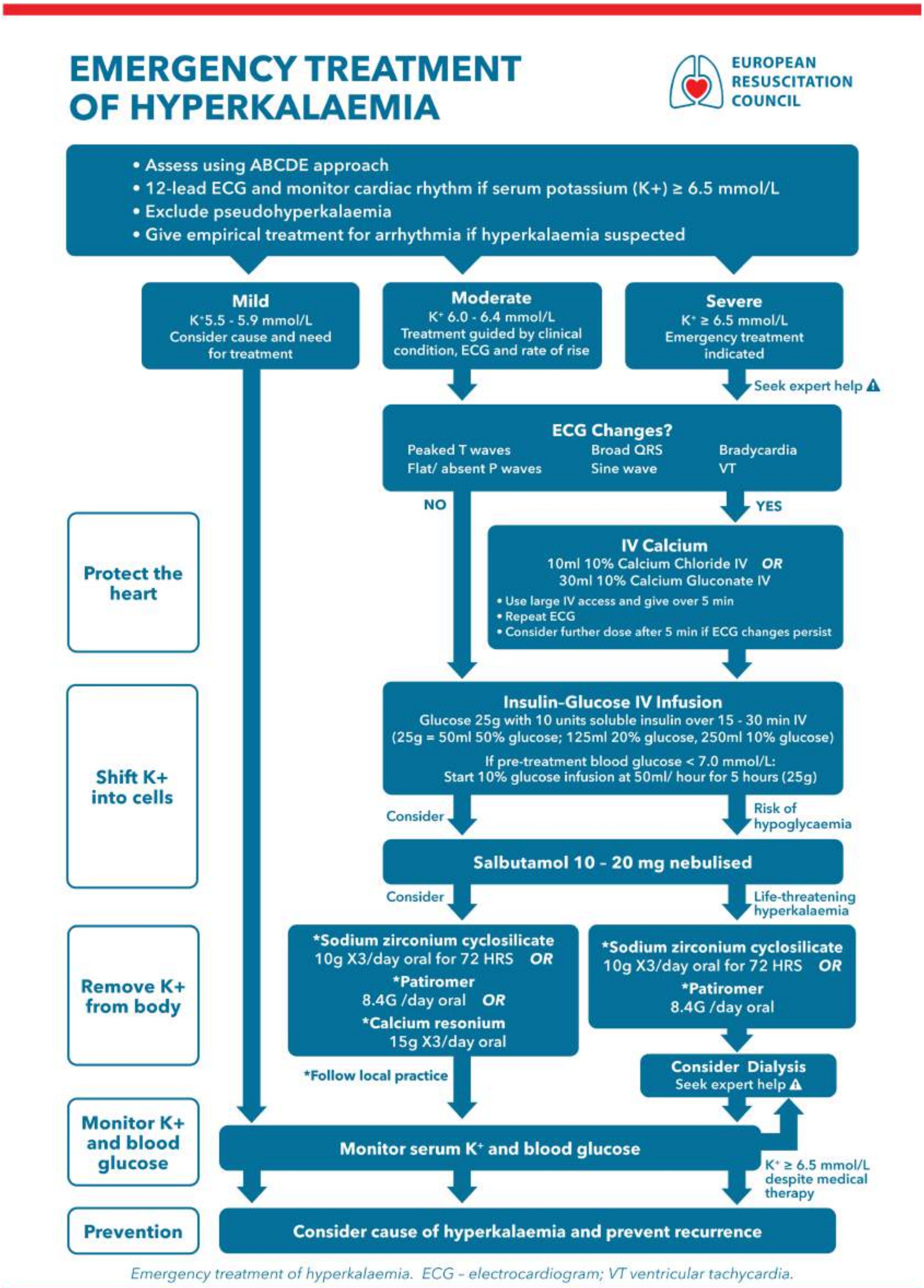


Fig. 4 – Treatment algorithm for management of hyperkalaemia in adults (adapted from the UK Renal Association Hyperkalaemia guideline 2020 https://renal.org/treatment-acute-hyperkalaemia-adults-updated-guide-line-released/).

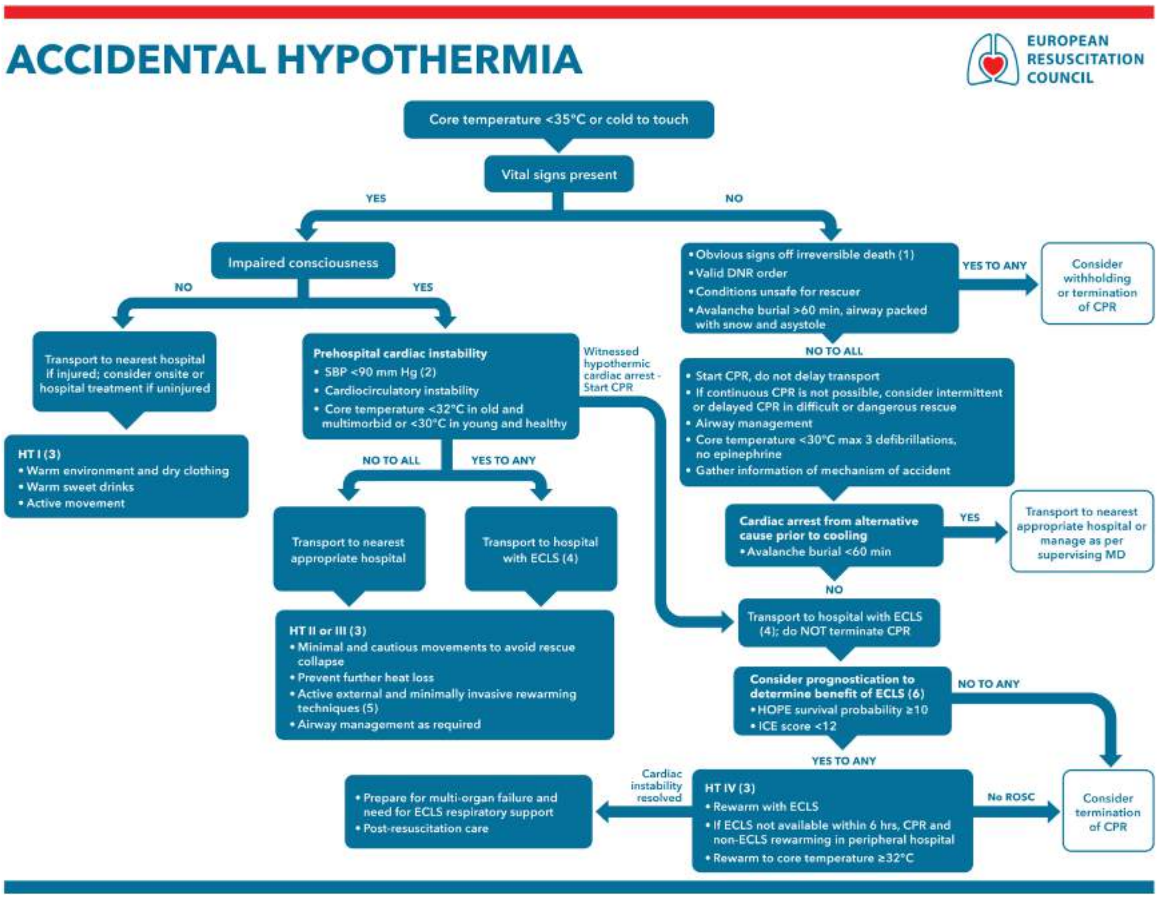


Fig. 5 – Management in accidental hypothermia.[4,5] (1) Decapitation; truncal transection; whole body decomposed or whole body frozen solid (chest wall not compressible).[6,7] (2) SBP < 90 mmHg is a reasonable prehospital estimate of cardiocirculatory instability but for in-hospital decisions, the minimum sufficient circulation for a deeply hypothermic patient (e.g., <28 °C) has not been defined. (3) Swiss staging of accidental hypothermia. (4) Direct transport to an ECMO 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 ECLS centre (e.g. 6 h). (5) 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 ECLS. (6) If the decision is made to stop at an intermediate hospital to measure serum potassium, a hospital en route to an ECLS centre should be chosen. HOPE and ICE scores should not be used in children, instead consider expert consultation.
CPR denotes cardiopulmonary resuscitation, DNR do-not- resuscitate, ECLS extracorporeal life support, HT hypothermia, MD medical doctor, ROSC return of spontaneous circulation, SBP systolic blood pressure.

- In-hospital prognostication of successful rewarming should be based on the HOPE or ICE score. The traditional in-hospital serum potassium prognostication is less reliable.
- In hypothermic cardiac arrest rewarming should be performed with ECLS, preferably with extra-corporeal membrane oxygenation (ECMO) over cardiopulmonary bypass (CPB).
- Non-ECLS rewarming should be initiated in a peripheral hospital if an ECLS centre cannot be reached within hours (e.g. 6 h).

Avalanche rescue

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

Hyperthermia and malignant hyperthermia

Hyperthermia

- Measurement of core temperature should be available to guide treatment (Fig. 8).
- Heat syncope – remove patient to a cool environment, cool passively and provide oral isotonic or hypertonic fluids.
- Heat exhaustion – remove patient to a cool environment, lie them flat, administer IV isotonic or hypertonic fluids, consider additional

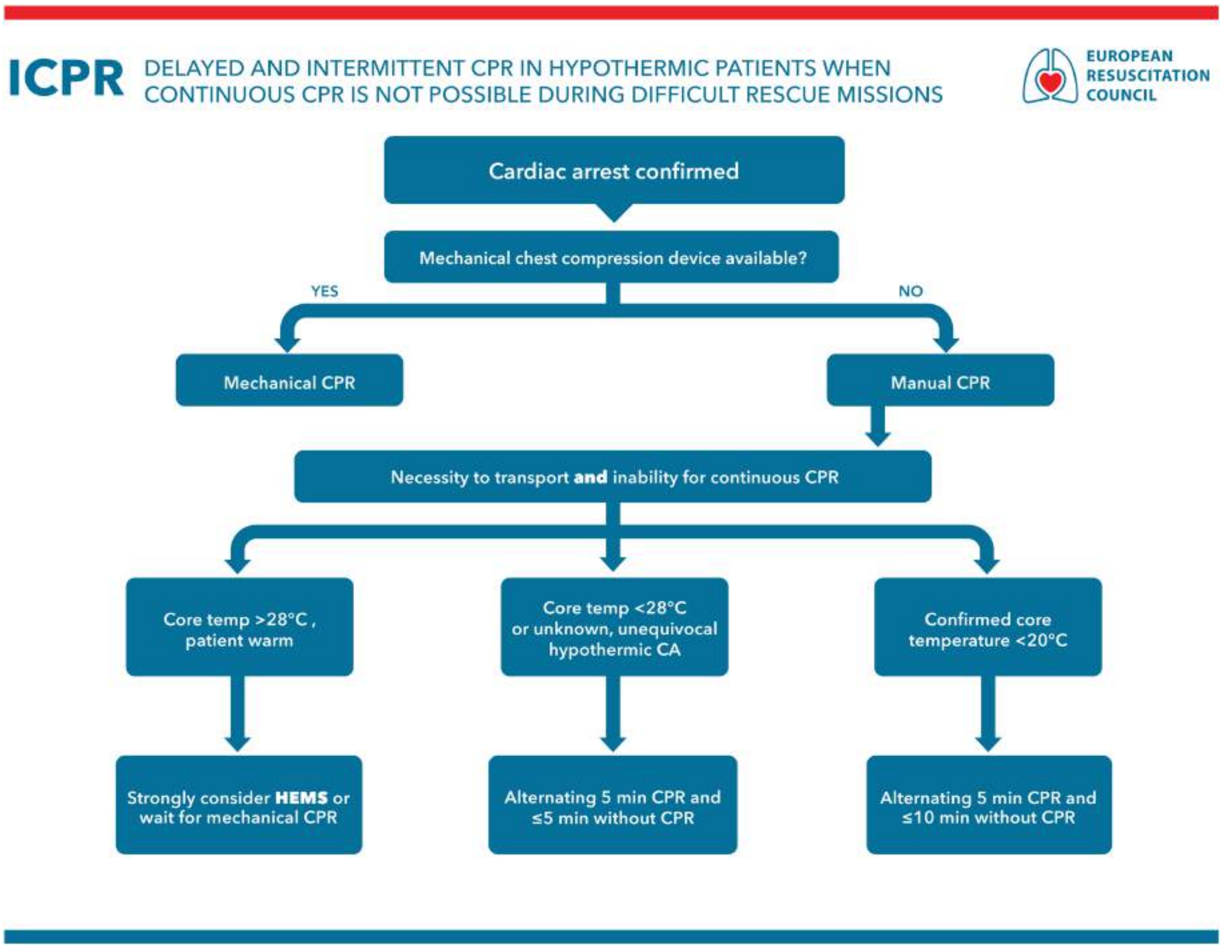


Fig. 6 – Delayed and intermittent CPR in hypothermic patients when continuous CPR is not possible during dangerous or difficult rescue.[8,190].

electrolyte replacement therapy with isotonic fluids. Replacement of 1–2 l crystalloids at 500 ml/h is often adequate.

- Simple external cooling measures are usually not required but may involve conductive, convective and evaporative measures (see section 10 First Aid).
- Heat stroke – a 'cool and run' approach is recommended:
  - Remove patient to a cool environment.
  - Lie them flat.
  - Immediately active cool using whole body (from neck down) water immersion technique (1–26 °C) until core temperature <39 °C.
  - Where water immersion is not available use immediately any active or passive technique that provides the most rapid rate of cooling.
  - Administer IV isotonic or hypertonic fluids (with blood sodium ≤130 mmol/l up to 3 × 100 ml NaCl 3%).
  - Consider additional electrolyte replacement with isotonic fluids. Substantial amounts of fluids may be required.
  - In exertional heat stroke a cooling rate faster than 0.10 °C/min is safe and desirable.
  - Follow the ABCDE approach in any patient with deteriorating vital signs.

Malignant hyperthermia

- Stop triggering agents immediately.
- Provide oxygen.
- Aim for normocapnia using hyperventilation.
- Consider correction of severe acidosis with bicarbonate (1–2 mmol kg^{-1}).
- Treat hyperkalaemia (calcium, glucose/insulin, hyperventilation) (see hyperkalaemia guideline).
- Give dantrolene (2.5 mg/kg initially, and 10 mg/kg as required).
- Start active cooling.
- Follow the ALS algorithm in cardiac arrest and continue cooling.
- After return of spontaneous circulation (ROSC) monitor the patient closely for 48–72 h, as 25% of patients experience relapse.
- Contact an expert malignant hyperthermia centre for advice and follow-up.

Thrombosis

Pulmonary embolism

Cardiac arrest prevention

- Follow the ABCDE approach.

Airway

- Treat life-threatening hypoxia with high-flow oxygen.

Breathing

- Consider pulmonary embolism (PE) in all patients with sudden onset of progressive dyspnoea and absence of known

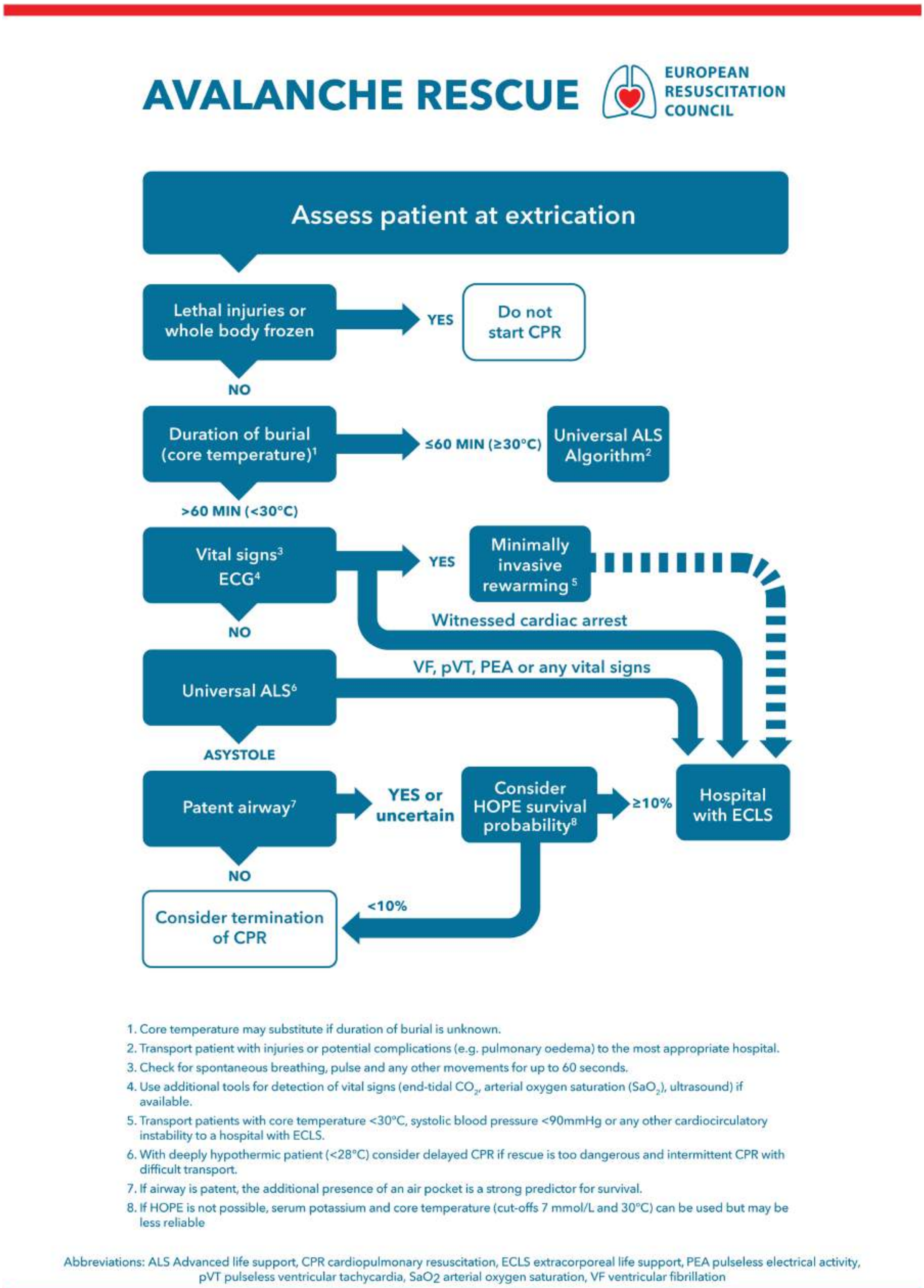


Fig. 7 – Avalanche accident algorithm. Management of completely buried victims.

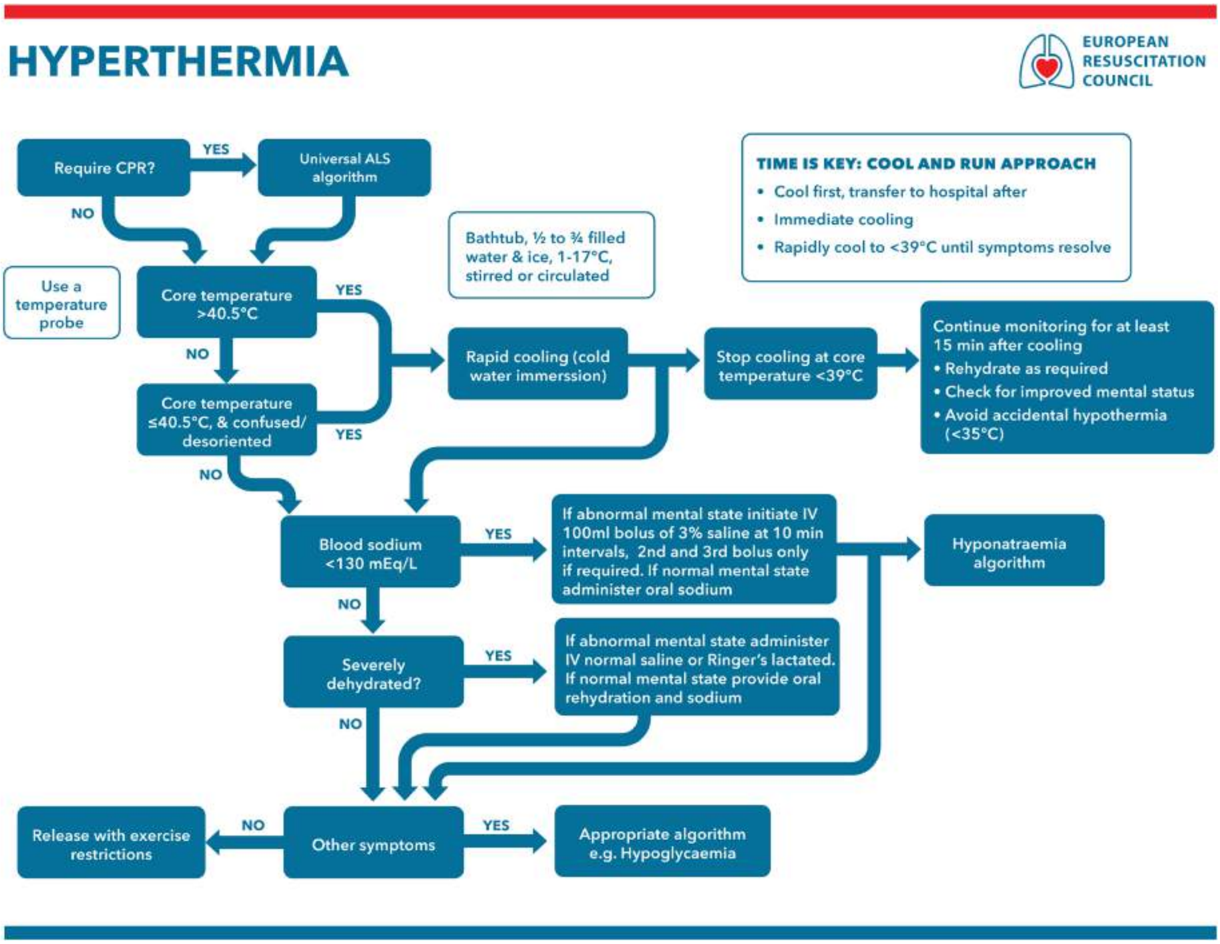


Fig. 8 – Treatment of hyperthermia (figure adapted from Racinais S, et al. www.ephysiol.com/toolbox/).

pulmonary disease (always exclude pneumothorax and anaphylaxis).

Circulation

- Obtain 12-lead ECG (exclude acute coronary syndrome, look for right ventricle strain).
- Identify haemodynamic instability and high-risk PE.
- Perform bedside echocardiography.
- Initiate anticoagulation therapy (heparin 80 IU/kg IV) during diagnostic process, unless signs of bleeding or absolute contraindications.
- Confirm diagnosis with computed tomographic pulmonary angiography (CTPA).
- Set-up a multidisciplinary team for making decisions on management of high-risk PE (depending on local resources).
- Give rescue thrombolytic therapy in rapidly deteriorating patients.
- Consider surgical embolectomy or catheter-directed treatment as alternative to rescue thrombolytic therapy in rapidly deteriorating patients.

Exposure

- Request information about past medical history, predisposing factors, and medication that may support diagnosis of pulmonary embolism:
  - Previous pulmonary embolism or deep venous thrombosis (DVT).
  - Surgery or immobilisation within the past four weeks.
  - Active cancer.
  - Clinical signs of DVT.
  - Oral contraceptive use or hormone replacement therapy.
  - Long-distance flights.

Cardiac arrest management

- Cardiac arrest commonly presents as PEA.
- Low E_TCO_2 readings (below 1.7 kPa/13 mmHg) while performing high-quality chest compressions may support a diagnosis of pulmonary embolism, although it is a non-specific sign.
- Consider emergency echocardiography performed by a qualified sonographer as an additional diagnostic tool.
- Administer thrombolytic drugs for cardiac arrest when PE is the suspected cause of cardiac arrest.
- When thrombolytic drugs have been administered, consider continuing CPR attempts for at least 60–90 min before termination of resuscitation attempts.
- Use thrombolytic drugs or surgical embolectomy or percutaneous mechanical thrombectomy for cardiac arrest when PE is the known cause of cardiac arrest.
- Consider ECPR as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it can be implemented.

Coronary thrombosis

Prevent and be prepared (Fig. 9 and Supplementary Fig. S1):

- Encourage cardiovascular prevention to reduce the risk of acute events.
- Endorse health education to reduce delay to first medical contact.
- Promote layperson basic life support to increase the chances of bystander CPR.
- Ensure adequate resources for better management.
  - Improve quality management systems and indicators for better quality monitoring.

Detect parameters suggesting coronary thrombosis and activate the ST-elevation myocardial infarction (STEMI) network (Supplementary Fig. S2):

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

Resuscitate and treat possible causes (establish reperfusion strategy):

- Patients with sustained ROSC
  - STEMI patients:
    - ➢ Primary percutaneous coronary intervention (PCI) strategy ≤120 min from diagnosis: activate catheterisation laboratory and transfer patient for immediate PCI.
    - ➢ Primary PCI not possible in ≤120 min: perform pre-hospital thrombolysis and transfer patient to PCI centre (Fig. 9).
  - Non STEMI patients: individualise decisions considering patient characteristics, OHCA setting and ECG findings.
    - ➢ Consider quick diagnostic work-up (discard non-coronary causes and check patient condition).
    - ➢ Perform urgent coronary angiography (≤120 min) if ongoing myocardial ischaemia is suspected or the patient is hemodynamically/electrically instable.
    - ➢ Consider delayed coronary angiography if there is no suspected ongoing ischaemia and the patient is stable
- Patients with no sustained ROSC: Assess setting and patient conditions and available resources
  - Futile: Stop CPR.
  - Not-futile: Consider patient transfer to a percutaneous coronary intervention (PCI) centre with on-going CPR (Fig. 9).
    - ➢ Consider mechanical compression and ECPR.
    - ➢ Consider coronary angiography.

Cardiac tamponade

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

Tension pneumothorax

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

Toxic agents

Prevention

- Poisoning rarely causes cardiac arrest.
- Manage hypertensive emergencies with benzodiazepines, vasodilators and pure alpha-antagonists.
- Drug induced hypotension usually responds to IV fluids.
- Use specific treatments where available in addition to the ALS management of arrhythmias.
- Provide early advanced airway management.
- Administer antidotes, where available, as soon as possible.

Cardiac arrest treatment

- Have a low threshold to ensure your personal safety (Fig. 10).
- Consider using specific treatment measures as antidotes, decontamination and enhanced elimination.
- Do not use mouth-to-mouth ventilation in the presence of chemicals such as cyanide, hydrogen sulphide, corrosives and organophosphates.
- Exclude all reversible causes of cardiac arrest, including electrolyte abnormalities which can be indirectly caused by a toxic agent.
- Measure the patient's temperature because hypo- or hyperthermia may occur during drug overdose.
- Be prepared to continue resuscitation for a prolonged period of time. The toxin concentration may fall as it is metabolised or excreted during extended resuscitation measures.
- Consult regional or national poison centres for information on treatment of the poisoned patient.
- Consider ECPR as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it can be implemented.

Special settings

Healthcare facilities

Cardiac arrest in the operating room (OR)

- Recognise cardiac arrest by continuous monitoring.
- Inform the surgeon and the theatre team. Call for help and the defibrillator.
- Initiate high-quality chest compressions and effective ventilation.
- Follow the ALS algorithm with a strong focus on reversible causes, especially hypovolaemia (anaphylaxis, bleeding), hypoxia, tension-pneumothorax, thrombosis (pulmonary embolism).
- Use ultrasound to guide resuscitation.
- Adjust the height of the OR table to enable high-quality CPR.
- Check the airway and review the $EtCO_2$ tracing.
- Administer oxygen with a FiO_2 1.0.
- Open cardiac compression should be considered as an effective alternative to closed chest compression.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

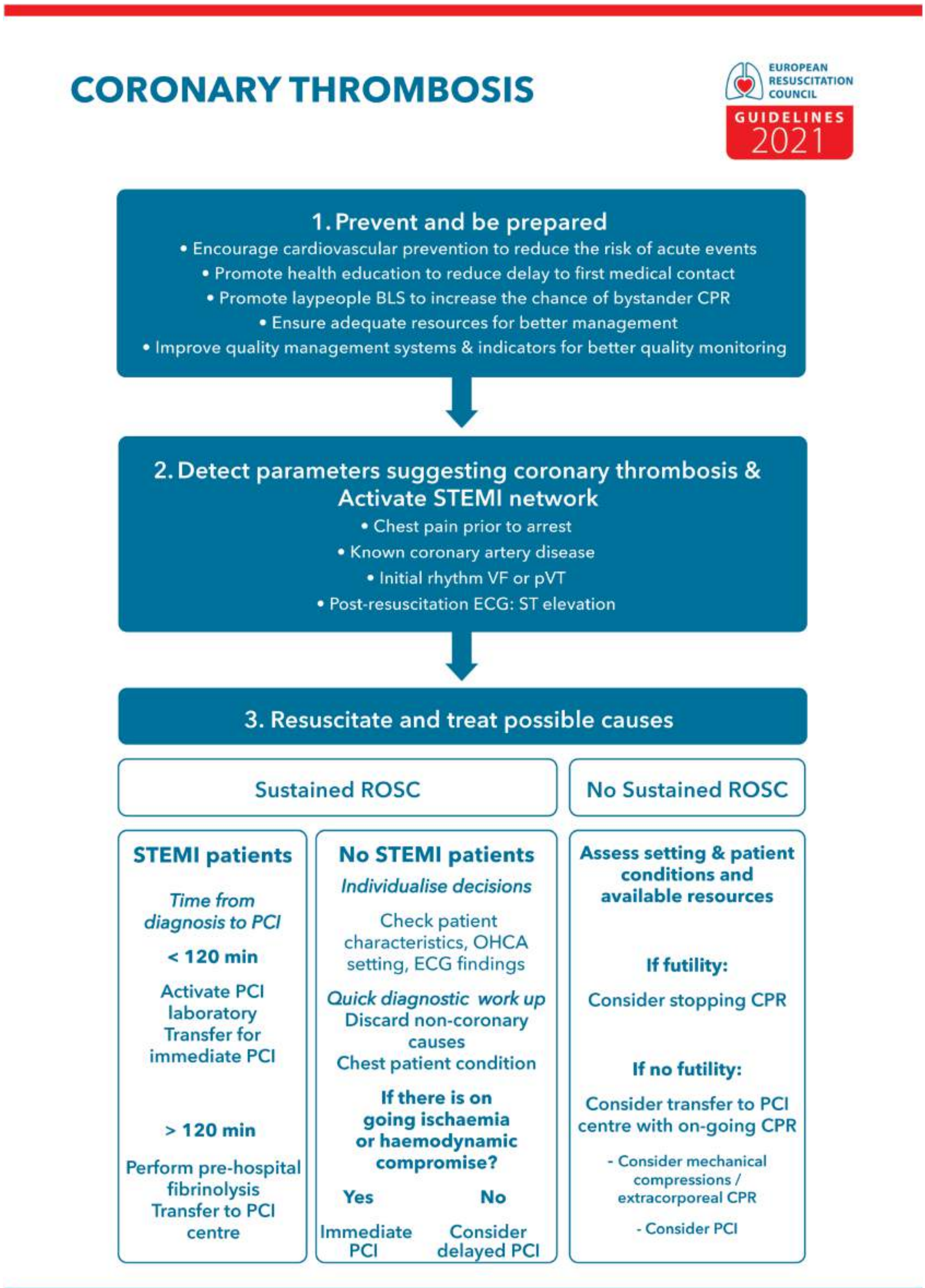


Fig. 9 – Management of out-of-hospital cardiac arrest in the setting of suspected coronary thrombosis. *Note that prolonged or traumatic resuscitation is a relative contraindication for fibrinolysis. **Individualised decision based on careful evaluation of the benefit/futility ratio, available resources and team expertise.
Abbreviations: OHCA, out-of-hospital cardiac arrest; STEMI, ST-elevation myocardial infarction; ROSC, return of spontaneous circulation; PCI, percutaneous coronary intervention; CPR: cardiopulmonary resuscitation.

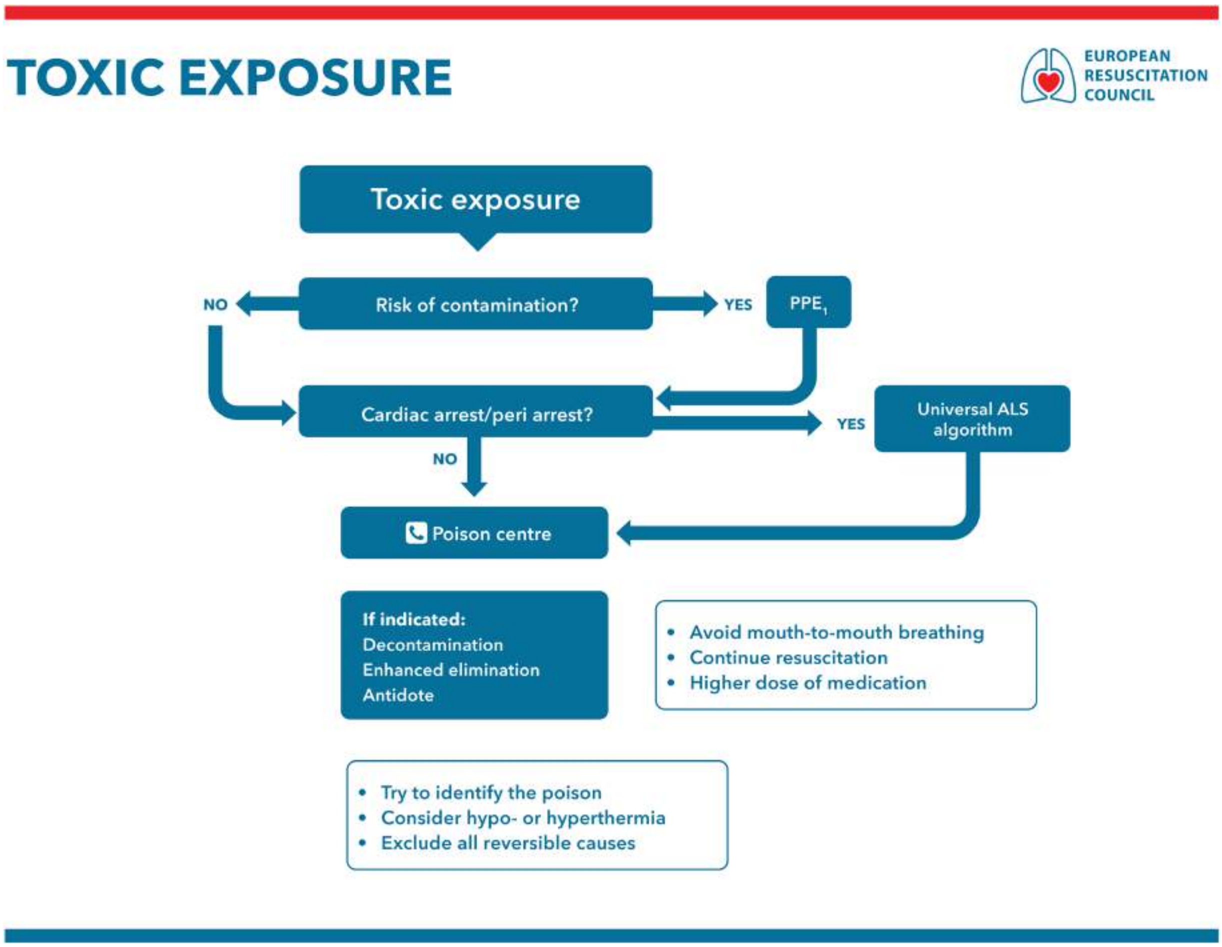


Fig. 10 – Toxic exposure algorithm. PPE—personal protective equipment.

- Consider ECPR as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing.

Cardiac surgery

Prevent and be prepared

- Ensure adequate training of the staff in resuscitation technical skills and ALS (Fig. 11).
- Ensure equipment for emergency re-sternotomy is available in the ICU.
- Use safety checklists.

Detect cardiac arrest and activate cardiac arrest protocol:

- Identify and manage deterioration in the postoperative cardiac patient.
- Consider echocardiography.
- Confirm cardiac arrest by clinical signs and pulseless pressure waveforms.
- Shout for help and activate cardiac arrest protocol.

Resuscitate and treat possible causes:

- Resuscitate according to ALS MODIFIED algorithm:
  - ➢ VF/pVT → Defibrillate: apply up to 3 consecutive shocks (<1 min).
  - ➢ Asystole/extreme bradycardia → Apply early pacing (<1 min).
  - ➢ PEA → Correct potentially reversible causes. If paced rhythm, turn off pacing to exclude VF.

→ No ROSC:
- o Initiate chest compression and ventilation.
- o Perform early resternotomy (< 5 min).
- o Consider circulatory support devices and ECPR (Fig. 11).

Catheterisation laboratory

Prevent and be prepared (Fig. 12):

- Ensure adequate training of the staff in resuscitation technical skills and ALS.
- Use safety checklists.

Detect cardiac arrest and activate cardiac arrest protocol:

- Check patient's status and monitored vital signs periodically.
- Consider cardiac echocardiography in case of haemodynamic instability or suspected complication.
- Shout for help and activate cardiac arrest protocol.

Resuscitate and treat possible causes:

- Resuscitate according to the MODIFIED ALS algorithm:
  - ◦ VF/pVT cardiac arrest → Defibrillate (apply up to 3 consecutive shocks) → no ROSC → resuscitate according to ALS algorithm.
  - ◦ Asystole/ PEA → resuscitate according to ALS algorithm.
- Check and correct potentially reversible causes, including the use of echocardiography and angiography.
- Consider mechanical chest compression and circulatory support devices (including ECPR).

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

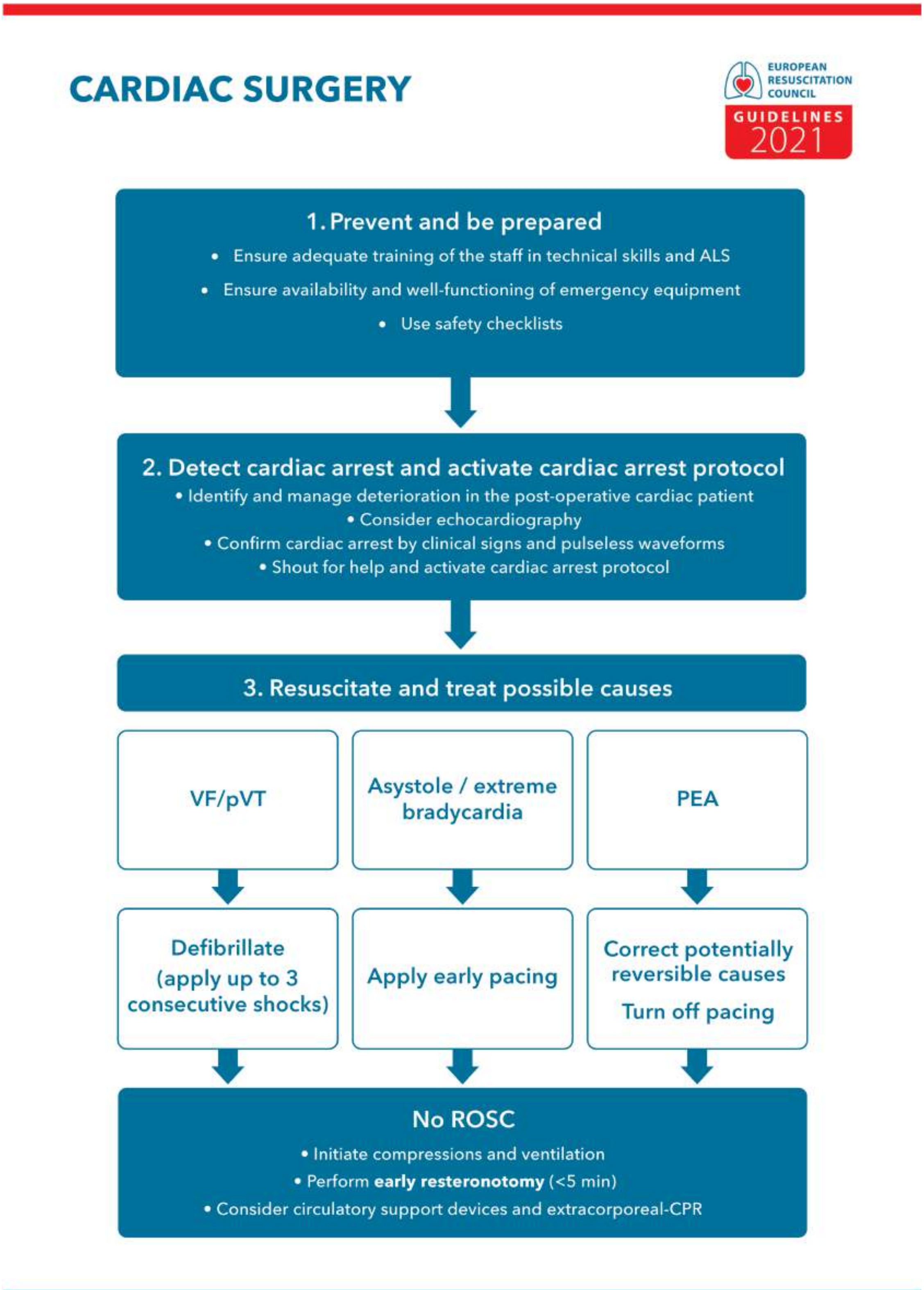


Fig. 11 – Advanced life support (ALS) algorithm for postoperative cardiac arrest following cardiac surgery. ALS, advanced life support, VF, ventricular fibrillation; PVT: pulseless ventricular tachycardia; CPR, cardiopulmonary resuscitation; ROSC, return of spontaneous circulation; PEA: pulseless electrical activity. ** Consider IABP to support CPR or extracorporeal life support as an alternative if resternotomy is not feasible or fails to revert cardiac arrest.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

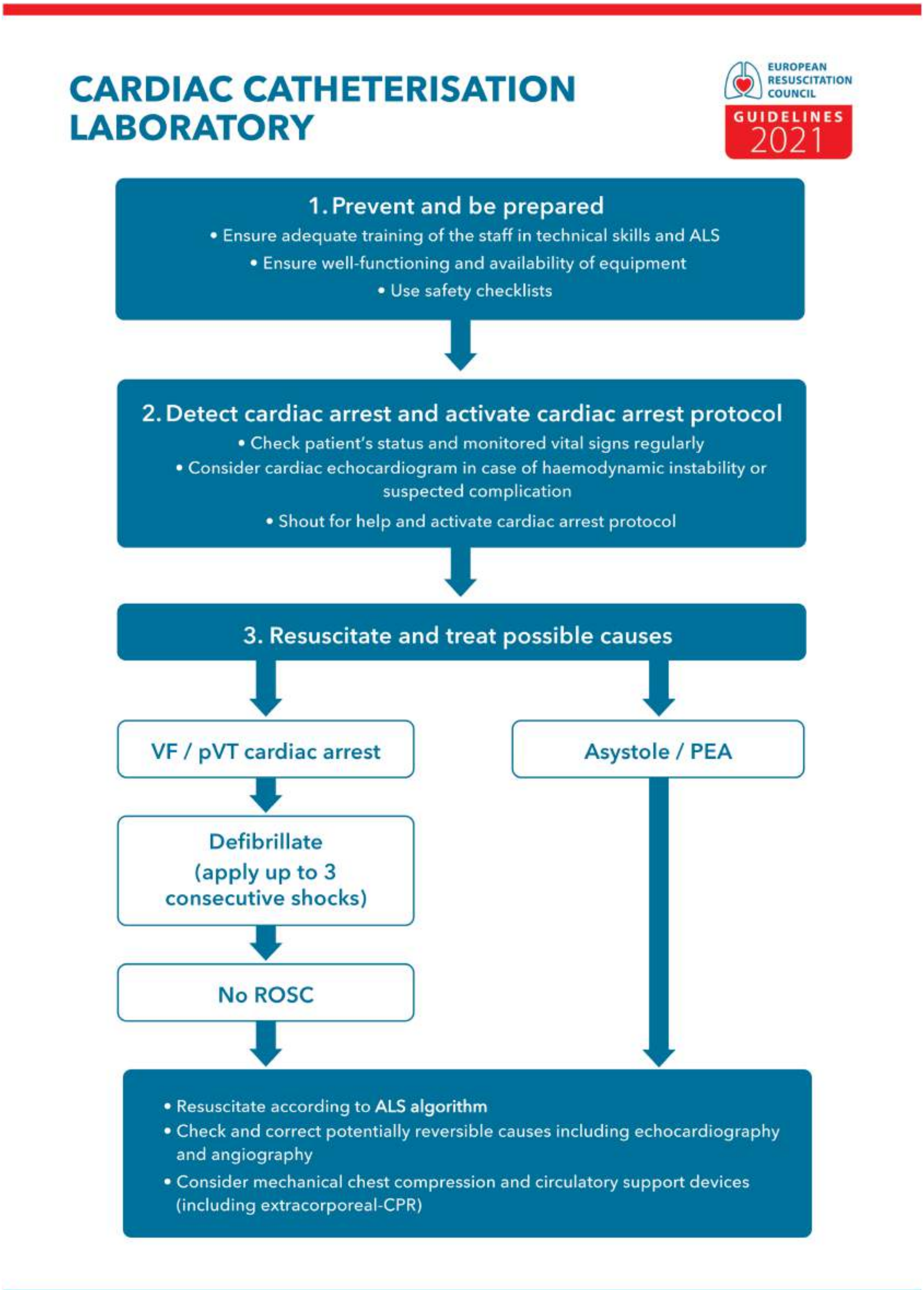


Fig. 12 – Management of cardiac arrest in the catheterisation laboratory. ALS, advanced life support, VF, ventricular fibrillation; PVT: pulseless ventricular tachycardia; CPR, cardiopulmonary resuscitation; ROSC, return of spontaneous circulation; PEA: pulseless electrical activity.

Dialysis unit

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

Dentistry

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

Transportation

Inflight cardiac arrest

- Medical professional help should be sought (in-flight announcement).
- The rescuer should kneel in the leg-space in front of the aisle seats to perform chest compressions if the patient cannot be transferred within a few seconds to an area with adequate floor space (galley).
- 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.
- Consider risks of diversion if ROSC is unlikely and give appropriate recommendations to the flight crew.
- If CPR is terminated (no ROSC) a flight diversion should not usually be performed.

Helicopter emergency medical services (HEMS) and air ambulances

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

Cruise ship

- Use all medical resources immediately (personal, equipment).
- Activate HEMS if close to the coastline.
- Consider early telemedicine support.
- Have all equipment needed for ALS available on board.
- In case of insufficient number of health care professionals to treat CA, call for further medical staff via an on-board announcement.

Cardiac arrest in sport

Planning

- All sports and exercise facilities should undertake a medical risk assessment of the risk of sudden cardiac arrest.
- Where there is a raised risk, mitigation must include resuscitation planning to include:
  - Staff and members training in the recognition and management of cardiac arrest.
  - Direct provision of an AED or clear directions to the nearest public access AED.

Implementation

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

Prevention

- Do not undertake exercise, especially extreme exercise or competitive sport, if feeling unwell.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

- Follow medical advice in relation to the levels of exercise or sport competition.
- Consider cardiac screening for young athletes undertaking high level competitive sport.

Drowning

Initial rescue

- Undertake a dynamic risk assessment considering feasibility, chances of survival and risks to the rescuer:
  - Submersion duration is the strongest predictor of outcome.
  - Salinity has an inconsistent effect on outcome.

- Assess consciousness and breathing:
  - If conscious and/or breathing normally, aim to prevent cardiac arrest.
  - If unconscious and not breathing normally, start resuscitation.

Cardiac arrest prevention

Airway

- Ensure a patent airway.
- Treat life threatening hypoxia with 100% inspired oxygen until the arterial oxygen saturation or the partial pressure of arterial oxygen can be measured reliably.
- Once SpO_2 can be measured reliably or arterial blood gas values are obtained, titrate the inspired oxygen to achieve an arterial oxygen saturation of 94–98% or arterial partial pressure of oxygen (PaO_2) of 10 –13 kPa (75–100 mmHg).

Breathing

- Assess respiratory rate, accessory muscle use, ability to speak in full sentences, pulse oximetry, percussion and breath sounds; request chest X-ray.
- Consider non-invasive ventilation if respiratory distress and safe to do so.
- Consider invasive mechanical ventilation if respiratory distress and unsafe or unable to initiate non-invasive ventilation.
- Consider extracorporeal membrane oxygenation if poor response to invasive ventilation.

Circulation

- Assess heart rate and blood pressure, attach ECG.
- Obtain IV access.
- Consider IV fluids and/or vasoactive drugs to support the circulation.

Disability

- Assess using AVPU or GCS.

Exposure

- Measure core temperature.
- Initiate hypothermia algorithm if core temperature <35 °C.

Cardiac arrest

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

Mass casualty incidents

- Identify hazards and immediately request assistance if necessary.
- Use adequate personal protection equipment (PPE) (e.g. bulletproof vest, respirator, long-sleeved gown, eye and face protection) depending on specific risks on scene.
- Reduce secondary risks to other patients and providers.
- Use a locally established triage system to prioritise treatment.
- Perform life-saving interventions in patients triaged as “immediate” (highest priority) to prevent cardiac arrest.
- Consider assigning a higher triage risk level to elderly and to survivors of high-energy trauma in order to reduce preventable deaths.
- Healthcare professionals must be regularly trained to use the triage protocols during simulations and live exercises.

Special patients

Asthma and COPD

Cardiac arrest prevention

Airway

- Ensure a patent airway.
- Treat life threatening hypoxia with high flow oxygen (Fig. 13).
- Titrate subsequent oxygen therapy with pulse oximetry (SpO_2 94–98% for asthma; 88–92% for chronic obstructive pulmonary disease (COPD)).

Breathing

- Assess respiratory rate, accessory muscle use, ability to speak in full sentences, pulse oximetry, percussion and breath sounds; request chest X-ray.
- Look for evidence of pneumothorax/tension pneumothorax.
- Provide nebulised bronchodilators (oxygen driven for asthma, consider air driven for COPD).
- Administer steroids (Prednisolone 40–50 mg or hydrocortisone 100 mg).
- Consider IV magnesium sulphate for asthma.
- Seek senior advice before giving IV aminophylline or salbutamol.

Circulation

- Assess heart rate and blood pressure, attach ECG.
- Obtain vascular access.
- Consider IV fluids.

Cardiac arrest treatment

- Administer high concentration oxygen.
- Ventilate with respiratory rate (8–10 min^{-1}) and sufficient tidal volume to cause the chest to rise.

Management of acute asthma in adults in hospital

Features of acute severe asthma

- Peak expiratory flow (PEF) 33–50% of best (use % predicted if recent best unknown)
- Can't complete sentences in one breath
- Respiration ≥25 breaths/min
- Pulse ≥110 beats/min

Life-threatening features

- PEF <33% of best or predicted
- SpO_2 <92%
- Silent chest, cyanosis, or poor respiratory effort
- Arrhythmia or hypotension
- Exhaustion, altered consciousness

If a patient has any life-threatening feature, measure arterial blood gases. No other investigations are needed for immediate management.

Blood gas markers of a life-threatening attack:

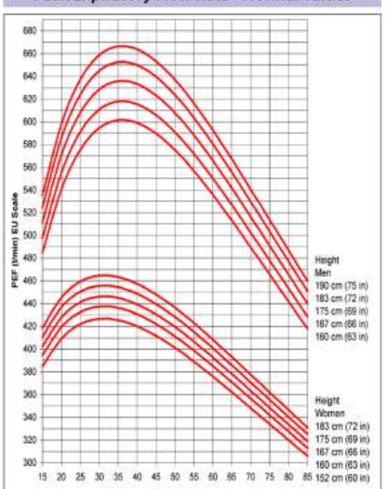
- 'Normal' (4.6–6 kPa, 35–45 mmHg) $PaCO_2$
- Severe hypoxia: PaO_2 <8 kPa (60 mmHg) irrespective of treatment with oxygen
- A low pH (or high H^+)

Caution: Patients with severe or life-threatening attacks may not be distressed and may not have all these abnormalities. The presence of any should alert the doctor.

Near-fatal asthma

- Raised $PaCO_2$
- Requiring mechanical ventilation with raised inflation pressures

Peak Expiratory Flow Rate - Normal Values



Adapted by Clement Clarke for use with EN13826 / EU scale peak flow meters from Nunn AJ Gregg I, Br Med J 1989:298;1068-70

IMMEDIATE TREATMENT

- Oxygen to maintain SpO_2 94–98%
- β_2 bronchodilator (salbutamol 5 mg) via an oxygen-driven nebuliser
- Ipratropium bromide 0.5 mg via an oxygen-driven nebuliser
- Prednisolone tablets 40–50 mg or IV hydrocortisone 100 mg
- No sedatives of any kind
- Chest X-ray if pneumothorax or consolidation are suspected or patient requires mechanical ventilation

IF LIFE-THREATENING FEATURES ARE PRESENT:

- Discuss with senior clinician and ICU team
- Consider IV magnesium sulphate 1.2–2 g infusion over 20 minutes (unless already given)
- Give nebulised β_2 bronchodilator more frequently eg salbutamol 5 mg up to every 15-30 minutes or 10 mg per hour via continuous nebulisation (requires special nebuliser)

SUBSEQUENT MANAGEMENT

IF PATIENT IS IMPROVING continue:

- Oxygen to maintain SpO_2 94–98%
- Prednisolone 40–50mg daily or IV hydrocortisone 100 mg 6 hourly
- Nebulised β_2 bronchodilator with ipratropium 4–6 hourly

IF PATIENT NOT IMPROVING AFTER 15–30 MINUTES:

- Continue oxygen and steroids
- Use continuous nebulisation of salbutamol at 5–10 mg/hour if an appropriate nebuliser is available. Otherwise give nebulised salbutamol 5 mg every 15–30 minutes
- Continue ipratropium 0.5 mg 4–6 hourly until patient is improving

IF PATIENT IS STILL NOT IMPROVING:

- Discuss patient with senior clinician and ICU team
- Consider IV magnesium sulphate 1.2–2 g over 20 minutes (unless already given)
- Senior clinician may consider use of IV β_2 bronchodilator or IV aminophylline or progression to mechanical ventilation

MONITORING

- Repeat measurement of PEF 15–30 minutes after starting treatment
- Oximetry: maintain SpO_2 >94–98%
- Repeat blood gas measurements within 1 hour of starting treatment if:
  - initial PaO_2 <8 kPa (60 mmHg) unless subsequent SpO_2 >92% or
  - $PaCO_2$ normal or raised or
  - patient deteriorates
- Chart PEF before and after giving β_2 bronchodilator and at least 4 times daily throughout hospital stay

Transfer to ICU accompanied by a doctor prepared to intubate if:

- Deteriorating PEF, worsening or persisting hypoxia, or hypercapnia
- Exhaustion, altered consciousness
- Poor respiratory effort or respiratory arrest

DISCHARGE

When discharged from hospital, patients should have:

- Been on discharge medication for 12–24 hours and have had inhaler technique checked and recorded
- PEF >75% of best or predicted and PEF diurnal variability <25% unless discharge is agreed with respiratory physician
- Treatment with **oral steroids (prednisolone 40–50 mg until recovery - minimum 5 days) and inhaled steroids** in addition to bronchodilators
- Own PEF meter and **written asthma action plan**
- GP follow up arranged within **2 working days**
- Follow-up appointment in respiratory clinic **within 4 weeks**

Patients with severe asthma (indicated by need for admission) **and adverse behavioural or psychosocial features are at risk of further severe or fatal attacks.**

- Determine reason(s) for exacerbation and admission
- Send details of admission, discharge and potential best PEF to GP

Fig. 13 – Management of acute asthma in adults in hospital. 2019. (SIGN publication no. 158). Reproduced with permission from the Scottish Intercollegiate Guidelines Network (SIGN). Edinburgh: SIGN; available from URL: http://www.sign.ac.uk.

- Intubate the trachea if able to do so safely.
- Check for signs of tension pneumothorax and treat accordingly.
- Disconnect from positive pressure ventilation if relevant and apply pressure to manually reduce hyper-inflation.
- Consider IV fluids.
- Consider E-CPR in accordance with local protocols if initial resuscitation efforts are unsuccessful.

Neurological disease

- There are no modifications required in the BLS and ALS management of cardiac arrest from a primary neurological cause.
- Following ROSC, consider clinical features such as young age, female sex, non-shockable rhythm and neurological antecedents such as headache, seizures, and focal neurological deficit when suspecting a neurological cause of cardiac arrest.
- Early identification of a neurological cause can be achieved by performing a brain CT-scan at hospital admission, before or after coronary angiography.
- In the absence of signs or symptoms suggesting a neurological cause (e.g. headache, seizures or neurological deficits) or if there is clinical or ECG evidence of myocardial ischaemia, coronary angiography is undertaken first, followed by CT scan in the absence of causative lesions.

Obesity

- Delivery of effective CPR in obese patients may be challenging due to a number of factors:
  - patient access and transportation
  - vascular access
  - airway management
  - quality of chest compressions
  - efficacy of vasoactive drugs
  - efficacy of defibrillation

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

Pregnancy

Prevention of cardiac arrest in the deteriorating pregnant patient

- Use a validated obstetric early warning scoring system when caring for the ill-pregnant patient.
- Use a systematic ABCDE approach to assess and treat the pregnant patient.
- Place the patient in the left lateral position or manually and gently displace the uterus to the left to relieve aortocaval compression.
- Give oxygen guided by pulse oximetry to correct hypoxaemia.
- Give a fluid bolus if there is hypotension or evidence of hypovolaemia.
- Immediately re-evaluate the need for any drugs being given.
- Seek expert help early – obstetric, anaesthetic, critical care and neonatal specialists should be involved early in the resuscitation.
- Identify and treat the underlying cause of cardiac arrest, e.g. control of bleeding, sepsis.
- Give intravenous tranexamic acid 1 g IV for postpartum haemorrhage.

Modification for advanced life support in the pregnant patient

- Call for expert help early (including an obstetrician and neonatologist).
- Start basic life support according to standard guidelines.
- Use the standard hand position for chest compressions on the lower half of the sternum if feasible.
- If over 20 weeks pregnant or the uterus is palpable above the level of the umbilicus:
  - Manually displace the uterus to the left to remove aortocaval compression.
  - If feasible, add left lateral tilt – the chest should remain on supported on a firm surface (e.g. in the operating room). The optimal angle of tilt is unknown. Aim for a tilt between 15 and 30 degrees. Even a small amount of tilt may be better than no tilt. The angle of tilt used needs to enable high-quality chest compressions and if needed allow caesarean delivery of the fetus.
- Prepare early for emergency hysterostomy early – the fetus will need to be delivered if immediate (within 4 min) resuscitation efforts fail.
- If over 20 weeks pregnant or the uterus is palpable above the level of the umbilicus and immediate (within 4 min) resuscitation is unsuccessful, deliver the fetus by emergency caesarean section aiming for delivery within 5 min of collapse.
- Place defibrillator pads in the standard position as far as possible and use standard shock energies.
- Consider early tracheal intubation by a skilled operator.
- Identify and treat reversible causes (e.g. haemorrhage). Focused ultrasound by a skilled operator may help identify and treat reversible causes of cardiac arrest.
- Consider extracorporeal CPR (ECPR) as a rescue therapy if ALS measures are failing.

Preparation for cardiac arrest in pregnancy

Healthcare settings dealing with cardiac arrest in pregnancy should:

- have plans and equipment in place for resuscitation of both the pregnant woman and the newborn.
- ensure early involvement of obstetric, anaesthetic, critical care and neonatal teams.
- ensure regular training in obstetric emergencies.

Evidence informing the guidelines

Special causes

Hypoxia

Cardiac arrest caused by pure hypoxaemia is uncommon. It is seen more commonly as a consequence of asphyxia, which accounts for most of the non-cardiac causes of cardiac arrest. There are many causes of asphyxial cardiac arrest (Table 1); although there is usually a combination of hypoxaemia and hypercarbia, it is the hypoxaemia that ultimately causes cardiac arrest.[9] In an epidemiological study of

44,000 OHCAs in Osaka, Japan, asphyxia accounted for 6% of cardiac arrests with a resuscitation attempt, hanging 4.6% and drowning 2.4%.[10]

Evidence for the treatment of asphyxial cardiac arrest is based mainly on observational studies. There are very few data comparing different therapies for the treatment of asphyxial cardiac arrest although there are data comparing standard CPR with compression-only CPR. The Guidelines for clinical practice are based largely on expert opinion.

Pathophysiological mechanisms

If breathing is completely prevented by airway obstruction or apnoea, consciousness will be lost when oxygen saturation in the arterial blood reaches about 60%. The time taken to reach this concentration is difficult to predict, but based on mathematical modelling is likely to be of the order 1−2 min.[11] Based on animal experiments of cardiac arrest caused by asphyxia, pulseless electrical activity (PEA) will occur in 3−11 min. Asystole will ensue several minutes later.[12] In comparison with simple apnoea, the exaggerated respiratory movements that frequently accompany airway obstruction will increase oxygen consumption resulting in more rapid arterial blood oxygen desaturation and a shorter time to cardiac arrest. Complete airway obstruction after breathing air will result in PEA cardiac arrest in 5−10 min.[9] An initial monitored rhythm of VF occurs rarely after asphyxial cardiac arrest − in two of the largest series of hanging-associated out-of-hospital cardiac arrests (OHCAs), one from Melbourne, Australia, and the other from Osaka, Japan, just 20 (0.6%) of 3320 patients were in VF.[10,13]

Compression-only versus conventional CPR

ILCOR and the ERC suggest, that bystanders who are trained, able, and willing to give rescue breaths and chest compressions do so for all adult patients in cardiac arrest (weak recommendation, very-low-certainty evidence).[14,15] Observational studies suggest conventional CPR even more where there is a non-cardiac cause of cardiac arrest.[16,17]

Outcome

Survival after cardiac arrest from asphyxia is rare and most survivors sustain severe neurological injury. The Osaka study documented respectively one-month survival and neurologically favourable outcome after cardiac arrest following: asphyxia 14.3% and 2.7%; hanging 4.2% and 0.9%; and drowning 1.1% and 0.4%.[10]

Table 1 – Causes of asphyxial cardiac arrest.

Trauma
Hanging
Chronic obstruction pulmonary disease
Asthma
Airway obstruction, soft tissues (coma), laryngospasm, aspiration
Drowning
Central hypoventilation − brain or spinal cord injury
Impaired alveolar ventilation from neuromuscular disease
Traumatic asphyxia or compression asphyxia (e.g. crowd crush), tension pneumothorax
Pneumonia
High altitude
Avalanche burial
Anaemia

Of eight published series that included a total of 4189 patients with cardiac arrest following hanging where CPR was attempted, the overall survival rate was 4.3%; there were just 45 (1.1%) survivors with a favourable neurological outcome (CPC 1 or 2); 135 other survivors were documented to be CPC 3 or 4.[10,13,18−23] When resuscitating these patients, rescuers are frequently able to achieve ROSC but subsequent neurologically intact survival is rare. Those who are unconscious but have not progressed to a cardiac arrest are much more likely to make a good neurological recovery.[19,23,24]

Hypovolaemia

Hypovolaemia is a potentially treatable cause of cardiac arrest that usually results from a reduced intravascular volume (i.e. haemorrhage), but relative hypovolaemia may also occur in patients with severe vasodilation (e.g. anaphylaxis, sepsis, spinal cord injury). Hypovolaemia from mediator-activated vasodilation and increased capillary permeability is a major factor causing cardiac arrest in severe anaphylaxis.[25] Hypovolaemia from blood loss, is a leading cause of death in traumatic cardiac arrest.[26] External blood loss is usually obvious, e.g. trauma, haematemesis, haemoptysis, but may be more challenging to diagnose when occult, e.g. gastrointestinal bleeding or rupture of an aortic aneurysm. Patients undergoing major surgery are at high-risk from hypovolaemia due to post-operative haemorrhage and must be appropriately monitored (see perioperative cardiac arrest). Depending on the suspected cause, initiate volume therapy with warmed blood products and/or crystalloids, in order to rapidly restore intravascular volume. At the same time, initiate immediate intervention to control haemorrhage, e.g. surgery, endoscopy, endovascular techniques,[27] or treat the primary cause (e.g. anaphylactic shock). In the initial stages of resuscitation use any crystalloid solution that is immediately available, if haemorrhage is likely aim for early blood transfusion and vasopressor support. If there is a qualified sonographer able to perform ultrasound with minimum interruption to chest compressions, it may be considered as an additional diagnostic tool in hypovolaemic cardiac arrest. Treatment recommendations for cardiac arrest and peri arrest situations in trauma, anaphylaxis and sepsis are addressed in separate sections because of the need for specific therapeutic approaches.

Traumatic cardiac arrest (TCA)

Traumatic cardiac arrest (TCA) carries a very high mortality, but in those where ROSC can be achieved, neurological outcome in survivors appears to be much better than in other causes of cardiac arrest.[28,29] The response to TCA is time-critical and success depends on a well-established chain of survival, including advanced prehospital and specialised trauma centre care. Immediate resuscitative efforts in TCA focus on simultaneous treatment of reversible causes, which takes priority over chest compressions.

This section is based on an evidence update on TCA produced by recent systematic reviews and focused on scoping reviews addressing the questions:[28,30−32]

- Chest compressions in hypovolemic cardiac arrest/peri-arrest? (1291 titles screened/120 abstracts screened/8 publications selected).
- Chest compressions versus open cardiac massage (808 titles screened/43 abstracts screened/29 publications selected).
- Needle thoracocentesis versus resuscitative thoracotomy in pericardial tamponade (572 titles screened/29 abstracts screened/7 publications selected).

- Needle decompression in traumatic tension pneumothorax? (214 titles screened/7abstracts screened/5 publications selected).
- REBOA versus aortic occlusion of the descending aorta in TCA or peri-arrest (1056 titles screened/156 abstracts screened/11 publications selected).

Epidemiology and pathophysiology

Traumatic cardiac arrest (TCA) carries a high mortality. Registry data for survival range from 1.6% to 32%.[33–37] The considerable variation in reported survival mainly reflects heterogeneity in entry criteria but also in case mix and care in different systems.

In survivors, the neurological outcome appears to be much better than in other causes of cardiac arrest.[26,29,35,37] The reversible causes of TCA are uncontrolled haemorrhage (48%); tension pneumothorax (13%); asphyxia (13%); pericardial tamponade (10%).[26] The prevalent initial heart rhythms found in TCA are either PEA or asystole, depending on the time interval between circulatory arrest and the first electrocardiogram (ECG) recording PEA (66%); asystole (30%); VF (6%).[26]

Diagnosis

Patients with TCA will usually present with loss of consciousness, agonal or absent spontaneous breathing and absence of a central pulse. A peri-arrest state is characterised by cardiovascular instability, hypotension, loss of peripheral pulses and a deteriorating conscious level, without obvious underlying central nervous system problems. If untreated this state is likely to progress to cardiac arrest. The use of ultrasound may help to verify the cause of the TCA and direct the resuscitative efforts accordingly.[38]

Prognostic factors and withholding resuscitation

There are no reliable predictors of survival for TCA. Factors that are associated with survival include the presence of reactive pupils, respiratory activity, spontaneous movements and an organised ECG rhythm.[39,40] Short duration of CPR, short prehospital times,[41] penetrating chest injury,[42] witnessed arrest and the presence of a shockable rhythm are also associated with positive outcomes.[43,44] Children presenting with TCA have a better outcome than adults.[28,29]

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.[45] However, neurologically intact survivors initially presenting in this state have been reported.[29] We therefore recommend the following approach:

Consider withholding resuscitation in TCA in any of the following conditions:

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

We suggest termination of resuscitative efforts if there is:

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

Initial management steps

Pre-hospital care

The key decision to be made in the prehospital environment is establish whether the cardiac arrest is caused by trauma or by an underlying medical problem. If TCA cannot be confirmed, standard ALS guidelines apply. Short pre-hospital times are associated with increased survival rates for major trauma and traumatic cardiac arrest.[38]

Hospital care

Successful treatment of TCA requires a team approach with all measures carried out in parallel rather than sequentially. The emphasis lies on rapid treatment of all potentially reversible causes. Fig. 2 shows the traumatic cardiac (peri-)arrest algorithm of the European Resuscitation Council (ERC), which is based on the universal ALS algorithm.

Effectiveness of chest compressions

In cardiac arrest caused by hypovolaemia, cardiac tamponade or tension pneumothorax, chest compressions are unlikely to be as effective as in normovolaemic cardiac arrest and may reduce residual spontaneous cardiac output.[46–48] Therefore, chest compressions take a lower priority than addressing the reversible causes. Chest compressions must not delay immediate treatment of reversible causes. A retrospective cohort study analysing data from the Trauma Quality Improvement Program (TQIP) database, a nationwide trauma registry in the USA, between 2010 and 2016 compared open cardiac compressions to close chest compressions in IHCA patients admitted with signs of life. Results in this specific patient group showed a favourable outcome for the patients receiving open cardiac compressions versus closed chest compressions.[49]

Hypovolaemia

The treatment of severe hypovolaemic shock has several elements. The main principle is to achieve immediate haemostasis. Temporary haemorrhage control can be lifesaving.[38] In hypovolemic TCA, immediate restoration of the circulating blood volume with blood products is mandatory. Prehospital transfusion of fresh plasma and packed red cells yields a significant survival benefit if the journey time to the receiving hospital exceeds 20 min.[50,51]

Compressible external haemorrhage can be treated with direct or indirect pressure, pressure dressings, tourniquets and topical haemostatic agents.[52] 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 to surgical haemorrhage control.

- Immediate aortic occlusion is recommended as a last resort measure in patients with exsanguinating and uncontrollable infra-diaphragmatic torso haemorrhage. This can be achieved through Resuscitative Thoracotomy (RT) and cross-clamping the descending aorta or Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA). There is no evidence for one technique being superior over the other.[30]
- Neurogenic shock as a sequel of spinal cord injury (SCI) can aggravate hypovolemia due to blood loss in trauma patients. Even moderate blood loss can cause cardiac arrest in the presence of SCI due to the limited compensatory capacity. Indicators for SCI in severely injured patients are warm peripheries and loss of reflexes below the injured segment, severe hypotension and a low heart rate. The cornerstones of treatment are fluid replacement and IV vasopressors.[53]

Hypoxia

In TCA, hypoxaemia can be caused by airway obstruction, traumatic asphyxia or impact brain apnoea.[54] Impact brain apnoea is an underestimated cause of morbidity and mortality in trauma but not necessarily associated with an un-survivable brain injury.[55] Impact brain apnoea may aggravate the course of traumatic brain injury and can lead to asphyxiation if left untreated. Effective airway management and ventilation can prevent and reverse hypoxic cardiac arrest.

However, controlled ventilation in circulatory compromised patients is associated with major risks related to the side effect of anaesthetics and increase in intrathoracic pressure[56] which may lead to:[57] further decrease in residual cardiac output by impeding venous return to the heart, particularly in severely hypovolaemic patients.

- reduced diastolic filling in cardiac tamponade
- conversion of pneumothorax into a tension pneumothorax
- increase in blood loss from venous bleeding sites

Low tidal volumes may help optimise cardiac preload. Ventilation should be monitored with capnography and adjusted to achieve normocapnia.[38,58]

Tension pneumothorax

To decompress the chest in TCA, perform bilateral thoracostomies in the 4th intercostal space (ICS), allowing extension to a clamshell thoracotomy if required. Alternatively, a needle thoracocentesis can be attempted (see corresponding guideline section). In the presence of positive pressure ventilation, thoracostomies are likely to be more effective than needle thoracocentesis and quicker than inserting a chest tube.[59–62]

Cardiac tamponade

Cardiac tamponade is a frequent cause of cardiac arrest in penetrating chest trauma and immediate resuscitative thoracotomy (RT) via a clamshell or left anterolateral incision, is indicated to restore circulation.[63,64] The chance of survival is about 4 times higher in cardiac stab wounds than in gunshot wounds.[65]

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

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

If any of the four criteria is not met, RT is futile and exposes the team to unnecessary risks. RT is also a viable therapeutic option in the prehospital environment.[31,32,66,67]

Subsequent management and treatment

The principle of 'damage control resuscitation' has been adopted in trauma resuscitation for uncontrolled haemorrhage. Damage control resuscitation combines permissive hypotension and haemostatic resuscitation with damage control surgery. Limited evidence and general consensus have supported a conservative approach to IV fluid infusion, with permissive hypotension until surgical haemostasis is achieved.[68] Permissive hypotension allows IV fluid administration to a volume sufficient to maintain a radial pulse. Caution is advised patients with traumatic brain injury were a raised intracranial pressure may require a higher cerebral perfusion pressure. The duration of hypotensive resuscitation should not exceed 60 min, because the risks of irreversible organ damage then exceed its intended benefits.[69] Haemostatic resuscitation is the very early use of blood products as primary resuscitation fluid to prevent exsanguination and trauma-induced coagulopathy.[70–72] Tranexamic acid (TXA) (loading dose 1 g IV over 10 min followed by infusion of 1 g over 8 h) increases survival from traumatic haemorrhage. It is most effective when administered within the first hour and certainly within the first three hours following trauma.[73] TXA should not be started any later than four hours after the injury as it then may increase mortality.

Diagnostics

Sonography should be used in the evaluation of the compromised trauma patient to target lifesaving interventions if the cause of shock cannot be diagnosed clinically. Haemoperitoneum, haemo- or pneumothorax and cardiac tamponade can be diagnosed within minutes.[38,74]

Anaphylaxis

This guideline is specific for the initial treatment of adult patients with anaphylaxis or suspected anaphylaxis by clinicians. A precise definition of anaphylaxis is not important for its emergency treatment. Anaphylaxis is a serious systemic allergic reaction that is rapid in onset and may cause death.[75] The incidence of anaphylaxis is increasing globally, whereas the case fatality rate has remained stable or decreased, with an overall population risk of death of about 0.5–1 per million.[76,77] Foods (especially in children), drugs and insect bites are the commonest triggers.[76]

This anaphylaxis guidance is based on the most recent First Aid ILCOR CoSTR,[52] guidelines and updates from the World Allergy Organisation Anaphylaxis Committee,[78] European Academy of Allergy and Clinical Immunology (EAACI),[25] North American Practice Parameter,[79] Australasian Society of Clinical Immunology and Allergy (ASCIA) (https://www.allergy.org.au/hp//papers/acute-management-of-anaphylaxis-guidelines, accessed 10 August 2020), recent guidance on perioperative allergic reactions,[80] the findings from the UK National Audit Project of perioperative anaphylaxis,[81] and our understanding of the pathophysiology of anaphylaxis.[82] We completed a focused literature search up to July 2020 to identify any new relevant studies. The evidence supporting specific interventions for the treatment of anaphylaxis is limited with few RCTs. The majority of recommendations are based on observational data, good practice statements and expert consensus.[79,83]

Recognition of anaphylaxis

Anaphylaxis causes life threatening airway (swollen lips, tongue, uvula), breathing (dyspnoea, wheeze, bronchospasm, stridor, reduced peak flow, hypoxaemia) and circulation problems (hypotension, cardiac arrest) with or without skin or mucosal changes (generalised urticaria, flushing or itching) as part of an allergic reaction.[25,52,75,84] Skin and mucosal changes are not always present or obvious to the rescuer and severe bronchospasm, hypotension, or rarely sudden cardiac arrest can be the first features.[77,81] Knowledge of the patient's allergy history and triggers can help make the diagnosis, but this will not always be known.

Remove or stop the trigger if possible

Based on expert consensus, stop any drug suspected of causing anaphylaxis. Remove the stinger after a bee sting – early removal is more important than the method of removal.[85,86] Do not delay definitive treatment if removing the trigger is not feasible.

Give intramuscular adrenaline early and repeat after 5 min if necessary

Adrenaline is the most important drug for the treatment of anaphylaxis and is the first line treatment according to all current guidelines for anaphylaxis based on both its alpha- (vasoconstrictor) and beta-(bronchodilator, inotropic, mast cell stabilisation) agonist properties. Intramuscular (IM) adrenaline works within minutes and adverse effects are extremely rare with the correct doses. The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The available evidence for adrenaline and the recommended doses is weak and based on observational data and expert consensus.[78,83,87] The EAACI suggests Intramuscular adrenaline (1 mg/ml) should be given at a dose of 0.01 ml kg^{-1} of body weight to a maximum total dose of 0.5 ml.[25] These ERC guidelines recommend a dose of 0.5 mg IM in adults based on expert opinion. Adrenaline auto-injectors are also available – auto-injector devices are manufacturer specific for preparation, mechanism of injection and dose delivery (0.3 mg and 0.15 mg are the commonest doses). These can be used as an alternative to a syringe, needle and ampoule – follow the manufacturer instructions on how to use them. This ERC guideline does not address the choice, prescription, dosing, and instructions for self-use of adrenaline auto-injectors by those at risk of anaphylaxis.

Based on the available evidence[52,88] time to effect, the variability in response to the first dose of adrenaline, the observed need for a second dose reported to be in about 10–30% of cases,[89,90] variable EMS response times, and exiting international guideline recommendations, we suggest repeating the IM adrenaline dose if there is no improvement in the patient's condition after about 5-min.

Ensure the patient is lying and do not suddenly sit or stand the patient up

Observational data from a detailed review of 214 individual cases of death from anaphylaxis referred to Coroner's in the UK observed cardiovascular collapse occurred when some individuals with out-of-hospital anaphylaxis who had clinical signs of a low blood pressure sat- or stood-up or were sat- or stood-up by rescuers.[91] Based on this limited evidence, expert consensus and existing guidelines we suggest:

- Patients with Airway and Breathing problems may prefer to sit up as this will make breathing easier.
- Lying flat with or without leg elevation is helpful for patients with a low blood pressure.
- Patients who are breathing and unconscious should be placed on their side (recovery position).
- Pregnant patients should lie on their left side to prevent aortocaval compression.

Give intravenous fluids

Anaphylaxis can cause hypotension due to vasodilation, redistribution of blood between vascular compartments, and fluid extravasation and correcting for fluid losses in addition to adrenaline is based on experience of managing shock in other settings such as sepsis.[82,92] In line with these guidelines we suggest the use of either balanced crystalloids or 0.9% sodium chloride bolus doses and further doses based on haemodynamic response. The first resuscitation fluid bolus should be about 500 ml over 5–10 min.[93] Expert opinion suggests patients with refractory anaphylaxis may need large volumes of fluid resuscitation.[80,81] Emerging observational evidence suggests that anaphylaxis can impair stroke volume and that improves with early use of fluids.[94]

Give oxygen

Oxygen therapy to correct hypoxaemia is a standard part of resuscitation. As for other emergency conditions, high flow oxygen should be given early and once an oxygen saturation can be measured reliably with a pulse oximeter the inspired oxygen should be titrated to target an oxygen saturation of 94–98%.[95]

Intravenous (IV) adrenaline in specialist settings

Intravenous adrenaline should be used only by those experienced in the use and titration of vasopressors in their normal clinical practice (based on expert opinion and exiting guidelines) Patients who are given IV adrenaline must be monitored – continuous ECG and pulse oximetry and frequent non-invasive blood pressure measurements as a minimum. Titrate IV adrenaline using a 20–50 μg bolus according to response.[80,84] If repeated adrenaline doses are needed, start an IV adrenaline infusion.[80,81,84]

Other drugs to support the circulation

Several guidelines based on expert opinion recommend glucagon 1–2 mg IV is considered for anaphylaxis refractory to adrenaline in patients who are taking beta-blockers.[80,84] In addition based on expert opinion when anaphylaxis is refractory other vasopressors can be considered as a bolus dose or infusion including vasopressin, noradrenaline, metaraminol, phenylephrine.[80,81,84] (Australian guideline website - https://www.allergy.org.au/images/ASCIA_HP_Guidelines_Acute_Management_Anaphylaxis_2020.pdf – accessed 10 August 2020).

Role of steroids and antihistamines in the immediate management of anaphylaxis

There is no evidence that supports the routine use of either steroids or antihistamines in the initial resuscitation of a patient with anaphylaxis.[79,83,96–98] They do not appear to alter the progress of anaphylaxis or prevent biphasic reactions.[77,99,100] Steroids should be considered if there are ongoing asthma-like symptoms or in the setting of refractory shock in accordance with guidelines for asthma and shock states.

Considerations for cardiac arrest in anaphylaxis

There are no specific studies of advanced life support for anaphylaxis. Based on expert opinion follow standard ALS guidelines for cardiac arrest care including use of IV adrenaline and correction of potentially reversible causes (fluids, oxygen) (see ALS Guidelines).[101] Areas of controversy is the effectiveness of chest compressions in patients with vasodilatory cardiac arrest and when should chest compressions start in closely monitored patients.[80,102] In a case series of peri-operative cardiac arrest caused by anaphylaxis 31 of 40 patients (77.5%) survived with ALS interventions, and 67% of survivors required an adrenaline or vasopressor infusion after ROSC.[81] The cardiac arrest rhythm was PEA in 34 (85%), VF in 4 (10%) and asystole in 2 (5%).

Role of extracorporeal life support and extracorporeal CPR in anaphylaxis

The ILCOR ALS Task Force suggests that ECPR may be considered as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it can be implemented (weak recommendation, very low-certainty evidence).[2,103] Expert opinion, case reports and clinical experience supports the use of emergency ECLS or ECPR in the peri-arrest or refractory cardiac arrest setting in select patients.

Follow up and further investigations

The ongoing care of patients with anaphylaxis should follow existing guidelines from the World Allergy Organisation Anaphylaxis Committee,[78] European Academy of Allergy and Clinical Immunology (EAACI),[25] North American Practice Parameter[79] Australasian Society of Clinical Immunology and Allergy (ASCIA). (https://www.allergy.org.au/hp//papers/acute-management-of-anaphylaxis-guidelines, accessed 10 August 2020) Mast cell tryptase measurement can help diagnose anaphylaxis. The consensus on optimal timing for measurement is that ideally three timed samples should be taken:[104]

- First sample as soon as feasible after resuscitation has started – do not delay resuscitation to take sample.
- Second sample at 1–2 h after the start of symptoms
- Third sample either after 24 h or in convalescence. This provides baseline tryptase levels – some individuals have an elevated baseline level.

Sepsis

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Septic shock is a subset of sepsis with circulatory and cellular/metabolic dysfunction associated with a higher risk of mortality.[92]

This section is written on the care of the adult patient based on the Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock (2016) and National Institute of Clinical Excellence (2016).[92,105] Please refer to paediatric and neonatal guidelines on sepsis. An update to guidelines was published by Surviving Sepsis Campaign in 2018 which combined initial resuscitation into Hour 1 Bundle.[106] This was revised in 2019, the starting time was defined as time when sepsis is recognised (Fig. 3).

Cardiac arrest prevention in sepsis

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Septic shock is a subset of sepsis with circulatory and cellular/metabolic dysfunction associated with a higher risk of mortality.[92]

Key steps in the initial treatment and management of severe sepsis to prevent cardiac arrest in adults are summarized in Fig. 3. The ERC recommends assessment using the ABCDE approach while controlling the underlying source of infection is critical in the prevention of shock, multi-organ failure and cardiorespiratory arrest. Once immediate life-threatening problems have been addressed, initial resuscitation in patients should begin immediately. High-flow oxygen should be given to optimize oxygen delivery to tissues. Blood cultures should be obtained followed by the administration of broad-spectrum antibiotics. Lactate levels should be measured. Hypotension or a lactate measurement of $\geq$4 mmol/l should be treated with the rapid administration of 30 ml kg^{-1} crystalloid. Hourly urine output should be measured to help guide IV fluid therapy. Vasopressors should be started if the patient remains hypotensive during or after fluid resuscitation to maintain a mean arterial pressure $\geq$65 mmHg.

Cardiac arrest treatment due to sepsis

Cardiac arrest in a person with severe sepsis can be a result of hypoxia and cardiovascular collapse. Treat cardiac arrest in a patient with sepsis or suspected sepsis according to standard ALS guidelines.

Hypoxia

The correction of hypoxia in cardiac arrest due to sepsis may require high flow oxygenation, intubation and mechanical ventilation. Correcting hypoxia and hypotension will optimise oxygen delivery to tissues and vital organs.

Hypovolaemia

Sepsis-induced tissue hypoperfusion or septic shock requires prompt and effective fluid resuscitation. Initial fluid resuscitation should begin immediately following the recognition of a patient with sepsis and/or hypotension and elevated lactate, and completed within 3 h of recognition. A minimum of 30 ml kg^{-1} of IV crystalloid fluid is recommended based on data from observational studies.[107,108] Based on the lack of evidence to support the use of colloid compared with crystalloid solutions, the guidelines make a strong recommendation for the use of crystalloid solutions in the initial resuscitation of patients with sepsis and septic shock.

The goal of resuscitation is to restore adequate perfusion pressure to the vital organs. If patient remains hypotensive after initial fluid resuscitation, then vasopressors should be started within the first hour to achieve a mean arterial pressure (MAP) of $\geq$65 mmHg.[109,110]

Post resuscitation care

Sources of infection should be identified and treated accordingly. Serum lactate is a surrogate for tissue perfusion and can be used to guide resuscitation.[111,112] Careful clinical assessment is required beyond the initial resuscitation stages to assess fluid responsiveness and avoid potentially harmful sustained positive fluid balance.[113–115]

Early initiation of treatment is required to prevent organ dysfunction and cardiac arrest. Follow standard ALS guidelines for cardiac arrest in a patient with sepsis or suspected sepsis. Correct hypoxia and treat hypovolaemia and look for other potentially reversible causes using the 4Hs and 4Ts approach. In post resuscitation care, avoid sustained positive fluid balance. Serum lactate may be useful in guiding therapy.

Hypo-/hyperkalaemia and other electrolyte disorders

Electrolyte abnormalities are recognised causes of arrhythmias and cardiac arrest. Potassium disorders, hyperkalaemia and hypokalaemia are the most common electrolyte disturbances associated with life-threatening arrhythmias, whilst calcium and magnesium disorders occur less commonly. The primary focus in this section is the recognition, treatment and prevention of hyperkalaemia.

This section is based on the UK Renal Association Hyperkalaemia guideline 2020 which used the GRADE approach for quality of evidence.[116] There remains sparse evidence for the drug treatments for hyperkalaemia (i.e. intravenous calcium and insulin-glucose) in cardiac arrest. A review did not identify any other relevant, high quality guidelines on the management of hyperkalaemic cardiac arrest or the initiation of dialysis during CPR. A scoping review was performed using keywords 'hyperkalaemia', 'treatment', 'ECG', and 'Cardiac arrest' in PubMed (1960–2020), Ovid Medline (1946–2020), EMBASE (1974–2020) and The Cochrane Library (1995–2020). Websites searches included National Institute for Health and Care Excellence (NICE), Scottish Medicines Consortium (SMC), Healthcare Improvement Scotland, Medicines and Healthcare products Regulatory Agency (MHRA) and European Medicines Agency (EMA).

Hyperkalaemia

Hyperkalaemia occurs in 1–10% of hospitalised patients, most often in patients with pre-existing kidney disease or in the context of an acute kidney injury.[117–119] People receiving long-term haemodialysis (HD) are most at risk of hyperkalaemia. They are also susceptible to cardiac disease and together with hyperkalaemia, may account for the high risk of sudden cardiac death in patients on maintenance dialysis. The reported incidence varies from 1% to 13% of in-hospital cardiac arrests (IHCA).[120–122]

Definition

There is no universal definition. We have defined hyperkalaemia as a serum potassium (K^+) concentration greater than 5.5 mmol/l, although in clinical practice, hyperkalaemia is a continuum. The severity of hyperkalaemia guides response to treatment. Hyperkalaemia may be categorised as 'mild' (K^+ 5.5–5.9 mmol/l), 'moderate' (K^+ 6.0–6.4 mmol/l) or 'severe' (K^+ ≥6.5 mmol/l).

Causes

The main causes of hyperkalaemia are:

- Renal failure (i.e. acute kidney injury (AKI), chronic kidney disease (CKD) or end-stage renal disease (ESRD)).
- Drugs (e.g. angiotensin converting enzyme inhibitors (ACE-I), angiotensin II receptor antagonists (ARB), mineralocorticoid receptor antagonists (MRA), non-steroidal anti-inflammatory drugs, non-selective beta-blockers, trimethoprim, suxamethonium).
- Endocrine disorders (e.g. diabetic ketoacidosis, Addison's disease).
- Tissue breakdown (e.g. rhabdomyolysis, tumour lysis syndrome, haemolysis).
- Diet (high potassium intake in patients with advanced CKD).
- Spurious – consider pseudo-hyperkalaemia in the presence of normal renal function, normal ECG and/or history of haematological disorder.[123] Pseudo-hyperkalaemia is detected when the *serum* (clotted blood) K^+ level exceeds that of the *plasma* (non-clotted blood) by more than 0.4 mmol/l on simultaneous samples.[124] Difficult venepuncture, prolonged transit time and poor storage conditions can contribute to spurious K^+ levels.

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

Diagnosis

Consider hyperkalaemia in all patients with an arrhythmia or cardiac arrest, especially in the patient groups at risk (e.g. renal failure, heart failure, diabetes mellitus, rhabdomyolysis).

Symptoms may be absent or over-shadowed by the primary illness causing hyperkalaemia, but the presence of limb weakness, flaccid paralysis or paraesthesia are indicators of severe hyperkalaemia. Confirm the presence of hyperkalaemia using point-of-care testing (i.e. blood gas analyser) if available. Formal laboratory samples will take some time, therefore clinical decisions can be made on the results using point-of-care testing.[19,125–128] The ECG is used to assess cardiac toxicity and risk of arrhythmias in patients with known or suspected hyperkalaemia and may be the most readily available diagnostic tool. When the diagnosis of hyperkalaemia can be established based on the ECG, treatment can be initiated even before serum biochemistry is available.

The ECG signs of hyperkalaemia are usually progressive and include:

- First degree heart block (prolonged PR interval >0.2 s).
- Flattened or absent P waves.
- Tall, peaked (tented) T waves (i.e. T wave larger than R wave in more than 1 lead).
- ST-segment depression.
- Widened QRS (>0.12 s).
- VT.
- Bradycardia.
- Cardiac arrest (PEA, VF/pVT, asystole).

The incidence of ECG changes appears to correlate with severity of hyperkalaemia. In patients with severe hyperkalaemia (K^+ ≥6.5 mmol/l), arrhythmias or cardiac arrest have been shown to occur in 15% of patients within 6 h of the presenting ECG prior to initiation of treatment.[129] However, it is also recognised that the ECG may be normal even in the presence of severe hyperkalaemia and the first ECG sign of hyperkalaemia may be an arrhythmia or cardiac arrest.[116]

Treatment

There are five key steps in the treatment of hyperkalaemia:[116]

1. Protect the heart;
2. Shift potassium into cells;
3. Remove potassium from the body;
4. Monitor serum potassium and glucose levels;
5. Prevent recurrence of hyperkalaemia.

Follow a systematic approach as outlined in the hyperkalaemia treatment algorithm (Fig. 4). Assess the patient using the ABCDE approach and check the severity of hyperkalaemia with urgent bloods and an ECG. Treatment is guided by the severity of hyperkalaemia and the presence of ECG changes. Cardiac monitoring is essential for patients with severe hyperkalaemia. Consider the need for early specialist or critical care referral. The treatment of mild hyperkalaemia is out with the scope of this guideline.

Intravenous calcium salts (calcium chloride or gluconate) are indicated in severe hyperkalaemia in the presence of ECG changes. Although this therapy lacks a strong evidence base, it is widely accepted that it prevents arrhythmias and cardiac arrest.[130] The main risk of this treatment is tissue necrosis secondary to extravasation, therefore ensure secure vascular access prior to administration.

Insulin and glucose is the most effective and reliable therapy for lowering serum K^+ by shifting K^+ into cells. However, there is growing evidence to highlight the risk of hypoglycaemia with an incidence rate of up to 28%. Studies comparing low dose (5 units) to conventional dose (10 units) insulin reported hypoglycaemia in 8.7–19.7% of patients. Two studies have also reported an apparent dose-dependent effect with 10 units insulin showing greater efficacy than 5 units insulin.[131] The risk of hypoglycaemia is reduced by the administration of 50 g glucose.[132] Continuous delivery of glucose has also been shown to reduce hypoglycaemic events, therefore this strategy has been applied to the patient group most at risk. A low pre-treatment blood glucose level appears to be a consistent risk factor for development of hypoglycaemia.[133–139] Treat moderate or severe hyperkalaemia with 10 units insulin and 25 g, glucose followed by a continuous infusion of 10% glucose over 5 h (25 g glucose) in patients with a baseline glucose <7.0 mmol/l to reduce the risk of hypoglycaemia.[116]

Salbutamol is a beta-2 adrenoceptor agonist and promotes the intracellular shift of K^+. Its effect is dose-dependent, but a lower dose is recommended in patients with heart disease. Salbutamol may be ineffective in some patients including those receiving non-selective beta-blockers and in up to 40% of patients with ESRD, therefore it should not be used as monotherapy. The combination of salbutamol with insulin-glucose is more effective than either treatment alone.[140–142]

The novel potassium binders SZC[143] and the cation exchange resin Patiromer[144] are approved by NICE in the UK for the treatment of life-threatening hyperkalaemia ($K^+ \geq 6.5$ mmol/l) (enteral application). SZC works within 1 h[145] and lowers serum K^+ by 1.1 mmol/l within 48 h.[146] Efficacy increases with severity of hyperkalaemia. In patients with a serum $K^+ > 6.0$ mmol/l, SZC lowers serum K^+ by 1.5 mmol/l within 48 h.[146] SZC normalises serum K^+ in 66% of patients within 24 h, 75% within 48 h and in 78% of patients within 72 h.[147] Patiromer works more slowly with an onset of action within 4–7 h and lowers serum K^+ by 0.36 mmol/l within 72 h.[148] Pilot studies for both drugs in the acute setting have been inconclusive.[149,150] Both drugs may be used in patients with persistent moderate hyperkalaemia. However, NICE has recommended restricted use for patients with CKD 3b-5 (not on dialysis) or heart failure who are being treated with a sub-optimal dose of an ACE-I or ARB. Resins, e.g. calcium resonium, may be considered for patients who do not meet these criteria. Follow local guidelines for use of potassium binders.

Serial monitoring of the serum K^+ and blood glucose are essential to assess efficacy of treatment and to detect hypoglycaemia. Insulin-glucose and salbutamol are effective for 4–6 h, thereafter, be alert for a rebound of hyperkalaemia.

Indications for dialysis

Dialysis is the most definitive treatment for hyperkalaemia. The main indications for dialysis in patients with hyperkalaemia are:

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

Several dialysis modalities have been used safely and effectively in cardiac arrest, but requires expert help and equipment.[151–155] The procedure for dialysis initiation during cardiac arrest is outlined in the Renal Association (UK) Hyperkalaemia Guideline (2020).[116] Following dialysis, rebound hyperkalaemia may occur.

Hypokalaemia

Hypokalaemia is a common electrolyte disorder in clinical practice. It is associated with a higher in-hospital mortality and an increased risk of ventricular arrhythmias.[156–158] The risk of adverse events is increased in patients with pre-existing heart disease and in those treated with digoxin.[158–160]

Definition

Hypokalaemia is defined as a serum $K^+ < 3.5$ mmol/l. Clinical manifestations and treatment is guided by severity: mild (K^+ 3.0–3.4 mmol/l), moderate (K^+ 2.5–2.9 mmol/l) or severe ($K^+ < 2.5$ mmol/l or symptomatic).[160]

Causes

The main causes of hypokalaemia are:

- Gastrointestinal loss (e.g. diarrhoea, laxative abuse, villous adenoma of colon);
- Drugs (e.g. diuretics, laxatives, steroids);
- Therapies for hyperkalaemia (insulin/ glucose, salbutamol);
- Renal losses (e.g. renal tubular disorders, diabetes insipidus);
- Dialysis losses (e.g. peritoneal dialysis, post haemodialysis therapy);
- Magnesium depletion;
- Metabolic alkalosis;
- Endocrine disorders (e.g. Cushing's syndrome, primary hypoaldosteronism);
- Poor dietary intake.

Recognition

Consider hypokalaemia for all patients with an arrhythmia or cardiac arrest. As serum K^+ level falls, the nerves and muscles are predominantly affected causing fatigue, weakness, leg cramps and constipation. Mild hypokalaemia is usually asymptomatic, however, in severe cases ($K^+ < 2.5$ mmol/l), rhabdomyolysis, ascending paralysis, respiratory difficulties and arrhythmias may occur.[160]

ECG features of hypokalaemia:

- U waves;
- T wave flattening;
- Prolonged PR interval;
- ST segment changes (ST depression, T-wave inversion);
- Arrhythmias (increased risk on patients taking digoxin);
- Cardiac arrest (PEA, VF/pVT, asystole).

Treatment

There are 4 key steps in treating hypokalaemia:

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

Treatment is guided by the severity of hypokalaemia and presence of symptoms and/or ECG abnormalities. Slow replacement of potassium is preferable, but in an emergency, more rapid IV replacement is required.

- The standard rate of infusion of potassium is 10 mmol/h. The maximum rate is 20 mmol/h, but more rapid infusion (e.g. 2 mmol/min for 10 min, followed by 10 mmol over 5–10 min) is indicated for unstable arrhythmias when cardiac arrest is imminent.
- Continuous ECG monitoring is essential, ideally in a high dependency area.
- Monitor potassium level closely and titrate rate of replacement according to the level.

Magnesium is important for potassium uptake and for the maintenance of intracellular potassium concentration, particularly in the myocardium. Magnesium deficiency is common in patients with hypokalaemia. Repletion of magnesium will facilitate more rapid correction of hypokalaemia.[161] If hypokalaemia occurs concurrently with hypomagnesaemia, give 4 ml magnesium sulphate 50% (8 mmol) diluted in 10 ml NaCl 0.9% over 20 min, followed by potassium replacement (40 mmol KCL in 1000 ml 0.9% NaCl at a rate guided by urgency for correction as above). Follow with further magnesium replacement.[160]

Calcium and magnesium disorders

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

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

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	Primary or tertiary hyperparathyroidism Malignancy Sarcoidosis Drugs	Confusion Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Short QT interval Prolonged QRS interval Flat T waves AV block Cardiac arrest	Fluid replacement IV Furosemide 1 mg/kg IV Hydrocortisone 200–300 mg IV Pamidronate 30–90 mg IV Treat underlying cause
Hypocalcaemia Calcium <2.1 mmol/l	Chronic kidney disease Acute pancreatitis Calcium channel blocker overdose Toxic shock syndrome Rhabdomyolysis Tumour lysis syndrome	Paraesthesia Tetany Seizures AV-block Cardiac arrest	Prolonged QT interval T wave inversion Heart block Cardiac arrest	Calcium chloride 10% 10–40 ml IV Magnesium sulphate 50% 4–8 mmol IV (if necessary)
Hypermagnesaemia Magnesium >1.1 mmol/l	Renal failure Iatrogenic	Confusion Weakness Respiratory depression AV-block Cardiac arrest	Prolonged PR and QT intervals T wave peaking AV block Cardiac arrest	Consider treatment when magnesium >1.75 mmol/l: Calcium chloride 10% 5–10 ml IV repeated if necessary Saline diuresis –0.9% saline with furosemide 1 mg/kg IV Haemodialysis Ventilatory support if necessary
Hypomagnesaemia Magnesium <0.6 mmol/l	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: 2 g magnesium sulphate 50% (4 ml; 8 mmol) IV over 15 min Torsades de pointes: 2 g magnesium sulphate 50% (4 ml; 8 mmol) IV over 1–2 min Seizure: 2 g magnesium sulphate 50% (4 ml; 8 mmol) IV over 10 min

Hypothermia

Accidental hypothermia is the involuntary drop in core temperature <35 °C. Severe hypothermia may reduce vital functions until the occurrence of cardiac arrest. In hypothermic patients with spontaneous circulation insulation, hospital triage followed by transfer and rewarming are key interventions. In hypothermic patients with cardiac arrest, continuous CPR and ECPR rewarming may result in good neurological outcome even with prolonged no-flow or low-flow (i.e. CPR) time, provided that hypothermia ensued before cardiac arrest. These guidelines help to improve prehospital triage, transport and treatment as well as in-hospital management of hypothermic patients.

A scoping review was performed using the PubMed search engine on February 22nd 2020 using the keywords "avalanche" AND "rescue" (n = 100). Four systematic reviews were identified.[162–165] Relevant articles from the systematic reviews were included and references lists crosschecked for further articles.

Accidental hypothermia

Prevention from cardiac arrest

Accidental hypothermia is the involuntary drop in the core temperature <35 °C.[5,6] Primary hypothermia is induced by exposure to cold, while secondary hypothermia is induced by illness and other external causes. Primary hypothermia is prominent in outdoor (athletes and lost persons) and urban environments (homeless and intoxicated persons), while secondary hypothermia is rapidly increasing among old and multimorbid persons in the indoor environment.[166,167]

Assess temperature with a low reading thermometer (Fig. 5):[168,169]

- tympanically in spontaneously breathing patients,
- oesophageal in patients who are endotracheal intubated or instrumented with a 2nd generation supraglottic airway,
- If the ear is not well cleaned from snow and cold water or not insulated against the cold environment the reading could be mistakenly low by several °C.[170,171]

Accidental hypothermia gradually reduces vital functions until they finally cease completely (Table 3). 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. Rewarming is not feasible during short transportation times (e.g. <1 h).[6,172,173] Hypothermic patients with signs of prehospital cardiac instability (i.e. systolic blood pressure <90 mmHg, ventricular arrhythmia, core temperature <30 °C) should be rewarmed in hospital using minimally invasive techniques. Where possible they should be directly transferred to a hospital with stand-by extracorporeal life support (ECLS). ECLS should only be established if patients arrest or deteriorate (e.g. decreasing blood pressure, increasing acidosis).[6,84] Primary ECLS rewarming should also be considered in patients with $etCO_2 < 10$ mmHg or a systolic blood pressure ≤60 mmHg.[174]

Management of cardiac arrest

The lowest temperature from which successful resuscitation and rewarming has been achieved is currently 11.8 °C[176] or accidental hypothermia and 4.2 °C for induced hypothermia.[177] A recent systematic review reported only five patients (28–75 years of age) who had arrested at a core temperature >28 °C, suggesting that cardiac arrest due to primary hypothermia at >28 °C is possible but unlikely.[164] Some may still have minimal vital signs at a core temperature <24 °C.[175] This does not preclude resuscitation attempts at even lower temperatures if clinical judgment suggests the possibility of successful resuscitation.

A deeply hypothermic person may appear dead but still survive with resuscitation. Check for signs of life for one minute – not only by clinical examination but also by using ECG, $EtCO_2$ and ultrasound.[6,84] In hypothermic cardiac arrest information should be collected to estimate the survival probability from hypothermic cardiac arrest with HOPE, ICE or the traditional potassium triage:[162,163,178,179]

- Core temperature,
- Mechanism of hypothermia induction,
- Duration of CPR,
- Sex,
- Witnessed/unwitnessed cardiac arrest,
- First cardiac rhythm,
- Trauma (to decide whether to start ECLS rewarming with heparin),
- Serum potassium (in hospital).

This information is crucial for prognostication and to decide whether to rewarm the patient with ECLS. For hypothermic cardiac arrest HOPE (Hypothermia Outcome Prediction after ECLS rewarming for hypothermic arrested patients) has been best validated. The website to calculate HOPE can be accessed via: https://www.hypothermiascore.org.

Hypothermic patients in witnessed and unwitnessed cardiac arrest have good chances of neurological recovery if hypothermia developed before hypoxia and cardiac arrest and if the chain of survival is functioning well.[6,164,180–183] 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.[184] A recent systematic review on witnessed hypothermic cardiac arrest patients (n = 214) reported a survival to hospital-discharge rate of 73%, 89% had a favourable outcome. Another systematic review on hypothermic patients with unwitnessed cardiac arrest (n = 221) reported a survival rate of 27%, 83% had a neurologically intact outcome. Of note, the first rhythm was asystole in 48% of survivors.[165] Hypothermic cardiac arrest patients should receive continuous CPR until circulation has been re-established. Chest compression and ventilation rate should follow the standard ALS algorithm as for normothermic patients. Hypothermic cardiac arrest is often refractory to defibrillation and adrenaline. Defibrillation attempts have been successful in patients with a core temperature >24 °C, however, ROSC tends to be unstable with lower temperature.[185]

The hypothermic heart may be unresponsive to cardioactive drugs, attempted electrical pacing and defibrillation. Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drug given.[6] The evidence for the efficacy of drugs in severe hypothermia is limited and based mainly on animal studies. For instance, in severe hypothermic cardiac arrest, the efficacy of amiodarone is reduced.[186] Adrenaline may be effective in increasing coronary perfusion pressure, but not survival.[187,188] Vasopressors may also increase the chances of successful defibrillation, but with a core temperature <30 °C, sinus rhythm often degrades back into VF. Given that defibrillation and adrenaline may induce myocardial injury, it is reasonable to withhold adrenaline, other CPR drugs and shocks until the patient has been warmed to a core temperature ≥30 °C. Once 30 °C has been reached, the intervals between drug doses should be doubled when compared to normothermia (i.e. adrenaline every 6–10 min). As normothermia (≥35 °C) is approached, standard drug protocols become effective again.[5,6]

Arrested hypothermic patients should, where possible, be directly transferred to an ECLS centre for rewarming. In primary hypothermia an unwitnessed cardiac arrest with asystole as first rhythm is not a contraindication to ECLS rewarming.[165] In hypothermic cardiac arrest rewarming should be performed with ECLS, preferably with ECMO rather than CPB.[6,189,190] If ECLS is not available within 6 h, non-ECLS rewarming may be used.[183,191,192] 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 (Fig. 6).[8]

In-hospital prognostication of successful rewarming should be based on the HOPE or ICE score (Table 4), the traditional in-hospital serum potassium prognostication is less reliable.[162,163,178]

A post-resuscitation care bundle is recommended following successful resuscitation. Emergency medical services (EMS) and hospitals should install structured protocols to improve prehospital triage, transport and treatment as well as in-hospital management of hypothermic patients.

Avalanche rescue

Most avalanche victims die from asphyxia.[193,194] Avalanche victims in an unwitnessed cardiac arrest have a poor chance of survival.[193–195] The chance of a good outcome are improved if there is a ROSC in the

Table 3 – Staging of accidental hypothermia.[6].

Stage	Clinical findings	Core temperature (°C) (if available)
Hypothermia I (mild)	Conscious, shivering[a]	35–32 °C
Hypothermia II (moderate)	Impaired consciousness[a]; may or may not be shivering	<32–28 °C
Hypothermia III (severe)	Unconscious[a]; vital signs present	<28°C
Hypothermia IV (severe)	Apparent death; Vital signs absent	Variable[b]

[a] Shivering and consciousness may be impaired by comorbid illness (i.e. trauma, CNS pathology, toxic ingestion, etc.) or drugs (i.e. sedatives, muscle relaxants, narcotics etc.) independent of core temperature.

[b] The risk of cardiac arrest increases <32 °C, older and sicker are at higher risk, alternative causes should be considered. Some still have vital signs <24 °C.[175].

first minutes of CPR.[162,163,194] In arrested patients five ventilations should initially be provided because hypoxia is the most likely cause of the cardiac arrest. Avalanche victims with OHCA and duration of burial <60 min should be managed like normothermic patients (Fig. 7). Standard ALS should be provided for at least 20 min.[84,196,197] Avalanche victims with duration of burial >60 min without evidence of an un-survivable injury undertake full resuscitative measures, including ECLS rewarming.[6,84,197] CPR should be considered as futile in cardiac arrest with a burial time >60 min and evidence of an obstructed airway.[84,197] In-hospital prognostication of successful rewarming should be based on the HOPE score.[162,163] The traditional triage with based on serum potassium and the core temperature (cut-off 7 mmol/l and 30 °C, respectively) may be less reliable.[178] A post-resuscitation care bundle is recommended following successful resuscitation.

Hyperthermia and malignant hyperthermia

Hyperthermia is a condition when the temperature of the body increases above normothermia (36.5–37.5 °C) because of failed thermoregulation. Heat stroke is an emerging health concern due to a soaring environmental temperature caused by increasing greenhouse gas emissions.[198] Heatwaves kill more people than any other extreme weather condition.[199] In 2003, an extreme heatwave killed 70,000 people in Europe.[200] The lack of ability to sweat is the main risk factor for hyperthermia. Extremes of age and multimorbidity confer additional risks.[199,201] The easiest modifiable risk factor is sufficient hydration.[202] Hyperthermia is a continuum of heat-related conditions, ranging from heat stress progressing to heat exhaustion, finally to exertional and non-exertional (caused by environmental heat) heat stroke and potentially progressing to multiple organ dysfunction and cardiac arrest (>40 °C).

Malignant hyperthermia (MH) 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 and succinylcholine. 3,4-Methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines may also cause a condition similar to MH. After exposure a rapid and uncontrolled influx of calcium into the cytoplasm of the skeletal muscle cell results in a hypermetabolic cascade involving sustained muscular contractures, depletion of adenosine triphosphate (ATP) and muscular cell death. The resulting clinical signs include hypercapnia, masseter muscle, generalized muscle rigidity, acidosis, hyperkalaemia, peaked T waves and hyperthermia.

This section is based on an ILCOR systematic review, two recent reviews and an additional scoping review (February 22nd 2020).[52,199,202,203]

Hyperthermia

Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds values normally maintained by homeostatic mechanisms.[199,202] Hyperthermia may be primarily induced by environmental conditions, or secondary due to endogenous heat production.[199] Environment-related hyperthermia occurs where heat, usually in the form of radiant energy, is absorbed by the body at a rate faster than can be lost by thermoregulatory mechanisms. Hyperthermia is a continuum of heat-related conditions, starting with heat syncope, progressing to heat exhaustion, then to heat stroke with a compensable and a non-compensable state and finally to multiple organ dysfunction and cardiac arrest. Importantly, the heat stroke-triggered inflammatory response may resemble the systemic inflammatory response syndrome (SIRS) and be misdiagnosed and a critical delay may result in providing the appropriate treatment.[199] A rectal or oesophageal temperature probe should be available to measure core temperature and to guide treatment (Fig. 8).[168]

Heat syncope is a mild form of hyperthermia.[202] Treatment includes removing patients to a cool environment, passive cooling and administration of oral isotonic or hypertonic fluids.

Heat exhaustion is caused by mild to moderate hyperthermia due to exposure to high environmental heat or excessive exercise.[202] Symptoms include intense thirst, weakness, discomfort, anxiety, dizziness, syncope, core temperature may be normal or >37 °C. Treatment includes removing patients to a cool environment, lying them flat and administering IV isotonic or hypertonic fluids, consider additional electrolyte replacement therapy with isotonic fluids. Oral rehydration may not be effective in rapidly replacing electrolytes but may be a more practical treatment. Replacement of 1–2 l crystalloids at 500 ml/h is often adequate. Simple external cooling measures are usually not required but may involve conductive (cold floor, ice sheets; commercial ice packs to hand, feet and cheek), convective (cold water immersion, cold shower) and evaporative measures (spraying cold water, fanning the undressed).

Heat stroke is primarily a clinical diagnosis based on the 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 or exertional hyperthermia).[204] Symptoms include central nervous

Table 4 – Hypothermia Outcome Prediction after ECLS (HOPE) for hypothermic cardiac arrest patients, description of parameters affecting HOPE with regard to estimation of the survival probability. CPR denotes cardiopulmonary resuscitation, ECLS extracorporeal life support.

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

system dysregulation (e.g. altered mental state, seizure, coma), tachycardia, tachypnoea and arterial hypotension.[199] Mortality is approximately 10% and when combined with hypotension it approaches 33%.[202] The outcome worsens if the body temperature is sustained at >40.5 °C.

It is essential to rapidly cool the patient to <39 °C, preferably <38.5 −38.0 °C as quickly as possible. Treatment involves removing patients to a cool environment, lying them flat. Cold-water immersion (from neck down) or full body conductive cooling should be used, cooling rates of 0.2−0.35 °C/min can be achieved.[205] 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).[203] Alternatively, a combination of simple cooling techniques could be used including conductive, convective and evaporative measures, although no comparative studies exist to guide the best option.[203] One systematic review concluded that water immersion (1 −17 °C water) lowers body temperature more effective compared to passive cooling.[203] Misting and fanning cooling techniques are marginally faster than passive cooling and cold showers (20.8 °C) cool faster than passive cooling.[203] Isotonic or hypertonic fluids should be administered (with blood sodium >130 mEq/l up to 3 × 100 ml 3% NaCl at 10 min intervals).[206] If mental state is abnormal 3% NaCl should be administered IV, if mental state is normal it can be administered orally.[206] Additional electrolyte replacement with isotonic fluids should be considered and substantial amounts of fluids may be required. For exertional heatstroke, a cooling rate faster than 0.10 °C/min is safe and desirable. 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.[199,207] There may be a requirement for advanced cooling techniques including external or internal devices used for targeted temperature management. There are no specific drugs lowering core temperature.

Malignant hyperthermia

Most MH associated variants are caused by mutation of the RYR1 gene. RYR1 encodes the skeletal muscle ryanodine receptor protein, that regulates the movement of calcium from the sarcoplasmic reticulum into the cytoplasm of the muscle cell.[208,209] Drugs such as 3,4-methylenedioxymethamphetamine (MDMA, ‘ecstasy’) and amphetamines may also cause a condition similar to MH and the use of dantrolene may be helpful.[210] If cardiac arrest occurs, follow the universal ALS algorithm and continue to cool the patient. Attempt defibrillation using standard energy levels. Apply the same cooling techniques as for post-resuscitation care targeted temperature management.

Give dantrolene (2.5 mg/kg initially, 10 mg/kg as required). Ryanodex® is a lyophilized nanosuspension of dantrolene sodium with substantially improved pharmacological properties (fewer vials 1:12.5, administration time is 1 instead of 20 min). The introduction in European countries is pending.

It is essential to contact an expert MH centre for ongoing advice once the patient has been stabilized. Due the increased metabolic rate, outcome is poor compared with normothermic cardiac arrest.[211] Unfavourable neurological outcome increases by 2.26 odds ratio for 1 °C of body temperature >37 °C.[212]

Thrombosis

This section refers to pulmonary and to coronary thrombosis as potential reversible causes of cardiac arrest.

Pulmonary embolism

Cardiac arrest from acute pulmonary embolism is the most serious clinical presentation of venous thromboembolism, in most cases originating from a DVT.[213] The reported incidence of cardiac arrest caused by pulmonary embolism is 2−7% of all OHCAs,[214,215] and 5 −6% of all IHCAs,[120,216] but this is likely to be an underestimation. Overall survival is low.[215,217] Specific treatments for cardiac arrest resulting from pulmonary embolism include administration of fibrinolytics, surgical embolectomy and percutaneous mechanical thrombectomy.

The updated 2020 ILCOR systematic review explored the influence of specific treatments (e.g. fibrinolytics, or any other) yielding favourable outcomes.[2] The 2019 ILCOR summary statement reviewed the use of ECPR for cardiac arrest in adults.[103] Additional evidence was identified from the updated ESC guideline on pulmonary embolism,[213] pertaining articles were included and references lists crosschecked for further articles.

Diagnosis

Diagnosis of acute pulmonary embolism during cardiac arrest is difficult. One study has reported correct recognition of the underlying causes in up to 85% of all in-hospital resuscitation attempts,[216] but accurate prehospital diagnosis of acute pulmonary embolism is particularly challenging.[218−220] The 2019 European Society of Cardiology Guidelines on the diagnosis and management of acute pulmonary embolism define ‘confirmed pulmonary embolism’ as a probability of pulmonary embolism high enough to indicate the need for specific treatment.[213] Clinical history and assessment, capnography and echocardiography (if available) can all assist in the diagnosis of acute pulmonary embolism during CPR with varying degrees of specificity and sensitivity. Cardiac arrest commonly presents as PEA.[217] Low $ETCO_2$ readings (about 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.[219,221]

Common symptoms preceding cardiac arrest are sudden onset of dyspnoea, pleuritic or substernal chest pain, cough, haemoptysis, syncope and signs of DVT in particular (unilateral lower extremity swelling) Information about past medical history, predisposing factors, and medication that may support diagnosis of pulmonary embolism should be obtained, although none of these are specific.[84,213] In as many as 30% of the patients with pulmonary embolism, no risk factors are apparent.[222] If a 12-lead ECG can be obtained before onset of cardiac arrest, changes indicative of right ventricular strain may be found:[213,223]

- 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.

Acute PE can cause right ventricle (RV) pressure overload and dysfunction and these signs can be seen on echocardiography. Unfortunately, there is no individual echocardiographic parameter that provides fast and reliable information on RV size or function. Echocardiographic criteria for the diagnosis of PE differ between studies, the negative predictive value is only 40−50%.[213] Signs of right ventricular overload or dysfunction may also be caused by other cardiac or pulmonary disease.[224]

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

Prevention of cardiac arrest

Airway

Low cardiac output results in desaturation of the mixed venous blood. Although no studies were found which examined the role of oxygen versus any other gas, the writing group considered hypoxaemia as confounding risk factor for cardiac arrest and recommends administration of high-flow oxygen until goal-directed therapy could be established.

Breathing

Hypoxaemia and hypocapnia are frequently encountered in patients with PE, but they are of moderate severity in most cases. PE should be considered in all patients with sudden onset of progressive dyspnoea, especially in patients without pre-existing pulmonary disease. Other reversible causes of cardiovascular deterioration and dyspnoea have to be excluded, e.g. (tension) pneumothorax and anaphylaxis (anaphylactic shock). Hypoxaemia is usually reversed with administration of oxygen.

When mechanical ventilation is required, care should be taken to limit its adverse haemodynamic effects. In particular, the positive intrathoracic pressure induced by mechanical ventilation may reduce venous return and worsen RV failure.[213]

Circulation

The clinical classification of the severity of an episode of acute PE is based on the estimated in-hospital or 30-day mortality. High-risk PE is suspected or confirmed in the presence of shock or persistent arterial hypotension. Suspected high-risk PE is an immediately life-threatening situation.

Acute right ventricle (RV) failure is the leading cause of death in patients with high-risk PE. Aggressive volume expansion is of no benefit and may even worsen RV function by causing mechanical overstretch. On the other hand, modest ($\leq$500 ml) fluid challenge may help to increase cardiac index in patients with PE, low cardiac index, and normal BP. Use of vasopressors and/or inotropes is frequently needed.[213]

Reperfusion

Parenteral anticoagulation should be initiated whilst awaiting the results of diagnostic tests. Intravenous unfractionated heparin (UFH) is recommended for patients with shock and hypotension, and in whom primary reperfusion is considered. Thrombolytic treatment of acute PE restores pulmonary perfusion more rapidly than anticoagulation with UFH alone. A review of RCTs indicated that thrombolysis may be associated with a reduction in mortality or recurrent PE in high-risk patients who present with haemodynamic instability.[225] Surgical embolectomy or percutaneous catheter-directed treatment are recommended as alternative to rescue thrombolytic therapy in rapidly deteriorating patients if expertise and resources are available on site. Treatment decisions should be made by an interdisciplinary team involving a thoracic surgeon or interventional cardiologist.[213,226]

Modifications to ALS for PE

Thrombolysis

When PE is the suspected cause of cardiac arrest thrombolytic drugs should be administered (weak recommendation, very low certainty of evidence).[2] For ROSC two studies provided very-low-certainty evidence of no difference with thrombolysis versus without,[227,228] one study showed benefit associated with the use of thrombolytic drugs compared with no thrombolytic drugs in patients with PE.[217] One study showed benefit with thrombolysis for survival at 24 h whereas another study showed no difference with versus without thrombolysis.[214,227] Three observational studies showed no benefit for survival to discharge.[217,227,228] For survival with favourable neurologic outcome at 30 days one RCT compared thrombolytics with placebo in 37 patients with confirmed PE, finding no difference between groups,[215] another observational study with/without thrombolysis found no difference.[214] There is insufficient evidence to recommend any optimal drug and dosing strategy for thrombolysis during CPR.[2] When thrombolytic drugs have been administered, consider continuing CPR attempts for at least 60–90 min before termination of resuscitation attempts.[229–231]

Surgical embolectomy

When PE is the known cause of cardiac arrest the use of fibrinolytic drugs or surgical embolectomy or percutaneous mechanical thrombectomy is recommended (weak recommendation, very low certainty of evidence).[2] The method is reported in 2 case series without control group in cardiac arrest patients.[232,233]

Percutaneous mechanical thrombectomy

This method is reported in 1 case series in cardiac arrest patients.[234]

Extracorporeal CPR

ECPR should be considered as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it can be implemented (weak recommendation, very low certainty of evidence).[103] ECPR is increasingly used to support circulation in patients with cardiac arrest refractory to conventional CPR.[235] Some observational studies suggest the use of extracorporeal life support (ECLS) if cardiac arrest is associated with PE.[236,237] ECPR maintains vital organ perfusion while potential reversible causes of the cardiac arrest can be identified and treated. ECPR can be considered in select patients when rapid expert deployment is possible; however, the optimal patient selection and timing of the therapy are not well defined. The recommendations on ECPR derive from heterogeneous individual studies that are difficult to interpret, mainly because of confounding. Randomised controlled trials (RCTs) are not available. This recent weak recommendation takes the extremely high mortality rate of patients with cardiac arrest, particularly when the arrest is refractory to standard advanced cardiac life support interventions (i.e., cardiac arrest when conventional CPR is failing) in account. Therefore, the potential for benefit and the value of this intervention remain despite the overall low certainty of supporting evidence and lack of randomised trials.[238]

Coronary thrombosis

Obstructive coronary artery disease (CAD) is the most common condition underlying OHCA in adults.[239,240] The clinical spectrum of CAD includes ventricular arrhythmias due to acute ischaemia, those presenting during the acute and convalescent stages of myocardial infarction and arrhythmias related with post-myocardial infarction scar or ischaemic remodelling.[241] Significant or acute coronary stenosis are especially prevalent in the presence of shockable rhythms (VF/pVT) and ST-elevation in the post-arrest ECG, although a significant percentage of acute coronary lesions has been reported among patients with ROSC and no ST-elevation (Table 5). Conversely, the presence and role of CAD in non-shockable rhythms (PEA or asystole) remains uncertain, since coronary angiography is less frequently performed in such cases.[242]

Evidence based recommendations for the suspicion and management of coronary thrombosis as the cause of OHCA derive from the 2019 ILCOR CoSTR summary,[238] the 2015 ILCOR CoSTR on acute coronary syndromes (ACS),[243] the European Society of Cardiology Guidelines,[244–246] the consensus statement from the European Association for Percutaneous Cardiovascular Interventions (EAPCI)[247] and the American Heart Association (AHA) scientific statement.[242] Guidelines were assessed according to the AGREE-II framework. Additionally, focused literature search for an evidence update was performed and recommendations were established by consensus within the writing group.

Prevent and be prepared

Encourage cardiovascular prevention

Tackling the onset and progression of CAD by means of primary and secondary cardiovascular prevention, including healthy lifestyles and adherence to evidence-based medications may be the first step to reduce the risk of acute cardiovascular events, including OHCA.[246,248] Cardiac rehabilitation for patients after acute coronary syndrome (ACS) has proven effective at reducing the risk of subsequent events,[249] but it remains underutilised and should be encouraged.[250,251]

Endorse health education

Reducing time from symptom onset to first medical contact is a well-known area for improvement. This could be attained by educational campaigns to increase public awareness on the importance of recognising symptoms of myocardial infarction (chest pain) and early alerting the EMS.[245]

Promote layperson basic life support (BLS)

Initiatives promoting BLS training among the general public might improve awareness and the likelihood of bystander intervention in OHCA, increasing the chances of good outcome. Further information is provided in section 12 (education). Particularly, training should involve high-risk groups such as relatives of patients with previous ACS.[252,253] A systematic review of studies addressing BLS training for family members of high-risk cardiac patients reported adequate disposition and capacity to learn,[254] but reaching this group may be challenging. Additional later studies have shown positive results of implementing BLS training targeted at patients and their relatives within cardiac rehabilitation programs.[255,256]

Ensure adequate resources

Regional STEMI networks have contributed to reduce reperfusion times and improve patient outcomes.[257–262] These initiatives should be encouraged and provided with sufficient human and material resources and adequate training of the staff (including ECG interpretation and advanced life support).[263]

Improve quality management systems and indicators

Data concerning OHCA in the setting of ACS have irregularly been reported or excluded from mortality analyses. Seeking quality standards similar to those established for non-OHCA ACS might enhance quality monitoring and lead to better outcomes.[245] However, the particular characteristics of OHCA-ACS patients may require categorising these cases separately and pursuing appropriate and reliable quality indicators.[264]

Detect parameters suggesting coronary thrombosis and activate STEMI network

Post-arrest 12-lead ECG may present unspecific alterations that hinder interpretation, and criteria suggesting coronary occlusion (i.e. bundle branch block, hyperacute T-waves, diffuse ST depression with V1/aVR ST-elevation) if symptoms of ischaemia are present [245] [Ibanez 2018, 119] may not necessarily apply to OHCA patients. Given the limitations of a post-arrest ECG to predict coronary occlusion,[265,266] all available information should be gathered to make decisions, including specific clinical features such as chest pain prior to arrest or known history of CAD. Once clinical suspicion is established, the STEMI network should be activated to facilitate early reperfusion.

Resuscitate and treat possible causes (establish reperfusion strategy)

Patients with sustained ROSC

Although prospective randomised trials are lacking, there is general consensus that successfully resuscitated STEMI patients should undergo immediate reperfusion, as extrapolated from recommendations regarding non-OHCA STEMI patients.[242,243,245,247] Primary percutaneous coronary intervention (PCI) is the strategy of choice and should be performed in $\leq$120 min from diagnosis.[242,243,245,247] Pre-hospital fibrinolysis may be administered if a greater delay is expected, unless resuscitation efforts were prolonged or traumatic or other contraindications are present.[242,243,245,247] Resuscitated STEMI patients who remain comatose after ROSC constitute a highly heterogeneous subgroup with a poorer prognosis, but there is no current evidence to discourage urgent coronary angiography.[243]

In patients with ROSC and no-STEMI criteria, two systematic reviews reported benefits of performing PCI,[267,268] although the timing of coronary angiography remains controversial. The COACT trial showed no benefit of emergent compared to delayed coronary angiography in 90-

Table 5 – Prevalence of significant coronary artery disease among patients with ventricular fibrillation/pulseless electrical tachycardia out-of-hospital cardiac arrest.

	ROSC		Refractory VF/pVT
	ST elevation	No ST elevation	
Prevalence of significant CAD	70–95%	25–50%	75–85%
Prevalence of acute lesions	70–80%	25–35%	60–65%

days survival among patients with initial shockable rhythm and no STEMI or another non-cardiac cause for OHCA who remained unconscious after ROSC.[269] However, the higher survival than expected in both groups may have lessened the power of the trial. Until new evidence from ongoing randomised trials is available, an individualised approach considering patient characteristics, ECG findings and haemodynamic condition is recommended.[244] Brief evaluation in the emergency department or intensive care unit may be considered to exclude obvious non-coronary causes of OHCA and check patient's status. If ongoing myocardial ischaemia is suspected or the patient is haemodynamically or electrically unstable, early coronary angiography ($\leq$120 min) should be performed. In resuscitated stable patients without STEMI, a delayed angiography strategy would be considered.[242–245,247,270]

Patients with no sustained ROSC

Decisions regarding patients who do not achieve sustained ROSC despite resuscitation are challenging, and should be individualised considering patient and the setting conditions and the available resources. Further information on termination of resuscitation decisions is provided in the ERC Guidelines on Ethics.[271] Consistent with the principles outlined in those guidelines, routine coronary angiography cannot be recommended in this subset of patients with refractory OHCA, and should be individualised after careful evaluation of the benefit/futility ratio, available resources and expertise of the team.[247]

Although mechanical CPR has not proven superiority to conventional CPR, it may facilitate delivering high-quality chest compressions during transportation of patients or during coronary angiography. Special attention must be paid to minimising interruption of compressions and any delay to defibrillation.[272] A recent systematic review on ECPR showed no conclusive evidence to support or discourage its use for IHCA or OHCA for both adults and children.[273] Accordingly, ILCOR considers ECPR for selected patients when conventional CPR is failing (weak recommendation, very-low certainty of evidence).[103,238] However, a later large registry study including 13,191 patients with OHCA found no association between ECPR and improved outcome compared with conventional CPR, although certain features (initial shockable rhythm, transient ROSC) leading to better outcomes in the ECPR group were identified.[274] Randomised trials of ECPR initiated out of hospital (clinicaltrials.gov NCT02527031) and in hospital (clinicaltrials.gov NCT03101787 and NCT01511666) will contribute to increase evidence on patient selection, risk-benefit and cost-effectiveness.

Cardiac tamponade

Cardiac tamponade occurs when the pericardial sac is filled with fluid under pressure, which leads to compromise of cardiac function and ultimately cardiac arrest. The condition most commonly occurs after penetrating trauma and cardiac surgery. Mortality is high and immediate decompression of the pericardium is required to give any chance of survival. Evidence base for diagnosis (ultrasound/ALS) and treatment (trauma/Special circumstances – TCA; cardiac surgery/Special circumstances – cardiac surgery) is given in more detail in the respective sections of the 2020 guidelines.

The literature review for an evidence update did not result in any new evidence compared to the ERC guidelines 2015.

Diagnosis

Echocardiographic evaluation of cardiac tamponade is important for timely and appropriate diagnosis and management. Use of point of care cardiac ultrasound is described in detail in the ALS chapter.

Treatment

Thoracotomy

The criteria and prerequisites for resuscitative thoracotomy in patients with penetrating trauma to the chest or epigastrium are described in the section on traumatic cardiac arrest. Treatment of cardiac tamponade following cardiac surgery is addressed in the section on cardiac arrest following cardiac surgery.

Pericardiocentesis

If thoracotomy is not possible, consider ultrasound guided pericardiocentesis to treat cardiac arrest associated with suspected traumatic or non-traumatic cardiac tamponade. Non-image guided pericardiocentesis is an alternative, only if ultrasound is not available.[84]

Tension pneumothorax

Tension pneumothorax is a reversible cause of cardiac arrest and must be excluded during CPR. It may lead to cardiac arrest by obstructing venous return through mediastinal shift. Tension pneumothorax may be caused by trauma, severe asthma and other respiratory disease, but can also be iatrogenic following invasive procedures, e.g. central line insertion. Institution of positive pressure ventilation can convert a pneumothorax into a tension pneumothorax.[275] The prevalence of tension pneumothorax is approximately 0.5% in all major trauma patients treated in the prehospital setting and 13% of those developing TCA.[26]

Recommendations in this section are based on focused literature search for evidence update and 1 systematic review.[275]

Diagnosis

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, chest crepitations and subcutaneous emphysema and mediastinal shift (tracheal deviation and jugular venous distention).[275] During CPR presentation 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 if the expertise is available.[276]

Treatment recommendations

Needle decompression

Needle chest decompression is rapid and within the skill set of most ambulance personnel. It is frequently carried out with standard IV cannulas. However, a significant proportion of patients have a chest wall thickness which makes needle decompression with a standard length 14-gauge cannula ineffective.[61] A needle length of at least 7 cm is required to reach the pleural space at mid-clavicular position in the 2nd intercostal space in 90% of all attempts in an average population.[62] Needle decompression in the 4th/5th ICS at anterior axillary line has the lowest predicted failure rate compared to the 4th/5th ICS midaxillary line and the 2nd ICS MCL.[277]

Cannulae are also prone to kinking and blockage.[278] Any attempt at needle decompression under CPR must be followed by an open thoracostomy or a chest tube if the expertise is available.

Thoracostomy

In TCA patients, chest decompression effectively treats tension pneumothorax and takes priority over all other measures. Open thoracostomy is simple to perform and used routinely by several prehospital services.[279] The thoracostomy is the first stage of standard chest tube insertion – a simple incision and rapid dissection into the pleural space (see traumatic cardiac arrest). Chest drain insertion can be carried out following successful resuscitation.

Toxic agents

Overall, poisoning rarely causes cardiac arrest or death,[280] although the latest reports show that, among human exposure to toxic agents, those with more serious outcomes (moderate, major or death) have increased 4.45% per year since 2000.[281] The top 5 poisoning substance classes in 2018 were analgesics, household cleaning substances, cosmetics and personal care products, sedatives, hypnotics, antipsychotics and antidepressants.[281] Intentional (i.e. suicide) and accidental poisoning from pesticides are both significant causes of mortality.[282] Poisoning is an important cause of OHCA in younger age groups.[283] Inappropriate drug dosing, drug interactions and other medication errors can also cause harm. Accidental poisoning is more common in children than in adults.[284,285] Homicidal poisoning is uncommon. Industrial accidents, warfare or terrorism can also cause exposure to toxins.

Recommendations in this section are based on systematic reviews, using a dual review approach. For opioid toxicity ILCOR published an evidence update.[2] Given the infrequent nature of most poisonings, clinical effectiveness of many interventions often is based on low-certainty evidence including animal studies and human case series or case reports, with significant publication bias. The likelihood of confirmatory RCTs to prove effectiveness of such results is poor. Hence, most of the following updates and related recommendations are weak and based on low level of evidence.

Cardiovascular emergencies in acute poisoning

Toxic agents can produce cardiovascular emergencies via indirect (mediated by metabolic disorders) or direct mechanisms. In the latter case, toxic agents can modify blood pressure, myocardial contractility and conductivity. Hypertensive emergencies can occur during acute poisonings with adrenergic agonists such as cocaine or amphetamines. The best management consists of sedation with benzodiazepines, vasodilators and pure alpha-antagonists.

Hypotension can be caused by many toxic agents which lead to hypovolaemia due to acute losses (pesticides, mushrooms, lithium, diuretics, cholinomimetics) or to vasodilation (alcohol, antihypertensive medications, anticholinergics, tricyclic antidepressants, calcium channel blockers, opioids). Toxic agents can also cause tachy- or bradyarrhythmia (anticholinergics, sympathomimetics, anti-arrhythmic drugs, halogenated hydrocarbons, etc). Medications with quinidine-like effects should be treated with sodium bicarbonate (1–2 $mmol\,kg^{-1}$) IV. It is important to keep in mind specific treatments where available (calcium channel blocker and beta-blocker, digoxin intoxications) on top of the ALS management of arrythmias.[101]

Neurological emergencies in acute poisoning

Toxic agents can also be responsible for neurological emergencies, such as reduced levels of consciousness, seizures and movement disorders. Clinically, in metabolic (or toxic) comas, oculo-cephalic and oculo-vestibular reflexes are usually preserved and motor response is usually symmetrical. Pupillary size can guide the diagnosis (miosis being typical of opioid overdose and mydriasis of anticholinergic overdose). Many medications can cause seizures via direct effect (anti-histamine, antidepressant, antipsychotics, antibiotics, lithium, caffeine, cocaine, amphetamines, pesticides, carbon monoxide). Treatment of such emergencies must follow the ALS algorithm including early advanced airway management (see ALS Guidelines).[101]

Prevention of cardiac arrest

Assess the patient using the systematic ABCDE approach. Airway obstruction and respiratory arrest secondary to a decreased conscious level is a common cause of death after self-poisoning (benzodiazepines, alcohol, opiates, tricyclics, barbiturates).[286,287] Early tracheal intubation of the unconscious patient by trained personnel may decrease the risk of aspiration. Drug-induced hypotension usually responds to IV fluids, but occasionally vasopressor support is required. Measure serum electrolytes (particularly potassium), blood glucose and arterial blood gases. Retain samples of blood and urine for toxin analysis. Patients with severe poisoning should be cared for in a critical care setting.[287] If available, once the patient has been stabilised, check for any history that can provide information on the toxic agent involved. If an antidote is available administer it as soon as possible in order to improve outcome. The causative agent has been shown to be strongly associated with outcome in poisoning-induced OHCAs.[288]

Modifications to resuscitation

In cardiac arrest caused by toxic agents specific treatment measures such as antidotes, decontamination and enhanced elimination should be considered. There are several specific precautions regarding resuscitation of intoxicated patients. Personal safety is most important. A careful approach to the patient must be considered in suspicious cases, unexpected cardiac arrests or in cases with more than one casualty. Mouth-to-mouth ventilation in the presence of chemicals such as cyanide, hydrogen sulphide, corrosives and organophosphates should be avoided as it might lead to poisoning of the rescuer.

The toxin(s) need to be identified as early as possible. Relatives, friends and ambulance crews can provide useful information. Examination of the patient may reveal diagnostic clues such as odours, needle marks, pupil abnormalities and signs of corrosion in the mouth.

All reversible causes of cardiac arrest should be excluded in cardiac arrest patients due to toxic agents. Life-threatening tachyarrhythmias can be caused by toxic agents directly or indirectly, e.g. due to electrolyte abnormalities. Hypo- or hyperthermia may occur during drug overdose as well. It might be necessary to continue resuscitation for a prolonged time period, particularly in young patients, as the poison may be metabolised or excreted during extended resuscitation measures.

There are a number of alternative approaches which may be effective in severely poisoned patients including higher doses of medication than in standard protocols (e.g. high-dose insulin euglycemia);[289] non-standard drug therapies (e.g. IV lipid emulsion);[290–292] ECPR;[293,294] and haemodialysis.[294]

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

Helpful websites:

- https://pubchem.ncbi.nlm.nih.gov/.
- https://chem.nlm.nih.gov/chemidplus/chemidlite.jsp.

Specific therapeutic measures

There are a few specific therapeutic measures for poisoning – decontamination, enhancing elimination and the use of specific antidotes. Many of these interventions should only be used based on expert advice. For up-to-date guidance in severe or uncommon poisonings, seek advice from a poisons centre.

Decontamination

Decontamination is a process of removal of the toxic agent from the body dependent on the route of exposure:

For dermal exposure clothing should be removed and copious irrigation with water for at least 15 min should be commenced. Neutralising chemical substances should not be used, as these might cause further tissue damage.

For ocular lesions immediate copious irrigation with normal saline for at least 30 min in the most severe cases should be commenced. Topical medication should not be applied before an expert evaluation has taken place.[52]

Gastric lavage should not be performed routinely, if at all, for the treatment of poisoned patients. In the rare instances in which gastric lavage is indicated, it should only be performed by individuals with proper training and expertise. It is only indicated in case of assumption of a potentially lethal amount of toxic agent and only within one hour of ingestion.[295] Gastric lavage may be associated with life-threatening complications, e.g. aspiration pneumonitis, aspiration pneumonia, oesophageal or gastric perforation, fluid and electrolyte imbalances or arrythmias. It is contraindicated if the airway is not protected and if a hydrocarbon with a high risk of aspiration potential or a corrosive substance has been ingested.[296]

The preferred method of gastrointestinal decontamination in patients with an intact or protected airway is activated charcoal, but the evidence that active charcoal improves outcome is limited.[287] It is most effective if given within 1 h of the time of ingestion.[297] The recommended dose is 0.5–1 g kg^{-1} both in paediatric and adult patients. Activated charcoal does not bind lithium, heavy metals and toxic alcohols. Most common side effects are vomiting and constipation. It is contraindicated if the airway is not protected, in case of ingestion of corrosive, irritant agents or hydrocarbons with a high potential of aspiration.

Whole bowel irrigation (WBI) can be considered for potentially toxic ingestions of sustained-release or enteric-coated drugs particularly for those patients presenting later than 2 h after drug ingestion when activated charcoal is less effective. WBI can be considered for patients who have ingested substantial amounts of iron, lithium, or potassium as the morbidity is high and there is a lack of other potentially effective options for gastrointestinal decontamination. WBI can be considered for removal of ingested packets of illicit drugs in "body packers." However, controlled data documenting improvement in clinical outcome after WBI are lacking. WBI is contraindicated in patients with bowel obstruction, perforation, or ileus, and in patients with hemodynamic instability or compromised unprotected airways. It should be used cautiously in debilitated patients and in unstable patients. The concurrent administration of activated charcoal and WBI might decrease the effectiveness of the charcoal.[298]

Routine administration of laxatives (cathartics) must be avoided, emetics should not be used as well (e.g. ipecac syrup).[299]

Enhanced elimination

The aim of this technique is to accelerate elimination of substances that have already been absorbed. Non-invasive strategies include multiple-dose activated charcoal (MDAC) and forced diuresis (with) urine alkalisation. Invasive techniques include haemodialysis, hemofiltration, plasmapheresis.

MDAC administered over several hours can increase elimination for certain drugs,[300,301] especially in high doses of toxic agents, drugs that tend to form bezoars, agents that slow the GI motility, sustained release or toxic agents with elevated biliary excretion and entero-hepatic circulation. The initial dose is 1 g kg^{-1}, followed by 0.25–0.5 g kg^{-1} every 2–4 h.

Forced diuresis is a very useful technique for drugs with elevated renal excretion, low protein binding and low volume of distribution. Indications are poisonings from amanita phalloides (death cap fungus), phenobarbital, salicylates and ethylene glycol.

Urinary alkalinisation (urine pH $\geq$7.5) involves an IV sodium bicarbonate infusion.[302] It is most commonly performed in patients with salicylate intoxication who do not need dialysis. Consider urine alkalinisation in addition with forced diuresis (3–6 ml kg^{-1} h^{-1}) in severe poisoning by phenobarbital and herbicides. Hypokalaemia is the most common complication.[303]

Haemodialysis removes drugs or metabolites with low molecular weight, low protein binding, small volumes of distribution and high water-solubility. In hypotension, use continuous veno-venous hemofiltration (CVVH) or continuous veno-venous haemodialysis (CVVHD). Indications for haemodialysis include: worsening despite standard treatment; lethal blood levels of a toxic agents or certain history of lethal dose; patients with alterations of normal excretion systems or kidney injury secondary to the intoxication; poisonings with substances that produce highly toxic metabolites. Main indications for haemodialysis are poisonings with ethylene glycol, methanol, lithium, barbiturates, salicylates, paraquat.[304]

Antidotes

Antidotes interact with the toxic agent by means of different mechanisms, they make it less effective and decrease or stops its biological effects. Although basic supportive care remains the key treatment of poisonings, antidotes can be sometimes life-saving or may reduce morbidity as well as medical and other resources required in the care of a patient. In areas remote from hospital or in developing countries where facilities for supportive care are often limited and transport to treatment centres may take a long time, the availability of antidotes is even more essential.[305,306] Nano-antidotes have shown efficacy in proof-of-concept studies, but require clinical validation (Table 6).[307]

Specific toxic agents

Special settings

Healthcare facilities

Cardiac arrest in the operating room (OR)

Cardiac arrest in the operating room (OR) is a rare but a potentially life-limiting event with a mortality rate of more than 50%.[392,393] In the event of cardiac arrest in the OR, follow the ALS algorithmwith appropriate

Table 6 – Specific toxic agents.

Drugs	First Line	Consider	Avoid
Cardiovascular and neurological medication			
Digoxin	Lidocaine – ventricular arrhythmias	Digoxin-Fab 80 mg, repeated as required[308,309]	Calcium channel blockers Class 1a antiarrhythmic drugs
Calcium channel blockers	IV calcium 1–2 g every 10–20 min/ 0.02–0.04 g/kg/h High-dose insulin euglycemic therapy Catecholamines Atropine[289,310–323]	Pacing VA-ECMO Intravenous lipid emulsion[324,325]	
Beta-blockers	High-dose insulin euglycemic therapy Catecholamines[326–328]	Glucagon Intravenous lipid emulsion phosphodiesterase inhibitors[329–332]	
Tricyclic antidepressants	Sodium bicarbonate - broad complex ventricular arrhythmias: 1-2 mmol kg^{-1}, target pH 7.45–7.55[333–339]	Intravenous lipid emulsion[290]	
Neuroleptics	Sodium bicarbonate - broad complex ventricular arrhythmias: 1–2 mmol kg^{-1} Dantrolene, Bromocriptine - neuroleptic malignant syndrome[340]		Dopamine Adrenaline Dobutamine[341]
Anticonvulsants	Sodium bicarbonate - broad complex ventricular arrhythmias: 1–2 mmol kg^{-1} Dantrolene Carnitine, Naloxone – valproic acid[342]	Haemodialysis ECLS – carbamazepine[343,344]	
Benzodiazepines		Flumazenil[345,346]	
Local anaesthetics	Intravenous lipid emulsion: 20% lipid emulsion, 1.5 ml kg^{-1} over 1 min followed by an infusion at 0.25 ml kg^{-1} min^{-1} for up to 60 min. 2 bolus repetitions, max cumulative dose 12 ml kg^{-1}.[290,347–353]		
Drugs of abuse			
Opioids	Naloxone 0.4–2 mg, repeat every 2–3 min (strong recommendation, very low- quality evidence)[354,355]		
Cocaine	Benzodiazepines - seizure control[356,357]	Alpha-blockers, calcium channel blockers, nitro-glycerine – hypertension[358–361]	Beta-blockers not as first line management[362–364]
Amphetamines	Benzodiazepines - seizure control	Cyproheptadine, chlorpromazine, ziprasidone – serotoninergic syndrome[365–368]	
Systemic asphyxiants			
Cyanide	Hydroxycobolamin 70 mg/kg/1–3 min[369,370]	Sodium thiosulfate[371]	Amyl nitrite, sodium nitrite – avoid if smoke inhalation[372,373]
Carbon monoxide	Oxygen	Hyperbaric oxygen[374–379]	
Hydrogen sulphide	Nitrite Hydroxycobolamin[380–384]		
Local asphyxiants (Irritant gases)		*N*-Acetylcysteine – phosgene[385]	
Organic solvents and halogenated hydrocarbons		Beta-blockers – arrhythmias *N*-Acetylcysteine – hepatotoxicity[386,387]	
Biotoxins			
Botulinum toxin	Antitoxin[388,389]		
Viper envenomation	Antivenom	Polyvalent immune Fab[390]	
Marine biotoxins	Antivenom, magnesium – jellyfish[391]		

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

modifications. The incidence of perioperative cardiac arrest is higher in children, especially newborns and infants as well as in older patients.[394]

Strong predictors of Intraoperative Cardiac Arrest (IOCA) are associated with higher American Society of Anesthesiologists (ASA) physical status, current sepsis, urgent/emergency case, anaesthetic technique and age.[392,395] In addition, there are also several factors such as hypoxia, acute blood loss with shock, pulmonary embolism, myocardial infarction, arrhythmia or electrolyte disturbances, which all can be the cause or confounding factors in an intraoperative cardiac arrest.[392,396,397] Additional risk factors for intraoperative cardiac arrest for patients in prone position, such as major spinal surgery, can include air embolism, wound irrigation with hydrogen peroxide and occluded venous return.

This section is based on recent European Society of Anaesthesiology and Intensive Care (ESAIC) and ERC guideline process (27 PICO questions; 28,221 titles screened/452 publications selected).

Early recognition of intraoperative cardiac arrest

In many cases of intraoperative cardiac arrest, physiological deterioration is gradual and the cause of the cardiac arrest is known and hence the arrest anticipated.[398] In those where this is not the case, follow the ALS algorithm and and prioritise the reversible causes. If the patient deteriorates, call for help immediately. Inform the perioperative team of the deterioration and a possible impending cardiac arrest. Ensure that sufficient skilled assistance is present.

High-risk patients will often have invasive blood pressure monitoring (IABP), which is invaluable for recognition and treatment of cardiac arrest. If cardiac arrest is a strong possibility, a defibrillator should be on standby. Apply self-adhesive defibrillation electrodes before induction of anaesthesia, ensure adequate venous access, and prepare resuscitation drugs and fluids. Use fluid warmers and forced air warmers to limit perioperative hypothermia and monitor the patient's temperature.

Chest compressions and defibrillation

In adult IOCA patients with shockable rhythm, immediate defibrillation should be performed. A high incidence of reversible causes is to be expected. This could be hypoxemia due to airway problems, bronchospasm or equipment failure, intoxications caused by drug error, hypovolemia due to blood loss, anaphylactic reactions, thromboembolism including air embolism and tension pneumothorax or even cardiac tamponade after central line insertion.

The majority of events is covered by standard ALS measures. However, closed chest compressions are not very effective in hypovolemia, cardiac tamponade or tension pneumothorax (see corresponding section). Therefore closed chest compressions should not delay addressing these particular reversible causes. To optimise closed chest compressions the position and height of the operating table or trolley should be adjusted. CPR is ideally carried out in the supine position of the patient, but is possible in patients in prone position as well.[399,400] Open cardiac compressions should be considered early as an effective alternative to closed chest compressions in the operating room environment.[398]

Airway management

Advanced airway management (if not already undertaken) and ventilation with 100% oxygen should be performed as soon as possible.[397]

Reversible causes

Hypovolaemia

Depending on the suspected cause, initiate volume therapy with warmed blood products and/or crystalloids, in order to rapidly restore intravascular volume. At the same time, initiate immediate haemorrhage control, e.g. surgery, endoscopy, endovascular techniques.[27] Chest compressions are only of use if the circulating volume is replaced simultaneously. In the initial stages of resuscitation crystalloid solutions are acceptable. In case of massive blood loss immediate transfusion of blood products is required. A focused ultrasound examination can help to confirm the course of cardiac arrest and target resuscitative interventions. (see hypovolaemia section).

Anaphylaxis

The incidence of immune-mediated anaphylaxis during anaesthesia ranges from 1 in 10,000 to 1 in 20,000. Neuromuscular blocking drugs are the commonest cause, being associated with 60% of cases. The associated morbidity and mortality are high, particularly if there are delays in the diagnosis and management.[401] Initial management of anaphylaxis starts with removal of the allergen if possible and then follows the ABCDE approach and the management principles outlined in the chapter on anaphylaxis. Adrenaline is the most effective drug in anaphylaxis and should be given as early as possible. In contrast to alternative anaphylaxis scenarios it might be appropriate for anaesthetists to give adrenaline by the IV route. Repeated doses of adrenaline maybe necessary (see anaphylaxis section below).

Systemic toxicity of local anaesthetic

Cardiac arrest is a rare but well recognized complication of local anaesthetic (LA) overdose, especially following inadvertent intravascular injection. Direct action of the LA on cardiac myocytes causes cardiovascular collapse, usually within 1–5 min of injection, but onset may range from 30 s to as long as 60 min.[402] Significant hypotension, dysrhythmias, and seizures are typical manifestations, but the diagnosis maybe one of exclusion.[292] Intravenous lipid therapy has been used as a rescue therapy to treat cardio-vascular collapse and cardiac arrest, but its efficacy is controversial.[403] In the absence of documented harm, guidelines recommend that 20% lipid emulsion should be available for use wherever patients receive large doses of LA (e.g. operating rooms, labour wards and the emergency department).[404] Stop injecting the LA and call for help. Secure and maintain the airway and, if necessary, intubate the trachea. Give 100% oxygen and ensure adequate ventilation (hyperventilation may help by increasing plasma pH in the presence of metabolic acidosis). Control seizures using a benzodiazepine, thiopentone or propofol. Give an initial IV bolus injection of 20% lipid emulsion at 1.5 ml kg^{-1} over 1 min and then start an infusion at 15 ml kg^{-1} h^{-1}. 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-min intervals until ROSC has been achieved. Do not exceed a maximum cumulative dose of 12 ml kg^{-1}.[405,406] If the patient does not respond to treatment ECPR should be considered.

Crew resource management

Every resuscitation event should have a designated team leader who directs and coordinates all staff and the components of the resuscitation, with a central focus on minimising no-flow times and addressing the reversible causes simultaneously. Operative surgery needs to be stopped unless it is addressing a reversible cause of the cardiac arrest. Patient access and resuscitation tasks may necessitate covering the surgical field and withdrawing the surgical team from the patient. Team tasks should be prioritised, good quality BLS should be ensured, relevant reversible causes should be identified and non-priority tasks avoided. If the patient is not responding to resuscitative efforts (i.e. E_TCO_2 <2.7 kPa/20 mmHg), the quality of CPR needs to be improved.[407]

Successful management of intraoperative cardiac arrest requires not only individual technical skills and a well-organized team response, but also an institutional safety culture embedded in everyday practice through continuous education, training and multidisciplinary cooperation. Corresponding institutional protocols (e.g. massive transfusion protocols) and checklists help to optimise the response to cardiac arrest in the operating room environment.

Post resuscitation care

There is lack of evidence to support the use of immediate hypothermia versus no hypothermia after adult intraoperative cardiac with only one single case report showed complete neurological recovery and data suggest improved neurological outcome.[408] Targeted temperature management should be applied according to general post resuscitation care.

Cardiac surgery

The incidence of cardiac arrest following cardiac surgery has been reported around 2–5% in recent series, with higher survival rates (around 50%) compared to other scenarios.[409–412] This is largely justified by the fact that many causes are reversible; Major causes of cardiac arrest in this setting include ventricular fibrillation (VF), accounting for up to 50% of cases, followed by cardiac tamponade and major bleeding, which often present as PEA.

Evidence based recommendations for the management of cardiac arrest following cardiac surgery derive from the 2019 and 2018 ILCOR CoSTR documents,[103,413] the European Association for Cardio-Thoracic Surgery (EACTS) guidelines [414,415] and the Society of Thoracic Surgeons (STS) expert consensus document for the resuscitation of patients who arrest after cardiac surgery.[416] Additional focused literature search was conducted for evidence update and consensus was reached within the writing group to establish recommendations.

Prevent and be prepared

Ensure adequate training of the staff in resuscitation technical skills and ALS (Figs. 11 and S3).

Staff involved in the care of post-operative cardiac patients should receive adequate training with periodic refreshers. This should comprise resuscitation technical skills and ALS, including training to perform an emergency resternotomy. Roles should be previously allocated to staff in the intensive care unit (ICU) to favour coordination in case this procedure is required.[417]

Ensure availability and well-functioning of emergency equipment

All emergency equipment should be located, adequately marked and periodically checked, including small resternotomy sets containing only the essential elements to open the chest.[415,416]

Use safety checklists

First introduced in the surgical environment by the World Health Organization, safety checklists have proven to reduce complications and mortality of non-cardiac surgery, and should be implemented.[418] Specific checklists developed for cardiothoracic surgery, including checks on preparations for bleeding, perfusions and ICU preparations should be considered to enhance prevention.[414]

Detect cardiac arrest and activate cardiac arrest protocol

Identify and manage deterioration in the postoperative cardiac patient

Early signs of deterioration can be identified in the monitored postoperative patient after careful examination. Hypotension is a common observation to several different complications (Table 7).[419–421] Echocardiography should be performed in case of haemodynamic instability, considering transoesophageal application for more precise diagnosis.[422] Continuous ECG monitoring allows early identification of arrhythmias; supraventricular tachycardias are the most frequent in this setting.[423]

Confirm cardiac arrest by clinical signs and pulseless pressure waveforms

Cardiac arrest can be detected by checking rhythm in the ECG monitor, identifying absent circulation by clinical examination and monitoring of vital signs, including pulseless pressure waveforms (arterial, central venous and pulmonary artery pressures, and pulse oximetry) and rapid decrease in the end-tidal capnography.[415,416]

Shout for help and activate cardiac arrest protocol

Once recognised, immediately getting help and activating the cardiac arrest protocol are mandatory.

Resuscitate and treat possible causes

Modifications to the standard ALS algorithm include immediate correction of reversible causes and emergent resternotomy if this is not successful.[415,416]

Restore pulsatile cardiac rhythm

In patients with VF/pVT defibrillation of up to three stacked shocks should be prioritised and justifies delaying external chest compressions for as long as one minute.[424,425] If these fail, immediate resternotomy is advised.[425] In case of asystole or extreme bradycardia, epicardial pacing (DDD mode at 80–100 beats min^{-1} and at maximum output voltages) or transcutaneous pacing should be attempted for one minute before initiating chest compressions. PEA should trigger immediate external chest compressions, searching for reversible causes and preparing for early resternotomy. In the presence of a pulseless stimulated rhythm, pacing should be paused to eventually unmask underlying VF and, if indicated, a defibrillation should be provided.[415,416]

Compressions and ventilations

If ROSC is not achieved following defibrillation or pacing, or in case of PEA, compressions and ventilations should be initiated while preparing for emergency resternotomy. External compressions should be performed at 100–120 beats min^{-1}, aimed to reach a systolic blood pressure >60 mmHg; failure to attain this value despite adequate performance may indicate tamponade or severe haemorrhage, requiring emergency resternotomy.[415,416] Compared with external compressions, internal cardiac massage provides better coronary and systemic perfusion pressure and this sole fact may justify chest reopening.[426,427] Airway management in this setting

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

Table 7 – Common causes of patient deterioration after cardiac surgery and management.

Haemorrhage • "Medical" bleeding: post-operative coagulopathy • "Surgical" bleeding: operative trauma	• Correct hypothermia and hypertension, avoid haemodilution • Consider blood products transfusion and use of haemostatic agents guided by haematological tests • Check drains to detect 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

follows the usual indications for ALS.[101] In mechanically ventilated patients the position and patency of the tracheal tube should be checked, inspiratory oxygen increased at 100% and positive end-expiratory pressure removed. If a tension pneumothorax is suspected, emergent decompression is advocated.[415,416]

Drugs during resuscitation

As a general principle previous infusion other than needed for resuscitation should be stopped, with the possible exception of sedatives. Amiodarone (300 mg) or lidocaine (100 mg) may be administered intravenously after three failed shocks to treat VF/pVT, although this recommendation is extrapolated from research in OHCA (weak recommendation, low certainty evidence).[413,416] Conversely, using adrenaline (1 mg) shortly after cardiac surgery is controversial. The European Association of Cardio-Thoracic Surgery (EACTS) and the Society of Thoracic Surgeons (STS) discourage the routine use of adrenaline[415,416] based on the concern that intense hypertension induced by adrenaline may cause bleeding or disruption of surgical anastomoses after ROSC, although lower doses (50–300 mcg boluses) may be considered in peri-arrest situations (expert consensus).[416,421,428] The 2019 ILCOR CoSTR Summary included a specific section on the role of vasopressor drugs for adult cardiac arrest, based on an ILCOR-commissioned systematic review and meta-analysis.[238,429] The recommendation to administer 1 mg adrenaline during CPR was maintained (strong recommendation, low-moderate certainty of evidence), based on increased ROSC and survival at hospital discharge. However, limited RCT evidence on its use for IHCA was acknowledged, with recommendations being extended from the OHCA setting. In summary, although there is insufficient evidence to establish recommendations on the use of adrenaline in the postoperative cardiac patient, considering the potential risks of adrenaline-induced intense hypertension in this particular setting, 1 mg adrenaline should be avoided in patients who collapse shortly after cardiac surgery, if defibrillation and early resternotomy are likely to revert cardiac arrest. However, lower doses may be considered in peri-arrest situations.[415,416,428]

Early resternotomy

Refractory cardiac arrest requires performing resternotomy within 5 min, in order to perform internal massage or defibrillation and eventually correct underlying causes. This has shown to be a safe procedure in the ICU,[430] leading to significantly higher survival rates, especially if performed with minimum delay and in the presence of surgically repairable problem on reopening.[431] Resternotomy should be conceived as part of the resuscitation protocol of postoperative cardiac patients until at least day 10 after surgery.[416]

Circulatory support devices

In patients supported by intra-aortic balloon pump who present cardiac arrest, the device may contribute to improve coronary and brain perfusion if coordinated with cardiac massage (1:1 ratio, with maximal amplification). The ECG trigger of the balloon is not reliable during resuscitation and should be switched to pressure trigger mode, or to internal mode at 100 beats min^{-1} if massage is interrupted for a significant interval. ECPR may be considered if resternotomy fails to revert cardiac arrest or as an alternative for patients undergoing minimally invasive cardiac surgery (i.e. thoracotomy) or who arrest >10 days after initial sternotomy.[416] However, there is limited data addressing this specific scenario, since most studies have studied its usefulness to treat cardiogenic shock or have focused on paediatric populations. A small series of twenty-four adult patients who received ECPR support for postoperative cardiac arrest reported overall successful weaning from extracorporeal membrane oxygenation (ECMO) in sixteen patients (66.7%), eight of whom survival to hospital discharge (33.3%) with most of patients dying because of multiple organ failure.[432]

Catheterisation laboratory

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

Updated robust data on the global incidence of cardiac arrest in the catheterisation laboratory are lacking; registries mostly refer to PCI and show incidence rates highly dependent on patient pre-procedural risk.[433,434]

Evidence based recommendations derive from the ILCOR CoSTR documents[238,435,436] and ILCOR systematic reviews,[273] expert consensus statements from the European Association of Percutaneous Cardiovascular Interventions (EAPCI),[437] the Society for Cardiovascular Angiography and Interventions (SCAI),[438] and the International ECMO Network and The Extracorporeal Life Support Organization (ELSO),[439] plus focused literature search for evidence update. Where insufficient quality of evidence was obtained, recommendations were established by consensus expert within the writing group.

Prevent and be prepared

Ensure adequate training of the staff in technical skills and ALS

Staff working in the catheterisation laboratory should be adequately trained in resuscitation technical skills and ALS, including team and leadership training (Figs. 12 and S1).[435] Protocols for specific emergency procedures (e.g. initiation of mechanical CPR, emergency transcutaneous or transvenous pacing, pericardiocentesis, ventricular assist devices) should be established. On-site emergency drills should be considered to facilitate implementation and familiarisation of the staff.[438]

Ensure availability and well-functioning of emergency equipment

Emergency equipment should be clearly identified and the staff should be aware of its location to minimise delays if needed. Proper functioning should be regularly tested.

Use safety checklists

The use of safety checklists to minimise human factors should be encouraged,[437,438,440] since their use has been associated with fewer procedural complications and improved team communication.[441]

Detect cardiac arrest and activate cardiac arrest protocol

Check patient's status and monitored vital signs periodically

Continuous monitoring of vital signs (invasive blood pressure, heart rate and rhythm, pulse oximetry, capnography) facilitates early recognition and management of complications to prevent further deterioration and should be periodically checked. For example, high-degree atrioventricular block can occur during PCI, septal alcohol ablation or transaortic valve replacement (TAVR); chest pain, haemodynamic instability and ST-elevation in the ECG may be an alert for acute stent thrombosis during PCI or coronary ostium occlusion during TAVR; sudden hypotension requires ruling out pericardial tamponade (due to coronary perforation, atrial/ ventricular wall perforation or annulus rupture during balloon valvotomy or TAVR) or hypovolaemia in case of vascular complications. Defibrillation pads should be attached to at least all STEMI patients and considered in cases of complex PCI or high-risk patients.[438]

Consider cardiac echocardiography in case of haemodynamic instability or suspected complication

Cardiac echocardiography can help to detect complications and should rapidly be performed in case of haemodynamic instability. In procedures performed under transoesophageal echocardiography monitoring, this may provide better quality imaging for quicker and more precise identification of complications.[422]

Shout for help and activate cardiac arrest protocol

Once cardiac arrest is confirmed, the resuscitation team should be called immediately. Even if staff in the catheterisation laboratory should initiate resuscitation without delay, additional support may be required to allow on-going resuscitation while specific procedures to treat possible causes of arrest are performed (i.e. PCI, pericardiocentesis, invasive pacing). Leadership and roles during resuscitation should be clearly identified especially if new rescuers take over, to ensure coordinated and effective performance.

Resuscitate and treat possible causes

Resuscitate according to the modified ALS algorithm

Cardiac arrest in the catheterisation laboratory should be managed according to the ALS protocol, with some modifications.[101] In the presence of monitored VF/pVT, consider immediate defibrillation with up to three stacked shocks before starting chest compressions. In case of asystole/PEA, CPR and drugs should be administered according to the ALS protocol.

Check for reversible causes, including the use of echocardiography and angiography.

Identifying reversible causes is especially critical in non-shockable rhythms, for which cardiac echocardiography should be performed, and angiography considered if appropriate. Point of care ultrasonography (POCUS) can help to identify reversible causes of cardiac arrest, although attention should be paid to minimising pauses in chest compression.[442–444] In this regard, transoesophageal echocardiography may be helpful to enable continuous, higher-quality imaging assessment without interfering with resuscitation efforts.[445,446]

Consider mechanical compressions and percutaneous circulatory support devices

A Cochrane review including 11 trials comparing mechanical CPR versus manual chest compressions during CPR for adult patients suffering IHCA or OHCA arrest failed to prove superiority of mechanical over conventional CPR. However, the role of mechanical CPR was recognised as a reasonable alternative in settings where high-quality chest compressions are not possible or dangerous for the provider.[272] Delivering quality manual CPR in the catheterisation laboratory may be challenging due to the presence of the X-ray tube, and may expose the rescuer harmful radiation; for this reason, mechanical CPR should be considered.

Percutaneous ventricular assist devices such as intra-aortic balloon pump, Impella® [447] or TandemHeart® may provide circulatory support while performing rescue procedures during cardiac arrest, although their use in this setting has not been extensively evaluated. Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) offers both circulatory and pulmonary support and may be used in cardiac arrest (extracorporeal life support: ECPR), but there is insufficient evidence to systematically recommend such strategy.[238] A recent systematic review comparing ECPR to manual or mechanical CPR reported positive outcomes of ECPR in seven studies assessing their use for adult IHCA, although these were handicapped by their observational nature and limited internal validity.[273] Other smaller series have reported successful use of ECPR for in-hospital refractory cardiac arrest due to acute myocardial infarction[448] or complicating PCI or TAVR.[449] Should ECPR be considered, rapid initiation rather than waiting for complete failure of conventional measures is recommended,[439,450] since shorter conventional CPR (low-flow) time is a key factor for success.[451] Until randomised trials provide more consistent evidence, decisions to use ECPR or other ventricular assist devices should be adapted to the case, availability and expertise of the team.

Dialysis unit

Patients receiving long-term HD are one of the highest risk groups for out-of-hospital cardiac arrest (OHCA), which includes events occurring within dialysis clinics. OHCA occurs 20 times more frequently in dialysis patients compared with the general population.[452] Cardiac arrests occurring within a dialysis clinic are predominantly witnessed events and may occur before, during or after dialysis treatment. Studies investigating the timing of cardiac arrest in relation to dialysis have reported that 70–80% of cardiac arrests occurred during HD treatment.[453–455] Several risk factors for cardiac arrest in patients receiving long-term HD have been postulated including hyperkalaemia, excessive volume shifts during dialysis, the 2-day inter-dialytic interval, low potassium dialysate fluid, cardiac disease, and non-compliance with diet and dialysis regimen.[456–461] Although HD patients are susceptible to cardiac arrest in the first 12 h from the start of the HD session,[456] the highest risk period is the latter end of the 2-day inter-dialytic interval (e.g. weekend break) as potassium level rises and fluid accumulates.[456,458] Historically, the outcome of IHCA in HD patients was deemed to be poor,[462] but this may in part relate to the resuscitation strategy and perceived futility. Previous studies have shown a lower survival after IHCA in dialysis patients compared with the general population.[463,464] However, a recent study has shown a higher incidence of ROSC (69% compared with 62%), similar survival to hospital discharge (23% compared with 22%), and a slightly higher frequency of favourable neurological status (17% compared with 16%) in HD patients compared with non-dialysis patients.[458] Shortfalls in resuscitation practice have been found in both OHCA and IHCA with respect to initiation of CPR and timely first defibrillation for a shockable rhythm in the dialysis patient.[458] There are also some special considerations in the approach to resuscitation in the setting of a dialysis clinic, particularly if the event occurs during dialysis.[116]

Recommendations are based on a scoping review and the recently updated the UK Renal Association Hyperkalaemia Guidelines.[116] The scoping review screened multiple databases – PubMed (1960–2019), Ovid MEDLINE (1946–2019), EMBASE (1974–2019), Science Direct (1995–2019), The Cochrane Library (1995–2019), Web of Knowledge (2001–2019) for all human studies published in English pertaining to the cardiac arrest and haemodialysis. Websites searches included National Institute for Health and Care Excellence (NICE), Scottish Medicines Consortium (SMC), Healthcare Improvement Scotland, Medicines and Healthcare products Regulatory Agency (MHRA) and European Medicines Agency (EMA). No randomised controlled trials were identified. Evidence was drawn from observational studies. The UK Renal Association Hyperkalaemia guideline (2019) utilised this scoping review and expert consensus opinion to develop recommendations for the treatment of cardiac arrest in dialysis units.

Prevention of cardiac arrest in dialysis patients

Hyperkalaemia and volume overload are common causes of cardiac arrest in dialysis patients, but prevention largely relies on dietary and fluid restrictions and dialysis-related factors. Although the delivery of maintenance dialysis three times a week is difficult to overcome, careful dialysis prescription may reduce the risk of cardiac arrest.[453,457,465]

Treatment of cardiac arrest

Initial steps

Resuscitation should be started following the standard ALS algorithm. A trained dialysis nurse should be assigned to operate the HD machine. The HD machine should be stopped and blood volume returned to the patient with a fluid bolus. As long as the HD machine is not defibrillation-proof it should be disconnected from the patient in accordance with the International Electrotechnical Committee (IEC) standards. The dialysis access should be kept open to use for drug administration.

Modifications to cardiopulmonary resuscitation

Defibrillation

Dialysis clinics are predominantly nurse-led units. An automated external defibrillator (AED) is the standard for delivery of defibrillation in HD facilities but staff training and confidence may influence the rate of nurse-led defibrillation.[466] A three-fold increase in odds to hospital discharge with favourable neurological status, has been shown when CPR is initiated by dialysis staff rather than awaiting the arrival of emergency services. Although nurse-led AED placement occurred in only 52.3% of patients, this study also showed a trend towards improved survival with staff-initiated defibrillation in patients with a shockable rhythm.[457] Given the higher chance of survival with shockable rhythm, steps are required to avoid delay in defibrillation in dialysis units.

Vascular access

Use dialysis access in life-threatening situations and cardiac arrest.

Potential reversible causes

All of the standard reversible causes (4 Hs and 4Ts) apply to dialysis patients. Electrolyte imbalances and fluid shifts during dialysis are common causes. For management of hyperkalaemic cardiac arrest, refer to the hyperkalaemia section of this chapter.

Post resuscitation care

Dialysis may be required in the early post resuscitation period guided by fluid status and serum biochemistry. Patient transfer to an area with dialysis facilities (i.e. intensive care unit or renal high dependency unit) is essential.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

Dentistry

Medical emergencies in a dental office include a variety of situations ranging from psychosomatic disorders precipitated by fear and anxiety to life-threatening situations. The most frequent medical emergencies include vasovagal (pre-) syncope, orthostatic hypotension, hypertensive crisis, hyperventilation, seizures, moderate allergic reactions, hypoglycaemia, and angina.[467,468] Life-threatening emergencies commonly arise from myocardial infarction, seizures or exacerbation of asthma. Cardiac arrest in primary dental practice is a rare event with an incidence of 0.002−0.011 cases per dentist per year.[468,469]

A PubMed scoping review was performed on March 27th, 2020 using the keywords "dentistry" OR "dental surgery" AND "cardiac arrest or heart arrest" OR "resuscitation or cardiopulmonary resuscitation" in the last 5 years (n = 271). There were neither RCTs nor systematic reviews identified on this topic. Thus, recommendations are based on the evidence already informing the ERC guidelines 2015. Recommendation on modification to chest compression is based on some case reports that described effectiveness of chest compression in a patient left on a dental chair.[470,471] Small simulation studies comparing the effectiveness of CPR on a dental chair and on the floor reported either lower or equivalent CPR quality.[472−475] Recent simulation study verified the efficacy of a stool as a stabilizer in different types of dental chairs and confirmed feasibility of ERC guidelines 2015.[84,476] Expert consensus was provided by the Resuscitation Council UK in May 2020 as a part of Quality Standards for primary dental care. (https://www.resus.org.uk/library/quality-standards-cpr/quality-standards-acute-care).

Causes of cardiac arrest

Causes of cardiac arrest usually relate to pre-existing comorbidities, complications of the procedure or allergic reactions.

Airway and breathing

Dental procedures may cause loss of airway patency related to the primary pathology or complications of the procedure (e.g. bleeding, secretions, tissue swelling). The occurrence of choking is low, with a reported incidence of 0.07−0.09 cases per dentist per year.[468,469] The addition of sedation is a contributory risk in these cases, although provision of dental treatment under both local anaesthesia and sedation has an excellent safety record.[477,478]

Circulation

Although life-threatening anaphylaxis is rare, it is a documented cause of death during dental procedures. In additional to chlorhexidine mouthwash, other common causes may include local anaesthetic agents and latex. True anaphylaxis occurs in only 0.004−0.013 cases per dentist per year, while coronary symptoms (angina or myocardial infarction) are reported more frequently: 0.15−0.18 cases per year.[468,469]

Treatment of cardiac arrest

The patient's mouth should be checked and all solid materials from the oral cavity (e.g. retractor, suction tube, tampons etc.) removed. Prevention of foreign body airway obstruction should precede positioning.

The dental chair should be reclined into a fully horizontal position with a stool placed under the backrest for its stabilization.[470,473,476] If reduced venous return or vasodilation has caused loss of consciousness (e.g. vasovagal syncope, orthostatic hypotension), cardiac output might be restored with no need for CPR.

If breathing is not normal following opening of the airway, assume a cardiac arrest until proven otherwise. Chest compressions should be started immediately with the patient lying flat on the chair. Moving the patient down onto the floor should be considered once that could be achieved with the help of sufficient personnel (injury prevention), when space allows, and without delaying CPR.[470,471] If access to either side of chest is limited over-head CPR should be considered.[479,480]

Equipment and training

Specific resuscitation equipment, including suction, oropharyngeal airway, self-inflating bag with face masks, oxygen, emergency drug kit, should be available immediately. This equipment list should be standardised on the national level (https://www.resus.org.uk/library/quality-standards-cpr/quality-standards-acute-care).[481] All dental practices delivering clinical care should have immediate access to an AED, with all staff trained in its use. The role of early defibrillation should be emphasized to increase availability of AEDs in dental offices, which is still unsatisfactory, ranging from a reported 1.7−2.6% in Europe,[467,482] to 11% in the US.[483]

Medical professionals working in a dental office have an obligation to provide CPR in case of cardiac arrest, and to ensure that staff are trained and updated regularly. (https://www.resus.org.uk/library/quality-standards-cpr/quality-standards-acute-care).[484] There is a public expectation that dental practitioners and all other dental care professionals should be competent in treating cardiorespiratory arrest. However, only 0.2−0.3% dentists have a real experience,[467,468,485] and CPR training varies significantly between countries.[468,469,485,486] Maintaining knowledge and competence to deal with medical emergencies must be an important part of training of the dentists.

Special settings

Inflight cardiac arrest

According to prognosis provided before the COVID pandemic the number of passengers travelling by plane will rise to 9 billion in the year 2040 (Association International Air Transport (2016) http://www.iata.org/pressroom/facts_figures/fact_sheets/Documents/fact-sheet-industry-facts.pdf accessed 20 Jul 20). Although air travel is safe in general, passenger demographics, pre-existing medical conditions as well as the number of passengers aboard larger aircraft and flights over very long distances raise the probability of in-flight emergencies per flight.[487] Between 1 out of 14,000 and 50,000 passengers will experience acute medical problems/emergencies during a flight with cardiac arrest accounting for 0.3% of all in-flight medical emergencies.[488−490]

Early recognition and calling for help, early defibrillation, high-quality cardiopulmonary resuscitation (CPR) with minimal interruption of chest compressions and treatment of reversible causes, are the most important interventions. Especially in the remote environment of an aircraft, treatment of cardiac arrest requires adaption, modification, and supplementation to ensure the best possible outcome for patients.

Recommendations are based on one treatment guideline from the German society of aerospace medicine (DGLRM), a scoping review and expert consensus within the writing group.[491]

Modifications of ALS

Chest compressions

Bystander CPR enhances the survival rate significantly and should be performed as soon as possible. If a cardiac arrest is recognised, the cabin crew should commence resuscitation and medical professional help should be sought immediately. The easiest and most effective way is an on-board announcement. Ideally, CPR is performed by at least two people according to the universal CPR guidelines. Optimally, the rescuer should kneel in the leg-space in front of the aisle seats to perform chest compressions. A second rescuer can sit/knee in the aisle performing ventilation or attaching the AED.[492] [Charles 2011, 582] In situations where it is not possible to perform standard CPR according to the CPR guidelines, over-the-head (OTH) CPR may be considered as a suitable alternative.[480]

Defibrillation

Every commercial passenger aircraft should be equipped with an AED. An AED and a first-aid kit should be requested immediately from the cabin crew, since time to defibrillation is one of the most important factors for survival after cardiac arrest.[492]

Airway management

Adapted to the aircraft environment, the use of SGA may be superior for airway management in inflight resuscitation.[493] The use of capnometry/capnography might be helpful during an in-flight cardiac arrest. A (simple) qualitative capnometer should be available.[493]

Environment

Emergency equipment location should be clearly signposted. Brief information how to act in case of cardiac arrest should be printed on the seat pocket safety instruction card. A standardised medical documentation form must be available. Infrastructure and fast access to emergency equipment can reduce the potential delay to adequate therapeutic attempts, and substantially decrease no-flow-time. Since all passengers and crew members on-board are potential bystanders, all should know whom to contact in case of a cardiac arrest. Besides some general information in the pocket safety cards, the location of the emergency equipment should be mentioned in the pre-flight safety announcement.[494] Cabin crew must be trained in CPR and AED defibrillation and should be re-trained every six months.[493]

Diversion and post-resuscitation care

A typical scenario to perform an emergency diversion before ROSC is when leaving land and expecting a flight over open-water during an ongoing CPR event. Furthermore, when near an airport, an early diversion might also be useful. However, there are reasons for avoiding a diversion as long as ROSC is not achieved. For a patient presenting with a non-shockable rhythms, available evidence suggests that the time required for diversion may be futile. An aircraft diversion also includes additional risks: emergency landings, potential need to dump fuel, landing with overweight aircraft, altered flight patterns, landing in poor weather, high costs, and landing in unfamiliar conditions all increase the operational risk. If a person is found to be life extinct or CPR has been terminated, a diversion is not recommended.[493,495] If telemedicine support is available, it should be used to receive recommendations and to discuss the further course.

Helicopter emergency medical services (HEMS) and air ambulances

Air ambulance services operate either a helicopter or a fixed wing aircraft that routinely transport critically ill or injured patients directly to specialty centers. They also perform secondary transfers between hospitals. Cardiac arrest may occur during flight, both in patients being transported from an accident site (primary missions) and also in critically ill patients being transported between hospitals (secondary missions).[496,497] The extent of treatment available onboard an air ambulance varies and depends on medical, technical, and personal factors, e.g., crew competences and configuration, cabin size, and equipment. Ideally, all interventions should be performed before flight to avoid the need for unplanned treatments during flight.[492]

This section is based on an evidence update on Cardiac arrest in HEMS and air ambulance produced by recent (randomised) clinical trials or systematic reviews and focused on scoping reviews addressing the questions:

- General recommendations for Cardiac arrest in HEMS and air ambulance (30 titles screened/28 abstracts screened/7 publications selected).
- Method of Chest compressions for Cardiac arrest in HEMS and air ambulance (28 titles screened/17 abstracts screened/4 publications selected).
- Airway management for Cardiac arrest in HEMS and air ambulance (28 titles screened/20 abstracts screened/7 publications selected).

Pre-flight evaluation

When preparing the transport of a critically ill or injured patient, ensure that all necessary equipment is functioning, easily accessible, and that all necessary drugs and technical equipment are available within an arm-length during the flight. A standardized documentation form should be available in order to check pre-flight medical status.[493] Consider the patients fitness to fly. Long-haul flights lasting 12–14 h can cause a more significant adverse effect on vulnerable passengers. HEMS or aircraft cruising heights can vary between 100 and 13,000 m (300–41,000 ft) above sea level. The passenger cabin pressure is maximum equivalent to an altitude of approx. 2500 m (8000 ft).[498] Arterial oxygen partial pressure (PaO_2) can decrease from 95 mmHg to as low as 60 mmHg at the lowest level of cabin pressure.[499]

Due to high levels of stress (noise, movement etc.) and environmental alteration evaluate patient's current health status according to following:

- Recent post-surgery of a large body cavity
- Recent or current pneumothorax
- Cerebrovascular accident
- Acute psychotic mental illness
- Neonates or prematurely born infants
- Acute Myocardial infarction or unstable angina
- Recent cardiac surgery

Diagnosis

Usually patients transported by HEMS or fixed wing airplane are monitored, so asystole and shockable rhythms (VF/pVT) can be immediately identified. However, noise levels and flight helmets usually prevent acoustic alarm recognition. Recognition of PEA may be challenging, especially under sedation or general anaesthesia. Loss of consciousness, change in the ECG pattern, and loss of the pulse oximeter signal should provoke a breathing/pulse and patient check. A sudden decrease in $EtCO_2$ values in those being ventilated or loss of a waveform in those breathing spontaneously with $EtCO_2$ monitoring are excellent indicators of cardiac arrest.

Modifications to ALS

When a cardiac arrest is recognised, communication within the medical and flight teams should occur immediately. In situations where it is not possible to perform standard CPR according to the CPR guidelines, over-the-head CPR may be considered as a suitable alternative.[480] According to cabin size, chest compressions in a helicopter might not be possible. Consider installing a mechanical CPR device on the patient before flight where there is a risk of cardiac arrest.[500]

In the case of an unexpected cardiac arrest during flight, immediate landing should be considered to initiate high quality resuscitation. Use of a SGA should be considered if the patient was not ventilated previously.[493,501,502] For VT/VF during flight consider three stacked shocks.[503]

Cruise ship

Outcome from cardiac arrest on cruise ships is worse compared to the overall population, as access to healthcare facilities is more complicated and transfers can be prolonged. Furthermore, some environments overseas are harsher than urban oversea territories (e.g. cold, windy, wet, ice and snow).[504] Austere and isolated environments (such as polar regions) will not provide any possibility to return rapidly to the next harbour, so autonomous management of a cardiac arrest patient might be necessary.[505]

This section is based on an evidence update on Cardiac arrest on a cruise ship produced by recent (randomized) clinical trials or systematic reviews and focused on scoping reviews addressing the questions:

- General recommendations for Cardiac arrest on a cruise ship (16 titles screened/8 abstracts screened/6 publications selected).
- Recommendations for Post-resuscitation care for Cardiac arrest Cardiac arrest on a cruise ship (5 titles screened/5 abstracts screened/2 publications selected).

Cardiac arrest on a cruise ship

If a cardiac arrest is recognised on a cruise ship, all medical resources should be used immediately. 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, since time to defibrillation is one of the most important factors for survival after cardiac arrest.[506] Where there are insufficient numbers of crew health care professionals, an onboard announcement should be made to call for further medical professional help.[507] Depending on the resources available telemedicine should be used as early as possible.[508] Qualified medical air transportation is an option to cover long distances to medical facilities.

Cardiac arrest in sport

The incidence of sudden cardiac death (SCD) associated with sport or exercise in the general population is 0.46 per 100,000 person-years.[509] There is a wide range in the incidence of SCD in those below 35 years of age (1.0−6.4 cases per 100,000 participant-years)[510] depending on the study parameters and the incidence is markedly higher in those susceptible to cardiac arrythmias during or shortly after participating in sport.[511] In a recent study involving 18.5 million person-years the incidence of SCA of sport related cardiac arrest was 0.76 cases per 100,000 athlete years.[512] The same authors reported the highest incidence as being in runners and in soccer players for athletes in competition and for running and gym exercise during non-competition. There have been many reports of higher risks associated with strenuous sports such as racquet sports[513] downhill skiing[514] marathon running,[515] triathlon participation[516] and high-intensity sports activities such as basketball.[517]

It is important to recognise that the absolute risk of experiencing a cardiac event or SCD during physical exercise is small.[518] It is estimated that the absolute risk in male athletes during vigorous exercise is 1 SCD in 1.51 million episodes.[519] In population-based studies the incidence of SCD is reported as 0.46 per 100 ,000 person-years in France[520] and 0.31 per 100,000 person-years in Japan.[521] In a Dutch study, with a reported incidence of 2.1 per 100,000 person-years, there was a higher survival after exercise or sport related incidents than after non-exercise related incidents (42.1% compared with 17.2%).[522] However, in the United States it has been estimated that in younger age groups there is an approximately 4.5-fold higher risk of sudden cardiac arrest (SCA) or SCD in competing athletes when compared with recreational athletes of a similar age.[509]

Recommendations in this section are based on a literature review for an evidence update, including one recent AHA scientific statement,[518] hand searching of specific topics and expert opinions from sports medicine and pre-hospital emergency medicine practitioners.

Management

SCA during sport or exercise requires rapid recognition and effective treatment if the individual is to survive. Reports of improved survival for SCA during sport with survival rates improving from 8.0% in the general population to 22.8% in sport related events[509] and even reaching as high as 71% in US High schools[523] are attributed to the majority of the events being witnessed, the implementation of prompt resuscitation and the availability of a defibrillator. In marathon running it has been reported that 50% of SCDs occurred in the last mile with the highest prediction of survival being early bystander resuscitation and the use of an AED.[518]

Therefore, there is strong evidence in favour of planning, adhering and implementing standard resuscitation procedures that include basic life support and the use of an AED in sport related cardiac events.

Prevention

Historically, cardiac screening has been the recommended strategy for the prevention of cardiac events in sport. However there remains differences between the European Society of Cardiology who recommend the use of a 12-lead ECG as a screening tool for all young athletes[524] and the AHA/American College of Cardiology who found insufficient evidence to support this screening tool.[525] Currently the International Olympic Committee and many International Sport Federations recommend cardiac screening for athletes.[526]

For older participants in sports and exercise, a medical evaluation should include the individual's current level of physical activity, their known cardiovascular, metabolic, or renal disease, the presence of the signs or symptoms suggestive of cardiovascular disease and the desired or anticipated exercise intensity.[527]

Commotio cordis

Whereas most cardiac events in sport are not associated with contact or trauma, commotio cordis is the exception. Commotio cordis, the disruption of cardiac rhythm by a blow to the precordium, has a quoted incidence of 3%.[528] The striking object must strike the chest within the cardiac silhouette within a 20 ms window of the upstroke of the T-wave.[529] The overall survival rate from commotio cordis is reported to have improved with survival rates of up to 58% reported in recent years.[530] This has been attributed to rapid recognition of the collapse, early basic life support and the availability of AEDs resulting in prompt defibrillation.

Drowning

Drowning is the third leading cause of unintentional injury death worldwide, accounting for over 360,000 deaths annually [https://www.who.int/news-room/fact-sheets/detail/drowning]. Care of a submersion victim in high-resource countries often involves a multiagency approach, with several different organizations being independently responsible for different phases of the patient's care, beginning with the initial aquatic rescue, through on-scene resuscitation and transfer to hospital and in-hospital and rehabilitative care. Attempting to rescue a submerged patient has substantial resource implications and may place rescuers at risk themselves. The major sequalae of drowning is hypoxia caused by respiratory impairment secondary to the aspiration of fluid into the lungs. If severe or prolonged, this can cause cardiac arrest. Early effective intervention is critical to improve survival and reduce morbidity.

Recommendations in this section follow the updated 2020 ILCOR systematic review and ILCOR scoping review.[3,14]

Initial rescue

The updated 2020 ILCOR systematic review explored the influence of key prognostic factors on the likelihood of search and rescue operations yielding favourable outcomes. The review found moderate certainty evidence that submersion duration was the strongest predictor of outcome and recommended its use when making decisions surrounding search and rescue resource management/operations.[14] The CoSTR suggested against the use of age, EMS response time, water type (fresh or salt), water temperature, and witness status when making prognostic decisions (very low certainty evidence). Feedback during the public consultation on the guidelines highlighted a potential role for drones to reduce submersion duration or provide flotation aids.[531–533]

Cardiac arrest prevention

Insights from a scoping review identified limited evidence from observational studies and manikin studies to inform the treatment of the drowning victim.[3] A summary of the key findings of the review is presented in Table 8. The ERC recommendations for the treatment of drowning are therefore based on expert consensus from the writing group, informed by evidence from the scoping review.

Cardiac arrest

Similar to the cardiac arrest prevention section, limited evidence to inform practice guidelines was identified during the scoping review.[3] Key findings are summarised in Table 9. The ERC recommendations for the treatment of cardiac arrest related to drowning are therefore based on expert consensus from the writing group, informed by evidence from the scoping review. Given that the duration of submersion and duration of cardiac arrest are key prognostic indicators, initiating resuscitation as early as it is safe and practical to do so is strongly supported by the writing group.

Mass casualty incidents

Mass casualty incidents (MCIs), characterized by greater demand for medical care than available resources, are rare events. Among the 19.8 million yearly EMS activations in the United States, only 0.3% had an MCI dispatch code of which less than a half were confirmed on scene.[535] The MCI can be caused by variety of chemical, biological, radiological or nuclear (CBRN) incidents, but traumatic incidents (e.g. traffic accidents, acts of crime, or natural and industrial disasters) play a leading role in developed countries.[536] Key themes were identified recently to improve future practice of prehospital providers: tactical emergency medical support may harmonise inner cordon interventions, a need for inter-service education on effective haemorrhage control (trauma specific), the value of senior triage operators and the need for regular mass casualty incident simulation.[537]

A PubMed scoping review was performed on March 27th, 2020 using the keywords "mass-casualty incident" AND "cardiac arrest or heart arrest" OR "resuscitation or cardiopulmonary resuscitation" in the last 5 years (n = 47). There were no systematic reviews and RCTs identified on this topic related to CPR. There are few RCTs exploring different educational and managerial strategies during MCIs including use of modern technologies, e.g. unmanned aerial vehicle (UAV) or Smart Glasses providing telemedical connection from the scene.[538] Available resources did not provide any evidence for change of resuscitation practice when compared to the ERC guidelines 2015.[84]

Modifications to CPR during pandemics of highly contagious infectious disease have been addressed in the separately published ERC COVID-19 guidelines (April 2020).[539] Although pandemics did not fulfil MCI definition 'per se', some healthcare systems were facing shortages of personnel and equipment limiting capacity of critical care. Decisions on allocation of resources, including provision of CPR, during pandemics had to be made locally on the level of individual healthcare systems. However, the COVID-19 guidelines have emphasised importance of generally applicable safety precautions.

Safety

Potential hazards should be identified and assistance should be requested immediately. The presence of multiple victims should always alert rescuers to the possibility of a CBRN incident. Never approach the victims unless the area is safe. High risks are present at crime scenes (e.g. shootings, bomb attacks), or places polluted by noxious substances (e.g. carbon monoxide, industrial cyanides or other chemicals).

Adequate personal protection equipment (PPE) (e.g. bulletproof vest, respirator, long-sleeved gown, eye and face protection) should be used depending on specific risks on scene. Healthcare providers are required to don (put-on) PPE before approaching casualties even if time-critical interventions are required. It is acknowledged that this could cause delay to treatment.[539] Wearing PPE may also adversely affect performance of interventions and limit the standards of care. Simulation studies have shown reduced success rate of advanced airway techniques, prolonged time for securing IV and intraosseous access, and difficulties with drug preparation.[540–542]

Secondary risks to patients and providers should be avoided. During sarin attacks in Japan, 10% of 1363 EMS technicians developed poisoning, mostly from primary victims in poorly ventilated ambulances.[543]

Triage

Initial triage of casualties enables identification of patient care priorities. Unlike normal circumstances, CPR is not usually initiated in MCI, in order to avoid delay potentially effective treatment for salvageable victims. This critical decision depends on available resources in relation to the number of casualties.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

Table 8 – Cardiac arrest prevention in drowning.

Topic	Evidence identified	Key findings
Oxygen administration	No studies identified which directly addressed this question. 4 observational studies, indirectly address this question.	Insufficient evidence to guide the pre-hospital use of oxygen therapy in drowning. Pragmatically, consider treating the hypoxic patient with high flow oxygen prior to arrival in hospital where direct measurement of arterial oxygenation can be performed to enable controlled oxygen therapy. Further research to guide on the optimal mode for delivery and optimising pre-hospital monitoring is required.
Airway management	No studies identified which directly addressed this question.	The studies reviewed show that that intubation is a feasible intervention following a submersion incident. The association between intubation and poor outcomes is almost certainly confounded by the need for intubation being an intervention limited to more severe drowning.
	Indirect evidence from 15 observational studies.	In the absence of data supporting an alternative strategy, adoption of the ALS Task Force recommendations for airway management is reasonable.[2]
Ventilation strategies	4 observational studies	NIV appears feasible as a treatment for moderate to severe lung injury caused by drowning. The published experience involves mostly patients with higher GCS, who are haemodynamically stable. Patients appear to respond within 12–24 h. The indications for the optimal time to transition to invasive ventilation if NIV is unsuccessful requires further research. Given the absence of direct evidence for any particular invasive ventilation strategy in drowning, the writing group advocates the adoption of evidence based strategies for the management of acute respiratory distress syndrome.[534]
ECMO	13 observational studies	The evidence identified for severe respiratory failure, is consistent with guidelines suggesting the use of ECMO in selected patients with severe ARDS (weak recommendation, very low certainty of evidence).[534]

Locally established triage systems to prioritise treatment should be used.[544–546] There is not sufficient evidence to declare one of the triage protocols superior in all aspects to the others.[547] Advanced prehospital teams involved in the initial scene triage must avoid over triage. Repeated triage (re-triage) is needed at hospital admission and responsible personnel at all stages of emergency care must be familiar with the triage system used.

Life-saving interventions should be performed in patients triaged as "immediate" (highest priority) to prevent cardiac arrest:[545]

- open airway using basic techniques
- control bleeding
- decompress chest for tension pneumothorax
- use antidotes in auto-injectors
- consider initial rescue breaths in a non-breathing child.

Assigning a higher triage risk level to elderly and to survivors of high-energy trauma should be considered to reduce the number of preventable deaths. In the National Trauma Database (NTDB), patients in all triage levels were compared to mortality outcomes. There were 322,162 subjects assigned to the 'green' triage level of which 2046 died before hospital discharge. Age was the primary predictor of under triage.[544]

In children, special triage tapes or a paediatric-specific MCI triage system (e.g. JumpSTART) should be used.[548] If this is not available, any adult triage system can be used.

Decision to use an MCI triage sieve and withhold care to those with imminent death, (including victims without signs of life), is responsibility of a medical commander who is usually the most experienced EMS clinician on scene. Individual role allocations usually depend on local protocols. Modern technologies (e.g. UAVs or Smart Glasses) allow real-time video transmission from the triage site to the remote incident commanders or personnel at receiving hospitals.[549] Triage inaccuracy may have fatal consequences in patients with survivable injuries. Healthcare professionals must be regularly trained to use the triage protocols during simulations and live exercises.[550] Educational video games enhance learning and improve subsequent performance when compared to traditional educational methods.[551] Training allows fast and correct recognition of those requesting lifesaving procedures and reduces the risk of inappropriate care given to futile cases.

Special patients

Asthma and COPD

Evidence based recommendations for the management of acute life threatening asthma are provided by the British Thoracic Society, Scottish Intercollegiate Guidelines Network (Fig. 13) [https://www.sign.ac.uk/sign-158-british-guideline-on-the-management-of-asthma.html] and for chronic obstructive lung disease by the Global initiative for chronic obstructive lung disease (GOLD) (https://gold-copd.org/). The guidelines were assessed according to the AGREE-II framework and rated as high quality and consequently recommend the use of this guideline in practice.

The BTS/SIGN and GOLD guidelines do not contain specific information on the management of cardiac arrest. Our review did not identify any other relevant, high quality guidelines. Therefore, we undertook a scoping review and formed our guidelines based on expert consensus amongst the writing group.

The scoping review identified 352 papers of which 19 were relevant. No RCTs were identified. Evidence is therefore drawn from these observational studies, supplemented by studies identified in the 2015 guidelines. The recommendations are based on the expert consensus of the writing group.

Cardiac arrest prevention

A stepwise approach to the initial assessment and treatment, based on the ABCDE approach is recommended for patients at risk of cardiac arrest due to an exacerbation of obstructive lung disease (asthma/COPD).

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

Table 9 – Cardiac arrest management in drowning.

Topic	Evidence identified	Key findings
In-water resuscitation	1 observational study and 4 manikin studies	In-water resuscitation by highly trained rescue teams with water rescue equipment is feasible. If trained and capable rescue teams are available, initiate in-water resuscitation for the unconscious and not-breathing patient by performing up to 1 min of ventilations (≈10 ventilations) before attempting transfer to land. If breathing is not restored, patient should be towed to the shore/boat without more attempts of ventilations during water rescue. Outside of the setting of a highly trained rescue team, who are familiar with in-water resuscitation, transfer directly to land/boat before initiating resuscitation. One rescuer, although well trained in in-water resuscitation, without rescue equipment should also bring the patient directly to the shore.
CPR on a boat	2 observational studies and 4 manikin studies	Resuscitation in a boat seems feasible. Those providing resuscitation should focus on high quality CPR and be alert to the development of fatigue and consider switching CPR provider regularly.
Bystander CPR	18 observational studies	Bystander CPR in drowning is feasible and appears effective. The apparent superiority of conventional CPR which includes ventilation, has biological plausibility as cardiac arrest association with drowning is primarily due to hypoxia. The findings of this review are consistent with the ILCOR CoSTR which recommends that chest compressions be performed for all patients in cardiac arrest. ILCOR suggests that those who are trained, able and willing to give rescue breaths as well as chest compressions do so for all adult patients in cardiac arrest.[3]
AED use	No studies identified which directly addressed this question. Indirect evidence from 15 observational studies.	AED use in cardiac arrest due to drowning appears feasible and safe. The chances of a shockable rhythm is lower than for a primary cardiac cause. Given this, the writing group considered initiating rescue breaths and chest compressions should be prioritised. This is consistent with the ILCOR treatment recommendation which advises a short period of CPR while the defibrillator is prepared for analysis, which is likely to be particularly important where the cardiac arrest was caused by drowning.[3]
Airway management	No studies identified which directly addressed this question. Indirect evidence from 15 observational studies.	In the absence of data supporting an alternative strategy, adoption of the ALS Task Force recommendations for airway management is reasonable.[2] Start with basic airway techniques and progress stepwise according to the skills of the rescuer until effective ventilation is achieved. If an advanced airway is required, only rescuers with a high tracheal intubation success rate should use tracheal intubation.
ECPR	13 observational studies	Extracorporeal oxygenation to treat cardiac arrest or severe respiratory failure caused by drowning is feasible. The evidence identified supports the ILCOR treatment recommendation that suggests “extracorporeal cardiopulmonary resuscitation (ECPR) may be considered as a rescue therapy for selected patients with cardiac arrest when conventional cardiopulmonary resuscitation is failing in settings where this can be implemented (weak recommendation, very-low certainty of evidence)”.

Further steps in the treatment of acute severe asthma are summarized in Fig. 13.

For COPD, the GOLD 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 short acting beta-2 agonists with or without short-acting anticholinergics, systemic corticosteroids and antibiotics if a bacterial infection is suspected. Non-invasive ventilation (NIV) is recommended in the presence of respiratory acidosis ($PaCO_2 < 6$ kPa/35 mmHg and arterial pH <7.35); severe dyspnoea with clinical signs of fatigue and/or increased work of breathing. Escalation to invasive ventilation may be required in the event of NIV failure, the patient is intolerant to NIV, agitation or reduced conscious level, risk of aspiration, cardiovascular instability or life-threatening hypoxia. 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.[552]

Treatment of cardiac arrest caused by obstructive lung disease

Cardiac arrest in patients with obstructive lung disease may arise as a consequence of hypoxia, hypovolaemia, toxins (arrhythmias caused by stimulant drugs e.g. beta-adrenergic agonists, aminophylline), electrolyte disturbance, tension pneumothorax and/or the effects of gas trapping leading to reducing venous return and blood pressure.[553−557] Cardiac arrest in obstructive lung disease is usually associated with a non-shockable rhythm and therefore poor survival rates.[558,559]

Airway

Oxygen: Although no definitive studies were found which examined the role of oxygen versus any other gas in cardiac arrest due to obstructive lung disease, the writing group considered hypoxia, as the main cause of cardiac arrest, a high priority and recommend high concentration oxygen when providing assisted ventilation.

Advanced airway management: An observational study involving 12 patients recorded peak airway pressures in acute severe asthma (mean 67.8 + 11 cm H_2O) which are significantly higher than the normal lower oesophageal sphincter pressure.[560] There is a significant risk of gastric inflation and hypoventilation of the lungs when attempting to ventilate a severe asthmatic without a tracheal tube. During cardiac arrest this risk is even higher, because the lower oesophageal sphincter pressure is substantially lower than normal. The writing groups therefore suggest that the trachea is intubated as soon as possible during cardiac arrest caused by asthma. Consistent with the ALS airway management guidelines, intubation should only be performed by someone who is trained and competent to do so.[101]

Breathing

Check for signs of tension pneumothorax and treat accordingly: Patients with obstructive lung disease may develop tension pneumothorax, which, if left untreated, may cause cardiac arrest.[561–565] Check for signs of tension pneumothorax and treat accordingly, noting that needle decompression alone may be insufficient to relieve a tension pneumothorax.[561,566]

Disconnect from positive pressure ventilation if air-trapping and hyperinflation occurs and apply pressure to manually reduce the hyperinflation: Some case reports have reported ROSC in patients with air trapping when the tracheal tube was disconnected.[567–573] If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest while disconnecting the tracheal tube may relieve air trapping.[572,574] Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.[574,575]

Ventilate with respiratory rate (8–10 min) and sufficient tidal volume to cause the chest to rise. Respiratory rates of 8–10 breaths per minute and a tidal volume required for a normal chest rise during CPR should minimize dynamic hyperinflation of the lungs (air trapping).[576] Tidal volume depends on inspiratory time and inspiratory flow. Lung emptying depends on expiratory time and expiratory flow. In mechanically ventilated severe asthmatics, increasing the expiratory time (achieved by reducing the respiratory rate) provides only moderate gains in terms of reduced gas trapping when a minute volume of less than 10 l min^{-1} is used.[560]

Circulation

Consider IV fluids: No studies evaluating the use of IV fluids for cardiac arrest due to obstructive lung disease were identified. Expert consensus from the writing group suggests that IV fluids should be considered due to the risk of patients with obstructive lung disease becoming dehydrated due to reduced oral intake and/or increased insensible losses.

Consider standard dose IV adrenaline: In alignment with the ILCOR CoSTR on vasopressors in cardiac arrest, the ALS guidelines recommend that 1 mg adrenaline is given every 3–5 min during cardiac arrest based on moderate quality evidence.[2,101] The main trial informing these recommendations however excluded patients with asthma.[577] Some small observational studies, predominantly in younger patients suggest it may be administered in life threatening asthma without adverse sequalae.[578,579]

Consider ECPR: ECMO has been used successfully in patients with life threatening asthma.[580,581] Consistent with ALS guidelines E-CPR may be considered if conventional therapies fail and the health system has immediate access to this treatment.[2]

Neurological disease

Cardiac arrest associated with acute neurological disease is relatively uncommon and can occur with subarachnoid haemorrhage, intracerebral haemorrhage, epileptic seizures, and ischaemic stroke.[582] In a US post mortem study of 335 sudden cardiac deaths, 18 (5.4%) were sudden neurological deaths (intracranial haemorrhage, sudden unexpected death in epilepsy, aneurysmal subarachnoid haemorrhage, acute stroke, aspiration from Huntington Disease).[583] These deaths made up 14.9% of the 121 non-cardiac deaths in the study.

The evidence supporting this guideline is based on observational data and expert opinion, and guidelines for the care of common neurological conditions that may cause cardiac arrest. A focused literature search was carried out up to 10 August 2020 and identified observational 9 studies and a Cochrane review since the since the 2015 guideline.[84]

Prodromal signs

Certain features such as a younger age, female gender, non-shockable initial rhythm and neurological antecedents (e.g. headache, seizures, neurological deficits) suggest a neurological cause of cardiac arrest.[584] Other non-specific signs include syncope, shortness of breath and chest pain.[585]

Early brain imaging

Identifying a neurological cause of cardiac arrest after ROSC is part of the ERC post resuscitation care guideline.[270] The expert opinion based on observational data is that early identification of a neurological cause can be achieved by performing a brain CT-scan at hospital admission, before or after coronary angiography. In the absence of signs or symptoms suggesting a neurological cause (e.g. headache, seizures or neurological deficits) or if there is clinical or ECG evidence of myocardial ischaemia, coronary angiography is undertaken first, followed by CT scan in the absence of causative lesions on coronary angiography. A systematic review of the diagnostic yield of non-invasive imaging in patients following non-traumatic OHCA identified 9 observational studies of brain imaging.[586] The most common diagnoses were brain haemorrhage (16.9%, including intraparenchymal, intracranial or extra-axial haemorrhage) and acute stroke (11.8%). The indication for the scans was not entirely clear so the true rate of identifying neurological causes is uncertain.

Subarachnoid haemorrhage

Cardiac or respiratory arrest occurs in between 3–11% of patents with subarachnoid haemorrhage (SAH).[587] There is considerable regional variation in the incidence of SAH as a cause of cardiac arrest among those with sustained ROSC at hospital admission. Published case series report 16.2% in Japan,[588] 11.4% in Korea[589] and 7% in France.[590] In a Japanese study of patients with ROSC, SAH was most often associated with an initial non-shockable rhythm (95.7%), a prodromal headache before cardiac arrest (47.8%), and a negative cardiac troponin-T (94.7%).[588] Patients with SAH may have ECG changes that suggest an acute coronary syndrome.[585,591–593]

This can pose challenges after ROSC in terms of whether they should have an early brain scan or go directly for coronary angiography. The order of brain scanning versus early coronary angiography should be based on clinical suspicion (See Section X Post resuscitation care).[270] Prognosis is poor even in those with ROSC after a SAH.[585,591,594] This is probably because cardiac arrest tends to occur with larger more severe bleeds following a SAH.[595]

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

Sudden unexpected death in epilepsy

Sudden unexpected death in epilepsy (SUDEP) effects about 1 in every 100 people with epilepsy.[596] Data from the North American SUDEP registry showed cases of SUDEP had a median age of 26 years at death, 38% were female, 40% had generalized and 60% had focal epilepsy, most (93%) were unwitnessed, 70% occurred during apparent sleep, 69% of patients were prone, and only 37% of cases of SUDEP took their last dose of antiseizure medications. A Cochrane review found very low certainty evidence for interventions to prevent SUDEP addition to improving seizure control such as having a supervising person share a bedroom and use of monitoring devices.[597]

Stroke

Data from the Ontario stroke registry found that 3.9% of acute ischaemic stroke patients also had a cardiac arrest.[598] The risk of arrest was increased in older patients with increased stroke severity, diabetes, myocardial infarction, heart failure and atrial fibrillation. Mortality at 30 days was 82.1% in cardiac arrest stroke patients versus 9.3% in non-cardiac arrest stroke patients. Data from the all Japan Utstein registry reported that 7.7% of OHCA cases had a stroke related cardiac arrest.[599] This group had worse outcomes that patients who had a primary cardiac arrest.

Outcomes

Survival from sudden neurological death depends on the underlying cause and the Chain of survival (i.e., witnessed, early bystander CPR, ALS, and post resuscitation care). Survival is generally worse than for primary cardiac arrest.[1,582] Individuals who achieve ROSC after a primary neurological cause of cardiac arrest may not recover and have withdrawal of life sustaining treatments, or fulfil the criteria for death by neurological criteria. These patients should be considered for organ donation according to local legal and clinical criteria [see section X post-resuscitation care].[270]

Obesity

Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health. A crude population measure of obesity is the body mass index (BMI), a person's weight (in kg) divided by the square of his or her height (in metres). A person with a BMI of 30 kg m^{-2} or more is generally considered obese. In 2016, more than 1.9 billion (39%) adults were overweight, and of these over 600 million (13%) were obese. In the United States, the age-adjusted prevalence of obesity in 2013–2014 was 35.0% among men and 40.4% among women.[600]

Clinical and epidemiological evidence has linked obesity to a broad spectrum of cardiovascular diseases including coronary heart disease, heart failure, hypertension, stroke, atrial fibrillation and sudden cardiac death. Obesity can increase cardiovascular morbidity and mortality directly and indirectly. Direct effects are mediated by obesity-induced structural and functional adaptations of the cardiovascular system to accommodate excess body weight, as well as by adipokine effects on inflammation and vascular homeostasis. Indirect effects are mediated by co-existing risk factors such as insulin resistance, hyperglycaemia, hypertension and dyslipidaemia.[601–603]

A scoping review using the PubMed search engine was performed on March 27th, 2020 using the keywords "obesity" AND "cardiac arrest or heart arrest" OR "resuscitation or cardiopulmonary resuscitation" in the last 5 years (n = 122). There were two meta-analysis published on association between BMI and outcome after cardiac arrest.[604,605] Modification to chest compressions is based on 1 retrospective study evaluating chest compression depth in obese patients using computed tomography (CT).[606]

Treatment of cardiac arrest

No changes to the sequence of actions are recommended in resuscitation of obese patients, but delivery of effective CPR may be challenging. Physical and physiological factors related to obesity may adversely affect the delivery of CPR, including patient access and transportation, patient assessment, difficult IV access, airway management, quality of chest compressions, the efficacy of vasoactive drugs, and the efficacy of defibrillation because none of these measures are standardized to a patient's BMI or weight.[607]

Chest compressions

Healthcare providers should consider deeper chest compression in obese patients with a maximum depth of 6 cm using a feedback device, if available. Obese patients lying in a bed do not necessarily need to be moved onto the floor. Their heavy torso sinks into the mattress and leaves less potential for mattress displacement during chest compression.[608,609] Repositioning of obese patients may delay initiation of CPR, but also cause injuries to the patient and rescuers.

Rescuer providing chest compression should be changed more frequent compared to the standard two-minute interval to maintain sufficient compression depth (6 cm).[84]

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 obesity permagna patients. The upper limits include sternum height of 303 or 340 mm and chest width of 449 or 480 mm for piston devices; chest circumference of 130 cm, chest width of 380 mm and body weight of 136 kg for devices equipped with a load-distributing band.

Defibrillation

Defibrillation protocols for obese patients should follow those recommended for patients with a normal BMI with escalation of energies up to the maximum feasible for subsequent shocks if initial defibrillation attempts fail (expert opinion). Optimal defibrillation energy levels in obese patients are unknown. Modern biphasic defibrillators adjust their output according to the patient's impedance. Two small retrospective studies have demonstrated no apparent weight-based influence on defibrillation efficacy with a biphasic waveform of 150 J achieving high shock success rates without need for energy escalation.[610,611] An RCT evaluating cardioversion of atrial fibrillation in obese patients however reported lower success rate when using adhesive pads with standard energies. Use of paddles or manual pressure augmentation technique further improved success of the electrical therapy.[612]

Airway management and ventilation

Manual ventilation, using a bag-mask technique, should be performed by experienced staff using a two-person technique. The increased in abdominal size of obese individuals raises intra-abdominal pressure and repositions the diaphragm in cranial direction.[613] This requires higher inspiratory pressures for controlled ventilation increasing the risk of gastric insufflation and aspiration of gastric contents.

Experienced providers should intubate the trachea early so that the period of bag-mask ventilation is kept to a minimum. In all patients with morbid obesity, difficult intubation must be anticipated.[614–616] If intubation is not possible, use of a supraglottic airway device (SGA)

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

with sufficient pressure seal and oesophageal drainage tube should be considered as suitable option.[617,618]

Logistical considerations

Obesity must be considered when organizing pre-hospital resuscitation, especially concerning technical support and number of EMS staff.[619] Special vehicles modified to transport extremely obese patients, equipped with reinforced stretchers and specialized lifting gear, should be used if possible. Weight limits of both stretchers and hospital beds must be known prior to use.[620] Underestimation of the technical aspects of rescue operations may cause secondary trauma, or even make transportation to the hospital impossible.[619]

Cardiac arrest in pregnancy

Maternal mortality remains high with an estimated 295,000 deaths in 2017, the majority (94%) occurring in low and lower middle income countries. (WHO – https://www.who.int/news-room/fact-sheets/detail/maternal-mortality accessed 20 July 2020) A maternal cardiac arrest is a cardiac arrest that occurs at any stage in pregnancy and up to 6 weeks after birth. In a UK study the incidence of cardiac arrest was 1 in 36,000 maternities.[621] This section focuses on specific additional interventions for resuscitation during pregnancy and delivery.

This guideline has been informed by an ILCOR Evidence Update.[2] The majority of the guidance is unchanged from the ERC 2015 Special Circumstances.[84] In addition, this guidance has been informed by guidelines from the AHA,[622] UK Royal College of Obstetricians and Gynaecologists,[623] European Society of Cardiology Guidelines for management of cardiovascular disease during pregnancy.[624] Most guidance is based on expert opinion, our knowledge of the physiology of pregnancy, and observational data.

Causes of cardiac arrest associated with pregnancy

In 2015–17, 9.2 women per 100,000 died during pregnancy or up to six weeks after childbirth or the end of pregnancy.[625] The most common causes were heart disease (23%), thromboembolism (16%), epilepsy and stroke (13%), sepsis (10%), mental health conditions (10%), bleeding (8%), cancer (4%) and pre-eclampsia (2%). The risk increased with age, social deprivation and for ethnic minorities. A study of cardiac arrests in pregnancy between 2011 and 2014 identified 66 cardiac arrests of whom 28 died (42%).[621] Of these about 25% (16) of arrest were associated with anaesthesia (12 were obese) and all survived. Survival was poor for OHCA and if there was a delay in perimortem caesarean section. Most babies (46) survived, 32 to woman who survived and 14 to those that died.

Prevention and treatment of cardiac arrest in the pregnant patient

This should follow the standard ABCDE format identifying and treating problems as they are identified. Involving specialists in the care of the sick obstetric patient and neonate early is important in order to deliver specialist interventions. Expert consensus is that the use of validated obstetric specific early warning scores improve earlier recognition of deterioration and enable risk stratification of ill pregnant patients.[622,623]

Aortocaval compression

After 20 weeks' gestation, the pregnant woman's uterus can press down against the inferior vena cava and the aorta and reduces venous return and cardiac output by 3–40%.[626] This can cause pre-arrest hypotension or shock and, in the critically ill patient, may precipitate arrest.[627,628] After cardiac arrest, the compromise in venous return and cardiac output may limit the effectiveness of chest compressions. Manual left uterine displacement is the easiest way to reduce aortocaval compression and may be more effective than left lateral tilt.[629,630] This can be achieved by lifting the uterus up and leftward off the aortocaval vessels.[622] This maintains a supine position, allowing for continuous effective cardiac compressions if necessary.

Non-arrest studies show that left lateral tilt improves maternal blood pressure, cardiac output and stroke volume and improves fetal oxygenation and heart rate.[631–633] Non-cardiac arrest data show that the gravid uterus can be shifted away from the cava in most cases by placing the patient in 15 degrees of left lateral decubitus position.[634]

Unless the pregnant victim is on a tilting operating table, left lateral tilt is not easy to perform whilst maintaining high-quality chest compressions. A variety of methods to achieve a left lateral tilt have been described including placing the victim on the rescuer's knees. In a manikin study, the ability to provide effective chest compressions decreased as the angle of left lateral tilt increased and that at an angle of greater than 30° the manikin tended to roll.[635]

Chest compressions

Chest compressions should be according to BLS guidelines at a rate of 100–120 min^{-1} and depth of 5–6 cm on the lower half of the sternum.[15] The evidence for optimal hand position is conflicting. An MRI study showed no change in heart position[636] whereas a recent echocardiographic study suggested the enlarged uterus can push the diaphragm and heart upwards.[637] The current guideline based on expert opinion is to use the standard manual chest compression technique if feasible. The use of mechanical chest compression devices is not recommended in pregnancy.

Peri-mortem delivery of the fetus

Consider the need for an emergency hysterotomy or caesarean section as soon as a pregnant woman goes into cardiac arrest. In some circumstances immediate resuscitation attempts will restore a perfusing rhythm; in early pregnancy this may enable the pregnancy to proceed to term. Delivery will relieve aortocaval compression and may improve chances of maternal and fetal resuscitation.[638] The majority of evidence for early delivery comes from case reports and small observational studies.[84,639] A UK study of cardiac arrests in pregnancy between 2011 and 2014 identified 66 cardiac arrests of whom 49 (74%) had a perimortem caesarean section (PMCS).[621] In 61% this was within 5 min of collapse. The time from collapse to delivery in survivors was 7 min [interquartile range (IQR) 2.5–17.5] and 16 min (IQR 6.5–43.5) in non-survivors (P = 0.04). When PMCS was formed within 5 min 24 of 25 babies survived (96%). Seven of 10 babies (70%) survived when PMCS occurred after more than 5 min (P = 0.06).

Based on the available evidence and expert consensus the ERC guidelines remain unchanged – If over 20 weeks pregnant or the uterus is palpable above the level of the umbilicus and immediate (within 4 min) resuscitation is unsuccessful, deliver the fetus by emergency caesarean section aiming for delivery within 5 min of collapse. This requires that PMCS decision making occurs early and ideally takes place at the location of the cardiac arrest.

Extracorporeal life support

Starting ECLS before cardiac arrest or ECPR when traditional ALS measures are failing should be considered in pregnant patients in those settings where it is available. A retrospective analysis of peripartum patients who needed extracorporeal membrane oxygenation between 1997 and 2017 in the International Registry of Extracorporeal Life Support Organization identified 280 patients.[640] Overall survival was 70% that improved over the data collection period. Survival was better if ECLS was started prior to cardiac arrest. Forty-two patients had ECPR and 19/42 (45.2%) died in hospital.

Other modifications to advanced life support

Defibrillation

For cardiac arrest with a shockable rhythm attempt defibrillation as soon as possible. There is no change in transthoracic impedance during pregnancy, suggesting that standard shock energies for defibrillation attempts should be used in pregnant patients.[641] There is no evidence that shocks have adverse effects on the fetal heart. Left lateral tilt and large breasts will make it difficult to place an apical defibrillator pad.

Airway management

Pregnant patients have an increased risk of gastric regurgitation and aspiration, and have an increased risk of failed intubation.[642–644] The airway should be managed according to current ALS guidelines using a stepwise approach (bag-mask, supraglottic airway, tracheal tube, according to rescuer skills. Early intubation will make oxygenation and ventilation easier and protect against aspiration but this requires an expert intubator and be carried out according to current obstetric guidelines.[643]

Reversible causes

Rescuers should attempt to identify common and reversible causes of cardiac arrest in pregnancy during resuscitation attempts. The 4 Hs and 4Ts approach helps identify all the common causes of cardiac arrest in pregnancy.[623] Pregnant patients are at risk of all the other causes of cardiac arrest for their age group (e.g., anaphylaxis, drug overdose, trauma). Consider the use of abdominal ultrasound by a skilled operator to detect pregnancy and possible causes during cardiac arrest in pregnancy; however, do not delay other treatments and minimise interruptions to chest compressions.

Haemorrhage

Life-threatening haemorrhage can occur both antenatally and postnatally.[645] Associations include ectopic pregnancy, placental abruption, placenta praevia, placenta accreta, and uterine rupture.[646] A massive haemorrhage protocol must be available in all units and should be updated and rehearsed regularly in conjunction with the blood bank. Women at high risk of bleeding should be delivered in centres with facilities for blood transfusion, intensive care and other interventions, and plans should be made in advance for their management. Treatment is based on an ABCDE approach. Follow existing guidelines for management of massive haemorrhage obstetrics.[647,648] A large RCT showed that 1 g IV tranexamic acid reduced death from postpartum haemorrhage, especially if given within 3 h.[649]

Cardiovascular disease

Myocardial infarction and aneurysm or dissection of the aorta or its branches, and peripartum cardiomyopathy cause most deaths from acquired cardiac disease Patients with known cardiac disease need to be managed in a specialist unit. Pregnant women may develop an acute coronary syndrome, typically in association with risk factors such as obesity, older age, higher parity, smoking, diabetes, pre-existing hypertension and a family history of ischaemic heart disease.[84] Pregnant patients can have atypical features such as epigastric pain and vomiting. Percutaneous coronary intervention (PCI) is the reperfusion strategy of choice for ST-elevation myocardial infarction in pregnancy.[624] Thrombolysis should be considered if urgent PCI is unavailable. A review of 200 cases of thrombolysis for massive pulmonary embolism in pregnancy reported a maternal death rate of 1% and concluded that thrombolytic therapy is reasonably safe in pregnancy.[650]

Pre-eclampsia and eclampsia

Eclampsia is defined as the development of convulsions and/or unexplained coma during pregnancy or postpartum in patients with signs and symptoms of pre-eclampsia. The ERC recommends that existing guidance for pre-eclampsia and eclampsia is followed (e.g. Hypertension in pregnancy: diagnosis and management NICE guideline Published: 25 June 2019.[651]

Amniotic fluid embolism

Amniotic fluid embolism (AFE) usually presents around the time of delivery with sudden cardiovascular collapse, breathlessness, cyanosis, arrhythmias, hypotension and haemorrhage associated with disseminated intravascular coagulopathy.[652] Patients may have warning signs preceding collapse including breathlessness, chest pain, feeling cold, light-headedness, distress, panic, a feeling of pins and needles in the fingers, nausea, and vomiting. The UK Obstetric Surveillance System (UKOSS identified 120 cases of AFE between 2005 and 2014 with a total and fatal incidence estimated as 1.7 and 0.3 per 100 000, respectively, and association with older maternal age, multiple pregnancy, placenta praevia and induction of labour, instrumental vaginal and caesarean delivery.[653] Treatment is supportive, as there is no specific therapy based on an ABCDE approach and correction of coagulopathy.

Post resuscitation care

Post resuscitation care should follow standard guidelines. Targeted temperature management has been used safely and effectively in early pregnancy with fetal heart monitoring and resulted in favourable maternal and fetal outcome after a term delivery.[654,655]

Preparation for cardiac arrest in pregnancy

Advanced life support in pregnancy requires coordination of maternal resuscitation, Caesarean delivery of the fetus and newborn resuscitation ideally within 5 min. The evidence supporting this is largely based on observational data to achieve this, units likely to deal with cardiac arrest in pregnancy should:

- have plans and equipment in place for resuscitation of both the pregnant woman and newborn.
- ensure early involvement of obstetric, anaesthetic, critical care and neonatal teams.
- ensure regular training in obstetric emergencies.

The evidence to support this is largely based on expert opinion and observational data.[656–659]

Conflict of interest

JN reports funding from Elsevier for his role as Editor in Chief of the journals Resuscitation and Resuscitation Plus. He reports research funding from the National Institute for Health Research in relation to the PARAMEDIC2 trial and the AIRWAYS2 trial.

JS declares his role as an editor of Resuscitation; he declares institutional research funding for the Audit-7 project.

JH reports travel funding from Behring and Ambu

GDP reports funding from Elsevier for his role as an editor of the journal Resuscitation. He reports research funding from the National Institute for Health Research in relation to the PARAMEDIC2 trial.

JY declares research grants from National Institute for Health Research and Resuscitation Council UK.

KT reports Federal research funding for drone AED research.

Acknowledgements

GDP is supported by the National Institute for Health Research (NIHR) Applied Research Collaboration (ARC) West Midlands. The views expressed are those of the author(s) and not necessarily those of the NIHR or the Department of Health and Social Care.

Appendix A. ERC Special Circumstances Writing Group Collaborators

Gamal Eldin Abbas Khalifa[1], Efrén Álvarez[4], Roberta Barelli[2], Joost J. L.M. Bierens[1], Bernd Boettiger[1], Guttorm Brattebø[1], Douglas Browne[3], Hermann Brugger[1,3], Tomasz Darocha[3], Charles D. Deakin[1], Joel Dunning[1], Silvija Hunyadi-Anticevic[1], Rudolph W. Koster[1], David J. Lockey[1], Mathieu Pasquier[3], Jan Schmitz[5].

[1]Substantial contribution to guidelines 2015

[2]Substantial contribution to Toxic agents section

[3]Substantial contribution to Hypothermia section

[4]Substantial contribution to Coronary thrombosis, catheterisation laboratory and cardiac surgery sections

[5]Substantial contribution to cardiac arrest in the operating room, Inflight cardiac arrest, Helicopter emergency medical services (HEMS) and air ambulances and Cruise ship sections

Appendix B. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.resuscitation.2021.02.011.

REFERENCES

1. Nolan J, Soar J, Eikeland H. The chain of survival. Resuscitation 2006;71:270–1.
2. Soar J, Berg KM, Andersen LW, et al. Adult advanced life support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2020;156:A80–A119.
3. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult basic life support: International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2020;156:A35–79.
4. Perkins GD, Graesner JT, Semeraro F, et al. European Resuscitation Council Guidelines 2021—executive summary. Resuscitation 2021;161.
5. Brown DJ, Brugger H, Boyd J, Paal P. Accidental hypothermia. N Engl J Med 2012;367:1930–8.
6. Paal P, Gordon L, Strapazzon G, et al. Accidental hypothermia-an update: the content of this review is endorsed by the International Commission for Mountain Emergency Medicine (ICAR MEDCOM). Scand J Trauma Resusc Emerg Med 2016;24:111.
7. Paal P, Milani M, Brown D, Boyd J, Ellerton J. Termination of cardiopulmonary resuscitation in mountain rescue. High Altitude Med Biol 2012;13:200–8.
8. Gordon L, Paal P, Ellerton JA, et al. Delayed and intermittent CPR for severe accidental hypothermia. Resuscitation 2015;90:46–9.
9. Safar P, Paradis NA, Weil MH. Asphyxial cardiac arrest. In: Paradis NA, Halperin HR, Kern KB, Wenzel V, Chamberlain DA, editors. Cardiac arrest—the science and practice of resuscitation medicine. Cambridge: Cambride University Press; 2007. p. 969–93.
10. Kitamura T, Kiyohara K, Sakai T, et al. Epidemiology and outcome of adult out-of-hospital cardiac arrest of non-cardiac origin in Osaka: a population-based study. BMJ Open 2014;4:e006462.
11. Farmery AD, Roe PG. A model to describe the rate of oxyhaemoglobin desaturation during apnoea. Br J Anaesth 1996;76:284–91.
12. DeBehnke DJ, Hilander SJ, Dobler DW, Wickman LL, Swart GL. The hemodynamic and arterial blood gas response to asphyxiation: a canine model of pulseless electrical activity. Resuscitation 1995;30:169–75.
13. Deasy C, Bray J, Smith K, et al. Hanging-associated out-of-hospital cardiac arrests in Melbourne, Australia. Emerg Med 2013;30:38–42.
14. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult basic life support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Circulation 2020;142:S41–91.
15. Olasveengen TM, Semeraro F, Ristagno G, et al. European Resuscitation Council Guidelines 2021: basic life support. Resuscitation 2021;161.
16. Ogawa T, Akahane M, Koike S, et al. Outcomes of chest compression only CPR versus conventional CPR conducted by lay people in patients with out of hospital cardiopulmonary arrest witnessed by bystanders: nationwide population based observational study. BMJ 2011;342:c7106.
17. Riva G, Ringh M, Jonsson M, et al. Survival in out-of-hospital cardiac arrest after standard cardiopulmonary resuscitation or chest compressions only before arrival of emergency medical services: nationwide study during three guideline periods. Circulation 2019.
18. Escutnaire J, Ducrocq F, Singier A, et al. Can we define termination of resuscitation criteria in out-of-hospital hanging? Prehosp Emerg Care 2018;1–8.
19. Kim MJ, Yoon YS, Park JM, et al. Neurologic outcome of comatose survivors after hanging: a retrospective multicenter study. Am J Emerg Med 2016;34:1467–72.
20. Deasy C, Bray J, Smith K, et al. Paediatric hanging associated out of hospital cardiac arrest in Melbourne, Australia: characteristics and outcomes. Emerg Med 2011;28:411–5.
21. Wee JH, Park KN, Oh SH, et al. Outcome analysis of cardiac arrest due to hanging injury. Am J Emerg Med 2012;30:690–4.
22. Davies D, Lang M, Watts R. Paediatric hanging and strangulation injuries: a 10-year retrospective description of clinical factors and outcomes. Paediatr Child Health 2011;16:e78–81.
23. Penney DJ, Stewart AHL, Parr MJA. Prognostic outcome indicators following hanging injuries. Resuscitation 2002;54:27–9.
24. Wee JH, Park JH, Choi SP, Park KN. Outcomes of patients admitted for hanging injuries with decreased consciousness but without cardiac arrest. Am J Emerg Med 2013;31:1666–70.
25. Muraro A, Roberts G, Worm M, et al. Anaphylaxis: guidelines from the European Academy of Allergy and Clinical Immunology. Allergy 2014;69:1026–45.
26. Kleber C, Giesecke MT, Lindner T, Haas NP, Buschmann CT. Requirement for a structured algorithm in cardiac arrest following major trauma: epidemiology, management errors, and preventability of traumatic deaths in Berlin. Resuscitation 2014;85:405–10.

27. Brenner ML, Moore LJ, DuBose JJ, et al. A clinical series of resuscitative endovascular balloon occlusion of the aorta for hemorrhage control and resuscitation. J Trauma Acute Care Surg 2013;75:506–11.
28. Zwingmann J, Mehlhorn AT, Hammer T, et al. Survival and neurologic outcome after traumatic out-of-hospital cardiopulmonary arrest in a pediatric and adult population: a systematic review. Crit Care 2012;16:R117.
29. Leis CC, Hernandez CC, Blanco MJ, et al. Traumatic cardiac arrest: should advanced life support be initiated? J Trauma Acute Care Surg 2013;74:634–8.
30. Bulger EM, Perina DG, Qasim Z, et al. Clinical use of resuscitative endovascular balloon occlusion of the aorta (REBOA) in civilian trauma systems in the USA, 2019: a joint statement from the American College of Surgeons Committee on Trauma, the American College of Emergency Physicians, the National Association of Emergency Medical Services Physicians and the National Association of Emergency Medical Technicians. Trauma Surg Acute Care Open 2019;4:e000376.
31. Schimrigk J, Baulig C, Buschmann C, et al. Indications, procedure and outcome of prehospital emergency resuscitative thoracotomy-a systematic literature search. Unfallchirurg 2020;123:711–23.
32. Seamon MJ, Chovanes J, Fox N, et al. The use of emergency department thoracotomy for traumatic cardiopulmonary arrest. Injury 2012;43:1355–61.
33. Escutnaire J, Genin M, Babykina E, et al. Traumatic cardiac arrest is associated with lower survival rate vs. medical cardiac arrest—results from the French National Registry. Resuscitation 2018;131:48–54.
34. Beck B, Tohira H, Bray JE, et al. Trends in traumatic out-of-hospital cardiac arrest in Perth, Western Australia from 1997 to 2014. Resuscitation 2016;98:79–84.
35. Evans CC, Petersen A, Meier EN, et al. Prehospital traumatic cardiac arrest: management and outcomes from the resuscitation outcomes consortium epistry-trauma and PROPHET registries. J Trauma Acute Care Surg 2016;81:285–93.
36. Barnard E, Yates D, Edwards A, et al. Epidemiology and aetiology of traumatic cardiac arrest in England and Wales—a retrospective database analysis. Resuscitation 2017;110:90–4.
37. Zwingmann J, Lefering R, Feucht M, et al. Outcome and predictors for successful resuscitation in the emergency room of adult patients in traumatic cardiorespiratory arrest. Crit Care 2016;20:282.
38. Spahn DR, Bouillon B, Cerny V, et al. The European guideline on management of major bleeding and coagulopathy following trauma: fifth edition. Crit Care 2019;23:98.
39. Cera SM, Mostafa G, Sing RF, et al. Physiologic predictors of survival in post-traumatic arrest. Am Surg 2003;69:140–4.
40. Stratton SJ, Brickett K, Crammer T. Prehospital pulseless, unconscious penetrating trauma victims: field assessments associated with survival. J Trauma 1998;45:96–100.
41. Chen YC, Wu KH, Hsiao KY, et al. Factors associated with outcomes in traumatic cardiac arrest patients without prehospital return of spontaneous circulation. Injury 2019;50:4–9.
42. Seamon Mj, Haut Er, Van Arendonk K, et al. An evidence-based approach to patient selection for emergency department thoracotomy: a practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg 2015;79:159–73.
43. Djarv T, Axelsson C, Herlitz J, et al. Traumatic cardiac arrest in Sweden 1990-2016—a population-based national cohort study. Scand J Trauma Resusc Emerg Med 2018;26:30.
44. Israr S, Cook AD, Chapple KM, et al. Pulseless electrical activity following traumatic cardiac arrest: sign of life or death? Injury 2019;50:1507–10.
45. Millin MG, Galvagno SM, Khandker SR, et al. Withholding and termination of resuscitation of adult cardiopulmonary arrest secondary to trauma: resource document to the joint NAEMSP-ACSCOT position statements. J Trauma Acute Care Surg 2013;75:459–67.
46. Luna GK, Pavlin EG, Kirkman T, Copass MK, Rice CL. Hemodynamic effects of external cardiac massage in trauma shock. J Trauma 1989;29:1430–3.
47. Jeffcoach DR, Gallegos JJ, Jesty SA, et al. Use of CPR in hemorrhagic shock, a dog model. J Trauma Acute Care Surg 2016;81:27–33.
48. Watts S, Smith JE, Gwyther R, Kirkman E. Closed chest compressions reduce survival in an animal model of haemorrhage-induced traumatic cardiac arrest. Resuscitation 2019;140: 37–42.
49. Endo A, Kojima M, Hong ZJ, Otomo Y, Coimbra R. Open-chest versus closed-chest cardiopulmonary resuscitation in trauma patients with signs of life upon hospital arrival: a retrospective multicenter study. Crit Care 2020;24:541.
50. Sperry JL, Guyette FX, Brown JB, et al. Prehospital plasma during air medical transport in trauma patients at risk for hemorrhagic shock. N Engl J Med 2018;379:315–26.
51. Guyette FX, Sperry JL, Peitzman AB, et al. Prehospital blood product and crystalloid resuscitation in the severely injured patient: a secondary analysis of the prehospital air medical plasma trial. Ann Surg 2019.
52. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science with Treatment Recommendations. Resuscitation 2020156: A240–A82.
53. Yue JK, Tsolinas RE, Burke JF, et al. Vasopressor support in managing acute spinal cord injury: current knowledge. J Neurosurg Sci 2019;63:308–17.
54. Atkinson JL. The neglected prehospital phase of head injury: apnea and catecholamine surge. Mayo Clin Proc 2000;75:37–47.
55. Wilson MH, Hinds J, Grier G, et al. Impact brain apnoea—a forgotten cause of cardiovascular collapse in trauma. Resuscitation 2016;105:52–8.
56. Hudson AJ, Strandenes G, Bjerkvig CK, Svanevik M, Glassberg E. Airway and ventilation management strategies for hemorrhagic shock. To tube, or not to tube, that is the question!. J Trauma Acute Care Surg 2018;84:S77–82.
57. El-Sayed AA, Arafa SK, El-Demerdash AM. Pressure-controlled ventilation could decrease intraoperative blood loss and improve airway pressure measures during lumbar discectomy in the prone position: a comparison with volume-controlled ventilation mode. J Anaesthesiol Clin Pharmacol 2019;35:468–74.
58. Pepe PE, Roppolo LP, Fowler RL. The detrimental effects of ventilation during low-blood-flow states. Curr Opin Crit Care 2005;11:212–8.
59. Deakin CD, Davies G, Wilson A. Simple thoracostomy avoids chest drain insertion in prehospital trauma. J Trauma 1995;39: 373–4.
60. Escott ME, Gleisberg GR, Kimmel K, et al. Simple thoracostomy. Moving beyong needle decompression in traumatic cardiac arrest. JEMS 2014;39:26–32.
61. Clemency BM, Tanski CT, Rosenberg M, et al. Sufficient catheter length for pneumothorax needle decompression: a meta-analysis. Prehosp Disaster Med 2015;30:249–53.
62. Hecker M, Hegenscheid K, Volzke H, et al. Needle decompression of tension pneumothorax: population-based epidemiologic approach to adequate needle length in healthy volunteers in Northeast Germany. J Trauma Acute Care Surg 2016;80:119–24.
63. Flaris AN, Simms ER, Prat N, et al. Clamshell incision versus left anterolateral thoracotomy. Which one is faster when performing a resuscitative thoracotomy? The tortoise and the hare revisited. World J Surg 2015;39:1306–11.
64. Wise D, Davies G, Coats T, et al. Emergency thoracotomy: "how to do it". Emerg Med 2005;22:22–4.
65. Rhee PM, Acosta J, Bridgeman A, et al. Survival after emergency department thoracotomy: review of published data from the past 25 years. J Am Coll Surg 2000;190:288–98.
66. Burlew CC, Moore EE, Moore FA, et al. Western Trauma Association critical decisions in trauma: resuscitative thoracotomy. J Trauma Acute Care Surg 2012;73:1359–63.

67. Paulich S, Lockey D. Resuscitative thoracotomy. BJA Educ 2020;20:242–8.
68. National Institute for Clinical Excellence. Pre-hospital initiation of fluid replacement therapy for trauma. Technology appraisal guidance. London: National Institute for Clinical Excellence; 2004.
69. Harris T, Thomas GO, Brohi K. Early fluid resuscitation in severe trauma. BMJ 2012;345:e5752.
70. Jansen JO, Thomas R, Loudon MA, Brooks A. Damage control resuscitation for patients with major trauma. BMJ 2009;338: b1778.
71. Holcomb JB, Tilley BC, Baraniuk S, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA 2015;313:471–82.
72. Cannon JW, Khan MA, Raja AS, et al. Damage control resuscitation in patients with severe traumatic hemorrhage: a practice management guideline from the Eastern Association for the surgery of trauma. J Trauma Acute Care Surg 2017;82:605–17.
73. collaborators C-, Roberts I, Shakur H, et al. The importance of early treatment with tranexamic acid in bleeding trauma patients: an exploratory analysis of the CRASH-2 randomised controlled trial. Lancet 2011;377:1096–101 101 e1–2.
74. Ferrada P, Wolfe L, Anand RJ, et al. Use of limited transthoracic echocardiography in patients with traumatic cardiac arrest decreases the rate of nontherapeutic thoracotomy and hospital costs. J Ultrasound Med 2014;33:1829–32.
75. Sampson HA, Munoz-Furlong A, Campbell RL, et al. Second symposium on the definition and management of anaphylaxis: summary report—Second National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network symposium. J Allergy Clin Immunol 2006;117:391–7.
76. Turner PJ, Campbell DE, Motosue MS, Campbell RL. Global trends in anaphylaxis epidemiology and clinical implications. J Allergy Clin Immunol Pract 2020;8:1169–76.
77. Anagnostou K, Turner PJ. Myths, facts and controversies in the diagnosis and management of anaphylaxis. Arch Dis Child 2019;104:83–90.
78. Simons FE, Ebisawa M, Sanchez-Borges M, et al. 2015 update of the evidence base: World Allergy Organization anaphylaxis guidelines. World Allergy Organ J 2015;8:32.
79. Shaker MS, Wallace DV, Golden DBK, et al. Anaphylaxis—a 2020 practice parameter update, systematic review, and grading of recommendations, assessment, development and evaluation (GRADE) analysis. J Allergy Clin Immunol 2020;145:1082–123.
80. Garvey LH, Dewachter P, Hepner DL, et al. Management of suspected immediate perioperative allergic reactions: an international overview and consensus recommendations. Br J Anaesth 2019;123:e50–64.
81. Harper NJN, Cook TM, Garcez T, et al. Anaesthesia, surgery, and life-threatening allergic reactions: management and outcomes in the 6th National Audit Project (NAP6). Br J Anaesth 2018;121: 172–88.
82. Ebo DG, Clarke RC, Mertes PM, et al. Molecular mechanisms and pathophysiology of perioperative hypersensitivity and anaphylaxis: a narrative review. Br J Anaesth 2019;123:e38–49.
83. Dhami S, Panesar SS, Roberts G, et al. Management of anaphylaxis: a systematic review. Allergy 2014;69:168–75.
84. Truhlar A, Deakin CD, Soar J, et al. European Resuscitation Council Guidelines for resuscitation 2015: section 4. Cardiac arrest in special circumstances. Resuscitation 2015;95:148–201.
85. Visscher PK, Vetter RS, Camazine S. Removing bee stings. Lancet 1996;348:301–2.
86. Lee JA, Singletary E, Charlton N. Methods of honey bee stinger removal: a systematic review of the literature. Cureus 2020;12: e8078.
87. Sheikh A, Shehata YA, Brown SG, Simons FE. Adrenaline for the treatment of anaphylaxis: cochrane systematic review. Allergy 2009;64:204–12.
88. Singletary EM, Zideman DA, De Buck ED, et al. Part 9: first aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. Circulation 2015;132:S269–311.
89. Kelso JM. A second dose of epinephrine for anaphylaxis: how often needed and how to carry. J Allergy Clin Immunol 2006;117:464–5.
90. Gabrielli S, Clarke A, Morris J, et al. Evaluation of prehospital management in a canadian emergency department anaphylaxis cohort. J Allergy Clin Immunol Pract 20197: 2232-8 e3.
91. Pumphrey RSH. Fatal posture in anaphylactic shock. J Allergy Clin Immunol 2003;112:451–2.
92. Rhodes A, Evans LE, Alhazzani W, et al. Surviving sepsis campaign: International Guidelines for management of sepsis and septic shock: 2016. Intensive Care Med 2017;43:304–77.
93. Padhi S, Bullock I, Li L, et al. Intravenous fluid therapy for adults in hospital: summary of NICE guidance. BMJ 2013;347:f7073.
94. Ruiz-Garcia M, Bartra J, Alvarez O, et al. Cardiovascular changes during peanut-induced allergic reactions in human subjects. J Allergy Clin Immunol 2020.
95. O'Driscoll BR, Howard LS, Earis J, et al. BTS guideline for oxygen use in adults in healthcare and emergency settings. Thorax 2017;72: ii1–ii90.
96. Liyanage CK, Galappatthy P, Seneviratne SL. Corticosteroids in management of anaphylaxis; a systematic review of evidence. Eur Ann Allergy Clin Immunol 2017;49:196–207.
97. Nurmatov UB, Rhatigan E, Simons FE, Sheikh A. H2-antihistamines for the treatment of anaphylaxis with and without shock: a systematic review. Ann Allergy Asthma Immunol 2014;112:126–31.
98. Choo KJ, Simons E, Sheikh A. Glucocorticoids for the treatment of anaphylaxis: cochrane systematic review. Allergy 2010;65: 1205–11.
99. Alqurashi W, Ellis AK. Do corticosteroids prevent biphasic anaphylaxis? J Allergy Clin Immunol Pract 2017;5:1194–205.
100. Lee S, Bellolio MF, Hess EP, Campbell RL. Predictors of biphasic reactions in the emergency department for patients with anaphylaxis. J Allergy Clin Immunol Pract 2014;2:281–7.
101. Soar J, Carli P, Couper K, et al. European Resuscitation Council Guidelines 2021: advanced life support. Resuscitation 2021161:.
102. Harper NJN, Nolan JP, Soar J, Cook TM. Why chest compressions should start when systolic arterial blood pressure is below 50 mmHg in the anaesthetised patient. Br J Anaesth 2020;124:234–8.
103. Soar J, Maconochie I, Wyckoff MH, et al. 2019 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2019;145:95–150.
104. Soar J, Pumphrey R, Cant A, et al. Emergency treatment of anaphylactic reactions—guidelines for healthcare providers. Resuscitation 2008;77:157–69.
105. Excellence NIfHaC. Sepsis: recognition, assessment and eraly management. NICE Guideline 51. London: National Insitute for Health and Care Excellence; 2016.
106. Levy MM, Evans LE, Rhodes A. The surviving sepsis campaign bundle: 2018 update. Intensive Care Med 2018;44:925–8.
107. Levy MM, Dellinger RP, Townsend SR, et al. The surviving sepsis campaign: results of an international guideline-based performance improvement program targeting severe sepsis. Crit Care Med 2010;38:367–74.
108. Levy MM, Rhodes A, Phillips GS, et al. Surviving sepsis campaign: association between performance metrics and outcomes in a 7.5-year study. Crit Care Med 2015;43:3–12.
109. Asfar P, Meziani F, Hamel JF, et al. High versus low blood-pressure target in patients with septic shock. N Engl J Med 2014;370:1583–93.
110. Lamontagne F, Meade MO, Hebert PC, et al. Higher versus lower blood pressure targets for vasopressor therapy in shock: a multicentre pilot randomized controlled trial. Intensive Care Med 2016;42:542–50.
111. Gu WJ, Zhang Z, Bakker J. Early lactate clearance-guided therapy in patients with sepsis: a meta-analysis with trial sequential analysis of randomized controlled trials. Intensive Care Med 2015;41:1862–3.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

112. Simpson SQ, Gaines M, Hussein Y, Badgett RG. Early goal-directed therapy for severe sepsis and septic shock: a living systematic review. J Crit Care 2016;36:43–8.
113. Acheampong A, Vincent JL. A positive fluid balance is an independent prognostic factor in patients with sepsis. Crit Care 2015;19:251.
114. Brotfain E, Koyfman L, Toledano R, et al. Positive fluid balance as a major predictor of clinical outcome of patients with sepsis/septic shock after ICU discharge. Am J Emerg Med 2016;34:2122–6.
115. Mitchell KH, Carlbom D, Caldwell E, et al. Volume overload: prevalence, risk factors, and functional outcome in survivors of septic shock. Ann Am Thorac Soc 2015;12:1837–44.
116. Alfonzo A, Harris A, Baines R, Chu A, Mann S. Clinical practice guidelines treatment of acute hyperkalaemia in adults. London: The Renal Association; 2020.
117. Moore ML, Bailey RR. Hyperkalaemia in patients in hospital. N Z Med J 1989;102:557–8.
118. Shemer J, Modan M, Ezra D, Cabili S. Incidence of hyperkalemia in hospitalized patients. Isr J Med Sci 1983;19:659–61.
119. Conway R, Creagh D, Byrne DG, O'Riordan D, Silke B. Serum potassium levels as an outcome determinant in acute medical admissions. Clin Med (Lond) 2015;15:239–43.
120. Wallmuller C, Meron G, Kurkciyan I, et al. Causes of in-hospital cardiac arrest and influence on outcome. Resuscitation 2012;83:1206–11.
121. Wang CH, Huang CH, Chang WT, et al. The effects of calcium and sodium bicarbonate on severe hyperkalaemia during cardiopulmonary resuscitation: a retrospective cohort study of adult in-hospital cardiac arrest. Resuscitation 2016;98:105–11.
122. Saarinen S, Nurmi J, Toivio T, et al. Does appropriate treatment of the primary underlying cause of PEA during resuscitation improve patients' survival? Resuscitation 2012;83:819–22.
123. Smellie WS. Spurious hyperkalaemia. BMJ 2007;334:693–5.
124. Sevastos N, Theodossiades G, Archimandritis AJ. Pseudohyperkalemia in serum: a new insight into an old phenomenon. Clin Med Res 2008;6:30–2.
125. Ahn S, Kim WY, Sohn CH, et al. Potassium values in cardiac arrest patients measured with a point-of-care blood gas analyzer. Resuscitation 2011;82:e25–6.
126. Allardet-Servent J, Lebsir M, Dubroca C, et al. Point-of-care versus central laboratory measurements of hemoglobin, hematocrit, glucose, bicarbonate and electrolytes: a prospective observational study in critically ill patients. PLoS One 2017;12:e0169593.
127. Dashevsky M, Bernstein SL, Barsky CL, Taylor RA. Agreement between serum assays performed in ED point-of-care and hospital central laboratories. West J Emerg Med 2017;18:403–9.
128. Acikgoz SB, Genc AB, Sipahi S, et al. Agreement of serum potassium measured by blood gas and biochemistry analyzer in patients with moderate to severe hyperkalemia. Am J Emerg Med 2016;34:794–7.
129. Durfey N, Lehnhof B, Bergeson A, et al. Severe hyperkalemia: can the electrocardiogram risk stratify for short-term adverse events? West J Emerg Med 2017;18:963–71.
130. Batterink J, Lin J, Au-Yeung SH, Cessford T. Effectiveness of sodium polystyrene sulfonate for short-term treatment of hyperkalemia. Can J Hosp Pharm 2015;68:296–303.
131. Moussavi K, Nguyen LT, Hua H, Fitter S. Comparison of IV insulin dosing strategies for hyperkalemia in the emergency department. Crit Care Explor 2020;2:e0092.
132. Farina N, Anderson C. Impact of dextrose dose on hypoglycemia development following treatment of hyperkalemia. Ther Adv Drug Saf 2018;9:323–9.
133. Apel J, Reutrakul S, Baldwin D. Hypoglycemia in the treatment of hyperkalemia with insulin in patients with end-stage renal disease. Clin Kidney J 2014;7:248–50.
134. Coca A, Valencia AL, Bustamante J, Mendiluce A, Floege J. Hypoglycemia following intravenous insulin plus glucose for hyperkalemia in patients with impaired renal function. PLoS One 2017;12:e0172961.
135. Scott NL, Klein LR, Cales E, Driver BE. Hypoglycemia as a complication of intravenous insulin to treat hyperkalemia in the emergency department. Am J Emerg Med 2019;37:209–13.
136. Boughton CK, Dixon D, Goble E, et al. Preventing hypoglycemia following treatment of hyperkalemia in hospitalized patients. J Hosp Med 2019;14:284–7.
137. LaRue HA, Peksa GD, Shah SC. A comparison of insulin doses for the treatment of hyperkalemia in patients with renal insufficiency. Pharmacotherapy 2017;37:1516–22.
138. Garcia J, Pintens M, Morris A, et al. Reduced versus conventional dose insulin for hyperkalemia treatment. J Pharm Pract 2020; 33:262–6.
139. Pierce DA, Russell G, Pirkle Jr JL. Incidence of hypoglycemia in patients with low eGFR treated with insulin and dextrose for hyperkalemia. Ann Pharmacother 2015;49:1322–6.
140. Allon M, Copkney C. Albuterol and insulin for treatment of hyperkalemia in hemodialysis patients. Kidney Int 1990;38:869–72.
141. Ahmed J, Weisberg LS. Hyperkalemia in dialysis patients. Semin Dial 2001;14:348–56.
142. Lens XM, Montoliu J, Cases A, Campistol JM, Revert L. Treatment of hyperkalaemia in renal failure: salbutamol v. insulin. Nephrol Dial Transplant 1989;4:228–32.
143. Excellence NIfHaC. Sodium zirconium cyclosilicate for treating hyperkalaemia. Technology Appraisal Guidance TA599. London: National Institute for Health and Care Excellence; 2019.
144. Excellence NIfHaC. Patiromer for treating hyperkalaemia. Technology Appraisal Guidance TA623. London: National Institute for Health and Care Excellence; 2020.
145. Zeneca Astra. Lokelma (sodium zirconium cyclosilicate) for oral suspension: Summary of product characteristics. www.ema.europa.eu/ema/.
146. Kosiborod M, Rasmussen HS, Lavin P, et al. Effect of sodium zirconium cyclosilicate on potassium lowering for 28 days among outpatients with hyperkalemia: the HARMONIZE randomized clinical trial. JAMA 2014;312:2223–33.
147. Spinowitz BS, Fishbane S, Pergola PE, et al. Sodium zirconium cyclosilicate among individuals with hyperkalemia: A 12-month phase 3 study. Clin J Am Soc Nephrol 2019;14:798–809.
148. Meaney CJ, Beccari MV, Yang Y, Zhao J. Systematic review and meta-analysis of patiromer and sodium zirconium cyclosilicate: a new armamentarium for the treatment of hyperkalemia. Pharmacotherapy 2017;37:401–11.
149. Rafique Z, Liu M, Staggers KA, Minard CG, Peacock WF. Patiromer for treatment of hyperkalemia in the emergency department: a pilot study. Acad Emerg Med 2020;27:54–60.
150. Peacock WF, Rafique Z, Vishnevskiy K, et al. Emergency potassium normalization treatment including sodium zirconium cyclosilicate: a phase II, randomized, double-blind, placebo-controlled study (ENERGIZE). Acad Emerg Med 2020;27:475–86.
151. Lin JL, Lim PS, Leu ML, Huang CC. Outcomes of severe hyperkalemia in cardiopulmonary resuscitation with concomitant hemodialysis. Intensive Care Med 1994;20:287–90.
152. Kao KC, Huang CC, Tsai YH, Lin MC, Tsao TC. Hyperkalemic cardiac arrest successfully reversed by hemodialysis during cardiopulmonary resuscitation: case report. Changgeng Yi Xue Za Zhi 2000;23:555–9.
153. Chiu CC, Yen HH, Chen YL, Siao FY. Severe hyperkalemia with refractory ventricular fibrillation: successful resuscitation using extracorporeal membrane oxygenation. Am J Emerg Med 201432: 943 e5–6.
154. Kim Sh, Song Jh, Jung Kt. Combination of extracorporeal membrane oxygenation and inline hemofiltration for the acute hyperkalemic cardiac arrest in a patient with Duchenne muscular dystrophy following orthopedic surgery—a case report. Korean J Anesthesiol 2019;72:178–83.
155. Klingkowski U, Kropshofer G, Crazzolara R, Schachner T, Cortina G. Refractory hyperkalaemic cardiac arrest—what to do first: treat the reversible cause or initiate E-CPR? Resuscitation 2019;142:81.

156. Jensen HK, Brabrand M, Vinholt PJ, Hallas J, Lassen AT. Hypokalemia in acute medical patients: risk factors and prognosis. Am J Med 2015128: 60–7 e1.
157. Chen Y, Chang AR, McAdams DeMarco MA, et al. Serum potassium, mortality, and kidney outcomes in the atherosclerosis risk in communities study. Mayo Clin Proc 2016;91:1403–12.
158. Skogestad J, Aronsen JM. Hypokalemia-induced arrhythmias and heart failure: new insights and implications for therapy. Front Physiol 2018;9:1500.
159. Steiness E, Olesen KH. Cardiac arrhythmias induced by hypokalaemia and potassium loss during maintenance digoxin therapy. Br Heart J 1976;38:167–72.
160. Kardalas E, Paschou SA, Anagnostis P, et al. Hypokalemia: a clinical update. Endocr Connect 20187: R135–R46.
161. Huang CL, Kuo E. Mechanism of hypokalemia in magnesium deficiency. J Am Soc Nephrol 2007;18:2649–52.
162. Pasquier M, Hugli O, Paal P, et al. Hypothermia outcome prediction after extracorporeal life support for hypothermic cardiac arrest patients: the HOPE score. Resuscitation 2018;126:58–64.
163. Pasquier M, Rousson V, Darocha T, et al. Hypothermia outcome prediction after extracorporeal life support for hypothermic cardiac arrest patients: an external validation of the HOPE score. Resuscitation 2019;139:321–8.
164. Frei C, Darocha T, Debaty G, et al. Clinical characteristics and outcomes of witnessed hypothermic cardiac arrest: a systematic review on rescue collapse. Resuscitation 2019;137:41–8.
165. Podsiadlo P, Darocha T, Svendsen OS, et al. Outcomes of patients suffering unwitnessed hypothermic cardiac arrest rewarmed with extracorporeal life support: a systematic review. Artif Organs 2020.
166. Fujimoto Y, Matsuyama T, Morita S, et al. Indoor versus outdoor occurrence in mortality of accidental hypothermia in Japan: the J-point registry. Ther Hypothermia Temp Manag 2020;10:159–64.
167. Paal P, Rauch S. Indoor accidental hypothermia in the elderly: an emerging lethal entity in the 21st century. Emerg Med 2018;35: 667–8.
168. Strapazzon G, Procter E, Paal P, Brugger H. Pre-hospital core temperature measurement in accidental and therapeutic hypothermia. High Alt Med Biol 2014;15:104–11.
169. Pasquier M, Paal P, Kosinski S, et al. Esophageal temperature measurement. N Engl J Med 2020;383:e93.
170. Strapazzon G, Procter E, Putzer G, et al. Influence of low ambient temperature on epitympanic temperature measurement: a prospective randomized clinical study. Scand J Trauma Resusc Emerg Med 2015;23:90.
171. Muth CM, Shank E, Hauser B, et al. Infrared ear thermometry in water-related accidents-not a good choice. J Emerg Med 2010;38:417–21.
172. Henriksson O, Lundgren PJ, Kuklane K, et al. Protection against cold in prehospital care: wet clothing removal or addition of a vapor barrier. Wilderness Environ Med 2015;26:11–20.
173. Lundgren P, Henriksson O, Naredi P, Bjornstig U. The effect of active warming in prehospital trauma care during road and air ambulance transportation—a clinical randomized trial. Scand J Trauma Resusc Emerg Med 2011;19:59.
174. Darocha T, Kosinski S, Jarosz A, et al. Should capnography be used as a guide for choosing a ventilation strategy in circulatory shock caused by severe hypothermia? Observational case-series study. Scand J Trauma Resusc Emerg Med 2017;25:15.
175. Pasquier M, Zurron N, Weith B, et al. Deep accidental hypothermia with core temperature below 24 degrees c presenting with vital signs. High Alt Med Biol 2014;15:58–63.
176. Mroczek T, Gladki M, Skalski J. Successful resuscitation from accidental hypothermia of 11.8 degrees C: where is the lower bound for human beings? Eur J Cardiothorac Surg 2020;58:1091–2.
177. Stephen CR, Dent SJ, Hall KD, Smith WW. Physiologic reactions during profound hypothermia with cardioplegia. Anesthesiology 1961;22:873–81.
178. Brugger H, Bouzat P, Pasquier M, et al. Cut-off values of serum potassium and core temperature at hospital admission for extracorporeal rewarming of avalanche victims in cardiac arrest: a retrospective multi-centre study. Resuscitation 2019;139:222–9.
179. Saczkowski Rs, Brown Dja, Abu-Laban Rb, et al. Prediction and risk stratification of survival in accidental hypothermia requiring extracorporeal life support: an individual patient data meta-analysis. Resuscitation 2018;127:51–7.
180. Gordon L, Paal P. Normothermic and hypothermic cardiac arrest-Beware of Jekyll and Hyde. Resuscitation 2018129: e10–e1.
181. Pasquier M, Paal P, Blancher M, Darocha T. Higher survival chances of hypothermic vs. normothermic cardiac arrest patients with ECLS re-warming. Resuscitation 2019;134:161–2.
182. Althaus U, Aeberhard P, Schupbach P, Nachbur BH, Muhlemann W. Management of profound accidental hypothermia with cardiorespiratory arrest. Ann Surg 1982;195:492–5.
183. Lexow K. Severe accidental hypothermia: survival after 6 hours 30 minutes of cardiopulmonary resuscitation. Arctic Med Res 1991;50:112–4.
184. Wood S. Interactions between hypoxia and hypothermia. Annu Rev Physiol 1991;53:71–85.
185. Mair P, Gasteiger L, Mair B, Stroehle M, Walpoth B. Successful defibrillation of four hypothermic patients with witnessed cardiac arrest. High Alt Med Biol 2019;20:71–7.
186. Stoner J, Martin G, O'Mara K, Ehlers J, Tomlanovich M. Amiodarone and bretylium in the treatment of hypothermic ventricular fibrillation in a canine model. Acad Emerg Med 2003;10:187–91.
187. Krismer AC, Lindner KH, Kornberger R, et al. Cardiopulmonary resuscitation during severe hypothermia in pigs: does epinephrine or vasopressin increase coronary perfusion pressure? Anesth Analg 2000;90:69–73.
188. Kornberger E, Lindner KH, Mayr VD, et al. Effects of epinephrine in a pig model of hypothermic cardiac arrest and closed-chest cardiopulmonary resuscitation combined with active rewarming. Resuscitation 2001;50:301–8.
189. Mazur P, Kosinski S, Podsiadlo P, et al. Extracorporeal membrane oxygenation for accidental deep hypothermia-current challenges and future perspectives. Ann Cardiothorac Surg 2019;8:137–42.
190. Ruttmann E, Weissenbacher A, Ulmer H, et al. Prolonged extracorporeal membrane oxygenation-assisted support provides improved survival in hypothermic patients with cardiocirculatory arrest. J Thorac Cardiovasc Surg 2007;134:594–600.
191. Gruber E, Beikircher W, Pizzinini R, et al. Non-extracorporeal rewarming at a rate of 6.8 degrees C per hour in a deeply hypothermic arrested patient. Resuscitation 2014;85:e119–20.
192. Kuhnke M, Albrecht R, Schefold JC, Paal P. Successful resuscitation from prolonged hypothermic cardiac arrest without extracorporeal life support: a case report. J Med Case Rep 2019;13:354.
193. Boue Y, Payen JF, Brun J, et al. Survival after avalanche-induced cardiac arrest. Resuscitation 2014;85:1192–6.
194. Moroder L, Mair B, Brugger H, Voelckel W, Mair P. Outcome of avalanche victims with out-of-hospital cardiac arrest. Resuscitation 2015;89:114–8.
195. Metrailler-Mermoud J, Hugli O, Carron PN, et al. Avalanche victims in cardiac arrest are unlikely to survive despite adherence to medical guidelines. Resuscitation 2019;141:35–43.
196. Brugger H, Durrer B, Elsensohn F, et al. Resuscitation of avalanche victims: evidence-based guidelines of the international commission for mountain emergency medicine (ICAR MEDCOM): intended for physicians and other advanced life support personnel. Resuscitation 2013;84:539–46.
197. Van Tilburg C, Grissom CK, Zafren K, et al. Wilderness Medical Society Practice Guidelines for prevention and management of avalanche and nonavalanche snow burial accidents. Wilderness Environ Med 2017;28:23–42.
198. Kay JE. Early climate models successfully predicted global warming. Nature 2020;578:45–6.
199. Epstein Y, Yanovich R. Heatstroke. N Engl J Med 2019;380: 2449–59.

200. Robine JM, Cheung SL, Le Roy S, et al. Death toll exceeded 70,000 in Europe during the summer of 2003. C R Biol 2008;331:171–8.
201. Hayashida K, Shimizu K, Yokota H. Severe heatwave in Japan. Acute Med Surg 2019;6:206–7.
202. Lipman GS, Gaudio FG, Eifling KP, et al. Wilderness Medical Society Clinical Practice Guidelines for the prevention and treatment of heat illness: 2019 update. Wilderness Environ Med 2019;30:S33–46.
203. Douma MJ, Aves T, Allan KS, et al. First aid cooling techniques for heat stroke and exertional hyperthermia: a systematic review and meta-analysis. Resuscitation 2020;148:173–90.
204. Shapiro Y, Seidman DS. Field and clinical observations of exertional heat stroke patients. Med Sci Sports Exerc 1990;22:6–14.
205. McDermott BP, Casa DJ, Ganio MS, et al. Acute whole-body cooling for exercise-induced hyperthermia: a systematic review. J Athl Train 2009;44:84–93.
206. Hew-Butler T, Rosner MH, Fowkes-Godek S, et al. Statement of the 3rd International Exercise-Associated Hyponatremia Consensus Development Conference, Carlsbad, California, 2015. Br J Sports Med 2015;49:1432–46.
207. Bouchama A, Dehbi M, Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical recommendations. Crit Care 2007;11:R54.
208. Litman RS, Griggs SM, Dowling JJ, Riazi S. Malignant hyperthermia susceptibility and related diseases. Anesthesiology 2018;128: 159–67.
209. Riazi S, Kraeva N, Hopkins PM. Malignant hyperthermia in the post-genomics era: new perspectives on an old concept. Anesthesiology 2018;128:168–80.
210. Hall AP, Henry JA. Acute toxic effects of 'Ecstasy' (MDMA) and related compounds: overview of pathophysiology and clinical management. Br J Anaesth 2006;96:678–85.
211. Eshel G, Safar P, Sassano J, Stezoski W. Hyperthermia-induced cardiac arrest in dogs and monkeys. Resuscitation 1990;20:129–43.
212. Zeiner A, Holzer M, Sterz F, et al. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. Arch Intern Med 2001;161:2007–12.
213. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J 2020;41:543–603.
214. Javaudin F, Lascarrou JB, Le Bastard Q, et al. Thrombolysis during resuscitation for out-of-hospital cardiac arrest caused by pulmonary embolism increases 30-day survival: findings from the French National Cardiac Arrest Registry. Chest 2019;156:1167–75.
215. Bottiger BW, Arntz HR, Chamberlain DA, et al. Thrombolysis during resuscitation for out-of-hospital cardiac arrest. N Engl J Med 2008;359:2651–62.
216. Bergum D, Nordseth T, Mjolstad OC, Skogvoll E, Haugen BO. Causes of in-hospital cardiac arrest—incidences and rate of recognition. Resuscitation 2015;87:63–8.
217. Kurkciyan I, Meron G, Sterz F, et al. Pulmonary embolism as a cause of cardiac arrest: presentation and outcome. Arch Intern Med 2000;160:1529–35.
218. Pokorna M, Necas E, Skripsky R, et al. How accurately can the aetiology of cardiac arrest be established in an out-of-hospital setting? Analysis by "concordance in diagnosis crosscheck tables". Resuscitation 2011;82:391–7.
219. Aagaard R, Lofgren B, Caap P, et al. A low end-tidal CO_2/arterial CO_2 ratio during cardiopulmonary resuscitation suggests pulmonary embolism. Resuscitation 2018;133:137–40.
220. Group S-KS, Inokuchi S, Masui Y, et al. Investigation and treatment of pulmonary embolism as a potential etiology may be important to improve post-resuscitation prognosis in non-shockable out-of-hospital cardiopulmonary arrest: report on an analysis of the SOS-KANTO 2012 study. Acute Med Surg 2016;3:250–9.
221. Heradstveit BE, Sunde K, Sunde GA, Wentzel-Larsen T, Heltne JK. Factors complicating interpretation of capnography during advanced life support in cardiac arrest—a clinical retrospective study in 575 patients. Resuscitation 2012;83:813–8.
222. White RH. The epidemiology of venous thromboembolism. Circulation 2003;107:I4–8.
223. Geibel A, Zehender M, Kasper W, et al. Prognostic value of the ECG on admission in patients with acute major pulmonary embolism. Eur Respir J 2005;25:843–8.
224. Bova C, Greco F, Misuraca G, et al. Diagnostic utility of echocardiography in patients with suspected pulmonary embolism. Am J Emerg Med 2003;21:180–3.
225. Wan S, Quinlan DJ, Agnelli G, Eikelboom JW. Thrombolysis compared with heparin for the initial treatment of pulmonary embolism: a meta-analysis of the randomized controlled trials. Circulation 2004;110:744–9.
226. Goldhaber SZ, Haire WD, Feldstein ML, et al. Alteplase versus heparin in acute pulmonary embolism: randomised trial assessing right-ventricular function and pulmonary perfusion. Lancet 1993;341:507–11.
227. Janata K, Holzer M, Kurkciyan I, et al. Major bleeding complications in cardiopulmonary resuscitation: the place of thrombolytic therapy in cardiac arrest due to massive pulmonary embolism. Resuscitation 2003;57:49–55.
228. Yousuf T, Brinton T, Ahmed K, et al. Tissue plasminogen activator use in cardiac arrest secondary to fulminant pulmonary embolism. J Clin Med Res 2016;8:190–5.
229. Böttiger BW, Böhrer H, Bach A, Motsch J, Martin E. Bolus injection of thrombolytic agents during cardiopulmonary resuscitation for massive pulmonary embolism. Resuscitation 1994;28:45–54.
230. Wu JP, Gu DY, Wang S, et al. Good neurological recovery after rescue thrombolysis of presumed pulmonary embolism despite prior 100 minutes CPR. J Thorac Dis 2014;6:E289–93.
231. Summers K, Schultheis J, Raiff D, Dahhan T. Evaluation of rescue thrombolysis in cardiac arrest secondary to suspected or confirmed pulmonary embolism. Ann Pharmacother 2019;53:711–5.
232. Doerge HC, Schoendube FA, Loeser H, Walter M, Messmer BJ. Pulmonary embolectomy: review of a 15-year experience and role in the age of thrombolytic therapy. Eur J Cardiothorac Surg 1996;10:952–7.
233. Konstantinov IE, Saxena P, Koniuszko MD, Alvarez J, Newman MA. Acute massive pulmonary embolism with cardiopulmonary resuscitation: management and results. Tex Heart Inst J 2007;34:41–5 discussion 5–6.
234. Fava M, Loyola S, Bertoni H, Dougnac A. Massive pulmonary embolism: percutaneous mechanical thrombectomy during cardiopulmonary resuscitation. J Vasc Interv Radiol 2005;16: 119–23.
235. Conrad SA, Broman LM, Taccone FS, et al. The Extracorporeal Life Support Organization Maastricht Treaty for nomenclature in extracorporeal life support. A position paper of the Extracorporeal Life Support Organization. Am J Respir Crit Care Med 2018;198: 447–51.
236. Maj G, Melisurgo G, De Bonis M, Pappalardo F. ECLS management in pulmonary embolism with cardiac arrest: which strategy is better? Resuscitation 2014;85:e175–6.
237. Swol J, Buchwald D, Strauch J, Schildhauer TA. Extracorporeal life support (ECLS) for cardiopulmonary resuscitation (CPR) with pulmonary embolism in surgical patients—a case series. Perfusion 2016;31:54–9.
238. Soar J, Maconochie I, Wyckoff MH, et al. 2019 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations: summary from the basic life support; advanced life support; pediatric life support; neonatal life support; Education, Implementation, and Teams; and First Aid Task Forces. Circulation 2019140: e826–e80.
239. Hayashi M, Shimizu W, Albert CM. The spectrum of epidemiology underlying sudden cardiac death. Circ Res 2015;116:1887–906.
240. Myat A, Song KJ, Rea T. Out-of-hospital cardiac arrest: current concepts. Lancet 2018;391:970–9.
241. Myerburg RJ, Junttila MJ. Sudden cardiac death caused by coronary heart disease. Circulation 2012;125:1043–52.

242. Yannopoulos D, Bartos JA, Aufderheide TP, et al. The evolving role of the cardiac catheterization laboratory in the management of patients with out-of-hospital cardiac arrest: a scientific statement from the American Heart Association. Circulation 2019139: e530–e52.
243. Nikolaou NI, Welsford M, Beygui F, et al. Part 5: acute coronary syndromes: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2015;95:e121–46.
244. Roffi M, Patrono C, Collet JP, et al. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). Eur Heart J 2016;37:267–315.
245. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018;39:119–77.
246. Piepoli MF, Hoes AW, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts) seveloped with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). Eur Heart J 2016;37:2315–81.
247. Noc M, Fajadet J, Lassen JF, et al. Invasive coronary treatment strategies for out-of-hospital cardiac arrest: a consensus statement from the European association for percutaneous cardiovascular interventions (EAPCI)/stent for life (SFL) groups. EuroIntervention 2014;10:31–7.
248. Kotseva K, De Backer G, De Bacquer D, et al. Lifestyle and impact on cardiovascular risk factor control in coronary patients across 27 countries: results from the European Society of Cardiology ESC-EORP EUROASPIRE V registry. Eur J Prev Cardiol 2019;26:824–35.
249. Rauch B, Davos CH, Doherty P, et al. The prognostic effect of cardiac rehabilitation in the era of acute revascularisation and statin therapy: a systematic review and meta-analysis of randomized and non-randomized studies—the Cardiac Rehabilitation Outcome Study (CROS). Eur J Prev Cardiol 2016;23:1914–39.
250. Ruano-Ravina A, Pena-Gil C, Abu-Assi E, et al. Participation and adherence to cardiac rehabilitation programs. A systematic review. Int J Cardiol 2016;223:436–43.
251. Kotseva K, Wood D, De Bacquer D, Investigators E. Determinants of participation and risk factor control according to attendance in cardiac rehabilitation programmes in coronary patients in Europe: EUROASPIRE IV survey. Eur J Prev Cardiol 2018;25:1242–51.
252. Piepoli MF, Corra U, Dendale P, et al. Challenges in secondary prevention after acute myocardial infarction: a call for action. Eur J Prev Cardiol 2016;23:1994–2006.
253. Priori SG, Blomstrom-Lundqvist C, Mazzanti A, et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J 2015;36:2793–867.
254. Cartledge S, Bray JE, Leary M, Stub D, Finn J. A systematic review of basic life support training targeted to family members of high-risk cardiac patients. Resuscitation 2016;105:70–8.
255. Cartledge S, Finn J, Bray JE, et al. Incorporating cardiopulmonary resuscitation training into a cardiac rehabilitation programme: a feasibility study. Eur J Cardiovasc Nurs 2018;17:148–58.
256. Gonzalez-Salvado V, Abelairas-Gomez C, Gude F, et al. Targeting relatives: impact of a cardiac rehabilitation programme including basic life support training on their skills and attitudes. Eur J Prev Cardiol 2019;26:795–805.
257. Kalla K, Christ G, Karnik R, et al. Implementation of guidelines improves the standard of care: the Viennese registry on reperfusion strategies in ST-elevation myocardial infarction (Vienna STEMI registry). Circulation 2006;113:2398–405.
258. Ting HH, Rihal CS, Gersh BJ, et al. Regional systems of care to optimize timeliness of reperfusion therapy for ST-elevation myocardial infarction: the Mayo Clinic STEMI protocol. Circulation 2007;116:729–36.
259. Glickman SW, Lytle BL, Ou FS, et al. Care processes associated with quicker door-in-door-out times for patients with ST-elevation-myocardial infarction requiring transfer: results from a statewide regionalization program. Circ Cardiovasc Qual Outcomes 2011;4:382–8.
260. Cequier A, Ariza-Sole A, Elola FJ, et al. Impact on mortality of different network systems in the treatment of ST-segment elevation acute myocardial infarction. The Spanish experience. Rev Esp Cardiol (Engl Ed) 2017;70:155–61.
261. Jollis JG, Al-Khalidi HR, Roettig ML, et al. Impact of regionalization of ST-segment-elevation myocardial infarction care on treatment times and outcomes for emergency medical services-transported patients presenting to hospitals with percutaneous coronary intervention: mission: lifeline accelerator-2. Circulation 2018;137:376–87.
262. Filgueiras Filho NM, Feitosa Filho GS, Solla DJF, et al. Implementation of a regional network for ST-Segment-Elevation Myocardial Infarction (STEMI) care and 30-day mortality in a low- to middle-income city in Brazil: findings from Salvador's STEMI Registry (RESISST). J Am Heart Assoc 20187:.
263. Cequier A, Perez de Prado A, Moreno R, et al. Percutaneous cardiological intervention and cardiac surgery: patient-centered care. Position statement of the Spanish Society of Cardiology. Rev Esp Cardiol (Engl Ed) 2019;72:658–63.
264. Peberdy MA, Donnino MW, Callaway CW, et al. Impact of percutaneous coronary intervention performance reporting on cardiac resuscitation centers: a scientific statement from the American Heart Association. Circulation 2013;128:762–73.
265. Salam I, Hassager C, Thomsen JH, et al. Editor's choice-is the pre-hospital ECG after out-of-hospital cardiac arrest accurate for the diagnosis of ST-elevation myocardial infarction? Eur Heart J Acute Cardiovasc Care 2016;5:317–26.
266. Zanuttini D, Armellini I, Nucifora G, et al. Predictive value of electrocardiogram in diagnosing acute coronary artery lesions among patients with out-of-hospital-cardiac-arrest. Resuscitation 2013;84:1250–4.
267. Millin MG, Comer AC, Nable JV, et al. Patients without ST elevation after return of spontaneous circulation may benefit from emergent percutaneous intervention: a systematic review and meta-analysis. Resuscitation 2016;108:54–60.
268. Barbarawi M, Zayed Y, Kheiri B, et al. Optimal timing of coronary intervention in patients resuscitated from cardiac arrest without ST-segment elevation myocardial infarction (NSTEMI): a systematic review and meta-analysis. Resuscitation 2019;144:137–44.
269. Lemkes JS, Janssens GN, van der Hoeven NW, et al. Coronary angiography after cardiac arrest without ST-segment elevation. N Engl J Med 2019;380:1397–407.
270. Nolan JP, Böttiger BW, Cariou A, et al. European Resuscitation Council and European Society of Intensive Care Medicine Guidelines 2021: post-resuscitation care. Resuscitation 2021161:.
271. Mentzelopoulos SD, Couper K, Van de Voorde P, et al. European Resuscitation Council Guidelines 2021: Ethics of resuscitation and end of life decisions resuscitation. . p. 161.
272. Wang PL, Brooks SC. Mechanical versus manual chest compressions for cardiac arrest. Cochrane Database Syst Rev 2018;8:CD007260.
273. Holmberg MJ, Geri G, Wiberg S, et al. Extracorporeal cardiopulmonary resuscitation for cardiac arrest: a systematic review. Resuscitation 2018;131:91–100.

274. Bougouin W, Dumas F, Lamhaut L, et al. Extracorporeal cardiopulmonary resuscitation in out-of-hospital cardiac arrest: a registry study. Eur Heart J 2020;41:1961–71.
275. Roberts DJ, Leigh-Smith S, Faris PD, et al. Clinical presentation of patients with tension pneumothorax: a systematic review. Ann Surg 2015;261:1068–78.
276. Hilbert-Carius P, Wurmb T, Lier H, et al. Care for severely injured persons: update of the 2016 S3 guideline for the treatment of polytrauma and the severely injured. Anaesthesist 2017;66: 195–206.
277. Laan DV, Vu TD, Thiels CA, et al. Chest wall thickness and decompression failure: a systematic review and meta-analysis comparing anatomic locations in needle thoracostomy. Injury 2016;47:797–804.
278. Holcomb JB, McManus JG, Kerr ST, Pusateri AE. Needle versus tube thoracostomy in a swine model of traumatic tension hemopneumothorax. Prehosp Emerg Care 2009;13:18–27.
279. High K, Brywczynski J, Guillamondegui O. Safety and efficacy of thoracostomy in the air medical environment. Air Med J 2016;35:227–30.
280. Mowry JB, Spyker DA, Cantilena Jr LR, McMillan N, Ford M. 2013 annual report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 31st annual report. Clin Toxicol (Phila) 2014;52:1032–283.
281. Gummin DD, Mowry JB, Spyker DA, et al. 2018 annual report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 36th annual report. Clin Toxicol (Phila) 2019;57:1220–413.
282. Park JH, Shin SD, Song KJ, et al. Epidemiology and outcomes of poisoning-induced out-of-hospital cardiac arrest. Resuscitation 2012;83:51–7.
283. Safranek DJ, Eisenberg MS, Larsen MP. The epidemiology of cardiac arrest in young adults. Ann Emerg Med 1992;21:1102–6.
284. Engdahl J, Bang A, Karlson BW, Lindqvist J, Herlitz J. Characteristics and outcome among patients suffering from out of hospital cardiac arrest of non-cardiac aetiology. Resuscitation 2003;57:33–41.
285. Hess EP, Campbell RL, White RD. Epidemiology, trends, and outcome of out-of-hospital cardiac arrest of non-cardiac origin. Resuscitation 2007;72:200–6.
286. Yanagawa Y, Sakamoto T, Okada Y. Recovery from a psychotropic drug overdose tends to depend on the time from ingestion to arrival, the Glasgow Coma Scale, and a sign of circulatory insufficiency on arrival. Am J Emerg Med 2007;25:757–61.
287. Thompson TM, Theobald J, Lu J, Erickson TB. The general approach to the poisoned patient. Dis Mon 2014;60:509–24.
288. Kim M, Shin SD, Jeong S, Kwak YH, Suh GJ. Poisoning-induced out-of-hospital cardiac arrest and outcomes according to poison agent. J Korean Med Sci 2017;32:2042–50.
289. Graudins A, Lee HM, Druda D. Calcium channel antagonist and beta-blocker overdose: antidotes and adjunct therapies. Br J Clin Pharmacol 2016;81:453–61.
290. Gosselin S, Hoegberg LC, Hoffman RS, et al. Evidence-based recommendations on the use of intravenous lipid emulsion therapy in poisoning. Clin Toxicol (Phila) 2016;54:899–923.
291. Lam SH, Majlesi N, Vilke GM. Use of intravenous fat emulsion in the emergency department for the critically ill poisoned patient. J Emerg Med 2016;51:203–14.
292. Cao D, Heard K, Foran M, Koyfman A. Intravenous lipid emulsion in the emergency department: a systematic review of recent literature. J Emerg Med 2015;48:387–97.
293. de Lange DW, Sikma MA, Meulenbelt J. Extracorporeal membrane oxygenation in the treatment of poisoned patients. Clin Toxicol (Phila) 2013;51:385–93.
294. Masson R, Colas V, Parienti JJ, et al. A comparison of survival with and without extracorporeal life support treatment for severe poisoning due to drug intoxication. Resuscitation 2012;83:1413–7.
295. Benson BE, Hoppu K, Troutman WG, et al. Position paper update: gastric lavage for gastrointestinal decontamination. Clin Toxicol (Phila) 2013;51:140–6.
296. Seger D. Single-dose activated charcoal-backup and reassess. J Toxicol Clin Toxicol 2004;42:101–10.
297. Chyka PA, Seger D, Krenzelok EP, Vale JA. Position paper: single-dose activated charcoal. Clin Toxicol (Phila) 2005;43:61–87.
298. Thanacoody R, Caravati EM, Troutman B, et al. Position paper update: whole bowel irrigation for gastrointestinal decontamination of overdose patients. Clin Toxicol (Phila) 2015;53:5–12.
299. Krenzelok EP. Ipecac syrup-induced emesis . . . no evidence of benefit. Clin Toxicol (Phila) 2005;43:11–2.
300. Brahmi N, Kouraichi N, Thabet H, Amamou M. Influence of activated charcoal on the pharmacokinetics and the clinical features of carbamazepine poisoning. Am J Emerg Med 2006;24: 440–3.
301. Skinner CG, Chang AS, Matthews AS, Reedy SJ, Morgan BW. Randomized controlled study on the use of multiple-dose activated charcoal in patients with supratherapeutic phenytoin levels. Clin Toxicol (Phila) 2012;50:764–9.
302. Liss DB, Schwarz ES, Mullins ME. Sodium acetate infusion for serum and urine alkalinization. Ann Emerg Med 2017;70:601–2.
303. Proudfoot AT, Krenzelok EP, Vale JA. Position paper on urine alkalinization. J Toxicol Clin Toxicol 2004;42:1–26.
304. Sun X, Chen X, Lu J, et al. Extracorporeal treatment in children with acute severe poisoning. Medicine (Baltimore) 2019;98:e18086.
305. Haines JA, Jacobsen D, Meredith T, Pronczuk de Garbino J. International programme on chemical safety—antidotes project. J Toxicol Clin Toxicol 1997;35:125–6.
306. Betten DP, Vohra RB, Cook MD, Matteucci MJ, Clark RF. Antidote use in the critically ill poisoned patient. J Intensive Care Med 2006;21:255–77.
307. Forster V, Leroux JC. Nano-antidotes for drug overdose and poisoning. Sci Transl Med 2015;7:290ps14.
308. Eddleston M, Rajapakse S, Rajakanthan, et al. Anti-digoxin Fab fragments in cardiotoxicity induced by ingestion of yellow oleander: a randomised controlled trial. Lancet 2000;355:967–72.
309. Lapostolle F, Borron SW, Verdier C, et al. Digoxin-specific Fab fragments as single first-line therapy in digitalis poisoning. Crit Care Med 2008;36:3014–8.
310. Henry M, Kay MM, Viccellio P. Cardiogenic shock associated with calcium-channel and beta blockers: reversal with intravenous calcium chloride. Am J Emerg Med 1985;3:334–6.
311. Ramoska EA, Spiller HA, Winter M, Borys D. A one-year evaluation of calcium channel blocker overdoses: toxicity and treatment. Ann Emerg Med 1993;22:196–200.
312. Howarth DM, Dawson AH, Smith AJ, Buckley N, Whyte IM. Calcium channel blocking drug overdose: an Australian series. Hum Exp Toxicol 1994;13:161–6.
313. Strubelt O, Diederich KW. Studies of antidote therapy for nisoldipine intoxication in experimental animals. Arzneimittelforschung 1990;40:747–51.
314. Graudins A, Najafi J, Rur SM. Treatment of experimental verapamil poisoning with levosimendan utilizing a rodent model of drug toxicity. Clin Toxicol (Phila) 2008;46:50–6.
315. Graudins A, Wong KK. Comparative hemodynamic effects of levosimendan alone and in conjunction with 4-aminopyridine or calcium chloride in a rodent model of severe verapamil poisoning. J Med Toxicol 2010;6:85–93.
316. Greene SL, Gawarammana I, Wood DM, Jones AL, Dargan PI. Relative safety of hyperinsulinaemia/euglycaemia therapy in the management of calcium channel blocker overdose: a prospective observational study. Intensive Care Med 2007;33:2019–24.
317. Yuan TH, Kerns WPI, Tomaszewski CA, Ford MD, Kline JA. Insulin-glucose as adjunctive therapy for severe calcium channel antagonist poisoning. J Toxicol Clin Toxicol 1999;37:463–74.
318. Boyer EW, Shannon M. Treatment of calcium-channel-blocker intoxication with insulin infusion. N Engl J Med 2001;344: 1721–2.
319. Espinoza TR, Bryant SM, Aks SE. Hyperinsulin therapy for calcium channel antagonist poisoning: a seven-year retrospective study. Am J Ther 2013;20:29–31.

320. Kline JA, Leonova E, Raymond RM. Beneficial myocardial metabolic effects of insulin during verapamil toxicity in the anesthetized canine. Crit Care Med 1995;23:1251–63.
321. Kline JA, Raymond RM, Leonova ED, Williams TC, Watts JA. Insulin improves heart function and metabolism during non-ischemic cardiogenic shock in awake canines. Cardiovasc Res 1997;34:289–98.
322. Engebretsen KM, Kaczmarek KM, Morgan J, Holger JS. High-dose insulin therapy in beta-blocker and calcium channel-blocker poisoning. Clin Toxicol (Phila) 2011;49:277–83.
323. Holger JS, Stellpflug SJ, Cole JB, Harris CR, Engebretsen KM. High-dose insulin: a consecutive case series in toxin-induced cardiogenic shock. Clin Toxicol (Phila) 2011;49:653–8.
324. McGlinchey PG, McNeill AJ. Drug overdoses requiring temporary cardiac pacing; a study of six cases treated at Altnagelvin Hospital, Londonderry. Ulster Med J 1998;67:13–8.
325. Perichon D, Turfus S, Gerostamoulos D, Graudins A. An assessment of the in vivo effects of intravenous lipid emulsion on blood drug concentration and haemodynamics following oro-gastric amitriptyline overdose. Clin Toxicol (Phila) 2013;51:208–15.
326. Escajeda JT, Katz KD, Rittenberger JC. Successful treatment of metoprolol-induced cardiac arrest with high-dose insulin, lipid emulsion, and ECMO. Am J Emerg Med 201533: 1111 e1–4.
327. Kerns 2nd W, Schroeder D, Williams C, Tomaszewski C, Raymond R. Insulin improves survival in a canine model of acute beta-blocker toxicity. Ann Emerg Med 1997;29:748–57.
328. Cole JB, Arens AM, Laes JR, et al. High dose insulin for beta-blocker and calcium channel-blocker poisoning. Am J Emerg Med 2018;36:1817–24.
329. Fernandes CM, Daya MR. Sotalol-induced bradycardia reversed by glucagon. Can Fam Physician 1995;41:659–60 63-5.
330. Bailey B. Glucagon in beta-blocker and calcium channel blocker overdoses: a systematic review. J Toxicol Clin Toxicol 2003;41:595–602.
331. West PL, McKeown NJ, Hendrickson RG. Iatrogenic lipid emulsion overdose in a case of amlodipine poisoning. Clin Toxicol (Phila) 2010;48:393–6.
332. Kollef MH. Labetalol overdose successfully treated with amrinone and alpha-adrenergic receptor agonists. Chest 1994;105:626–7.
333. Sasyniuk BI, Jhamandas V, Valois M. Experimental amitriptyline intoxication: treatment of cardiac toxicity with sodium bicarbonate. Ann Emerg Med 1986;15:1052–9.
334. Knudsen K, Abrahamsson J. Epinephrine and sodium bicarbonate independently and additively increase survival in experimental amitriptyline poisoning. Crit Care Med 1997;25:669–74.
335. Bradberry SM, Thanacoody HK, Watt BE, Thomas SH, Vale JA. Management of the cardiovascular complications of tricyclic antidepressant poisoning: role of sodium bicarbonate. Toxicol Rev 2005;24:195–204.
336. Gunja N, Graudins A. Management of cardiac arrest following poisoning. EMA: Emerg Med Australas 2011;23:16–22.
337. Boehnert MT, Lovejoy Jr FH. Value of the QRS duration versus the serum drug level in predicting seizures and ventricular arrhythmias after an acute overdose of tricyclic antidepressants. N Engl J Med 1985;313:474–9.
338. Bou-Abboud E, Nattel S. Relative role of alkalosis and sodium ions in reversal of class I antiarrhythmic drug-induced sodium channel blockade by sodium bicarbonate. Circulation 1996;94:1954–61.
339. McCabe JL, Cobaugh DJ, Menegazzi JJ, Fata J. Experimental tricyclic antidepressant toxicity: a randomized, controlled comparison of hypertonic saline solution, sodium bicarbonate, and hyperventilation. Ann Emerg Med 1998;32:329–33.
340. Pileggi DJ, Cook AM. Neuroleptic malignant syndrome. Ann Pharmacother 2016;50:973–81.
341. Barelli A, Botti P, Della Puppa T. TBST (Toxicological Basic Support and Therapy). Bologna: Italian Resuscitation Council; 2010.
342. Roberge RJ, Francis 3rd EH. Use of naloxone in valproic acid overdose: case report and review. J Emerg Med 2002;22:67–70.
343. Sztajnkrycer MD. Valproic acid toxicity: overview and management. J Toxicol Clin Toxicol 2002;40:789–801.
344. Ghannoum M, Yates C, Galvao TF, et al. Extracorporeal treatment for carbamazepine poisoning: systematic review and recommendations from the EXTRIP workgroup. Clin Toxicol (Phila) 2014;52:993–1004.
345. Sivilotti ML. Flumazenil, naloxone and the ‘coma cocktail’. Br J Clin Pharmacol 2016;81:428–36.
346. Penninga EI, Graudal N, Ladekarl MB, Jurgens G. Adverse events associated with flumazenil treatment for the management of suspected benzodiazepine intoxication—a systematic review with meta-analyses of randomised trials. Basic Clin Pharmacol Toxicol 2016;118:37–44.
347. Hiller DB, Gregorio GD, Ripper R, et al. Epinephrine impairs lipid resuscitation from bupivacaine overdose: a threshold effect. Anesthesiology 2009;111:498–505.
348. Carreiro S, Blum J, Jay G, Hack JB. Intravenous lipid emulsion alters the hemodynamic response to epinephrine in a rat model. J Med Toxicol 2013;9:220–5.
349. Litz Rj, Popp M, Stehr Sn, Koch T. Successful resuscitation of a patient with ropivacaine-induced asystole after axillary plexus block using lipid infusion. Anaesthesia 2006;61:800–1.
350. Rosenblatt Ma, Abel M, Fischer Gw, Itzkovich Cj, Eisenkraft Jb. Successful use of a 20% lipid emulsion to resuscitate a patient after a presumed bupivacaine-related cardiac arrest. Anesthesiology 2006;105:217–8.
351. Ludot H, Tharin JY, Belouadah M, Mazoit JX, Malinovsky JM. Successful resuscitation after ropivacaine and lidocaine-induced ventricular arrhythmia following posterior lumbar plexus block in a child. Anesth Analg 2008;106:1572–4 table of contents.
352. American College of Medical Toxicology. ACMT position statement: interim guidance for the use of lipid resuscitation therapy. J Med Toxicol 2011;7:81–2.
353. Hicks SD, Salcido DD, Logue ES, et al. Lipid emulsion combined with epinephrine and vasopressin does not improve survival in a swine model of bupivacaine-induced cardiac arrest. Anesthesiology 2009;111:138–46.
354. Chou R, Korthuis PT, McCarty D, et al. Management of suspected opioid overdose with naloxone in out-of-hospital settings: a systematic review. Ann Intern Med 2017;167:867–75.
355. Gufford BT, Ainslie GR, White Jr JR, et al. Comparison of a new intranasal naloxone formulation to intramuscular naloxone: results from hypothesis-generating small clinical studies. Clin Transl Sci 2017;10:380–6.
356. Honderick T, Williams D, Seaberg D, Wears R. A prospective, randomized, controlled trial of benzodiazepines and nitroglycerine or nitroglycerine alone in the treatment of cocaine-associated acute coronary syndromes. Am J Emerg Med 2003;21:39–42.
357. Saland KE, Hillis LD, Lange RA, Cigarroa JE. Influence of morphine sulfate on cocaine-induced coronary vasoconstriction. Am J Cardiol 2002;90:810–1.
358. Lange RA, Cigarroa RG, Yancy Jr CW, et al. Cocaine-induced coronary-artery vasoconstriction. N Engl J Med 1989;321:1557–62.
359. Negus BH, Willard JE, Hillis LD, et al. Alleviation of cocaine-induced coronary vasoconstriction with intravenous verapamil. Am J Cardiol 1994;73:510–3.
360. Baumann BM, Perrone J, Hornig SE, Shofer FS, Hollander JE. Randomized, double-blind, placebo-controlled trial of diazepam, nitroglycerin, or both for treatment of patients with potential cocaine-associated acute coronary syndromes. Acad Emerg Med 2000;7:878–85.
361. Hollander JE, Hoffman RS, Gennis P, et al. Nitroglycerin in the treatment of cocaine associated chest pain—clinical safety and efficacy. J Toxicol Clin Toxicol 1994;32:243–56.
362. Pham D, Addison D, Kayani W, et al. Outcomes of beta blocker use in cocaine-associated chest pain: a meta-analysis. Emerg Med 2018;35:559–63.
363. Richards JR, Garber D, Laurin EG, et al. Treatment of cocaine cardiovascular toxicity: a systematic review. Clin Toxicol (Phila) 2016;54:345–64.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

364. King A, Dimovska M, Bisoski L. Sympathomimetic toxidromes and other pharmacological causes of acute hypertension. Curr Hypertens Rep 2018;20:8.
365. Graudins A, Stearman A, Chan B. Treatment of the serotonin syndrome with cyproheptadine. J Emerg Med 1998;16:615–9.
366. Mason PJ, Morris VA, Balcezak TJ. Serotonin syndrome. Presentation of 2 cases and review of the literature. Medicine (Baltimore) 2000;79:201–9.
367. Gillman PK. Serotonin syndrome treated with chlorpromazine. J Clin Psychopharmacol 1997;17:128–9.
368. Richelson E, Souder T. Binding of antipsychotic drugs to human brain receptors focus on newer generation compounds. Life Sci 2000;68:29–39.
369. Fortin JL, Desmettre T, Manzon C, et al. Cyanide poisoning and cardiac disorders: 161 cases. J Emerg Med 2010;38:467–76.
370. Bebarta VS, Tanen DA, Lairet J, et al. Hydroxocobalamin and sodium thiosulfate versus sodium nitrite and sodium thiosulfate in the treatment of acute cyanide toxicity in a swine (*Sus scrofa*) model. Ann Emerg Med 2010;55:345–51.
371. MacLennan L, Moiemen N. Management of cyanide toxicity in patients with burns. Burns 2015;41:18–24.
372. Hall AH, Kulig KW, Rumack BH. Suspected cyanide poisoning in smoke inhalation: complications of sodium nitrite therapy. J Toxicol Clin Exp 1989;9:3–9.
373. Kirk MA, Gerace R, Kulig KW. Cyanide and methemoglobin kinetics in smoke inhalation victims treated with the cyanide antidote kit. Ann Emerg Med 1993;22:1413–8.
374. Weaver LK. Clinical practice. Carbon monoxide poisoning. N Engl J Med 2009;360:1217–25.
375. Betterman K, Patel S. Neurologic complications of carbon monoxide intoxication. Handb Clin Neurol 2014;120:971–9.
376. Lin CH, Su WH, Chen YC, et al. Treatment with normobaric or hyperbaric oxygen and its effect on neuropsychometric dysfunction after carbon monoxide poisoning: a systematic review and meta-analysis of randomized controlled trials. Medicine (Baltimore) 2018;97:e12456.
377. Buckley NA, Juurlink DN, Isbister G, Bennett MH, Lavonas EJ. Hyperbaric oxygen for carbon monoxide poisoning. Cochrane Database Syst Rev 2011;CD002041.
378. Dries DJ, Endorf FW. Inhalation injury: epidemiology, pathology, treatment strategies. Scand J Trauma Resusc Emerg Med 2013;21:31.
379. Roderique JD, Josef CS, Feldman MJ, Spiess BD. A modern literature review of carbon monoxide poisoning theories, therapies, and potential targets for therapy advancement. Toxicology 2015;334:45–58.
380. Hoidal CR, Hall AH, Robinson MD, Kulig K, Rumack BH. Hydrogen sulfide poisoning from toxic inhalations of roofing asphalt fumes. Ann Emerg Med 1986;15:826–30.
381. Hall AH, Rumack BH. Hydrogen sulfide poisoning: an antidotal role for sodium nitrite? Vet Hum Toxicol 1997;39:152–4.
382. Cronican AA, Frawley KL, Ahmed H, Pearce LL, Peterson J. Antagonism of acute sulfide poisoning in mice by nitrite anion without methemoglobinemia. Chem Res Toxicol 2015;28: 1398–408.
383. Bebarta VS, Garrett N, Brenner M, et al. Efficacy of intravenous cobinamide versus hydroxocobalamin or saline for treatment of severe hydrogen sulfide toxicity in a swine (*Sus scrofa*) model. Acad Emerg Med 2017;24:1088–98.
384. Rumbeiha W, Whitley E, Anantharam P, Kim DS, Kanthasamy A. Acute hydrogen sulfide-induced neuropathology and neurological sequelae: challenges for translational neuroprotective research. Ann N Y Acad Sci 2016;1378:5–16.
385. Rendell R, Fairhall S, Graham S, et al. Assessment of *N*-acetylcysteine as a therapy for phosgene-induced acute lung injury. Toxicol Lett 2018;290:145–52.
386. Tormoehlen LM, Tekulve KJ, Nanagas KA. Hydrocarbon toxicity: a review. Clin Toxicol (Phila) 2014;52:479–89.
387. Dell'Aglio DM, Sutter ME, Schwartz MD, et al. Acute chloroform ingestion successfully treated with intravenously administered *N*-acetylcysteine. J Med Toxicol 2010;6:143–6.
388. Robinson RF, Nahata MC. Management of botulism. Ann Pharmacother 2003;37:127–31.
389. Sobel J. Diagnosis and treatment of botulism: a century later, clinical suspicion remains the cornerstone. Clin Infect Dis 2009;48:1674–5.
390. Gerardo CJ, Quackenbush E, Lewis B, et al. the efficacy of crotalidae polyvalent immune fab (ovine) antivenom versus placebo plus optional rescue therapy on recovery from copperhead snake envenomation: a randomized, double-blind, placebo-controlled, clinical trial. Ann Emerg Med 201770: 233-44 e3.
391. Currie BJ, Jacups SP. Prospective study of *Chironex fleckeri* and other box jellyfish stings in the "top end" of Australia's Northern Territory. Med J Aust 2005;183:631–6.
392. Hinkelbein J, Andres J, Thies KC, DE Robertis E. Perioperative cardiac arrest in the operating room environment: a review of the literature. Minerva Anestesiol 2017;83:1190–8.
393. Hur M, Lee HC, Lee KH, et al. The incidence and characteristics of 3-month mortality after intraoperative cardiac arrest in adults. Acta Anaesthesiol Scand 2017;61:1095–104.
394. Zuercher M, Ummenhofer W. Cardiac arrest during anesthesia. Curr Opin Crit Care 2008;14:269–74.
395. Kaiser HA, Saied NN, Kokoefer AS, et al. Incidence and prediction of intraoperative and postoperative cardiac arrest requiring cardiopulmonary resuscitation and 30-day mortality in non-cardiac surgical patients. PLoS One 2020;15:e0225939.
396. Andres J, Hinkelbein J, Bottiger BW. The stepchild of emergency medicine: sudden unexpected cardiac arrest during anaesthesia—do we need anaesthesia-centred advanced life support guidelines? Eur J Anaesthesiol 2013;30:95–6.
397. Moitra VK, Gabrielli A, Maccioli GA, O'Connor MF. Anesthesia advanced circulatory life support. Can J Anaesth 2012;59:586–603.
398. Moitra VK, Einav S, Thies KC, et al. Cardiac arrest in the operating room: resuscitation and management for the anesthesiologist: part 1. Anesth Analg 2018;126:876–88.
399. Brown J, Rogers J, Soar J. Cardiac arrest during surgery and ventilation in the prone position: a case report and systematic review. Resuscitation 2001;50:233–8.
400. Atkinson MC. The efficacy of cardiopulmonary resuscitation in the prone position. Crit Care Resusc 2000;2:188–90.
401. Mertes PM, Tajima K, Regnier-Kimmoun MA, et al. Perioperative anaphylaxis. Med Clin North Am 2010;94:761–89 xi.
402. Wolfe JW, Butterworth JF. Local anesthetic systemic toxicity: update on mechanisms and treatment. Curr Opin Anaesthesiol 2011;24:561–6.
403. Waring WS. Intravenous lipid administration for drug-induced toxicity: a critical review of the existing data. Expert Rev Clin Pharmacol 2012;5:437–44.
404. Neal JM, Mulroy MF, Weinberg GL, American Society of Regional A, Pain M.. American Society of Regional Anesthesia and Pain Medicine checklist for managing local anesthetic systemic toxicity: 2012 version. Reg Anesth Pain Med 2012;37:16–8.
405. Cave G, Harvey MG. Should we consider the infusion of lipid emulsion in the resuscitation of poisoned patients? Crit Care 2014;18:457.
406. Ozcan MS, Weinberg G. Intravenous lipid emulsion for the treatment of drug toxicity. J Intensive Care Med 2014;29:59–70.
407. Meaney PA, Bobrow BJ, Mancini ME, et al. Cardiopulmonary resuscitation quality: improving cardiac resuscitation outcomes both inside and outside the hospital: a consensus statement from the American Heart Association. Circulation 2013;128:417–35.
408. Cingi EC, McMahon LA, Prielipp RC. Novel resuscitation devices facilitate complete neurologic recovery after prolonged cardiac arrest in postanesthesia care unit. J Clin Anesth 2016;35:530–5.
409. Adam Z, Adam S, Everngam RL, et al. Resuscitation after cardiac surgery: results of an international survey. Eur J Cardiothorac Surg 2009;36:29–34.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

410. LaPar DJ, Ghanta RK, Kern JA, et al. Hospital variation in mortality from cardiac arrest after cardiac surgery: an opportunity for improvement? Ann Thorac Surg 2014;98:534–9 discussion 9-40.
411. Vakil K, Kealhofer JV, Alraies MC, et al. Long-term outcomes of patients who had cardiac arrest after cardiac operations. Ann Thorac Surg 2016;102:512–7.
412. Gupta P, Rettiganti M, Jeffries HE, et al. Risk factors and outcomes of in-hospital cardiac arrest following pediatric heart operations of varying complexity. Resuscitation 2016;105:1–7.
413. Soar J, Donnino MW, Maconochie I, et al. 2018 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations Summary. Resuscitation 2018;133:194–206.
414. Clark Sc, Dunning J, Alfieri Or, et al. EACTS guidelines for the use of patient safety checklists. Eur J Cardiothorac Surg 2012;41: 993–1004.
415. Dunning J, Fabbri A, Kolh PH, et al. Guideline for resuscitation in cardiac arrest after cardiac surgery. Eur J Cardiothorac Surg 2009;36:3–28.
416. Society of Thoracic Surgeons Task Force on Resuscitation After Cardiac S. The Society of Thoracic Surgeons expert consensus for the resuscitation of patients who arrest after cardiac surgery. Ann Thorac Surg 2017;103:1005–20.
417. Dunning J, Nandi J, Ariffin S, et al. The cardiac surgery advanced life support course (CALS): delivering significant improvements in emergency cardiothoracic care. Ann Thorac Surg 2006;81:1767–72.
418. Haynes AB, Weiser TG, Berry WR, et al. A surgical safety checklist to reduce morbidity and mortality in a global population. N Engl J Med 2009;360:491–9.
419. Lomivorotov VV, Efremov SM, Kirov MY, Fominskiy EV, Karaskov AM. Low-cardiac-output syndrome after cardiac surgery. J Cardiothorac Vasc Anesth 2017;31:291–308.
420. Pagano D, Milojevic M, Meesters Mi, et al. 2017 EACTS/EACTA Guidelines on patient blood management for adult cardiac surgery. Eur J Cardiothorac Surg 2018;53:79–111.
421. Brand J, McDonald A, Dunning J. Management of cardiac arrest following cardiac surgery. BJA Educ 2018;18:16–22.
422. Flachskampf FA, Wouters PF, Edvardsen T, et al. Recommendations for transoesophageal echocardiography: EACVI update 2014. Eur Heart J Cardiovasc Imaging 2014;15:353–65.
423. Peretto G, Durante A, Limite LR, Cianflone D. Postoperative arrhythmias after cardiac surgery: incidence, risk factors, and therapeutic management. Cardiol Res Pract 2014;2014: 615987.
424. Lockowandt U, Levine A, Strang T, Dunning J. If a patient arrests after cardiac surgery is it acceptable to delay cardiopulmonary resuscitation until you have attempted either defibrillation or pacing? Interact Cardiovasc Thorac Surg 2008;7:878–85.
425. Richardson L, Dissanayake A, Dunning J. What cardioversion protocol for ventricular fibrillation should be followed for patients who arrest shortly post-cardiac surgery? Interact Cardiovasc Thorac Surg 2007;6:799–805.
426. International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Part 4: advanced life support. Resuscitation 2005;67:213–47.
427. Twomey D, Das M, Subramanian H, Dunning J. Is internal massage superior to external massage for patients suffering a cardiac arrest after cardiac surgery? Interact Cardiovasc Thorac Surg 2008;7:151–6.
428. Tsagkataki M, Levine A, Strang T, Dunning J. Should adrenaline be routinely used by the resuscitation team if a patient suffers a cardiac arrest shortly after cardiac surgery? Interact Cardiovasc Thorac Surg 2008;7:457–62.
429. Holmberg MJ, Issa MS, Moskowitz A, et al. Vasopressors during adult cardiac arrest: a systematic review and meta-analysis. Resuscitation 2019;139:106–21.
430. Charalambous CP, Zipitis CS, Keenan DJ. Chest reexploration in the intensive care unit after cardiac surgery: a safe alternative to returning to the operating theater. Ann Thorac Surg 2006;81:191–4.
431. Mackay JH, Powell SJ, Osgathorp J, Rozario CJ. Six-year prospective audit of chest reopening after cardiac arrest. Eur J Cardiothorac Surg 2002;22:421–5.
432. Zhao Y, Xing J, Du Z, et al. Extracorporeal cardiopulmonary resuscitation for adult patients who underwent post-cardiac surgery. Eur J Med Res 2015;20:83.
433. Addala S, Kahn JK, Moccia TF, et al. Outcome of ventricular fibrillation developing during percutaneous coronary interventions in 19,497 patients without cardiogenic shock. Am J Cardiol 2005;96:764–5.
434. Mehta RH, Starr AZ, Lopes RD, et al. Incidence of and outcomes associated with ventricular tachycardia or fibrillation in patients undergoing primary percutaneous coronary intervention. JAMA 2009;301:1779–89.
435. Finn JC, Bhanji F, Lockey A, et al. Part 8: education, implementation, and teams: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2015;95:e203–24.
436. Olasveengen TM, de Caen AR, Mancini ME, et al. 2017 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations Summary. Resuscitation 2017;121:201–14.
437. Van de Walle S, Lerman A, Chevalier B, et al. Constructing a checklist for the prevention of complications during percutaneous coronary intervention. EuroIntervention 2008;4:189–92.
438. Naidu SS, Aronow HD, Box LC, et al. SCAI expert consensus statement: 2016 best practices in the cardiac catheterization laboratory: (endorsed by the Cardiological Society of India, and Sociedad Latino Americana de Cardiologia intervencionista; affirmation of value by the Canadian Association of Interventional Cardiology-Association canadienne de cardiologie d'intervention). Catheter Cardiovasc Interv 2016;88:407–23.
439. Abrams D, Garan AR, Abdelbary A, et al. Position paper for the organization of ECMO programs for cardiac failure in adults. Intensive Care Med 2018;44:717–29.
440. Cahill Tj, Clarke Sc, Simpson Ia, Stables Rh. A patient safety checklist for the cardiac catheterisation laboratory. Heart 2015;101:91–3.
441. Lindsay AC, Bishop J, Harron K, Davies S, Haxby E. Use of a safe procedure checklist in the cardiac catheterisation laboratory. BMJ Open Qual 2018;7:e000074.
442. Clattenburg EJ, Wroe PC, Gardner K, et al. Implementation of the Cardiac Arrest Sonographic Assessment (CASA) protocol for patients with cardiac arrest is associated with shorter CPR pulse checks. Resuscitation 2018;131:69–73.
443. Lien WC, Hsu SH, Chong KM, et al. US-CAB protocol for ultrasonographic evaluation during cardiopulmonary resuscitation: validation and potential impact. Resuscitation 2018;127:125–31.
444. Huis In't Veld MA, Allison MG, Bostick DS, et al. Ultrasound use during cardiopulmonary resuscitation is associated with delays in chest compressions. Resuscitation 2017;119:95–8.
445. Parker BK, Salerno A, Euerle BD. The use of transesophageal echocardiography during cardiac arrest resuscitation: a literature review. J Ultrasound Med 2019;38:1141–51.
446. Fair J, Mallin M, Mallemat H, et al. Transesophageal echocardiography: guidelines for point-of-care applications in cardiac arrest resuscitation. Ann Emerg Med 2018;71:201–7.
447. Vase H, Christensen S, Christiansen A, et al. The impella CP device for acute mechanical circulatory support in refractory cardiac arrest. Resuscitation 2017;112:70–4.
448. Garcia-Carreno J, Sousa-Casasnovas I, Devesa-Cordero C, et al. Cardiopulmonary resuscitation with percutaneous ECMO in refractory in-hospital cardiac arrest: a single-center experience. Rev Esp Cardiol (Engl Ed) 2019;72:880–2.

449. Arlt M, Philipp A, Voelkel S, et al. Early experiences with miniaturized extracorporeal life-support in the catheterization laboratory. Eur J Cardiothorac Surg 2012;42:858–63.
450. Guglin M, Zucker MJ, Bazan VM, et al. Venoarterial ECMO for adults: JACC scientific expert panel. J Am Coll Cardiol 2019;73:698–716.
451. D'Arrigo S, Cacciola S, Dennis M, et al. Predictors of favourable outcome after in-hospital cardiac arrest treated with extracorporeal cardiopulmonary resuscitation: a systematic review and meta-analysis. Resuscitation 2017;121:62–70.
452. Makar MS, Pun PH. Sudden cardiac death among hemodialysis patients. Am J Kidney Dis 2017;69:684–95.
453. Karnik JA, Young BS, Lew NL, et al. Cardiac arrest and sudden death in dialysis units. Kidney Int 2001;60:350–7.
454. Davis TR, Young BA, Eisenberg MS, et al. Outcome of cardiac arrests attended by emergency medical services staff at community outpatient dialysis centers. Kidney Int 2008;73:933–9.
455. Lafrance JP, Nolin L, Senecal L, Leblanc M. Predictors and outcome of cardiopulmonary resuscitation (CPR) calls in a large haemodialysis unit over a seven-year period. Nephrol Dial Transplant 2006;21:1006–12.
456. Bleyer AJ, Hartman J, Brannon PC, et al. Characteristics of sudden death in hemodialysis patients. Kidney Int 2006;69:2268–73.
457. Pun PH, Lehrich RW, Honeycutt EF, Herzog CA, Middleton JP. Modifiable risk factors associated with sudden cardiac arrest within hemodialysis clinics. Kidney Int 2011;79:218–27.
458. Starks MA, Wu J, Peterson ED, et al. In-hospital cardiac arrest resuscitation practices and outcomes in maintenance dialysis patients. Clin J Am Soc Nephrol 2020;15:219–27.
459. Bander Sj, Walters Ba. Hemodialysis morbidity and mortality: links to patient non-compliance. Curr Opin Nephrol Hypertens 1998;7:649–53.
460. Kovesdy CP, Regidor DL, Mehrotra R, et al. Serum and dialysate potassium concentrations and survival in hemodialysis patients. Clin J Am Soc Nephrol 2007;2:999–1007.
461. Foley RN, Gilbertson DT, Murray T, Collins AJ. Long interdialytic interval and mortality among patients receiving hemodialysis. N Engl J Med 2011;365:1099–107.
462. Moss AH, Holley JL, Upton MB. Outcomes of cardiopulmonary resuscitation in dialysis patients. J Am Soc Nephrol 1992;3:1238–43.
463. Wong SP, Kreuter W, Curtis JR, Hall YN, O'Hare AM. Trends in in-hospital cardiopulmonary resuscitation and survival in adults receiving maintenance dialysis. JAMA Intern Med 2015;175:1028–35.
464. Saeed F, Adil MM, Malik AA, Schold JD, Holley JL. Outcomes of in-hospital cardiopulmonary resuscitation in maintenance dialysis patients. J Am Soc Nephrol 2015;26:3093–101.
465. Jadoul M, Thumma J, Fuller DS, et al. Modifiable practices associated with sudden death among hemodialysis patients in the dialysis outcomes and practice patterns study. Clin J Am Soc Nephrol 2012;7:765–74.
466. Lehrich RW, Pun PH, Tanenbaum ND, Smith SR, Middleton JP. Automated external defibrillators and survival from cardiac arrest in the outpatient hemodialysis clinic. J Am Soc Nephrol 2007;18:312–20.
467. Muller MP, Hansel M, Stehr SN, Weber S, Koch T. A state-wide survey of medical emergency management in dental practices: incidence of emergencies and training experience. Emerg Med 2008;25:296–300.
468. Arsati F, Montalli VA, Florio FM, et al. Brazilian dentists' attitudes about medical emergencies during dental treatment. J Dent Educ 2010;74:661–6.
469. Girdler NM, Smith DG. Prevalence of emergency events in British dental practice and emergency management skills of British dentists. Resuscitation 1999;41:159–67.
470. Chapman Pj, Penkeyman Hw. Successful defibrillation of a dental patient in cardiac arrest. Aust Dent J 2002;47:176–7.
471. Absi EG. A cardiac arrest in the dental chair. Br Dent J 1987;163: 199–200.
472. Yokoyama T, Yoshida K, Suwa K. Efficacy of external cardiac compression in a dental chair. Resuscitation 2008;79:175–6.
473. Fujino H, Yokoyama T, Yoshida K, Suwa K. Using a stool for stabilization of a dental chair when CPR is required. Resuscitation 2010;81:502.
474. Laurent F, Segal N, Augustin P. Chest compression: not as effective on dental chair as on the floor. Resuscitation 2010;81:1729 author reply 30.
475. Lepere AJ, Finn J, Jacobs I. Efficacy of cardiopulmonary resuscitation performed in a dental chair. Aust Dent J 2003;48: 244–7.
476. Awata N, Hitosugi T, Miki Y, et al. Usefulness of a stool to stabilize dental chairs for cardiopulmonary resuscitation (CPR). BMC Emerg Med 2019;19:46.
477. Meechan JG, Skelly AM. Problems complicating dental treatment with local anaesthesia or sedation: prevention and management. Dent Update 1997;24:278–83.
478. Jowett NI, Cabot LB. Patients with cardiac disease: considerations for the dental practitioner. Br Dent J 2000;189:297–302.
479. Handley AJ, Handley JA. Performing chest compressions in a confined space. Resuscitation 2004;61:55–61.
480. Perkins GD, Stephenson BT, Smith CM, Gao F. A comparison between over-the-head and standard cardiopulmonary resuscitation. Resuscitation 2004;61:155–61.
481. Rosenberg M. Preparing for medical emergencies: the essential drugs and equipment for the dental office. J Am Dent Assoc 2010;141:14S–9S.
482. Laurent F, Augustin P, Zak C, Maman L, Segal N. Preparedness of dental practices to treat cardiac arrest: availability of defibrillators. Resuscitation 2011;82:1468–9.
483. Kandray DP, Pieren JA, Benner RW. Attitudes of Ohio dentists and dental hygienists on the use of automated external defibrillators. J Dent Educ 2007;71:480–6.
484. Breuer G, Knipfer C, Huber T, et al. Competency in managing cardiac arrest: a scenario-based evaluation of dental students. Acta Odontol Scand 2016;74:241–9.
485. Chapman PJ. A questionnaire survey of dentists regarding knowledge and perceived competence in resuscitation and occurrence of resuscitation emergencies. Aust Dent J 1995;40: 98–103.
486. Atherton GJ, Pemberton MN, Thornhill MH. Medical emergencies: the experience of staff of a UK dental teaching hospital. Br Dent J 2000;188:320–4.
487. Graf J, Stuben U, Pump S. In-flight medical emergencies. Dtsch Arztebl Int 2012;109:591–601 quiz 2.
488. Hinkelbein J. Significant more research required: no further progress without sound medical data and valid denominators for in-flight medical emergencies. J Travel Med 2015;22:355–6.
489. Hinkelbein J, Neuhaus C, Wetsch WA, et al. Emergency medical equipment on board German airliners. J Travel Med 2014;21:318–23.
490. Peterson DC, Martin-Gill C, Guyette FX, et al. Outcomes of medical emergencies on commercial airline flights. N Engl J Med 2013;368:2075–83.
491. Hinkelbein J, Neuhaus C. In-flight cardiac arrest and in-flight cardiopulmonary resuscitation during commercial air travel: consensus statement and supplementary treatment guideline from the German society of aerospace medicine (DGLRM): reply. Intern Emerg Med 2019;14:629–30.
492. Charles RA. Cardiac arrest in the skies. Singapore Med J 2011;52:582–5.
493. Hinkelbein J, Bohm L, Braunecker S, et al. In-flight cardiac arrest and in-flight cardiopulmonary resuscitation during commercial air travel: consensus statement and supplementary treatment guideline from the German Society of Aerospace Medicine (DGLRM). Intern Emerg Med 2018;13:1305–22.
494. Mahony PH, Griffiths RF, Larsen P, Powell D. Retention of knowledge and skills in first aid and resuscitation by airline cabin crew. Resuscitation 2008;76:413–8.
495. Brown AM, Rittenberger JC, Ammon CM, Harrington S, Guyette FX. In-flight automated external defibrillator use and consultation patterns. Prehosp Emerg Care 2010;14:235–9.

496. Skogvoll E, Bjelland E, Thorarinsson B. Helicopter emergency medical service in out-of-hospital cardiac arrest—a 10-year population-based study. Acta Anaesthesiol Scand 2000;44:972–9.
497. Lyon RM, Nelson MJ. Helicopter emergency medical services (HEMS) response to out-of-hospital cardiac arrest. Scand J Trauma Resusc Emerg Med 2013;21:1.
498. Hinkelbein J, Schmitz J, Glaser E. Pressure but not the fraction of oxygen is altered in the aircraft cabin. Anaesth Intensive Care 2019;47:209.
499. Verjee MA, Crone R, Ostrovskiy G. Medical issues in flight and updating the emergency medical kit. Open Access Emerg Med 2018;10:47–51.
500. Rauch S, Strapazzon G, Brodmann M, et al. Implementation of a mechanical CPR device in a physician staffed HEMS—a prospective observational study. Scand J Trauma Resusc Emerg Med 2018;26:36.
501. McIntosh SE, Swanson ER, McKeone A, Barton ED. Location of airway management in air medical transport. Prehosp Emerg Care 2008;12:438–42.
502. Das A, Majumdar S, Mukherjee A, et al. i-gel in ambulatory surgery: a comparison with LMA-ProSeal in paralyzed anaesthetized patients. J Clin Diagn Res 2014;8:80–4.
503. Ley SJ. Standards for resuscitation after cardiac surgery. Crit Care Nurse 2015;35:30–7 quiz 8.
504. Paal P, Ellerton J, Sumann G, et al. Basic life support ventilation in mountain rescue. Official recommendations of the International Commission for Mountain Emergency Medicine (ICAR MEDCOM). High Alt Med Biol 2007;8:147–54.
505. Elsensohn F, Soteras I, Resiten O, et al. Equipment of medical backpacks in mountain rescue. High Alt Med Biol 2011;12:343–7.
506. Doan TN, Schultz BV, Rashford S, Bosley E. Surviving out-of-hospital cardiac arrest: the important role of bystander interventions. Australas Emerg Care 2020;23:47–54.
507. Carron M, Emeyriat N, Levraut J, Blondeau N. Cruise ship pathologies in remote regions. Int Marit Health 2018;69:75–83.
508. Alves PM, Leigh R, Bartos G, et al. Cardiovascular events on board commercial maritime vessels: a two-year review. Int Marit Health 2010;62:137–42.
509. Marijon E, Bougouin W, Karam N, et al. Survival from sports-related sudden cardiac arrest: in sports facilities versus outside of sports facilities. Am Heart J 2015170: 339–45 e1.
510. Ackerman M, Atkins DL, Triedman JK. Sudden cardiac death in the young. Circulation 2016;133:1006–26.
511. Finocchiaro G, Papadakis M, Robertus JL, et al. Etiology of sudden death in sports: insights from a United Kingdom Regional Registry. J Am Coll Cardiol 2016;67:2108–15.
512. Landry CH, Allan KS, Connelly KA, et al. Sudden cardiac arrest during participation in competitive sports. N Engl J Med 2017;377:1943–53.
513. Northcote RJ, Flannigan C, Ballantyne D. Sudden death and vigorous exercise—a study of 60 deaths associated with squash. Br Heart J 1986;55:198–203.
514. Burtscher M, Pachinger O, Mittleman MA, Ulmer H. Prior myocardial infarction is the major risk factor associated with sudden cardiac death during downhill skiing. Int J Sports Med 2000;21:613–5.
515. Kim JH, Malhotra R, Chiampas G, et al. Cardiac arrest during long-distance running races. N Engl J Med 2012;366:130–40.
516. Harris KM, Creswell LL, Haas TS, et al. Death and cardiac arrest in U. S. triathlon participants, 1985 to 2016: a case series. Ann Intern Med 2017;167:529–35.
517. Link MS, Estes 3rd NA. Sudden cardiac death in the athlete: bridging the gaps between evidence, policy, and practice. Circulation 2012;125:2511–6.
518. Franklin BA, Thompson PD, Al-Zaiti SS, et al. Exercise-related acute cardiovascular events and potential deleterious adaptations following long-term exercise training: placing the risks into perspective-an update: a scientific statement from the American Heart Association. Circulation 2020141: e705–e36.
519. Albert CM, Mittleman MA, Chae CU, et al. Triggering of sudden death from cardiac causes by vigorous exertion. N Engl J Med 2000;343:1355–61.
520. Marijon E, Bougouin W, Jouven X. Sports-related sudden death: lessons from the French registry. Rev Prat 2015;65:919–23.
521. Kiyohara K, Nishiyama C, Kiguchi T, et al. Exercise-related out-of-hospital cardiac arrest among the general population in the era of public-access defibrillation: a population-based observation in Japan. J Am Heart Assoc 20176:.
522. Berdowski J, de Beus MF, Blom M, et al. Exercise-related out-of-hospital cardiac arrest in the general population: incidence and prognosis. Eur Heart J 2013;34:3616–23.
523. Drezner JA, Toresdahl BG, Rao AL, Huszti E, Harmon KG. Outcomes from sudden cardiac arrest in US high schools: a 2-year prospective study from the National Registry for AED use in sports. Br J Sports Med 2013;47:1179–83.
524. Mont L, Pelliccia A, Sharma S, et al. Pre-participation cardiovascular evaluation for athletic participants to prevent sudden death: position paper from the EHRA and the EACPR, branches of the ESC. Endorsed by APHRS, HRS, and SOLAECE. Eur J Prev Cardiol 2017;24:41–69.
525. Maron BJ, Friedman RA, Kligfield P, et al. Assessment of the 12-lead ECG as a screening test for detection of cardiovascular disease in healthy general populations of young people (12-25 years of age): a scientific statement from the American Heart Association and the American College of Cardiology. Circulation 2014;130:1303–34.
526. Wilson M.G.D., J.A.
527. Sharma S. IOC manual of sports cardiology. London: Wiley; 2016.
528. Armstrong M, Paternostro-Bayles M, Conroy MB, et al. Preparticipation screening prior to physical activity in community lifestyle interventions. Transl J Am Coll Sports Med 2018;3:176–80.
529. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980–2006. Circulation 2009;119:1085–92.
530. Maron BJ, Gohman TE, Kyle SB, Estes 3rd NA, Link MS. Clinical profile and spectrum of commotio cordis. Jama 2002;287:1142–6.
531. Maron BJ, Haas TS, Ahluwalia A, et al. Increasing survival rate from commotio cordis. Heart Rhythm 2013;10:219–23.
532. Seguin C, Blaquiere G, Loundou A, Michelet P, Markarian T. Unmanned aerial vehicles (drones) to prevent drowning. Resuscitation 2018;127:63–7.
533. Claesson A, Svensson L, Nordberg P, et al. Drones may be used to save lives in out of hospital cardiac arrest due to drowning. Resuscitation 2017;114:152–6.
534. Claesson A, Schierbeck S, Hollenberg J, et al. The use of drones and a machine-learning model for recognition of simulated drowning victims—a feasibility study. Resuscitation 2020;156:196–201.
535. Griffiths MJD, McAuley DF, Perkins GD, et al. Guidelines on the management of acute respiratory distress syndrome. BMJ Open Respir Res 2019;6:e000420.
536. El Sayed M, Tamim H, Mann NC. Description of procedures performed on patients by emergency medical services during mass casualty incidents in the United States. Am J Emerg Med 2015;33:1030–6.
537. Schenk E, Wijetunge G, Mann NC, et al. Epidemiology of mass casualty incidents in the United States. Prehosp Emerg Care 2014;18:408–16.
538. Turner CD, Lockey DJ, Rehn M. Pre-hospital management of mass casualty civilian shootings: a systematic literature review. Crit Care 2016;20:362.
539. Jain T, Sibley A, Stryhn H, Hubloue I. Comparison of unmanned aerial vehicle technology-assisted triage versus standard practice in triaging casualties by paramedic students in a mass-casualty incident scenario. Prehosp Disaster Med 2018;33:375–80.
540. Nolan JP, Monsieurs KG, Bossaert L, et al. European Resuscitation Council COVID-19 guidelines executive summary. Resuscitation 2020;153:45–55.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

541. Castle N, Pillay Y, Spencer N. Comparison of six different intubation aids for use while wearing CBRN-PPE: a manikin study. Resuscitation 2011;82:1548–52.
542. Lamhaut L, Dagron C, Apriotesei R, et al. Comparison of intravenous and intraosseous access by pre-hospital medical emergency personnel with and without CBRN protective equipment. Resuscitation 2010;81:65–8.
543. Castle N, Bowen J, Spencer N. Does wearing CBRN-PPE adversely affect the ability for clinicians to accurately, safely, and speedily draw up drugs? Clin Toxicol (Phila) 2010;48:522–7.
544. Tokuda Y, Kikuchi M, Takahashi O, Stein GH. Prehospital management of sarin nerve gas terrorism in urban settings: 10 years of progress after the Tokyo subway sarin attack. Resuscitation 2006;68:193–202.
545. Cross KP, Petry MJ, Cicero MX. A better START for low-acuity victims: data-driven refinement of mass casualty triage. Prehosp Emerg Care 2015;19:272–8.
546. SALT mass casualty triage: concept endorsed by the American College of Emergency Physicians, American College of Surgeons Committee on Trauma, American Trauma Society, National Association of EMS Physicians, National Disaster Life Support Education Consortium, and State and Territorial Injury Prevention Directors Association. Disaster Med Public Health Prep 2008;2:245–6.
547. Cone DC, Serra J, Burns K, et al. Pilot test of the SALT mass casualty triage system. Prehosp Emerg Care 2009;13:536–40.
548. Streckbein S, Kohlmann T, Luxen J, Birkholz T, Pruckner S. Triage protocols for mass casualty incidents: an overview 30 years after START. Unfallchirurg 2016;119:620–31.
549. Jones N, White ML, Tofil N, et al. Randomized trial comparing two mass casualty triage systems (JumpSTART versus SALT) in a pediatric simulated mass casualty event. Prehosp Emerg Care 2014;18:417–23.
550. Broach J, Hart A, Griswold M, et al. Usability and reliability of smart glasses for secondary triage during mass casualty incidents. Proc Annu Hawaii Int Conf Syst Sci 2018;2018:1416–22.
551. Risavi BL, Terrell MA, Lee W, Holsten Jr DL. Prehospital mass-casualty triage training-written versus moulage scenarios: how much do EMS providers retain? Prehosp Disaster Med 2013;28:251–6.
552. Knight JF, Carley S, Tregunna B, et al. Serious gaming technology in major incident triage training: a pragmatic controlled trial. Resuscitation 2010;81:1175–9.
553. Franklin C, Samuel J, Hu T-C. Life-threatening hypotension associated with emergency intubation and the initiation of mechanical ventilation. Am J Emerg Med 1994;12:425–8.
554. Lemaitre RN, Siscovick DS, Psaty BM, et al. Inhaled beta-2 adrenergic receptor agonists and primary cardiac arrest. Am J Med 2002;113:711–6.
555. Boucher A, Payen C, Garayt C, et al. Salbutamol misuse or abuse with fatal outcome: a case-report. Hum Exp Toxicol 2011;30:1869–71.
556. van den Berg ME, Stricker BH, Brusselle GG, Lahousse L. Chronic obstructive pulmonary disease and sudden cardiac death: a systematic review. Trends Cardiovasc Med 2016;26:606–13.
557. Salpeter SR, Ormiston TM, Salpeter EE. Cardiovascular effects of beta-agonists in patients with asthma and COPD: a meta-analysis. Chest 2004;125:2309–21.
558. Rosero SZ, Zareba W, Moss AJ, et al. Asthma and the risk of cardiac events in the Long QT syndrome. Long QT Syndrome Investigative Group. Am J Cardiol 1999;84:1406–11.
559. Granfeldt A, Wissenberg M, Hansen SM, et al. Severity of chronic obstructive pulmonary disease and presenting rhythm in patients with out-of-hospital cardiac arrest. Resuscitation 2018;126:111–7.
560. Herlitz J, Rosenfelt M, Bang A, et al. Prognosis among patients with out-of-hospital cardiac arrest judged as being caused by deterioration of obstructive pulmonary disease. Resuscitation 1996;32:177–84.
561. Leatherman JW, McArthur C, Shapiro RS. Effect of prolongation of expiratory time on dynamic hyperinflation in mechanically ventilated patients with severe asthma. Crit Care Med 2004;32:1542–5.
562. Hostetler MA, Davis CO. Bilateral localized tension pneumothoraces refractory to needle decompression. Pediatr Emerg Care 1999;15:322–4.
563. Williams-Johnson J, Williams EW, Hart N, et al. Simultaneous spontaneous bilateral pneumothoraces in an asthmatic. West Indian Med J 2008;57:508–10.
564. Komasawa N, Ueki R, Kusuyama K, et al. Case of tension pneumothorax associated with asthma attack during general anesthesia. Masui 2010;59:614–7.
565. Robert J, Derkenne C, Jost D, Tourtier JP. Out-of-hospital cardiac arrest: an underlying reversible cause. Circulation 2017;135:2564–6.
566. Burdett-Smith P, Jaffey L. Tension pneumoperitoneum. J Accid Emerg Med 1996;13:220–1.
567. Castle N, Tagg A, Owen R. Bilateral tension pneumothorax. Resuscitation 2005;65:103–5.
568. Martens P, Vandekerckhove Y, Mullie A. Restoration of spontaneous circulation after cessation of cardiopulmonary resuscitation. Lancet 1993;341:841.
569. Lapinsky SE, Leung RS. Auto-PEEP and electromechanical dissociation. N Engl J Med 1996;335:674.
570. Rogers PL, Schlichtig R, Miro A, Pinsky M. Auto-PEEP during CPR. An "occult" cause of electromechanical dissociation? Chest 1991;99:492–3.
571. Rosengarten PI, Tuxen Dv, Dziukas L, et al. Circulatory arrest induced by intermittent positive pressure ventilation in a patient with severe asthma. Anaesth Intensive Care 1991;19:118–21.
572. Sprung J, Hunter K, Barnas GM, Bourke DL. Abdominal distention is not always a sign of esophageal intubation: cardiac arrest due to "auto-PEEP". Anesth Analg 1994;78:801–4.
573. Harrison R. Chest compression first aid for respiratory arrest due to acute asphyxic asthma. Emerg Med 2010;27:59–61.
574. Fisher MM, Whaley AP, Pye RR. External chest compression in the management of acute severe asthma—a technique in search of evidence. Prehosp Disaster Med 2001;16:124–7.
575. Eason J, Tayler D, Cottam S, et al. Manual chest compression for total bronchospasm. Lancet 1991;337:366.
576. Fisher MM, Bowey CJ, Ladd-Hudson K. External chest compression in acute asthma: a preliminary study. Crit Care Med 1989;17:686–7.
577. Myles PS, Weeks AM. Cardiopulmonary resuscitation in obstructive airways disease. Lancet 1993;341:1217.
578. Perkins GD, Ji C, Deakin CD, et al. A randomized trial of epinephrine in out-of-hospital cardiac arrest. N Engl J Med 2018;379:711–21.
579. Smith D, Riel J, Tilles I, et al. Intravenous epinephrine in life-threatening asthma. Ann Emerg Med 2003;41:706–11.
580. Putland M, Kerr D, Kelly AM. Adverse events associated with the use of intravenous epinephrine in emergency department patients presenting with severe asthma. Ann Emerg Med 2006;47:559–63.
581. Yeo HJ, Kim D, Jeon D, et al. Extracorporeal membrane oxygenation for life-threatening asthma refractory to mechanical ventilation: analysis of the Extracorporeal Life Support Organization registry. Crit Care 2017;21:297.
582. Mikkelsen ME, Woo YJ, Sager JS, Fuchs BD, Christie JD. Outcomes using extracorporeal life support for adult respiratory failure due to status asthmaticus. ASAIO J 2009;55:47–52.
583. Hubner P, Meron G, Kurkciyan I, et al. Neurologic causes of cardiac arrest and outcomes. J Emerg Med 2014;47:660–7.
584. Kim AS, Moffatt E, Ursell PC, et al. Sudden neurologic death masquerading as out-of-hospital sudden cardiac death. Neurology 2016;87:1669–73.
585. Sandroni C, Dell'Anna AM. Out-of-hospital cardiac arrest from neurologic cause: recognition and outcome. Crit Care Med 2015;43:508–9.

586. Arnaout M, Mongardon N, Deye N, et al. Out-of-hospital cardiac arrest from brain cause: epidemiology, clinical features, and outcome in a multicenter cohort. Crit Care Med 2015;43:453–60.
587. Petek BJ, Erley CL, Kudenchuk PJ, et al. Diagnostic yield of non-invasive imaging in patients following non-traumatic out-of-hospital sudden cardiac arrest: a systematic review. Resuscitation 2019;135:183–90.
588. Skrifvars MB, Parr MJ. Incidence, predisposing factors, management and survival following cardiac arrest due to subarachnoid haemorrhage: a review of the literature. Scand J Trauma Resusc Emerg Med 2012;20:75.
589. Inamasu J, Miyatake S, Tomioka H, et al. Subarachnoid haemorrhage as a cause of out-of-hospital cardiac arrest: a prospective computed tomography study. Resuscitation 2009;80:977–80.
590. Shin J, Kim K, Lim YS, et al. Incidence and clinical features of intracranial hemorrhage causing out-of-hospital cardiac arrest: a multicenter retrospective study. Am J Emerg Med 2016;34: 2326–30.
591. Legriel S, Bougouin W, Chocron R, et al. Early in-hospital management of cardiac arrest from neurological cause: diagnostic pitfalls and treatment issues. Resuscitation 2018;132:147–55.
592. Mitsuma W, Ito M, Kodama M, et al. Clinical and cardiac features of patients with subarachnoid haemorrhage presenting with out-of-hospital cardiac arrest. Resuscitation 2011;82:1294–7.
593. Park I, Kim YJ, Ahn S, et al. Subarachnoid hemorrhage mimicking ST-segment elevation myocardial infarction after return of spontaneous circulation. Clin Exp Emerg Med 2015;2:260–3.
594. Zachariah J, Stanich JA, Braksick SA, et al. Indicators of subarachnoid hemorrhage as a cause of sudden cardiac arrest. Clin Pract Cases Emerg Med 2017;1:132–5.
595. Noritomi DT, de Cleva R, Beer I, et al. Doctors awareness of spontaneous subarachnoid haemorrhage as a cause of cardiopulmonary arrest. Resuscitation 2006;71:123–4.
596. Morris NA, Robinson D, Schmidt JM, et al. Hunt-Hess 5 subarachnoid haemorrhage presenting with cardiac arrest is associated with larger volume bleeds. Resuscitation 2018;123:71–6.
597. Thurman DJ, Hesdorffer DC, French JA. Sudden unexpected death in epilepsy: assessing the public health burden. Epilepsia 2014;55:1479–85.
598. Maguire MJ, Jackson CF, Marson AG, Nevitt SJ. Treatments for the prevention of Sudden Unexpected Death in Epilepsy (SUDEP). Cochrane Database Syst Rev 2020;4:CD011792.
599. Joundi RA, Rabinstein AA, Nikneshan D, et al. Cardiac arrest in acute ischemic stroke: incidence, predisposing factors, and clinical outcomes. J Stroke Cerebrovasc Dis 2016;25:1644–52.
600. Fukuda T, Ohashi-Fukuda N, Kondo Y, et al. Epidemiology, risk factors, and outcomes of out-of-hospital cardiac arrest caused by stroke: a population-based study. Medicine (Baltimore) 2016;95: e3107.
601. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in obesity among adults in the United States, 2005 to 2014. JAMA 2016;315:2284–91.
602. Koliaki C, Liatis S, Kokkinos A. Obesity and cardiovascular disease: revisiting an old relationship. Metabolism 2019;92:98–107.
603. Romero-Corral A, Montori VM, Somers VK, et al. Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. Lancet 2006;368:666–78.
604. Adabag S, Huxley RR, Lopez FL, et al. Obesity related risk of sudden cardiac death in the atherosclerosis risk in communities study. Heart 2015;101:215–21.
605. Kakavas S, Georgiopoulos G, Oikonomou D, et al. The impact of body mass index on post resuscitation survival after cardiac arrest: a meta-analysis. Clin Nutr ESPEN 2018;24:47–53.
606. Ma Y, Huang L, Zhang L, Yu H, Liu B. Association between body mass index and clinical outcomes of patients after cardiac arrest and resuscitation: a meta-analysis. Am J Emerg Med 2018;36:1270–9.
607. Lee H, Oh J, Lee J, et al. Retrospective study using computed tomography to compare sufficient chest compression depth for cardiopulmonary resuscitation in obese patients. J Am Heart Assoc 2019;8:e013948.
608. Jain R, Nallamothu BK, Chan PS, American Heart Association National Registry of Cardiopulmonary Resuscitation i. Body mass index and survival after in-hospital cardiac arrest. Circ Cardiovasc Qual Outcomes 2010;3:490–7.
609. Nishisaki A, Maltese MR, Niles DE, et al. Backboards are important when chest compressions are provided on a soft mattress. Resuscitation 2012;83:1013–20.
610. Holt J, Ward A, Mohamed TY, et al. The optimal surface for delivery of CPR: a systematic review and meta-analysis. Resuscitation 2020;155:159–64.
611. Bunch TJ, White RD, Lopez-Jimenez F, Thomas RJ. Association of body weight with total mortality and with ICD shocks among survivors of ventricular fibrillation in out-of-hospital cardiac arrest. Resuscitation 2008;77:351–5.
612. White RD, Blackwell TH, Russell JK, Jorgenson DB. Body weight does not affect defibrillation, resuscitation, or survival in patients with out-of-hospital cardiac arrest treated with a nonescalating biphasic waveform defibrillator. Crit Care Med 2004;32:S387–92.
613. Voskoboinik A, Moskovitch J, Plunkett G, et al. Cardioversion of atrial fibrillation in obese patients: results from the cardioversion-BMI randomized controlled trial. J Cardiovasc Electrophysiol 2019;30:155–61.
614. Sugerman H, Windsor A, Bessos M, Wolfe L. Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. J Intern Med 1997;241:71–9.
615. Schnittker R, Marshall Sd, Berecki-Gisolf J. Patient and surgery factors associated with the incidence of failed and difficult intubation. Anaesthesia 2020;75:756–66.
616. Holmberg TJ, Bowman SM, Warner KJ, et al. The association between obesity and difficult prehospital tracheal intubation. Anesth Analg 2011;112:1132–8.
617. Cook TM, Woodall N, Frerk C. Major complications of airway management in the UK: results of the Fourth National Audit Project of the Royal College of Anaesthetists and the Difficult Airway Society. Part 1: anaesthesia. Br J Anaesth 2011;106:617–31.
618. Timmermann A, Nickel EA, Puhringer F. Second generation laryngeal masks: expanded indications. Anaesthesist 2015;64: 7–15.
619. Zoremba M, Aust H, Eberhart L, Braunecker S, Wulf H. Comparison between intubation and the laryngeal mask airway in moderately obese adults. Acta Anaesthesiol Scand 2009;53:436–42.
620. Reminiac F, Jouan Y, Cazals X, et al. Risks associated with obese patient handling in emergency prehospital care. Prehosp Emerg Care 2014;18:555–7.
621. Kruska P, Kappus S, Kerner T. Obesity in prehospital emergency care. Anasthesiol Intensivmed Notfallmed Schmerzther 2012;47: 556–62.
622. Beckett VA, Knight M, Sharpe P. The CAPS study: incidence, management and outcomes of cardiac arrest in pregnancy in the UK: a prospective, descriptive study. BJOG 2017;124: 1374–1381.
623. Jeejeebhoy FM, Zelop CM, Lipman S, et al. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association. Circulation 2015;132:1747–73.
624. Chu J, Johnston TA, Geoghegan J, Royal College of O, Gynaecologists. Maternal collapse in pregnancy and the puerperium: green-top guideline no. 56. BJOG 2020;127:e14–52.
625. Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J, et al. 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy. Eur Heart J 2018;39:3165–241.
626. UK M-UMaBRRtAaCEat. MBRRACE-UK perinatal mortality surveillance report. UK perinatal deaths for births from January to December 2017. London: MBRRACE-UK; 2019.
627. Chesnutt AN. Physiology of normal pregnancy. Crit Care Clin 2004;20:609–15.

Please cite this article in press as: C. Lott, et al., European Resuscitation Council Guidelines 2021: Cardiac arrest in special circumstances, Resuscitation (2021), https://doi.org/10.1016/j.resuscitation.2021.02.011

628. Page-Rodriguez A, Gonzalez-Sanchez JA. Perimortem cesarean section of twin pregnancy: case report and review of the literature. Acad Emerg Med 1999;6:1072–4.
629. Cardosi RJ, Porter KB. Cesarean delivery of twins during maternal cardiopulmonary arrest. Obstet Gynecol 1998;92:695–7.
630. Humphries A, Mirjalili SA, Tarr GP, Thompson JMD, Stone P. The effect of supine positioning on maternal hemodynamics during late pregnancy. J Matern Fetal Neonatal Med 2019;32:3923–30.
631. Kundra P, Khanna S, Habeebullah S, Ravishankar M. Manual displacement of the uterus during caesarean section. Anaesthesia 2007;62:460–5.
632. Mendonca C, Griffiths J, Ateleanu B, Collis RE. Hypotension following combined spinal-epidural anaesthesia for caesarean section. Left lateral position vs. tilted supine position. Anaesthesia 2003;58:428–31.
633. Rees SG, Thurlow JA, Gardner IC, Scrutton MJ, Kinsella SM. Maternal cardiovascular consequences of positioning after spinal anaesthesia for caesarean section: left 15 degree table tilt vs. left lateral. Anaesthesia 2002;57:15–20.
634. Bamber JH, Dresner M. Aortocaval compression in pregnancy: the effect of changing the degree and direction of lateral tilt on maternal cardiac output. Anesth Analg 2003;97:256–8 table of contents.
635. Kinsella SM. Lateral tilt for pregnant women: why 15 degrees? Anaesthesia 2003;58:835–6.
636. Rees GA, Willis BA. Resuscitation in late pregnancy. Anaesthesia 1988;43:347–9.
637. Holmes S, Kirkpatrick ID, Zelop CM, Jassal DS. MRI evaluation of maternal cardiac displacement in pregnancy: implications for cardiopulmonary resuscitation. Am J Obstet Gynecol 2015213: 401 e1–5.
638. Delgado C, Dawson K, Schwaegler B, et al. Hand placement during chest compressions in parturients: a pilot study to identify the location of the left ventricle using transthoracic echocardiography. Int Jf Obstet Anesth 2020;43:31–5.
639. Einav S, Kaufman N, Sela HY. Maternal cardiac arrest and perimortem caesarean delivery: evidence or expert-based? Resuscitation 2012;83:1191–200.
640. Benson MD, Padovano A, Bourjeily G, Zhou Y. Maternal collapse: challenging the four-minute rule. EBioMedicine 2016;6:253–7.
641. Ramanathan K, Tan CS, Rycus P, et al. Extracorporeal membrane oxygenation in pregnancy: an analysis of the Extracorporeal Life Support Organization Registry. Crit Care Med 2020;48: 696–703.
642. Nanson J, Elcock D, Williams M, Deakin CD. Do physiological changes in pregnancy change defibrillation energy requirements? Br J Anaesth 2001;87:237–9.
643. Mushambi MC, Athanassoglou V, Kinsella SM. Anticipated difficult airway during obstetric general anaesthesia: narrative literature review and management recommendations. Anaesthesia 2020;75:945–61.
644. Mushambi MC, Kinsella SM. Obstetric Anaesthetists' Association/ Difficult Airway Society difficult and failed tracheal intubation guidelines—the way forward for the obstetric airway. Br J Anaesth 2015;115:815–8.
645. Kinsella SM, Winton AL, Mushambi MC, et al. Failed tracheal intubation during obstetric general anaesthesia: a literature review. Int J Obstet Anesth 2015;24:356–74.
646. Say L, Chou D, Gemmill A, et al. Global causes of maternal death: a WHO systematic analysis. Lancet Glob Health 2014;2:e323–33.
647. Lewis G. The Confidential Enquiry into Maternal and Child Health (CEMACH). Saving mothers' lives: Reviewing maternal deaths to make motherhood safer – 2003–2005. The seventh report of the confidential enquiries into maternal deaths in the United Kingdom. London: CEMACH; 2007.
648. Jauniaux E, Alfirevic Z, Bhide AG, et al. Vasa praevia: diagnosis and management: green-top guideline no. 27b. BJOG 2019;126: e49–61.
649. Gynaecologists RCoOa. Postpartum Haemorrhage, Prevention and Management (Green-top Guideline No. 52). London; 2016.
650. Collaborators WT. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. Lancet 2017;389:2105–16.
651. Ahearn GS, Hadjiliadis D, Govert JA, Tapson VF. Massive pulmonary embolism during pregnancy successfully treated with recombinant tissue plasminogen activator: a case report and review of treatment options. Arch Intern Med 2002;162:1221–7.
652. Excellence NIfHaC. Hypertension in pregnancy: diagnosis and management NICE guideline NG133. London: National Institute for Health and Care Excellence; 2019.
653. Conde-Agudelo A, Romero R. Amniotic fluid embolism: an evidence-based review. Am J Obstet Gynecol 2009201: 445 e1–13.
654. Fitzpatrick KE, Tuffnell D, Kurinczuk JJ, Knight M. Incidence, risk factors, management and outcomes of amniotic-fluid embolism: a population-based cohort and nested case-control study. BJOG 2016;123:100–9.
655. Rittenberger JC, Kelly E, Jang D, Greer K, Heffner A. Successful outcome utilizing hypothermia after cardiac arrest in pregnancy: a case report. Crit Care Med 2008;36:1354–6.
656. Song KH, Lee BK, Jeung KW, Lee SM. Safely completed therapeutic hypothermia in postpartum cardiac arrest survivors. Am J Emerg Med 201533: 861 e5–6.
657. Merien AE, van de Ven J, Mol BW, Houterman S, Oei SG. Multidisciplinary team training in a simulation setting for acute obstetric emergencies: a systematic review. Obstet Gynecol 2010;115:1021–31.
658. Leonardsen AL, Svendsen EJ, Heitmann GB, et al. Development and validation of a questionnaire to assess healthcare personnel competence in cardiac arrest and resuscitation in pregnancy. PLoS One 2020;15:e0232984.
659. Merriel A, Ficquet J, Barnard K, et al. The effects of interactive training of healthcare providers on the management of life-threatening emergencies in hospital. Cochrane Database Syst Rev 2019;9: CD012177.