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European Resuscitation Council Guidelines for Resuscitation 2015 Section 4. Cardiac arrest in special circumstances

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Introduction

Irrespective of the cause of cardiac arrest, early recognition and calling for help, including appropriate management of the deteriorating patient, early defibrillation, high-quality cardiopulmonary resuscitation (CPR) with minimal interruption of chest compressions and treatment of reversible causes, are the most important interventions.

In certain conditions, however, advanced life support (ALS) guidelines require modification. The following guidelines for resuscitation in special circumstances are divided into three parts: special causes, special environments 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 any resuscitation. 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; Hypo-/hyperkalaemia and other electrolyte disorders; Hypo-/hyperthermia; Hypovolaemia; Tension pneumothorax; Tamponade (cardiac); Thrombosis (coronary and pulmonary); Toxins (poisoning). The second part covers cardiac arrest in special environments, 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.

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Summary of changes since 2010 Guidelines

The main changes in the ERC Guidelines 2015 in comparison with the Guidelines 2010[1] are summarised below:

Special causes

- Survival after an asphyxia-induced cardiac arrest is rare and survivors often have severe neurological impairment. During CPR, early effective ventilation of the lungs with supplementary oxygen is essential.
- A high degree of clinical suspicion and aggressive treatment can prevent cardiac arrest from electrolyte abnormalities. The new algorithm provides clinical guidance to emergency treatment of life-threatening hyperkalaemia.
- Hypothermic patients without signs of cardiac instability (systolic blood pressure $\geq$90 mmHg, absence of ventricular arrhythmias or core temperature $\geq$28 °C) can be rewarmed externally using minimally invasive techniques (e.g. with warm forced air and warm intravenous fluid). Patients with signs of cardiac instability should be transferred directly to a centre capable of extracorporeal life support (ECLS).
- Early recognition and immediate treatment with intramuscular adrenaline remains the mainstay of emergency treatment for anaphylaxis.
- The mortality from traumatic cardiac arrest (TCA) is very high. The most common cause of death is haemorrhage. It is recognised that most survivors do not have hypovolaemia, but instead have other reversible causes (hypoxia, tension pneumothorax, cardiac tamponade) that must be immediately treated. The new treatment algorithm for TCA was developed to prioritise the sequence of life-saving measures. Chest compressions should not delay the treatment of reversible causes. Cardiac arrests of non-traumatic origin leading to a secondary traumatic event should be recognised and treated with standard algorithms.
- There is limited evidence for recommending the routine transport of patients with continuing CPR after out-of-hospital cardiac arrest (OHCA) of suspected cardiac origin. Transport may be beneficial in selected patients where there is immediate hospital access to the catheterisation laboratory and an infrastructure providing prehospital and in-hospital teams experienced in mechanical or haemodynamic support and percutaneous coronary intervention (PCI) with ongoing CPR.
- Recommendations for administration of fibrinolytics when pulmonary embolism is the suspected cause of cardiac arrest remain unchanged. Routine use of surgical embolectomy or mechanical thrombectomy when pulmonary embolism is the suspected cause of cardiac arrest is not recommended. Consider these methods only when there is a known diagnosis of pulmonary embolism.
- Routine use of gastric lavage for gastrointestinal decontamination in poisoning is no longer recommended. Reduced emphasis is placed on hyperbaric oxygen therapy in carbon monoxide poisoning.

Special environments

- The special environments section includes recommendations for treatment of cardiac arrest occurring in specific locations. These locations are specialised healthcare facilities (e.g. operating theatre, cardiac surgery, catheterisation laboratory, dialysis unit, dental surgery), commercial airplanes or air ambulances, field of play, outside environment (e.g. drowning, difficult terrain, high altitude, avalanche burial, lightning strike and electrical injuries) or the scene of a mass casualty incident.
- Patients undergoing surgical procedures involving general anaesthesia, particularly in emergencies, are at risk from perioperative cardiac arrest. A new section covers the common causes and relevant modification to resuscitative procedures in this group of patients.
- Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase. Key to successful resuscitation is recognition of the need to perform emergency resternotomy, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective. Resternotomy should be performed within 5 min if other interventions have failed.
- Cardiac arrest from shockable rhythms (Ventricular Fibrillation (VF) or pulseless Ventricular Tachycardia (pVT)) during cardiac catheterisation should immediately be treated with up to three stacked shocks before starting chest compressions. Use of mechanical chest compression devices during angiography is recommended to ensure high-quality chest compressions and reduce the radiation burden to personnel during angiography with ongoing CPR.
- In dental surgery, do not move the patient from the dental chair in order to start CPR. Quickly recline the dental chair into a horizontal position and place a stool under the head of the chair to increase its stability during CPR.
- The in-flight use of AEDs aboard commercial airplanes can result in up to 50% survival to hospital discharge. AEDs and appropriate CPR equipment should be mandatory on board of all commercial aircraft in Europe, including regional and low-cost carriers. Consider an over-the-head technique of CPR if restricted access precludes a conventional method, e.g. in the aisle.
- The incidence of cardiac arrest on board helicopter emergency medical services (HEMS) and air ambulances is low. Importance of pre-flight preparation and use of mechanical chest compression devices are emphasised.
- Sudden and unexpected collapse of an athlete on the field of play is likely to be cardiac in origin and requires rapid recognition and early defibrillation.
- The duration of submersion is a key determinant of outcome from drowning. Submersion exceeding 10 min is associated with poor outcome. Bystanders play a critical role in early rescue and resuscitation. Resuscitation strategies for those in respiratory or cardiac arrest continue to prioritise oxygenation and ventilation.
- The chances of good outcome from cardiac arrest in difficult terrain or mountains may be reduced because of delayed access and prolonged transport. There is a recognised role of air rescue and availability of AEDs in remote but often-visited locations.
- The cut-off criteria for prolonged CPR and extracorporeal rewarming of avalanche victims in cardiac arrest are more stringent to reduce the number of futile cases treated with extracorpoereal life support (ECLS). ECLS is indicated if the duration of burial is >60 min (instead of >35 min), core temperature at extrication is <30 °C (instead of <32 °C), and serum potassium at hospital admission is $\leq$8 mmol L^{-1} (instead of $\leq$12 mmol L^{-1}); otherwise standard guidelines apply.
- Safety measures are emphasised when providing CPR to the victim of an electrical injury.
- Recommendations for management of multiple victims should prevent delay of treatment available for salvageable victims during mass casualty incidents (MCIs). Safety at scene is paramount. A triage system should be used to prioritise treatment and, if the number of casualties overwhelms healthcare resources, withhold CPR for those without signs of life.

Special patients

- The section on special patients gives guidance for CPR in patients with severe comorbidities (asthma, heart failure with

ventricular assist devices, neurological disease, obesity) and those with specific physiological conditions (pregnancy, elderly people).

- The first line treatment for acute asthma is inhaled beta-2 agonists while intravenous beta-2 agonists are suggested only for those patients in whom inhaled therapy cannot be used reliably. Inhaled magnesium is no longer recommended.
- In patients with ventricular assist devices (VADs), confirmation of cardiac arrest may be difficult. If during the first 10 days after surgery, cardiac arrest does not respond to defibrillation, perform resternotomy immediately.
- Patients with subarachnoid haemorrhage may have ECG changes that suggest an acute coronary syndrome (ACS). Whether a computed tomography (CT) brain scan is done before or after coronary angiography will depend on clinical judgement regarding the likelihood of a subarachnoid haemorrhage versus acute coronary syndrome.
- No changes to the sequence of actions are recommended in resuscitation of obese patients, although delivery of effective CPR may be challenging. Consider changing rescuers more frequently than the standard 2-min interval. Early tracheal intubation by an experienced provider is recommended.
- For the pregnant woman in cardiac arrest, high-quality CPR with manual uterine displacement, early ALS and delivery of the fetus if early return of spontaneous circulation (ROSC) is not achieved remain key interventions.

A – SPECIAL CAUSES

Hypoxia

Introduction

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 4.1); although there is usually a combination of hypoxaemia and hypercarbia, it is the hypoxaemia that ultimately causes cardiac arrest.[2]

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 is likely to be of the order 1–2 min.[3] 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.[4] 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. According to Safar, complete airway obstruction after breathing air will result in PEA cardiac arrest in 5–10 min.[2] VF is rarely the first monitored rhythm after asphyxial cardiac arrest – in one of the largest series of hanging-associated out-of-hospital cardiac arrests (OHCAs), from Melbourne, Australia, just 7 (0.5%) of 1321 patients were in VF.[5]

Table 4.1
Causes of asphyxial cardiac arrest

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

Treatment

Treating the cause of the asphyxia/hypoxaemia is the highest priority because this is a potentially reversible cause of the cardiac arrest. Effective ventilation with supplementary oxygen is a particular priority in these patients. The better outcomes for OHCA victims receiving compression-only CPR[6] is not the case for asphyxial cardiac arrests, which have much better survival rates with conventional CPR.[7] Follow the standard ALS algorithm when resuscitating these patients.

Outcome

Survival after cardiac arrest from asphyxia is rare and most survivors sustain severe neurological injury. Of five published series that included a total of 286 patients with cardiac arrest following hanging where CPR was attempted (this was attempted in only about 16% of cases), there were just six (2%) survivors with a full recovery; 11 other survivors all had severe permanent brain injury.[5,8–11] In one third (89; 31%) of these 286 patients, rescuers were able to achieve ROSC – thus when CPR is attempted, ROSC is not uncommon 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.[11,12]

Hypo-/hyperkalaemia and other electrolyte disorders

Introduction

Electrolyte abnormalities can cause cardiac arrhythmias or cardiac arrest. Life-threatening arrhythmias are associated most commonly with potassium disorders, particularly hyperkalaemia, and less commonly with disorders of serum calcium and magnesium. Consider electrolyte disturbances in patient groups at risk – renal failure, severe burns, cardiac failure and diabetes mellitus.

The electrolyte values for definitions have been chosen as a guide to clinical decision-making. The precise values that trigger treatment decisions will depend on the patient's clinical condition and rate of change of electrolyte values. There is little or no evidence for the treatment of electrolyte abnormalities during cardiac arrest. Guidance during cardiac arrest is based on the strategies used in the non-arrest patient.

Prevention of electrolyte disorders

When possible, identify and treat life-threatening electrolyte abnormalities before cardiac arrest occurs. Monitor renal function in patients at risk and avoid combination of drugs that may exacerbate hyperkalaemia. Prevent recurrence of electrolyte disorders by removing any precipitating factors (e.g. drugs, diet).

Potassium disorders

Potassium homeostasis. Extracellular potassium concentration is regulated tightly between 3.5 and 5.0 mmol L^{-1}. A large concentration gradient normally exists between intracellular and extracellular fluid compartments. This potassium gradient across cell membranes contributes to the excitability of nerve and muscle cells, including the myocardium. Evaluation of serum potassium must take into consideration the effects of changes in serum pH. When serum pH decreases (acidaemia), serum potassium increases because potassium shifts from the cellular to the vascular space; a process that is reversed when serum pH increases (alkalaemia).

Hyperkalaemia. This is the most common electrolyte disorder associated with cardiac arrest. It is usually caused by impaired excretion by the kidneys, drugs or increased potassium release from cells and metabolic acidosis. Hyperkalaemia occurs in up to 10% of hospitalised patients.[13–15] Chronic kidney disease (CKD) is common in the general population and the incidence of hyperkalaemia increases from 2 to 42% as glomerular filtration rate (GFR) drops from 60 to 20 mL min^{-1}.[16] Patients with end-stage renal disease are particularly susceptible, particularly following an OHCA.[17] Prolonged hyperkalaemia is an independent risk factor for in-hospital mortality.[18] Acute hyperkalaemia is more likely than chronic hyperkalaemia to cause life-threatening cardiac arrhythmias or cardiac arrest.

Definition. There is no universal definition. We have defined hyperkalaemia as a serum potassium concentration higher than 5.5 mmol L^{-1}; in practice, hyperkalaemia is a continuum. As the potassium concentration increases above this value the risk of adverse events increases and the need for urgent treatment increases. Severe hyperkalaemia has been defined as a serum potassium concentration higher than 6.5 mmol L^{-1}.

Causes. The main causes of hyperkalaemia are:

- renal failure (i.e. acute kidney injury or chronic kidney disease);
- drugs (e.g. angiotensin converting enzyme inhibitors (ACE-I), angiotensin II receptor antagonists (ARB), potassium-sparing diuretics, non-steroidal anti-inflammatory drugs, beta-blockers, trimethoprim);
- tissue breakdown (e.g. rhabdomyolysis, tumour lysis, haemolysis);
- metabolic acidosis (e.g. renal failure, diabetic ketoacidosis);
- endocrine disorders (e.g. Addison's disease);
- diet (may be sole cause in patients with advanced chronic kidney disease) and
- spurious – pseudo-hyperkalaemia (suspect in cases with normal renal function, normal ECG and/or history of haematological disorder). Pseudo-hyperkalaemia describes the finding of a raised serum (clotted blood) K^+ value concurrently with a normal plasma (non-clotted blood) potassium value. The clotting process releases K^+ from cells and platelets, which increases the serum K^+ concentration by an average of 0.4 mmol/L. The most common cause of pseudo-hyperkalaemia is a prolonged transit time to the laboratory or poor storage conditions.[19,20]

The risk of hyperkalaemia is even greater when there is a combination of factors such as the concomitant use of angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers and potassium-sparing diuretics.

Recognition of hyperkalaemia. Exclude hyperkalaemia in all patients with an arrhythmia or cardiac arrest. Patients may present with weakness progressing to flaccid paralysis, paraesthesia, or depressed deep tendon reflexes. Alternatively, the clinical picture can be overshadowed by the primary illness causing hyperkalaemia. The first indicator of hyperkalaemia may also be the presence of ECG abnormalities, arrhythmias, or cardiac arrest. The use of a blood gas analyser to measure potassium can reduce delays in recognition.[21,22]

The effect of hyperkalaemia on the ECG depends on the absolute serum potassium as well as the rate of increase.[23] The reported frequency of ECG changes in severe hyperkalaemia is variable, but most patients appear to show ECG abnormalities at a serum potassium concentration higher than 6.7 mmol L^{-1}.[23,24] The presence of ECG changes strongly correlates with mortality.[25] In some cases, the ECG may be normal or show atypical changes including ST elevation.

The ECG changes associated with 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;
- S & T wave merging (sine wave pattern);
- widened QRS (>0.12 s);
- ventricular tachycardia;
- bradycardia;
- cardiac arrest (PEA, VF/pVT, asystole).

Treatment of hyperkalaemia. There are five key treatment strategies for hyperkalaemia[22]:

- cardiac protection;
- shifting potassium into cells;
- removing potassium from the body;
- monitoring serum potassium and blood glucose;
- prevention of recurrence.

When hyperkalaemia is strongly suspected, e.g. in the presence of ECG changes, start life-saving treatment even before laboratory results are available. The treatment strategy for hyperkalaemia has been reviewed extensively.[13,22,26] Follow the hyperkalaemia emergency treatment algorithm (Fig. 4.1).[22] Avoid salbutamol monotherapy, which may be ineffective. There is insufficient evidence to support the use of sodium bicarbonate to decrease serum potassium. Consider the need for early specialist or critical care referral.

The main risks associated with treatment of hyperkalaemia are:

- Hypoglycaemia following insulin-glucose administration (usually occurs within 1–3 h of treatment, but may occur up to 6 h after infusion).[27] Monitor blood glucose and treat hypoglycaemia promptly.
- Tissue necrosis secondary to extravasation of intravenous calcium salts. Ensure secure vascular access prior to administration.
- Intestinal necrosis or obstruction following use of potassium exchange resins. Avoid prolonged use of resins and give laxative.
- Rebound hyperkalaemia after the effect of drug treatment has worn off (i.e. within 4–6 h). Continue to monitor serum potassium for a minimum of 24 h after an episode.

Patient not in cardiac arrest
Assess patient:

- Use systematic ABCDE approach and correct any abnormalities, obtain IV access.
- Check serum potassium.
- Record an ECG.

Monitor cardiac rhythm in patients with severe hyperkalaemia. Treatment is determined according to severity of hyperkalaemia. Approximate values are provided to guide treatment. Follow hyperkalaemia emergency treatment algorithm (Fig. 4.1).

Mild elevation (5.5–5.9 mmol L^{-1}).

- Address cause of hyperkalaemia to correct and avoid further rise in serum potassium (e.g. drugs, diet).
- If treatment is indicated, remove potassium from the body: potassium exchange resins-calcium resonium 15–30 g, or sodium polystyrene sulfonate (Kayexalate) 15–30 g, given either orally or by retention enema/PR (per rectum) (onset in >4 h).

Moderate elevation (6.0–6.4 mmol L^{-1}) without ECG changes.

- Shift potassium intracellularly with glucose/insulin: 10 units short-acting insulin and 25 g glucose IV over 15–30 min (onset in 15–30 min; maximal effect at 30–60 min; duration of action 4–6 h; monitor blood glucose).
- Remove potassium from the body (see above; consider dialysis guided by clinical setting).

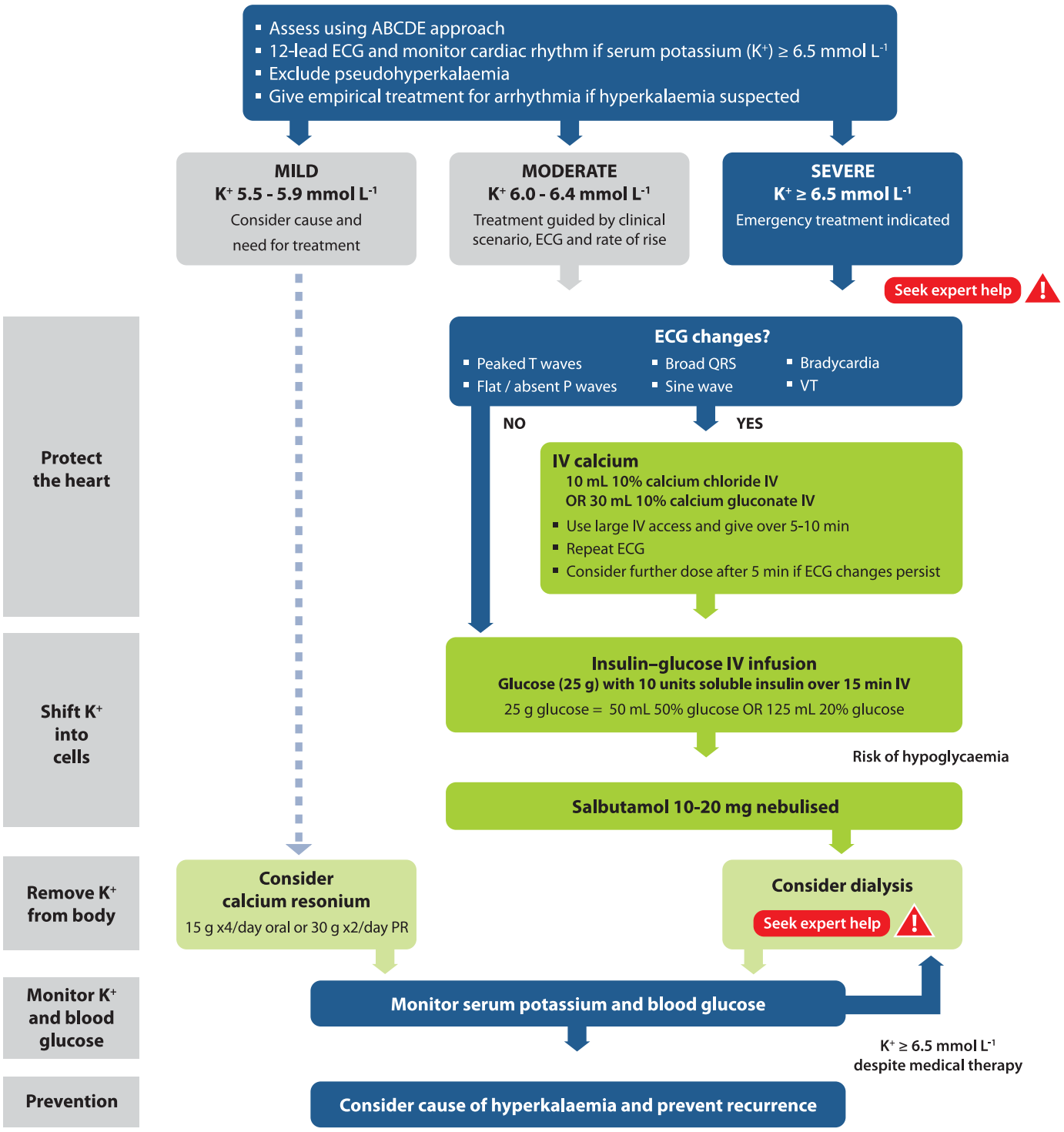

Fig. 4.1. Emergency treatment of hyperkalaemia. PR per rectum; ECG electrocardiogram; VT ventricular tachycardia.
Reproduced with permission from Renal Association and Resuscitation Council (UK).

Severe elevation (≥6.5 mmol L^{-1}) without ECG changes.

- Seek expert help.
- Give glucose/insulin (see above).
- Give salbutamol 10–20 mg nebulised (onset in 15–30 min; duration of action 4–6 h).
- Remove potassium from the body (consider dialysis).

Severe elevation (≥6.5 mmol L^{-1}) with toxic ECG changes.

- Seek expert help.
- Protect the heart with calcium chloride: 10 mL 10% calcium chloride IV over 2–5 min to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane. This protects the heart by reducing the risk of VF/pVT but does not lower serum potassium (onset in 1–3 min).
- Use shifting agents (glucose/insulin and salbutamol).
- Remove potassium from the body (consider dialysis at outset or if refractory to medical treatment).

Modifications to cardiopulmonary resuscitation. The following modifications to standard ALS guidelines are recommended in the presence of severe hyperkalaemia:

- Confirm hyperkalaemia using a blood gas analyser if available.
- Protect the heart: give 10 mL calcium chloride 10% IV by rapid bolus injection.
- Shift potassium into cells: Give glucose/insulin: 10 units short-acting insulin and 25 g glucose IV by rapid injection. Monitor blood glucose.
- Give sodium bicarbonate: 50 mmol IV by rapid injection (if severe acidosis or renal failure).
- Remove potassium from body: Consider dialysis for hyperkalaemic cardiac arrest resistant to medical treatment. Several dialysis modalities have been used safely and effectively in cardiac arrest, but this may only be available in specialist centres.[28] Consider use of a mechanical chest compression device if prolonged CPR is needed.

Indications for dialysis. 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 (<400 mL day^{-1} urine output);
- marked tissue breakdown (e.g. rhabdomyolysis).

Special considerations for management of cardiac arrest in a dialysis unit are addressed in the section Special environments (see cardiac arrest in a dialysis unit).

Hypokalaemia. Hypokalaemia is the most common electrolyte disorder in clinical practice.[29] It is seen in up to 20% of hospitalised patients.[30] Hypokalaemia increases the incidence of arrhythmias and sudden cardiac death (SCD).[31] The risk is increased in patients with pre-existing heart disease and in those treated with digoxin.

Definition. Hypokalaemia is defined as a serum potassium level <3.5 mmol L^{-1}. Severe hypokalaemia is defined as a serum potassium level <2.5 mmol L^{-1} and may be associated with symptoms.

Causes. The main causes of hypokalaemia include:

- gastrointestinal loss (e.g. diarrhoea);
- drugs (e.g. diuretics, laxatives, steroids);
- renal losses (e.g. renal tubular disorders, diabetes insipidus, dialysis);
- endocrine disorders (e.g. Cushing's syndrome, hyperaldosteronism);
- metabolic alkalosis;
- magnesium depletion;
- poor dietary intake.

Treatment strategies used for hyperkalaemia may also induce hypokalaemia.

Recognition of hypokalaemia. Exclude hypokalaemia in every patient with an arrhythmia or cardiac arrest. In dialysis patients, hypokalaemia may occur at the end of a haemodialysis session or during treatment with peritoneal dialysis.

As serum potassium concentration decreases, the nerves and muscles are predominantly affected, causing fatigue, weakness, leg cramps, constipation. In severe cases (serum potassium <2.5 mmol L^{-1}), rhabdomyolysis, ascending paralysis and respiratory difficulties may occur.

ECG features of hypokalaemia are:

- U waves;
- T wave flattening;
- ST segment changes;
- arrhythmias, especially if patient is taking digoxin;
- cardiac arrest (PEA, VF/pVT, asystole).

Treatment. This depends on the severity of hypokalaemia and the presence of symptoms and ECG abnormalities. Gradual replacement of potassium is preferable, but in an emergency, intravenous potassium is required. The maximum recommended IV dose of potassium is 20 mmol h^{-1}, but more rapid infusion (e.g. 2 mmol min^{-1} 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 during IV infusion and the dose should be titrated after repeated sampling of serum potassium levels.

Many patients who are potassium deficient are also deficient in magnesium. Magnesium is important for potassium uptake and for the maintenance of intracellular potassium values, particularly in the myocardium. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases of hypokalaemia.[32]

Calcium and magnesium disorders

The recognition and management of calcium and magnesium disorders is summarised in Table 4.2.

Hypo-/hyperthermia

Accidental hypothermia

Definition. Every year approximately 1500 people die of primary accidental hypothermia in the United States.[33] Accidental hypothermia is defined as an involuntary drop of the body core temperature <35 °C. The Swiss staging system is used to estimate core temperature at the scene. Its stages are based on clinical signs, which roughly correlate with the core temperature:

- hypothermia I; mild hypothermia (conscious, shivering, core temperature 35–32 °C);
- hypothermia II; moderate hypothermia (impaired consciousness without shivering, core temperature 32–28 °C);
- hypothermia III; severe hypothermia (unconscious, vitals signs present, core temperature 28–24 °C);
- hypothermia IV; cardiac arrest or low flow state (no or minimal vital signs, core temperature <24 °C);
- hypothermia V; death due to irreversible hypothermia (core temperature <13.7 °C).[34]

Diagnosis. Hypothermia is diagnosed in any patient with a core temperature <35 °C, or where measurement unavailable, a history of exposure to cold, or when the trunk feels cold.[33] Accidental hypothermia may be under-diagnosed in countries with a temperate climate. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia may follow a mild insult. The risk of hypothermia is increased by alcohol or drug ingestion, exhaustion, illness, injury or neglect especially when there is a decrease in the level of consciousness.

A low-reading thermometer is needed to measure the core temperature and confirm the diagnosis. The core temperature in the lower third of the oesophagus correlates well with heart temperature. Tympanic measurement using a thermistor technique is a reliable alternative but may be considerably lower than core temperature if the environment is very cold, the probe is not well insulated, or the external auditory canal is filled with snow or water[35,36] Widely available tympanic thermometers based on infrared technique do not seal the ear canal and are not designed for low core temperature readings.[37] The in-hospital core temperature measurement site should be the same throughout resuscitation and rewarming. Bladder and rectal temperatures lag behind core temperature;[38,39] for this reason, measurement of bladder and

Table 4.2
Calcium and magnesium disorders with associated clinical presentation, ECG manifestations and recommended treatment

Disorder	Causes	Presentation	ECG	Treatment
Hypercalcaemia				
Calcium > 2.6 mmol L^{-1}	Primary or tertiary hyperparathyroidism Malignancy Sarcoidosis Drugs	Confusion Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Short QT interval Prolonged QRS interval Flat T waves AV block Cardiac arrest	Fluid replacement IV Furosemide 1 mg kg^{-1} IV Hydrocortisone 200–300 mg IV Pamidronate 30–90 mg IV Treat underlying cause
Hypocalcaemia				
Calcium < 2.1 mmol L^{-1}	Chronic renal failure 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 Magnesium sulphate 50% 4–8 mmol (if necessary)
Hypermagnesaemia				
Magnesium > 1.1 mmol L^{-1}	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^{-1}* Calcium chloride 10% 5–10 mL repeated if necessary Ventilatory support if necessary Saline diuresis – 0.9% saline with furosemide 1 mg kg^{-1} IV Haemodialysis
Hypomagnesaemia				
Magnesium < 0.6 mmol L^{-1}	GI loss Polyuria Starvation Alcoholism Malabsorption	Tremor Ataxia Nystagmus Seizures Arrhythmias – torsade de pointes Cardiac arrest	Prolonged PR and QT intervals ST-segment depression T-wave inversion Flattened P waves Increased QRS duration Torsade de pointes	*Severe or symptomatic*: 2 g 50% magnesium sulphate (4 mL; 8 mmol) IV over 15 min *Torsade de pointes*: 2 g 50% magnesium sulphate (4 mL; 8 mmol) IV over 1–2 min *Seizure*: 2 g 50% magnesium sulphate (4 mL; 8 mmol) IV over 10 min

rectal temperature has been de-emphasised in patients with severe hypothermia.

Decision to resuscitate. Cooling of the human body decreases cellular oxygen consumption by about 6% per 1 °C decrease in core temperature.[40] At 28 °C, oxygen consumption is reduced by approximately 50% and at 22 °C by approximately 75%. At 18 °C the brain can tolerate cardiac arrest for up to 10 times longer than at 37 °C. This results in hypothermia exerting a protective effect on the brain and heart,[41] and intact neurological recovery may be possible even after prolonged cardiac arrest if deep hypothermia develops before asphyxia.

Beware of diagnosing death in a hypothermic patient because hypothermia itself may produce a very slow, small-volume, irregular pulse and unrecordable blood pressure. In a deeply hypothermic patient (hypothermia IV) signs of life may be so minimal that it is easy to overlook them. Therefore, look for signs of life for at least 1 min and use an ECG monitor to detect any electrical cardiac activity. Neurologically intact survival has been reported after hypothermic cardiac arrest with a core temperature as low as 13.7 °C[42] and CPR for as long as six and a half hours.[43]

Intermittent CPR, as rescue allows, may also be of benefit.[44] If continuous CPR cannot be delivered, a patient with hypothermic cardiac arrest and a core temperature <28 °C (or unknown), should receive 5 min of CPR, alternating with periods ≤5 min without CPR. Patients with a core temperature <20 °C, should receive 5 min of CPR, alternating with periods ≤10 min without CPR.[45]

In the prehospital setting, resuscitation should be withheld in hypothermic patients only if the cause of cardiac arrest is clearly attributable to a lethal injury, fatal illness, prolonged asphyxia, or if the chest is incompressible.[46] In all other hypothermic patients, the traditional guiding principle that 'no one is dead until warm and dead' should be considered. In remote areas, the impracticalities of achieving rewarming have to be considered. In the hospital setting involve senior doctors and use clinical judgement to determine when to stop resuscitating a hypothermic victim in cardiac arrest.

Modifications to cardiopulmonary resuscitation

- Do not delay careful tracheal intubation when it is indicated. The advantages of adequate oxygenation and protection from aspiration outweigh the minimal risk of triggering VF by performing tracheal intubation.[47]
- Check for signs of life for up to 1 min. Palpate a central artery and assess the cardiac rhythm (if ECG monitor available). Echocardiography, near-infrared spectroscopy or ultrasound with Doppler may be used to establish whether there is (an adequate) cardiac output or peripheral blood flow.[48,49] If there is any doubt, start CPR immediately.
- Hypothermia can cause stiffness of the chest wall, making ventilations and chest compressions difficult. Consider the use of mechanical chest compression devices.[50]
- Once CPR is under way, confirm hypothermia with a low-reading thermometer.
- 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.[51] 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.[52] Adrenaline may be effective in increasing coronary perfusion pressure, but not

survival.[53,54] 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, use standard drug protocols.

Treatment of arrhythmias. As core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation followed by VF and finally asystole.[55,56] Arrhythmias other than VF tend to revert spontaneously as core temperature increases, and usually do not require immediate treatment. Bradycardia is physiological in severe hypothermia. Cardiac pacing is not indicated unless bradycardia associated with haemodynamic compromise persists after rewarming. The temperature at which defibrillation should firstly be attempted, and how often it should be attempted in the severely hypothermic patient, has not been established. If VF is detected, defibrillate according to standard protocols. If VF persists after three shocks, delay further attempts until core temperature is ≥30 °C.[57] CPR and rewarming may have to be continued for several hours to facilitate successful defibrillation.

Insulation. General measures for all victims include removal from the cold environment, prevention of further heat loss and rapid transfer to hospital.[58] In the field, a patient with moderate or severe hypothermia (hypothermia ≥ II) should be immobilised and handled carefully, oxygenated adequately, monitored (including ECG and core temperature), and the whole body dried and insulated.[51]

Remove wet clothes while minimising excessive movement of the victim. Removal of wet clothing or use of a vapour barrier seems to be equally effective to limit heat loss.[59] Conscious victims (hypothermia I) can mobilise as exercise rewarms a person more rapidly than shivering.[60] Patients will continue cooling after removal from a cold environment (i.e. afterdrop), which may result in a life-threatening decrease in core temperature triggering a cardiac arrest during transport (i.e. 'rescue death'). Prehospitally, avoid prolonged investigations and treatment, as further heat loss is difficult to prevent. Patients who stop shivering (e.g. hypothermia II–IV, and sedated or anaesthetised patients) will cool faster.

Prehospital rewarming. Rewarming may be passive, active external, or active internal. In hypothermia I passive rewarming is appropriate as patients are still able to shiver. Passive rewarming is best achieved by full body insulation with wool blankets, aluminium foil, cap and a warm environment. In hypothermia II–IV the application of chemical heat packs to the trunk has been recommended. In conscious patients who are able to shiver, this improves thermal comfort but does not speed rewarming.[61] If the patient is unconscious and the airway is not secured, arrange the insulation around the patient lying in a recovery (lateral decubitus) position. Rewarming in the field with heated intravenous fluids and warm humidified gases is not feasible.[51] Intensive active rewarming must not delay transport to a hospital where advanced rewarming techniques, continuous monitoring and observation are available.

Transport. Transport patients with hypothermia stage I to the nearest hospital. In hypothermia stage II–IV, signs of prehospital cardiac instability (i.e. systolic blood pressure <90 mmHg, ventricular arrhythmia, core temperature <28 °C) should determine the choice of admitting hospital. If any signs of cardiac instability are present, transport the patient to an ECLS centre, contacting them well in advance to ensure that the hospital can accept the patient for extracorporeal rewarming. In hypothermia V, reasons for withholding or terminating CPR should be investigated (e.g. obvious signs of irreversible death, valid DNAR, conditions unsafe for rescuer, avalanche burial ≥60 min and airway packed with snow and asystole). In the absence of any of these signs, start CPR and transfer the patient to an ECLS centre.

In-hospital rewarming. Unless the patient goes into VF, rewarm using active external methods (i.e. with forced warm air) and minimally invasively methods (i.e. with warm IV infusions). With a core temperature <32 °C and potassium <8 mmol L^{-1}, consider ECLS rewarming.[33] Most ECLS rewarmings have been performed using cardiopulmonary bypass, but more recently, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) has become the preferred method due to its rapid availability, the need for less anticoagulation, and the potential to prolong cardiorespiratory support after rewarming.

If an ECLS centre is not available, rewarming may be attempted in hospital using a dedicated team and a combination of external and internal rewarming techniques (e.g. forced warm air, warm infusions, forced peritoneal lavage).[62]

Continuous haemodynamic monitoring and warm IV fluids are essential. Patients will require large volumes of fluids during rewarming, as vasodilation causes expansion of the intravascular space. Avoid hyperthermia during and after rewarming. Once ROSC has been achieved use standard post-resuscitation care.

Hyperthermia

Introduction. Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds that normally maintained by homeostatic mechanisms. Hyperthermia may be exogenous, caused by environmental conditions, or secondary to endogenous heat production.

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 stress, progressing to heat exhaustion, then to heat stroke and finally to multiple organ dysfunction and cardiac arrest.[63]

Malignant hyperthermia is a rare 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 depolarising muscle relaxants.[64,65]

Heat exhaustion

Definition. Heat exhaustion is a non-life-threatening clinical syndrome of weakness, malaise, nausea, syncope, and other non-specific symptoms caused by heat exposure. Thermoregulation is not impaired. Heat exhaustion is caused by water and electrolyte imbalance due to heat exposure, with or without exertion. Rarely, severe heat exhaustion after physical exertion may be complicated by rhabdomyolysis, myoglobinuria, acute renal failure, and disseminated intravascular coagulation (DIC).

Symptoms. Symptoms are often vague, and patients may not realise that heat is the cause. Symptoms may include weakness, dizziness, headache, nausea, and sometimes vomiting. Syncope due to standing for long periods in the heat (heat syncope) is common and may mimic cardiovascular disorders. On examination, patients appear tired and are usually sweaty and tachycardic. Mental status is typically normal, unlike in heatstroke. Temperature is usually normal and, when elevated, usually does not exceed 40 °C.

Diagnosis. Diagnosis is clinical and requires exclusion of other possible causes (e.g. hypoglycaemia, acute coronary syndrome, infections). Laboratory testing is required only if needed to rule out other disorders.

Treatment

Fluids and electrolyte replacement. Treatment involves removing patients to a cool environment, lying them flat, and giving IV fluids and electrolyte replacement therapy; oral rehydration may not be effective in rapidly replacing electrolytes, but may be a more practical treatment. Rate and volume of rehydration are guided by age, underlying disorders, and clinical response. Replacement of 1–2 L crystalloids at 500 mL h^{-1} is often adequate. External cooling measures are usually not required. Consider external cooling in patients with a core temperature of ≥40 °C.

Heat stroke

Definition. Heat stroke (HS) is defined as hyperthermia accompanied by a systemic inflammatory response with a core temperature >40 °C, accompanied by mental state change and varying levels of organ dysfunction.[63]

There are two forms of HS:

1. Classic (non-exertional) heat stroke (CHS) occurs during high environmental temperatures and often effects the elderly during heat waves.[66]
2. Exertional heat stroke (EHS) occurs during strenuous physical exercise in high environmental temperatures and/or high humidity and usually effects healthy young adults.[67]

Mortality from heat stroke ranges between 10 and 50%.[68]

Predisposing factors. The elderly are at increased risk for heat-related illness because of underlying illness, medication use, declining thermoregulatory mechanisms and limited social support. There are several risk factors: lack of acclimatisation, dehydration, obesity, alcohol, cardiovascular disease, skin conditions (psoriasis, eczema, scleroderma, burn, cystic fibrosis), hyperthyroidism, phaeochromocytoma and drugs (anticholinergics, diamorphine, cocaine, amphetamine, phenothiazines, sympathomimetics, calcium channel blockers, beta-blockers).

Symptoms. Heat stroke can resemble septic shock and may be caused by similar mechanisms.[69] A single centre case series reported 14 ICU deaths in 22 heat stroke patients admitted to ICU with multiple organ failure.[70] Features included:

- core temperature ≥40 °C;
- hot, dry skin (sweating present in about 50% of cases of exertional heat stroke);
- early signs and symptoms (e.g. extreme fatigue, headache, fainting, facial flushing, vomiting and diarrhoea);
- cardiovascular dysfunction including arrhythmias and hypotension[71];
- respiratory dysfunction including acute respiratory distress syndrome (ARDS)[72];
- central nervous system dysfunction including seizures and coma[73];
- liver and renal failure[74];
- coagulopathy;
- rhabdomyolysis.[75]

Other clinical conditions presenting with increased core temperature need to be considered, including drug toxicity, drug withdrawal syndrome, serotonin syndrome, neuroleptic malignant syndrome, sepsis, central nervous system infection, endocrine disorders (e.g. thyroid storm, phaeochromocytoma).

Treatment. The mainstay of treatment is supportive therapy and rapidly cooling the patient.[76–78] Start cooling in the prehospital setting if possible. Aim to rapidly reduce the core temperature to approximately 39 °C. Patients with severe heat stroke need to be managed in an ICU environment. Large volumes of fluids and correction of electrolyte abnormalities may be required (see hypo-/hyperkalaemia and other electrolyte disorders).

Cooling techniques. Several cooling methods have been described, but there are few formal trials to determine which is optimal. Simple cooling techniques include drinking cold fluids, fanning the completely undressed patient and spraying tepid water on the patient. Ice packs over areas where there are large superficial blood vessels (axillae, groins, neck) may also be useful. Surface cooling methods may cause shivering. In cooperative stable patients, immersion in cold water can be effective[79]; however, this may cause peripheral vasoconstriction, shunt blood away from the periphery and reduce heat dissipation. Immersion is also not practical in the sickest patients.

Further techniques to cool patients with hyperthermia are similar to those used for targeted temperature management after cardiac arrest (see post resuscitation care).[80] Cold intravenous fluids will decrease body temperature. Gastric, peritoneal,[81] pleural or bladder lavage with cold water will lower the core temperature. Intravascular cooling techniques include the use of cold IV fluids,[82] intravascular cooling catheters[83,84] and extracorporeal circuits,[85] e.g. continuous veno-venous haemofiltration or cardiopulmonary bypass.

Pharmacological treatment. There are no specific drug therapies in heat stroke that lower core temperature. There is no good evidence that antipyretics (e.g. non-steroidal anti-inflammatory drugs or paracetamol) are effective in heat stroke. Diazepam may be useful to treat seizures and facilitate cooling.[86] Dantrolene has not been shown to be beneficial.[87–89]

Malignant hyperthermia

Malignant hyperthermia is a life-threatening genetic sensitivity of skeletal muscles to halogenated volatile anaesthetics and depolarising neuromuscular blocking drugs, occurring during or after anaesthesia.[90] Stop triggering agents immediately; give oxygen, correct acidosis and electrolyte abnormalities. Start active cooling and give dantrolene.[91]

Other drugs such as 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and amphetamines also cause a condition similar to malignant hyperthermia and the use of dantrolene may be beneficial.[92]

Modifications to cardiopulmonary resuscitation. There are no specific studies of cardiac arrest in hyperthermia. If cardiac arrest occurs, follow standard guidelines and continue cooling the patient. Use the same cooling techniques as for targeted temperature management after cardiac arrest (see Section 5 Post-resuscitation care).[80] Attempt defibrillation using standard energy levels. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest.[93,94] The risk of unfavourable neurological outcome increases by 2.26 (odds ratio) for each degree of body temperature >37 °C.[95]

Hypovolaemia

Introduction

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). Hypovolaemia from mediator-activated vasodilation and increased capillary permeability is a major factor causing cardiac arrest in severe anaphylaxis.[96] Hypovolaemia from blood loss, is a leading cause of death in traumatic cardiac arrest.[97] 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,[98] or treat the primary cause (e.g. anaphylactic shock). In the initial stages of resuscitation use any crystalloid solution that is immediately available. If there is a qualified sonographer able to perform ultrasound without interruption to chest compressions, e.g. during rhythm check or ventilations, it may be considered as an additional diagnostic tool in hypovolaemic cardiac arrest.

Treatment recommendations for cardiac arrest and periarrest situations in anaphylaxis and trauma are addressed in separate sections because of the need for specific therapeutic approaches.

Anaphylaxis

Definition. A precise definition of anaphylaxis is not important for its emergency treatment.[99] The European Academy of Allergy and Clinical Immunology Nomenclature Committee proposed the following broad definition:[100] anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction. This is characterised by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.[1,96,101,102]

Epidemiology. Anaphylaxis is common and affects about 1 in 300 of the European population at some stage in their lives, with an incidence from 1.5 to 7.9 per 100,000 person-years.[103] Anaphylaxis can be triggered by any of a very broad range of triggers with food, drugs, stinging insects, and latex the most commonly identified triggers.[103] Food is the commonest trigger in children and drugs the commonest in adults.[104] Virtually any food or drug can be implicated, but certain foods (nuts) and drugs (muscle relaxants, antibiotics, nonsteroidal anti-inflammatory drugs and aspirin) cause most reactions.[105] A significant number of cases of anaphylaxis are idiopathic. Between 1992 and 2012 in the UK, admission and fatality rates for drug- and insect sting-induced anaphylaxis were highest in the group aged 60 years and older. In contrast, admissions due to food-triggered anaphylaxis were most common in young people, with a marked peak in the incidence of fatal food reactions during the second and third decades of life.[106]

The overall prognosis of anaphylaxis is good, with a case fatality ratio of less than 1% reported in most population-based studies. The European Anaphylaxis Registry reported that only 2% of 3333 cases were associated with cardiac arrest.[107] If intensive care unit admission is required, survival to discharge is over 90%. Over the period 2005–2009, there were 81 paediatric and 1269 adult admissions with anaphylaxis admitted to UK critical care units. Survival to discharge was 95% for children, and 92% for adults.[108]

Anaphylaxis and risk of death is increased in those with pre-existing asthma, particularly if the asthma is poorly controlled, severe or in asthmatics who delay treatment.[109,110] When anaphylaxis is fatal, death usually occurs very soon after contact with the trigger. From a case series, fatal food reactions cause respiratory arrest typically within 30–35 min; insect stings cause collapse from shock within 10–15 min; and deaths caused by intravenous medication occur most commonly within 5 min. Death never occurred more than 6 h after contact with the trigger.[101,111]

Recognition of an anaphylaxis. Anaphylaxis is the likely diagnosis if a patient who is exposed to a trigger (allergen) develops a sudden illness (usually within minutes) with rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes. The reaction is usually unexpected.

The European Academy of Allergy and Clinical Immunology's (EAACI) Taskforce on Anaphylaxis state that anaphylaxis is highly likely when any one of the following three criteria is fulfilled[96,112]:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g. generalised hives, pruritus or flushing, swollen lips–tongue–uvula) and at least one of the following:
   a. Respiratory compromise, e.g. dyspnoea, wheeze–bronchospasm, stridor, reduced peak expiratory flow (PEF), hypoxaemia.
   b. Reduced blood pressure or associated symptoms of end-organ dysfunction, e.g. hypotonia (collapse), syncope, incontinence.
2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):
   a. Involvement of the skin–mucosal tissue, e.g. generalised hives, itch-flush, swollen lips–tongue–uvula.
   b. Respiratory compromise, e.g. dyspnoea, wheeze–bronchospasm, stridor, reduced PEF, hypoxaemia.
   c. Reduced blood pressure or associated symptoms, e.g. hypotonia (collapse), syncope, incontinence.
   d. Persistent gastrointestinal symptoms, e.g. crampy abdominal pain, vomiting.
3. Reduced blood pressure after exposure to known allergen for that patient (minutes to several hours):
   a. Infants and children: low systolic blood pressure (<70 mmHg from 1 month to 1 year; <70 mmHg + (2 × age) from 1 year to 10 years; <90 mmHg from 11 to 17 years) or >30% decrease in systolic blood pressure.
   b. Adults: systolic blood pressure of <90 mmHg or >30% decrease from that person's baseline.

Treatment. The evidence supporting specific interventions for the treatment of anaphylaxis is limited.[113] A systematic ABCDE approach to recognise and treat anaphylaxis is recommended with immediate administration of intramuscular (IM) adrenaline (Fig. 4.2). Treat life-threatening problems as you find them. The basic principles of treatment are the same for all age groups. Monitor all patients who have suspected anaphylaxis as soon as possible (e.g. by ambulance crew, in the emergency department, etc.). Minimum monitoring includes pulse oximetry, non-invasive blood pressure and a 3-lead ECG.

Patient positioning. Patients with anaphylaxis can deteriorate and are at risk of cardiac arrest if made to sit up or stand up.[114] All patients should be placed in a comfortable position. 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.

Remove the trigger (if possible). Stop any drug suspected of causing anaphylaxis. Remove the stinger after a bee/wasp sting. Early removal is more important than the method of removal.[115] Do not delay definitive treatment if removing the trigger is not feasible.

Cardiac arrest following anaphylaxis. Start CPR immediately and follow current guidelines. Prolonged CPR may be necessary. Rescuers should ensure that help is on its way as early ALS is essential.

Airway obstruction. Anaphylaxis can cause airway swelling and obstruction. This will make airway and ventilation interventions (e.g. bag-mask ventilation, tracheal intubation, cricothyroidotomy) difficult. Consider early tracheal intubation before airway swelling makes this difficult. Call for expert help early.

Adrenaline (first line treatment). Adrenaline is the most important drug for the treatment of anaphylaxis.[116,117] Although there are no randomised controlled trials,[118] adrenaline is a logical treatment and there is consistent anecdotal evidence supporting its use to ease bronchospasm and circulatory collapse. As an

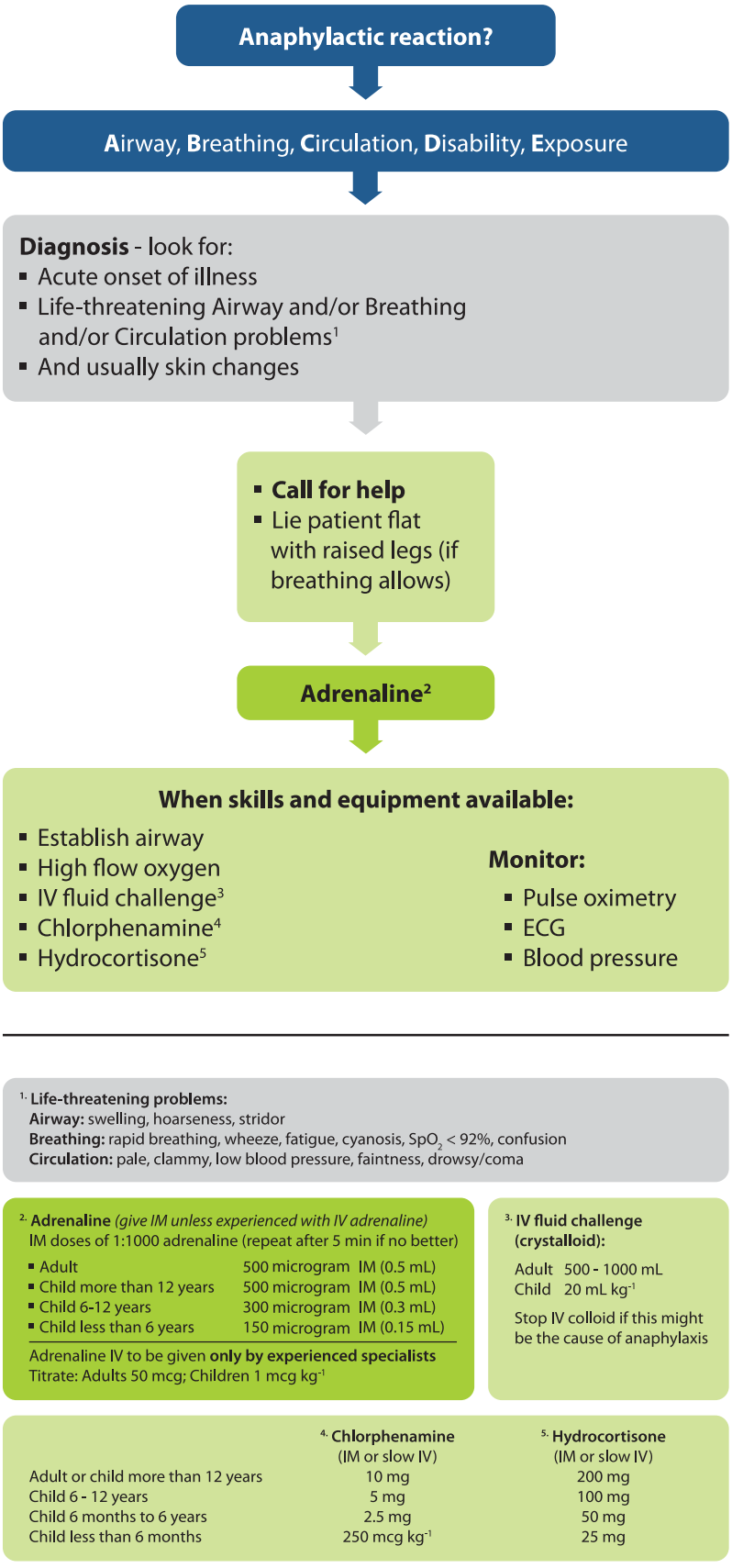

Fig. 4.2. Anaphylaxis treatment algorithm.[101]
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alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. Its beta-receptor activity dilates the bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release. Activation of beta-2 adrenergic receptors on mast cell surfaces inhibit their activation, and early adrenaline attenuates the severity of IgE-mediated allergic reactions. Adrenaline is most effective when given early after the onset of the reaction,[119] and adverse effects are extremely rare with correct IM doses.

Give adrenaline to all patients with life-threatening features. If these features are absent but there are other features of a systemic allergic reaction, the patient needs careful observation and symptomatic treatment using the ABCDE approach.

Intramuscular adrenaline. The intramuscular (IM) route is the best for most individuals who have to give adrenaline to treat anaphylaxis. Monitor the patient as soon as possible (pulse, blood pressure, ECG, pulse oximetry). This will help monitor the response to adrenaline. The IM route has several benefits:

- There is a greater margin of safety.
- It does not require intravenous access.
- The IM route is easier to learn.
- Patients with known allergies can self-administer IM adrenaline.

The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The needle for injection needs to be long enough to ensure that the adrenaline is injected into muscle.[120] The subcutaneous or inhaled routes for adrenaline are not recommended for the treatment of anaphylaxis because they are less effective than the IM route.[121–123]

Adrenaline intramuscular dose. The evidence for the recommended doses is limited. The EAACI suggests IM adrenaline ($1\ mg\ mL^{-1}$) should be given a dose of $10\ mcg\ kg^{-1}$ of body weight to a maximum total dose of 0.5 mg.[96]

The following doses are based on what is considered to be safe and practical to draw up and inject in an emergency (equivalent volume of 1:1000 adrenaline is shown in brackets):

>12 years and adults	500 microgram IM (0.5 mL)
>6–12 years	300 microgram IM (0.3 mL)
>6 months–6 years	150 microgram IM (0.15 mL)
<6 months	150 microgram IM (0.15 mL)

Repeat the IM adrenaline dose if there is no improvement in the patient's condition within 5 min. Further doses can be given at about 5-min intervals according to the patient's response.

Intravenous adrenaline (for specialist use only). There is a much greater risk of causing harmful side effects by inappropriate dosage or misdiagnosis of anaphylaxis when using intravenous (IV) adrenaline.[124] IV adrenaline should only be used by those experienced in the use and titration of vasopressors in their normal clinical practice (e.g. anaesthetists, emergency physicians, intensive care doctors). In patients with a spontaneous circulation, IV adrenaline can cause life-threatening hypertension, tachycardia, arrhythmias, and myocardial ischaemia. If IV access is not available or not achieved rapidly, use the IM route for adrenaline. Patients who are given IV adrenaline must be monitored – continuous ECG and pulse oximetry and frequent non-invasive blood pressure measurements as a minimum. Patients who require repeated IM doses of adrenaline may benefit from IV adrenaline. It is essential that these patients receive expert help early.

Adrenaline intravenous dose (for specialist use only).

- Adults: Titrate IV adrenaline using 50 microgram boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion.[125,126]
- Children: IM adrenaline is the preferred route for children having anaphylaxis. The IV route is recommended only in specialist paediatric settings by those familiar with its use (e.g. paediatric anaesthetists, paediatric emergency physicians, paediatric intensivists) and if the patient is monitored and IV access is already available. There is no evidence on which to base a dose recommendation – the dose is titrated according to response. A child may respond to a dose as small as $1\ mcg\ kg^{-1}$. This requires very careful dilution and checking to prevent dose errors.

Adrenaline intravenous/intraosseous dose (in cardiac arrest only). Cardiac arrest with suspected anaphylaxis should be treated with standard doses of IV or intraosseous (IO) adrenaline for cardiac arrest. If this is not feasible, consider IM adrenaline if cardiac arrest is imminent or has just occurred.

Oxygen (give as soon as available). Initially, give the highest concentration of oxygen possible using a mask with an oxygen reservoir.[127] Ensure high-flow oxygen (usually greater than $10\ L\ min^{-1}$ to prevent collapse of the reservoir during inspiration. If the patient's trachea is intubated, ventilate the lungs with high concentration oxygen using a self-inflating bag.

Fluids (give as soon as available). Large volumes of fluid may leak from the patient's circulation during anaphylaxis. There will also be vasodilation. If IV access has been gained, infuse IV fluids immediately. Give a rapid IV fluid challenge ($20\ mL\ kg^{-1}$) in a child or 500–1000 mL in an adult and monitor the response; give further doses as necessary. There is no evidence to support the use of colloids over crystalloids in this setting. Consider colloid infusion as a cause in a patient receiving a colloid at the time of onset of an anaphylaxis and stop the infusion. A large volume of fluid may be needed.

If IV access is delayed or impossible, the IO route can be used for fluids or drugs. Do not delay the administration of IM adrenaline while attempting IO access.

Antihistamines (give after initial resuscitation). Antihistamines are a second line treatment for anaphylaxis. The evidence to support their use is limited, but there are logical reasons for their use.[128] H_1-antihistamines help counter histamine-mediated vasodilation, bronchoconstriction, and particularly cutaneous symptoms. There is little evidence to support the routine use of an H_2-antihistamine (e.g. ranitidine, cimetidine) for the initial treatment of anaphylaxis.

Glucocorticosteroids (give after initial resuscitation). Corticosteroids may help prevent or shorten protracted reactions, although the evidence is limited.[129] In asthma, early corticosteroid treatment is beneficial in adults and children. There is little evidence on which to base the optimum dose of hydrocortisone in anaphylaxis.

Other drugs.

Bronchodilators. The presenting symptoms and signs of severe anaphylaxis and life-threatening asthma can be the same. Consider further bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), aminophylline (IV) or magnesium (IV) (see asthma). IV magnesium is a vasodilator and can make hypotension worse.

Cardiac drugs. Adrenaline remains the first line vasopressor for the treatment of anaphylaxis. There are animal studies and case reports describing the use of other vasopressors and inotropes (noradrenaline, vasopressin, terlipressin metaraminol, methoxamine, and glucagon) when initial resuscitation with adrenaline and fluids has not been successful.[130–142] Use these drugs only in specialist settings (e.g. ICU) where there is experience in their use. Glucagon can be useful to treat anaphylaxis in a patient taking a beta-blocker.[143] Some case reports of cardiac arrest suggest cardiopulmonary bypass[144,145] or mechanical chest compression devices may also be helpful.[146]

Investigations. Undertake the usual investigations appropriate for a medical emergency, e.g. 12-lead ECG, chest X-ray, urea and electrolytes, arterial blood gases, etc.

Mast cell tryptase. The specific test to help confirm a diagnosis of anaphylaxis is measurement of mast cell tryptase. Tryptase is the major protein component of mast cell secretory granules. In anaphylaxis, mast cell degranulation leads to markedly increased blood tryptase concentrations. Tryptase concentrations in the blood may not increase significantly until 30 min or more after the onset of symptoms, and peak 1–2 h after onset.[147] The half-life of tryptase is short (approximately 2 h), and concentrations may be back to normal within 6–8 h, so timing of any blood samples is very important. The time of onset of the anaphylaxis is the time when symptoms were first noticed.

(a) Minimum: one sample at 1–2 h after the start of symptoms.
(b) Ideally: Three timed samples:

- Initial 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 at 24 h or in convalescence (for example in a follow-up allergy clinic). This provides baseline tryptase levels – some individuals have an elevated baseline level.

Serial samples have better specificity and sensitivity than a single measurement in the confirmation of anaphylaxis.[148]

Discharge and follow-up. Patients who have had suspected anaphylaxis (i.e. an airway, breathing or circulation problem) should be treated and then observed in a clinical area with facilities for treating life-threatening ABC problems. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation. The exact incidence of biphasic reactions is unknown. Although studies quote an incidence of 1–20%, it is not clear whether all the patients in these studies actually had anaphylaxis or whether the initial treatment was appropriate.[149] There is no reliable way of predicting who will have a biphasic reaction. It is therefore important that decisions about discharge are made for each patient by an experienced clinician.

Before discharge from hospital, all patients must:

- Be reviewed by an allergy specialist and have a treatment plan based on their individual risk.
- Be given clear instructions to return to hospital if symptoms return.
- Be considered for an adrenaline auto-injector, or given a replacement[150–152] and ensured that appropriate training has been given.
- Have a plan for follow-up, including contact with the patient's general practitioner.

Patients need to know the allergen responsible (if identified) and how to avoid it. Patients need to be able to recognise the early symptoms of anaphylaxis, so that they can summon help quickly and prepare to use their emergency medication. Although there are no randomised clinical trials, there is evidence that individualised action plans for self-management should decrease the risk of recurrence.[153]

Traumatic cardiac arrest

Introduction. 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.[154,155] 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.

Diagnosis. The diagnosis of traumatic cardiac arrest is made clinically. The patient presents with agonal or absent spontaneous respiration and absence of a central pulse.

A peri-arrest state is characterised by cardiovascular instability, hypotension, loss of peripheral pulses in uninjured regions and a deteriorating conscious level without obvious central nervous system (CNS) cause. If untreated, this state is likely to progress to cardiac arrest. Rapid focused ultrasound assessment may be helpful in the immediate diagnosis and management, but should not delay resuscitative interventions.[156]

It is vital that a medical cardiac arrest is not misdiagnosed as a TCA and must be treated with the universal ALS algorithm. Cardiac arrest or other causes of sudden loss of consciousness (e.g. hypoglycaemia, stroke, seizures) may cause a secondary traumatic event. Some observational studies have reported that about 2.5% of non-traumatic OHCAs occur in cars.[157–159] In these cases, shockable rhythms (VF/pVT) are more common.[97] The primary cause of the cardiac arrest can be elucidated from information about past medical history, events preceding the accident (if possible), and a systematic post-ROSC assessment, including a 12-lead ECG.

Prognostic factors and withholding resuscitation. There are no reliable predictors of survival for traumatic cardiac arrest. Factors that are associated with survival include the presence of reactive pupils, an organised ECG rhythm and respiratory activity.[159,160] Short duration of CPR and prehospital times have also been associated with positive outcomes.[161]

A large systematic review reported an overall survival rate of 3.3% in blunt and 3.7% in penetrating trauma, with good neurological outcome in 1.6% of all cases.[154] Outcome is age dependent, with children having a better prognosis than adults.[97,154] There is considerable variation in reported mortality (range 0–27%) reflecting heterogeneity in casemix and care in different systems. PEA, which in TCA may initially be a low output state, and asystole are the prevalent heart rhythms in TCA. Ventricular fibrillation (VF) is rare but carries the best prognosis.[97,155]

One study reported good neurological outcome in 36.4% of TCA patients presenting with VF, but only in 7% with PEA and 2.7% of those in asystole,[155] but other studies of patients in non-shockable rhythms have reported 100% mortality.[159,162,163] 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.[164] However, neurologically intact survivors initially presenting in this state have been reported.[155] We therefore recommend the following approach:

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

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

We suggest termination of resuscitative efforts should be considered if there is:

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

Trauma care systems throughout Europe vary considerably and we recommend establishing regional guidelines for treatment of TCA and tailoring patient pathways to infrastructure and resources.

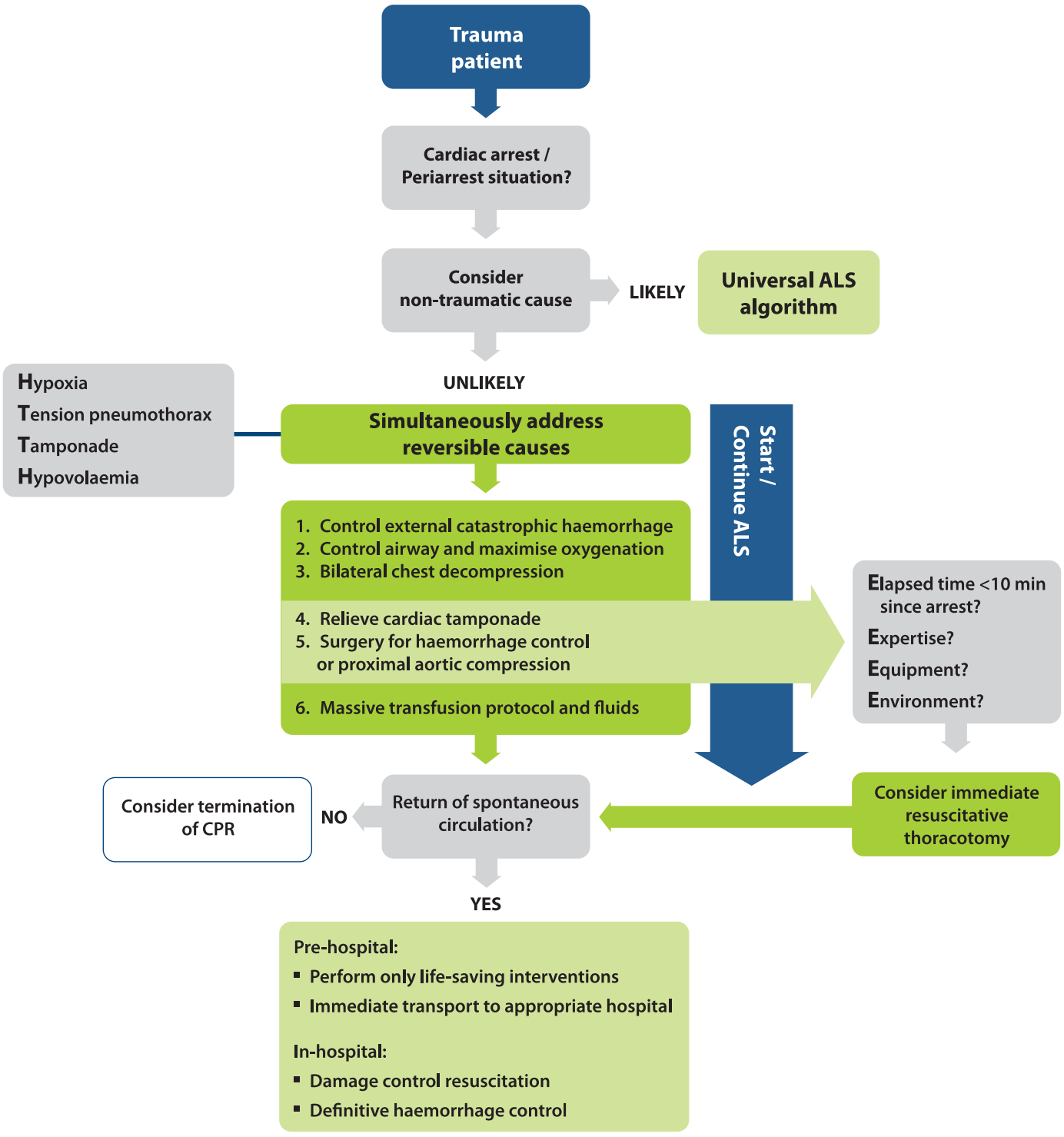

Fig. 4.3. Traumatic cardiac arrest algorithm.

Treatment. Emphasis on rapid treatment of all potentially reversible pathology is the basis of treatment guidelines. These principles are addressed in several treatment algorithms.[97,165–167] All algorithms attempt to rapidly address reversible causes of TCA in the prehospital and in-hospital phases of care. Fig. 4.3 shows a traumatic cardiac (peri-) arrest algorithm, which is based on the universal ALS algorithm.[168]

Effectiveness of chest compressions. Chest compressions are still the standard of care in patients with cardiac arrest, irrespective of aetiology. In cardiac arrest caused by hypovolaemia, cardiac tamponade or tension pneumothorax, chest compressions are unlikely to be as effective as in normovolaemic cardiac arrest.[169–172] Because of this fact, chest compressions take a lower priority than the immediate treatment of reversible causes, e.g. thoracotomy, controlling haemorrhage, etc. In an out-of-hospital setting, only essential life-saving interventions should be performed on scene followed by rapid transfer to the nearest appropriate hospital.

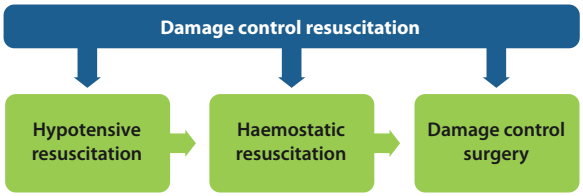

Fig. 4.4. Principles of damage control resuscitation in trauma.

Hypovolaemia. Uncontrolled haemorrhage is the cause of traumatic cardiac arrest in 48% of all TCA.[97] The treatment of severe hypovolaemic shock has several elements. The main principle is to achieve 'haemostasis without delay', usually with surgical or radiological intervention. Temporary haemorrhage control can be lifesaving:

- Treat compressible external haemorrhage with direct pressure (with or without a dressing), use tourniquets if needed and/or apply topical haemostatic agents.[173]
- Non-compressible haemorrhage is more difficult. Use splints (pelvic splint), blood products, intravenous fluids and tranexamic acid while moving the patient to surgical haemorrhage control.

Over the past 10 years 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[174] and general consensus have supported a conservative approach to intravenous fluid infusion, with permissive hypotension until surgical haemostasis is achieved. Permissive hypotension allows intravenous fluid administration to a volume sufficient to maintain a radial pulse.[175,176]

Haemostatic resuscitation is the very early use of blood products as primary resuscitation fluid to prevent exsanguination by trauma-induced coagulopathy.[177] The recommended ratio of packed red cells, fresh frozen plasma and platelets is 1:1:1.[178] Some services have also started using blood products in the prehospital phase of care.[179,180]

Simultaneous damage control surgery and haemostatic resuscitation using massive transfusion protocols (MTP)[173] are the principles of damage control resuscitation in patients with exsanguinating injuries (Fig. 4.4).[177]

Although the evidence for permissive hypotension during resuscitation is limited, particularly with regards to blunt trauma, permissive hypotension has been endorsed in both civilian and military care,[181] generally aiming for a systolic blood pressure of 80–90 mmHg. Caution is advised with this strategy in 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.[176]

Tranexamic acid (TXA) (loading dose 1 g over 10 min followed by infusion of 1 g over 8 h) increases survival from traumatic haemorrhage.[182] It is most effective when administered within the first hour and certainly within the first 3 h following trauma.[182] Give TXA in the prehospital setting when possible.

Hypoxaemia. Hypoxaemia due to airway obstruction and traumatic asphyxia has been reported as cause of traumatic cardiac arrest in 13% of all cases.[97] Effective airway management and ventilation can reverse hypoxic cardiac arrest and it is essential to establish and maintain oxygenation of trauma patients with a severely compromised airway. Tracheal intubation in trauma patients is a difficult procedure with a high failure rate if carried out by less experienced care providers.[183,184] Use basic airway manoeuvres and second-generation supraglottic airways to maintain oxygenation if tracheal intubation cannot be accomplished immediately.

Positive pressure ventilation worsens hypotension by impeding venous return to the heart, particularly in hypovolaemic patients.[185] Low tidal volumes and slow respiratory rates may help optimise cardiac preload. Monitor ventilation with continuous waveform capnography and adjust to achieve normocapnia.[173]

Tension pneumothorax. Thirteen percent of all cases of TCA are caused by tension pneumothorax.[97] To decompress the chest in TCA, perform bilateral thoracostomies in the 4th intercostal space, extending to a clamshell thoracotomy if required. In the presence of positive pressure ventilation, thoracostomies are likely to be more effective than needle thoracocentesis and quicker than inserting a chest tube (see tension pneumothorax).[186,187]

Cardiac tamponade and resuscitative thoracotomy. Cardiac tamponade is the underlying cause of approximately 10% of cardiac arrest in trauma.[97] Where there is TCA *and* penetrating trauma to the chest or epigastrium, immediate resuscitative thoracotomy (RT) via a clamshell incision[188] can be life saving.[189] The chance of survival is about 4 times higher in cardiac stab wounds than in gunshot wounds.[190]

Resuscitative thoracotomy is also applied for other life threatening injuries; the evidence was examined in 2012[191] and guidelines produced which recommend that, *after arrival in hospital*, the decision to proceed with RT should include the following criteria:

- blunt trauma patients with less than 10 min of prehospital CPR;
- penetrating torso trauma patients with less than 15 min of CPR.

These guidelines estimate survival rates for RT of approximately 15% for all patients with penetrating wounds and 35% for patients with a penetrating cardiac wound. In contrast, survival from RT following blunt trauma is dismal, with survival rates of 0–2% being reported.[191,192]

Successful RT is time critical. One UK service recommends that if surgical intervention cannot be accomplished within 10 min after loss of pulse in patients *with penetrating chest injury*, on scene RT should be considered.[10] Based on this approach, of 71 patients who underwent RT at scene, 13 patients survived and 11 of these made a good neurological recovery.

The prerequisites for a successful RT can be summarised as the 'four Es 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 10 min.

If any of the four criteria is not met, RT is futile and exposes the team to unnecessary risks.[193]

Needle aspiration of tamponade, with or without ultrasound guidance, is unreliable because the pericardium is commonly full of clotted blood.[194,195] If thoracotomy is not possible, however, consider ultrasound guided pericardiocentesis to treat TCA associated with suspected cardiac tamponade. Non-image guided pericardiocentesis is an alternative, only if ultrasound is not available. Placement of a pericardial drain may be beneficial in some patients.

Diagnostics. Ultrasonography should be used in the evaluation of the compromised trauma patient to target life-saving interventions if the cause of shock cannot be established clinically.[196,173] Haemoperitoneum, haemo- or pneumothorax and cardiac tamponade can be diagnosed reliably in minutes, even in the prehospital phase.[197] Early whole-body computed tomography (WBCT) scanning as part of the primary survey may improve outcome in major trauma.[198] WBCT is increasingly employed to identify the source of shock and to guide subsequent haemorrhage control.

Prehospital care. Short prehospital times are associated with increased survival rates for major trauma and TCA. The time elapsed between injury and surgical control of bleeding should therefore be minimised and the patient should be immediately transferred to a trauma centre for ongoing damage control resuscitation.[173] A 'scoop and run' concept for these patients may be life saving.

Tension pneumothorax

Introduction

Tension pneumothorax defined as haemodynamic compromise in a patient with an expanding intrapleural air mass is a treatable cause of cardiac arrest and should be excluded during CPR.[199] Tension pneumothorax can occur in a variety of clinical situations including trauma, asthma and other respiratory disease, but can also be iatrogenic following invasive procedures, e.g. attempts at central venous catheter insertion. It is more common and often more severe in patients undergoing positive pressure ventilation.[200] The incidence of tension pneumothorax is approximately 5% in major trauma patients treated in the prehospital setting (13% of those developing TCA), and less than 1% of adults admitted to ICU.[97,201,202]

Diagnosis

Diagnosis of tension pneumothorax in a patient with cardiac arrest or haemodynamic instability must be based on clinical examination. The symptoms include haemodynamic compromise (hypotension or cardiac arrest) in conjunction with signs suggestive of a pneumothorax (preceding respiratory distress, hypoxia, absent unilateral breath sounds on auscultation, subcutaneous emphysema) and mediastinal shift (tracheal deviation and jugular venous distention).[200] During CPR, presentation is not always classical, but when it is suspected in the presence of cardiac arrest or severe hypotension, chest decompression should be carried out immediately before radiographic confirmation.[201]

Treatment

Needle decompression. Needle chest decompression is rapid and within the skill set of most ambulance personnel but is of limited value.[203,204] A significant proportion of patients have chest wall thickness which makes needle decompression with a standard length 14-gauge cannula ineffective.[205] Cannulae are also prone to kinking and blockage.[206] Any attempt at needle decompression should be followed by insertion of a chest tube (see asthma).

Thoracostomy. Tracheal intubation, positive pressure ventilation and formal chest decompression effectively treats tension pneumothorax in patients with TCA. Simple thoracostomy is easy to perform and used routinely by several prehospital physician services.[187,207] This consists of the first stage of standard chest tube insertion – a simple incision and rapid dissection into the pleural space in the positive pressure ventilated patient (see traumatic cardiac arrest). Chest tube insertion is then carried out after the resuscitation phase. This requires additional equipment, takes longer to perform and creates a closed system that has the potential to re-tension. Chest drain tubes may become blocked with lung or blood clots and have the potential to kink.

Tamponade

Introduction

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.

Treatment

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

Thrombosis

Pulmonary embolism

Introduction. Cardiac arrest from acute pulmonary embolism is the most serious clinical presentation of venous thromboembolism, in most cases originating from a deep venous thrombosis (DVT).[208] The reported incidence of cardiac arrest caused by pulmonary embolism is 2–9% of all OHCAs,[209–212] and 5–6% of all in-hospital cardiac arrests.[213,214] but it is likely to be underestimated. Overall survival is low.[211,215] Specific treatments for cardiac arrest resulting from pulmonary embolism include administration of fibrinolytics, surgical embolectomy and percutaneous mechanical thrombectomy.

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,[214] but accurate prehospital diagnosis of acute pulmonary embolism is particularly challenging.[212,216]

The 2014 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.[208]

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:

- 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 low extremity swelling).[208] However, pulmonary embolism may not be symptomatic until it presents as sudden cardiac arrest.[217]
- Obtain information about past medical history, predisposing factors, and medication that may support diagnosis of pulmonary embolism, although none of these are specific, e.g.[208]
  - Previous pulmonary embolism or 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

In as many as 30% of the patients with pulmonary embolism, no risk factors are apparent.[218]

- If a 12-lead ECG can be obtained before onset of cardiac arrest, changes indicative of right ventricular strain may be found:
  - 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[208,219]
- Cardiac arrest commonly presents as PEA.[211]
- 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.[209]
- Consider emergency echocardiography performed by a qualified sonographer as an additional diagnostic tool to identify pulmonary embolism if it can be performed without interruptions to chest compressions, e.g. during rhythm check. Echocardiographic findings are evident after acute obstruction of more than 30% of the pulmonary arterial tree.[220] Common echocardiographic findings are an enlarged right ventricle with a flattened interventricular septum,[221,222] but absence of these features does not exclude pulmonary embolism.[223] Signs of right ventricular overload or dysfunction may also be caused by other cardiac or pulmonary disease.[224]
- More specific diagnostic methods, e.g. D-dimer testing, (computed tomographic) pulmonary angiography, lung scintigraphy, or magnetic resonance angiography, are not recommended for a cardiac arrest situation.

Modifications to cardiopulmonary resuscitation. A meta-analysis, which included patients with pulmonary embolism as a cause of cardiac arrest, concluded that fibrinolytics increased the rate of ROSC, survival to discharge and long-term neurological function.[225] A subgroup analysis of patients treated with thrombolytics compared with placebo in a randomised controlled trial[215] did not prove survival difference. However, this study was not designed for treatment of pulmonary embolism and not powered to reach significance in this small subgroup. Some other non-randomised studies have also documented use of thrombolytics in the treatment of cardiac arrest due to acute pulmonary embolism, but evidence for improved neurologically intact survival to hospital discharge is limited.[211,226]

In a cardiac arrest presumed to be caused by acute pulmonary embolism, follow the standard guidelines for ALS (see adult advanced life support).[168] The decision to treat for acute pulmonary embolism must be taken early, when a good outcome is still possible. The following treatment modifications are recommended:

- Consider administration of fibrinolytic therapy when acute pulmonary embolism is a known or suspected cause of cardiac arrest. Ongoing CPR is not a contraindication to fibrinolysis. Despite increased risk of severe bleeding, fibrinolysis may be an effective treatment, which can be initiated without delay, even outside specialised healthcare facilities. The potential benefit of fibrinolysis in terms of improved survival outweighs potential risks in a location where no alternative exists, e.g. in the prehospital setting.[211,227–231]
- Once a fibrinolytic drug is administered, continue CPR for at least 60–90 min before terminating resuscitation attempts.[227,232] Survival and good neurological outcome have been reported in cases requiring in excess of 100 min of CPR.[233]
- Consider the use of a mechanical chest compression device when maintenance of high quality chest compressions is needed for a prolonged time.

Extracorporeal CPR. Some observational studies suggest the use of extracorporeal life support (ECLS) if cardiac arrest is associated with pulmonary embolism.[234,235] The implementation of ECLS requires considerable resource and training. Its use should be considered as a rescue therapy for those patients in whom initial ALS measures are unsuccessful and/or to facilitate pulmonary thrombectomy.

Surgical embolectomy and mechanical thrombectomy. Survival of patients who underwent surgical embolectomy during CPR due to pulmonary embolism was reported as 13% and 71% in two case series,[229,236] but these results were not compared with standard treatment. Routine use of surgical embolectomy or mechanical thrombectomy for cardiac arrest from suspected pulmonary embolism is not recommended, but these methods may be considered when pulmonary embolism is the known cause of cardiac arrest.

Percutaneous pulmonary thrombectomy. In one case series, percutaneous pulmonary thrombectomy during CPR was successful in six of seven patients,[237,238] but larger studies are needed to validate this method.

Post-resuscitation care. In patients with sustained ROSC, exclude intra-abdominal and intra-thoracic CPR-related injuries, especially if a mechanical chest compression device was used simultaneously with administration of fibrinolytics.[239–241] Attempt to identify and treat the original cause of the pulmonary embolism. Evaluate the risks of a further pulmonary embolism and treat accordingly.

Coronary thrombosis

Coronary heart disease is the most frequent cause of OHCA. The peri-resuscitation management of acute coronary syndromes is addressed in a separate chapter (see Section 8 Initial management of acute coronary syndromes).[242] In cardiac arrest centres, coronary artery occlusion or high degree stenoses can be identified and treated. Of all patients in OHCA, however, at least half are not transported to hospital when ROSC is not achieved (see Section 10 Ethics of resuscitation and end-of-life decisions).[243] Although proper diagnosis of the cause may be difficult in a patient already in cardiac arrest, if the initial rhythm is VF it is most likely that the cause is coronary artery disease with an occluded large coronary vessel.

Consider transportation to hospital with ongoing CPR if treatment options are available that cannot be applied in the prehospital setting, such as immediate coronary angiography, primary percutaneous coronary intervention (PPCI) or other interventions such as (more rarely) pulmonary embolectomy (see pulmonary embolism). The decision to transport is complex and may depend on local circumstances. Prehospital initiation of extracorporeal cardiopulmonary life support (ECLS) requires specialised expertise and its feasibility on a wide-scale has not been established.[244–246] Mechanical chest compression devices maintain high quality CPR during transport and PCI (see cardiac arrest in HEMS and air ambulances).[247,248]

There is limited evidence for recommending routine transport to hospital with ongoing CPR. The decision will depend on patient selection, availability of optimal methods for mechanical or circulatory support during and after transport to the hospital, management of underlying pathology, treatment after ROSC, complication rate and outcome. There are no large outcome studies available, but small case series suggest benefit in selected cases.[249]

Before definitive recommendations can be made, controlled studies are needed.[250]

Transport with ongoing CPR and immediate access to the catheterisation laboratory may be considered if a prehospital and in-hospital infrastructure is available with teams experienced in mechanical or haemodynamic support and rescue PPCI with ongoing CPR. Excellent cooperation is required between prehospital and in-hospital teams. A decision to transport with ongoing CPR should take into consideration a realistic chance of survival (e.g. witnessed cardiac arrest with initial shockable rhythm (VF/pVT) and bystander CPR). Intermittent ROSC also strongly favours a decision to transport.[251]

Toxins

General considerations

Introduction. Overall, poisoning rarely causes cardiac arrest or death,[252] but hospital admissions are common, accounting for as many as 140,000 admissions each year in the UK.[252] Poisoning by therapeutic or recreational drugs and by household products are the main reasons for hospital admission and poison centre calls. Inappropriate drug dosing, drug interactions and other medication errors can also cause harm. Accidental poisoning is commonest in children. Homicidal poisoning is uncommon. Industrial accidents, warfare or terrorism can also cause exposure to toxins. Evidence for treatment consists primarily of animal studies, case reports and small case series.[253–255]

Prevention of cardiac arrest. Assess the patient using 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).[256,257] Early tracheal intubation of unconscious patients by trained personnel may decrease the risk of aspiration. Drug-induced hypotension usually responds to IV fluids, but occasionally vasopressor support (e.g. noradrenaline infusion) is required. Measure electrolytes (particularly potassium), blood glucose and arterial blood gases. Retain samples of blood and urine for analysis. Patients with severe poisoning should be cared for in a critical care setting.[257]

Modifications to resuscitation.

- Have a low threshold to ensure your personal safety where there is a suspicious cause or unexpected cardiac arrest. This is especially so when there is more than one casualty.
- Avoid mouth-to-mouth breathing in the presence of chemicals such as cyanide, hydrogen sulphide, corrosives and organophosphates.
- Treat life-threatening tachyarrhythmias with cardioversion according to the peri-arrest arrhythmia guidelines (see adult advanced life support).[168] This includes correction of electrolyte and acid-base abnormalities (see hypo-/hyperkalaemia and other electrolyte disorders).
- Try to identify the poison(s). 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.
- Measure the patient's temperature because hypo- or hyperthermia may occur after drug overdose (see hypo-/hyperthermia).
- Be prepared to continue resuscitation for a prolonged period, particularly in young patients, as the poison may be metabolised or excreted during extended resuscitation measures.
- Alternative approaches which may be effective in severely poisoned patients include: higher doses of medication than in standard protocols (e.g. high-dose insulin euglycemia)[258]; non-standard drug therapies (e.g. IV lipid emulsion)[259–262]; prolonged CPR, extracorporeal life support (ECLS),[263,264] and haemodialysis.
- Consult regional or national poisons centres for information on treatment of the poisoned patient. The International Programme on Chemical Safety (IPCS) lists poison centres on its website: http://www.who.int/ipcs/poisons/centre/en/.
- On-line databases for information on toxicology and hazardous chemicals may be helpful: http://toxnet.nlm.nih.gov/.

Specific therapeutic measures

There are few specific therapeutic measures for poisoning that are useful immediately and improve outcomes: decontamination, enhancing elimination, and the use of specific antidotes.[265–267] Many of these interventions should be used only 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 toxin from the body determined by the route of exposure:

- For dermal exposures initial management consists of clothing removal and copious irrigation with water, except in case of reactive alkali metals that can ignite.
- Routine use of gastric lavage for gastrointestinal decontamination is no longer recommended. In the rare instances (e.g. lethal ingestion with recent exposure), it should only be performed by individuals with proper training and expertise. Gastric lavage may be associated with life-threatening complications, e.g. aspiration pneumonitis, aspiration pneumonia, esophageal or gastric perforation, fluid and electrolyte imbalances, arrhythmia. It is contraindicated if the airway is not protected and if a hydrocarbon with high aspiration potential or a corrosive substance has been ingested.[267,268]
- The preferred method of gastrointestinal decontamination in patients with an intact or protected airway is activated charcoal. It is most effective if given within 1 h of the time of the ingestion.[269] Activated charcoal does not bind lithium, heavy metals and toxic alcohols. Most common side effects are vomiting and constipation. The evidence that active charcoal improves outcome is limited.[257]
- Based mainly on volunteer studies, consider whole-bowel irrigation in 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. It may be also used for the removal of substantial amounts of iron, lithium, potassium, or packets of illicit drugs. Whole-bowel irrigation is contraindicated in patients with bowel obstruction, perforation, ileus, and haemodynamic instability.[270]
- Avoid routine administration of laxatives (cathartics) and do not use emetics (e.g. ipecac syrup).[271–273]

Enhanced elimination. Modalities removing a toxin from the body once it has been absorbed include multiple-dose activated charcoal (MDAC), urinary alkalinisation and extracorporeal elimination techniques:

- MDAC, multiple doses of activated charcoal administered over several hours, can increase certain drug elimination.[274,275] Give an initial dose of 50–100 g in adults (25–50 g in children).
- Urinary alkalinisation (urine pH $\geq$ 7.5) involves an IV sodium bicarbonate infusion. It is most commonly performed in patients with salicylate intoxication who do not need dialysis. Consider urine alkalinisation with high urine flow (about 600 mL h^{-1}) in severe poisoning by phenobarbital and herbicides, e.g. 2,4-dichlorophenoxyacetic acid or methylchlorophenoxypropionic acid (mecoprop). Hypokalaemia is the most common complication.[265]

- Haemodialysis removes drugs or metabolites with low molecular weight, low protein binding, small volumes of distribution and high water solubility. In case of hypotension, use continuous veno-venous hemofiltration (CVVH) or continuous veno-venous haemodialysis (CVVHD) alternatively.[257]

Specific poisons

These guidelines address only some of the more common poisons causing cardiac arrest.

Benzodiazepines. Overdose of benzodiazepines can cause loss of consciousness, respiratory depression and hypotension. Flumazenil, a competitive antagonist of benzodiazepines, may be used for reversal of benzodiazepine sedation when there is no history or risk of seizures. Reversal of benzodiazepine intoxication with flumazenil can be associated with significant toxicity (seizure, arrhythmia, hypotension, and withdrawal syndrome) in patients with benzodiazepine dependence or co-ingestion of proconvulsant medications such as tricyclic antidepressants.[276–278] The routine use of flumazenil in the comatose overdose patient is not recommended.There are no specific modifications to the ALS algorithm required for cardiac arrest caused by benzodiazepines.[278–282]

Opioids. Opioid poisoning causes respiratory depression followed by respiratory insufficiency or respiratory arrest. The respiratory effects of opioids are reversed rapidly by the opiate antagonist naloxone.

In severe respiratory depression caused by opioids, there are fewer adverse events when airway opening, oxygen administration and ventilation are carried out before giving naloxone[283–289]; The use of naloxone can prevent the need for intubation. The preferred route for giving naloxone depends on the skills of the rescuer: intravenous (IV), intramuscular (IM), subcutaneous (SC), intraosseous (IO) and intranasal (IN) routes are all suitable.[290,291] The non-IV routes can be quicker because time is saved in not having to establish IV access, which may be difficult in an IV drug abuser. The initial doses of naloxone are 0.4–2 mg IV, IO, IM or SC, and may be repeated every 2–3 min. Additional doses may be needed every 20–60 min. Intranasal dosing is 2 mg IN (1 mg in each nostril) which may be repeated every 5 min. Titrate the dose until the victim is breathing adequately and has protective airway reflexes. Large opioid overdoses may require a total dose of up to 10 mg of naloxone.[283–285,290–300] All patients treated with naloxone must be monitored.

Acute withdrawal from opioids produces a state of sympathetic excess and may cause complications such as pulmonary oedema, ventricular arrhythmias and severe agitation. Use naloxone reversal of opioid intoxication with caution in patients suspected of opioid dependence.

There are no data on the use of any additional therapies beyond standard ALS guidelines in opioid-induced cardiac arrest. In respiratory arrest there is good evidence for the use of naloxone, but not for any other adjuncts or changes in interventions.[284]

Tricyclic antidepressants. This section addresses both tricyclic and related cyclic drugs (e.g. amitriptyline, desipramine, imipramine, nortriptyline, doxepin, and clomipramine). Self-poisoning with tricyclic antidepressants is common and can cause hypotension, seizures, coma and life-threatening arrhythmias. Cardiac toxicity mediated by anticholinergic and Na^+ channel-blocking effects can produce a wide complex tachycardia (VT). Hypotension is exacerbated by alpha-1 receptor blockade. Anticholinergic effects include mydriasis, fever, dry skin, delirium, tachycardia, ileus, and urinary retention. Most life-threatening problems occur within the first 6 h after ingestion.[301–303]

A widening QRS complex (>100 ms) and right axis deviation indicates a greater risk of arrhythmias.[304–306] Give sodium bicarbonate (1–2 mmol kg^{-1}) for the treatment of tricyclic-induced ventricular arrhytmias.[307–312] While no study has investigated the optimal target arterial pH with bicarbonate therapy, a pH of 7.45–7.55 is recommended.[255,257] Administration of bicarbonate may resolve arrhythmias and reverse hypotension even in the absence of acidosis.[312]

Intravenous lipid infusions in experimental models of tricyclic toxicity have suggested benefit but there are few human data.[313,314] Anti-tricyclic antibodies have also been beneficial in experimental models of tricyclic cardiotoxicity.[315–320] One small human study provided evidence of safety but clinical benefit has not been shown.[321]

There are no randomised controlled trials evaluating conventional versus alternative treatments for cardiac arrest caused by tricyclic toxicity. One small case series showed improvement with the use of sodium bicarbonate but the concomitant use of physostigmin prevents the ability to generalise its results.[322]

Cocaine. Sympathetic overstimulation associated with cocaine toxicity can cause agitation, tachycardia, hypertensive crisis, hyperthermia and coronary vasoconstriction causing myocardial ischaemia with angina.

In patients with severe cardiovascular toxicity, alpha blockers (phentolamine),[323] benzodiazepines (lorazepam, diazepam),[324,325] calcium channel blockers (verapamil),[326] morphine,[327] and sublingual nitroglycerine[328,329] may be used as needed to control hypertension, tachycardia, myocardial ischaemia and agitation. The evidence for or against the use of beta-blocker drugs,[330–333] including those beta-blockers with alpha blocking properties (carvedilol and labetolol) is limited.[334–336] The optimal choice of anti-arrhythmic drug for the treatment of cocaine-induced tachyarrhythmias is not known. If cardiac arrest occurs, follow standard resuscitation guidelines.[337]

Local anaesthetics. Systemic toxicity of local anaesthetics involves the central nervous and cardiovascular systems. Severe agitation, loss of consciousness, seizures, bradycardia, asystole or ventricular tachyarrhythmias can all occur. Toxicity typically occurs in the setting of regional anaesthesia, when a bolus of local anaesthetic inadvertently enters an artery or vein (see perioperative cardiac arrest).

Although there are many case reports and case series of patients who were resuscitated after administration of IV lipid emulsion, evidence for its benefit in treating local anaesthetic-induced cardiac arrest is limited. Despite the paucity of data, patients with both cardiovascular collapse and cardiac arrest attributable to local anaesthetic toxicity may benefit from treatment with intravenous 20% lipid emulsion in addition to standard ALS.[338–352] Give an initial intravenous bolus injection of 20% lipid emulsion 1.5 mL kg^{-1} over 1 min followed by an infusion at 15 mL kg^{-1} h^{-1}. Give up to a maximum of two repeat boluses at 5-min intervals and continue until the patient is stable or has received up to a maximum cumulative dose of 12 mL kg^{-1} of lipid emulsion.[259–262,353] Standard cardiac arrests drugs (e.g. adrenaline) should be given according to ALS guidelines, although animal studies provide inconsistent evidence for their role in local anaesthetic toxicity.[349,352,354–356]

Beta-blockers. Beta-blocker toxicity causes bradyarrhythmias and negative inotropic effects that are difficult to treat, and can lead to cardiac arrest.

Evidence for treatment is based on case reports and animal studies. Improvement has been reported with glucagon (50–150 mcg kg^{-1}),[357–370] high-dose insulin and glucose,[371–373] lipid emulsions,[374–377] phosphodiesterase inhibitors,[378,379]

extracorporeal and intra-aortic balloon pump support,[380–382] and calcium salts.[258,383]

Calcium channel blockers. Calcium channel blocker overdose is emerging as a common cause of prescription drug poisoning deaths.[384,385] Overdose of short-acting drugs can rapidly progress to cardiac arrest. Overdose by sustained-release formulations can result in delayed onset of arrhythmias, shock, and sudden cardiac collapse. The treatment for calcium channel blocker poisoning is supported by low-quality evidence.[386]

Give calcium chloride 10% in boluses of 20 mL (or equivalent dose of calcium gluconate every 2–5 min in severe bradycardia or hypotension followed by an infusion if needed.[255,257,258,386,387] While calcium in high doses can overcome some of the adverse effects, it rarely restores normal cardiovascular status. Haemodynamic instability may respond to high doses of insulin (1 unit kg^{-1} followed by an infusion of 0.5–2.0 units kg^{-1} h^{-1}) given with glucose supplementation and electrolyte monitoring in addition to standard treatments including fluids and vasopressors (e.g. dopamine, norepinephrine, vasopressin).[386–398] Extracorporeal life support (ECLS) was associated with improved survival in patients with severe shock or cardiac arrest at the cost of limb ischaemia, thrombosis, and bleeding.[264] Studies on decontamination, 4-aminopyridine, atropine, glucagon, pacemakers, levosimendan, and plasma exchange reported variable results.[386]

Digoxin. Although cases of digoxin poisoning are fewer than those involving calcium channel and beta-blockers, the mortality rate from digoxin is far greater. Other drugs including calcium channel blockers and amiodarone can also cause plasma concentrations of digoxin to rise. Atrioventricular conduction abnormalities and ventricular hyperexcitability due to digoxin toxicity can lead to severe arrhythmias and cardiac arrest.

Specific antidote therapy with digoxin-specific antibody fragments (digoxin-Fab) should be used if there are arrhythmias associated with haemodynamic instability.[257,399–401] Digoxin-Fab therapy may also be effective in poisoning from plants (e.g. oleander) and Chinese herbal medications containing cardiac glycosides.[399,402,403] Digoxin-Fab interfere with digoxin immunoassay measurements and can lead to overestimation of plasma digoxin concentrations. In acute poisoning, give an initial bolus of 2 vials digoxin-Fab (38 mg per vial) and repeat dose if necessary.[401] In a cardiac arrest, consider administration of 2 up to 10 vials IV over 30 min.

Cyanides. Cyanide is generally considered to be a rare cause of acute poisoning; however, cyanide exposure occurs relatively frequently in patients with smoke inhalation from residential or industrial fires. Cyanides are also used in several chemical and industrial processes. Its main toxicity results from inactivation of cytochrome oxidase (at cytochrome a3), thus uncoupling mitochondrial oxidative phosphorylation and inhibiting cellular respiration, even in the presence of adequate oxygen supply. Tissues with the highest oxygen needs (brain and heart) are the most severely affected by acute cyanide poisoning.

Patients with severe cardiovascular toxicity (cardiac arrest, cardiovascular instability, metabolic acidosis, or altered mental status) caused by known or suspected cyanide poisoning should receive cyanide antidote therapy in addition to standard resuscitation, incl. oxygen. Initial therapy should include a cyanide scavenger (either hydroxocobalamin 100 mg kg^{-1} IV or a nitrite – i.e. IV sodium nitrite and/or inhaled amyl nitrite), followed as soon as possible by IV sodium thiosulfate.[404–410] Hydroxocobalamin and nitrites are equally effective but hydroxocobalamin is safer because it does not cause methaemoglobin formation or hypotension.[411–413]

In the case of cardiac arrest caused by cyanide, standard treatment fails to restore spontaneous circulation as long as cellular respiration is blocked. Antidote treatment is needed for reactivation of cytochrome oxidase.

Carbon monoxide. Carbon monoxide poisoning is common. There were about 25,000 carbon monoxide related hospital admissions reported yearly in the United States.[414] Carbon monoxide levels do not correlate with the presence or absence of initial symptoms or with later outcomes.[415] Patients who develop cardiac arrest caused by carbon monoxide rarely survive to hospital discharge, even if ROSC is achieved.[413,416]

Give oxygen as soon as possible. The use of hyperbaric oxygen has been used to treat carbon monoxide exposure in order to reduce the incidence of adverse neurologic outcomes.[417] However, two Cochrane reviews failed to demonstrate convincing benefit from hyperbaric oxygen therapy for patients with carbon monoxide poisoning.[416,418] The role of carbon monoxide in nitric oxide release, reactive oxygen species formation, and its direct action on ion channels may be more significant than its higher affinity for haemoglobin, which is treated by oxygen therapies.[419] There is unproven benefit for transporting critically ill post-arrest patients to a hyperbaric facility and such decision must be considered on a case-by-case basis.[413,416,418,419] Patients who develop myocardial injury caused by carbon monoxide have an increased risk of cardiac and all-cause mortality lasting at least seven years after the event; it is reasonable to recommend cardiology follow-up for these patients.[413,420,421]

B – SPECIAL ENVIRONMENTS

Cardiac arrest in healthcare facilities

Perioperative cardiac arrest

Introduction. Although the safety of routine surgical procedures has increased over recent decades, the greater number of procedures being performed, particularly in more elderly patients and in emergency situations has resulted in a broadly stable incidence of perioperative cardiac arrests over the past decade.

Although the features of perioperative cardiac arrest are often different to those of cardiac arrests occurring in the general hospital population, the principles of treatment are similar. Perioperative cardiac arrest may be caused by the underlying condition being treated, physiological effects of the surgery, anaesthetic drugs and fluids, complications relating to existing co-morbidities, or adverse events.

Epidemiology. The overall incidence of perioperative cardiac arrest ranges from 4.3 to 34.6 per 10,000 procedures.[422–424] This wide range reflects differences in case-mix (some include neonates and/or cardiac surgery) and in the definition of perioperative. The incidence is higher in high-risk groups such as the elderly where it has been reported as 54.4 per 10,000 cases[425] and in patients undergoing emergency surgery where an incidence of 163 per 10,000 cases has been reported.[426] Young age (<2 years old), cardiovascular and respiratory comorbidities, increasing American Society of Anesthesiologists (ASA) physical status classification, preoperative shock, and surgery site have all been identified as risk factors for perioperative cardiac arrest.[426]

The incidence of cardiac arrest attributable primarily to anaesthesia is a relatively small proportion of this overall incidence and in recent studies is estimated to be 1.1–3.26 per 10,000 procedures.[425,427,428] Overall survival from perioperative cardiac arrest is higher than from OHCA, with survival to hospital discharge rates of 30–36.6% being reported recently.[422,424,428]

General versus regional anaesthesia. The incidence of perioperative cardiac arrest during general anaesthesia (GA) is higher than that of regional anaesthesia (RA). The incidence of cardiac arrest for patients receiving general anaesthesia in a study from the Mayo Clinic was higher (almost 3 times higher, at 4.3 per 10,000) than that for those receiving regional anaesthesia or monitored anaesthesia care. The incidence however decreased significantly over a 10-year period.[423]

Causes of cardiac arrest. Overall causes of cardiac arrest have been identified as:

- Hypovolaemia (e.g. bleeding).
- Cardiac-related.
- Other:
  - Drug-induced (e.g. muscle relaxants).
  - Anaesthesia related.
- Airway loss.
- Ventilation failure.
  - Anaphylaxis (drugs, blood products).

The commonest cause of anaesthesia-related cardiac arrest involves airway management.[427,428] Failure of ventilation, medication-related events, complications associated with central venous access, and perioperative myocardial infarction are also common.[423,429] In children, airway obstruction from laryngospasm, hypovolaemia from blood loss and hyperkalemia from transfusion of stored blood are additional causes.[430]

Cardiac arrest caused by bleeding had the highest mortality in non-cardiac surgery, with only 10.3% of these patients surviving to hospital discharge.[423] The primary arrest rhythms during perioperative cardiac arrest recorded in the Mayo Clinic series were asystole in 41.7%, VF in 35.4%, PEA in 14.4% and unknown in 8.5%. Contrary to studies of cardiac arrest in general, the rhythm associated with the best chance of survival to hospital discharge was asystole (43% survival).[423,431]

Management of perioperative cardiac arrest. Patients in the operating room are normally fully monitored and, as such, there should be little or no delay in diagnosing cardiac arrest. High-risk patients will often have invasive blood pressure monitoring, which is invaluable in the event of cardiac arrest. If cardiac arrest is a strong possibility, 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.

In the event of cardiac arrest, follow the ALS algorithm, but with appropriate modifications. Adjust the position and height of the operating table or trolley to optimise delivery of chest compressions. CPR is optimal in the supine position, but is possible in patients who are prone and where immediate turning to a supine position is not possible.[432,433] Risk factors for cardiac arrest in prone patients include cardiac abnormalities in patients undergoing major spinal surgery, hypovolaemia, air embolism, wound irrigation with hydrogen peroxide, occluded venous return.

Identification of causes. In many cases of perioperative cardiac arrest, physiological deterioration is gradual and the cause of the cardiac arrest is known and hence the arrest anticipated. In those where this is not the case, follow the standard ABC algorithm to identify and treat reversible causes. If patients deteriorate, call for senior help immediately. Inform the perioperative team of the deterioration and possible impending cardiac arrest, ensuring that sufficient skilled assistance is present.

C Catastrophic haemorrhage is usually obvious, but may be occult it if involves bleeding into body compartments (abdomen, chest) or into soft tissues in patients with multiple limb fractures. Pelvic and retroperitoneal haemorrhage can also cause rapid hypovolaemia and should be excluded, e.g. by ultrasound if pre-operative haemodynamic instability. In cases where direct surgical intervention is unable to control haemorrhage, early interventional radiography should be considered.

A Loss of the airway is a common cause of perioperative cardiac arrest. Assess the airway carefully before induction of anaesthesia. Prepare all equipment, including suction and an operating table or trolley that can be tipped head-down (Trendelenburg position). Ensure that difficult airway equipment is immediately available, and brief the team on a failed intubation drill if appropriate. Always use waveform capnography. Children are particularly prone to loss of the airway from laryngospasm; ensure an appropriate neuromuscular blocker is available and give before significant hypoxaemia has occurred in order to break the laryngospasm.

B Undiagnosed tension pneumothorax is a readily treatable cause of cardiac arrest. Although usually associated with trauma, consider early in the management of all patients who arrest, particularly those with chronic obstructive pulmonary disease and severe asthma. A sudden increase in airway pressures may indicate a tension pneumothorax or problems with the breathing tubing, but also consider asthma and anaphylaxis.

C Cardiovascular collapse has several causes, but in the context of perioperative cardiac arrest, common causes include hypovolaemia, anaphylaxis, and vagal stimulation. Use of transthoracic echocardiography is a useful tool to exclude cardiac tamponade (if suspected) and to assess myocardial contractility and filling.

Anaphylaxis. The incidence of immune-mediated anaphylaxis during anaesthesia ranges from 1 in 10,000 to 1 in 20,000.[434] 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. Initial management of anaphylaxis follows the ABC approach and the management principles outlined in the chapter on anaphylaxis. Adrenaline is the most effective drug in anaphylaxis and is given as early as possible. It is appropriate for anaesthetists to give adrenaline by the intravenous route. Repeated doses may be necessary.

If cardiac arrest ensues despite correct treatment for the anaphylaxis (see anaphylaxis), continue resuscitation using the standard ALS algorithm (see adult advanced life support).[168]

Systemic toxicity of local anaesthetics. Cardiac arrest is a rare but well recognised 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.[435] Significant hypotension, dysrhythmias, and seizures are typical manifestations, but diagnosis may be one of exclusion.[436]

IV lipid therapy has been used as a rescue therapy to treat cardiovascular collapse and cardiac arrest, but its efficacy is debated.[437] 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).[353,438] Stop injecting the LA and call for help. Secure and maintain the airway and, if necessary, intubate. 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, thiopental 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}.[259,260]

Diagnosis of cardiac arrest. Asystole and ventricular fibrillation (VF) will be detected immediately, but the onset of PEA might not be so obvious – loss of the pulse oximeter signal and very low end-tidal CO_2-values will be good clues and should provoke a pulse check. Do not waste time attempting to obtain a non-invasive blood pressure measurement.

Management of cardiac arrest. The management of a cardiac arrest follows the principles of the ALS algorithm. Chest compression in the prone position can be achieved with or without sternal counter-pressure. In one study of prone CPR with sternal counter pressure (provided by a sandbag) versus standard CPR, higher mean arterial pressures were achieved with the prone technique.[439] Consider open cardiac compressions in patients where the thorax is open or the heart can be easily accessed.

Ventricular fibrillation. In the case of VF, call for a defibrillator. If one is not immediately available, apply a precordial thump. If that is unsuccessful, give chest compressions and ventilation until the defibrillator arrives. Look for reversible causes immediately – hypoxaemia and hypovolaemia will be the most common in this setting.

Asystole/extreme bradycardia. Stop any surgical activity likely to be causing excessive vagal activity – if this is the likely cause – give 0.5 mg atropine IV/IO (not 3 mg). Start CPR and immediately look for other reversible causes. Exclude a completely straight line, which suggests an ECG monitoring lead has become detached.

Pulseless electrical activity. Start CPR while looking quickly for reversible causes of PEA. Give fluid unless you are certain that the intravascular volume is adequate. Stop administration of the anaesthetic. While a vasopressor will be required, in these circumstances 1 mg of adrenaline (as directed by the standard ALS guidelines) may be excessive. Give a smaller dose (e.g. 1 mcg kg^{-1}) of adrenaline, or another vasopressor initially; if this fails to restore the cardiac output, increase the dose while continuing to perform chest compressions and ventilation.

Monitoring and feedback during CPR. Unlike OHCAs where monitoring if often limited, patients arresting in the perioperative period can often be monitored with a greater degree of precision.

Monitoring enables assessment of rescuer performance and patient response:

- Rescuer CPR performance.

Feedback sensors (e.g. accelerometers) improve the delivery of effective chest compressions and enable the rescuer to tailor their performance accordingly. Their use should be considered whenever available. Performance feedback can be obtained from invasive and non-invasive patient monitoring and the rescuer should have direct visualisation of monitors displaying these data.

- Patient response.

Monitoring of the patient requires adequate lighting and patient exposure.Non-invasive blood pressure is unlikely to be of assistance until ROSC is achieved, but in patients with invasive arterial monitoring, aim for a diastolic blood pressure >25 mmHg,[440] titrating it to this level (after chest compressions are optimised) by administration of a vasopressor, if necessary. This goal is based on expert consensus derived from experimental and limited clinical data.[441–443]

Waveform capnography is a minimum monitoring standard during anaesthesia and therefore immediately available during a perioperative cardiac arrest. In addition to its use for patients with tracheal intubation where it is particularly valuable to confirm correct tracheal tube placement, it may also be used in patients with supraglottic airway devices (although an air leak may limit quantitative evaluation). An end-tidal carbon dioxide ($ETCO_2$) value <1.4 kPa/10 mmHg suggests a low cardiac output and rescuers may be able to adjust their technique to optimise this variable. An abrupt sustained increase to a normal value (4.7–5.4 kPa/35–40 mmHg) or even higher may be an indicator of ROSC. Optimise CPR to achieve an $ETCO_2$ >2.7 kPa/20 mmHg, while ventilating the lungs at about 10 breaths min^{-1}, with only minimal chest rise).[440]

Team working. 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 delivering high-quality CPR. Stop operative surgery 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. Prioritise team tasks, ensure good quality basic life support (BLS), identify reversible causes and avoid non-priority tasks.[440] If the patient is not responding to resuscitative efforts (i.e. $ETCO_2$ <2.7 kPa/20 mmHg), try to improve the quality of CPR by optimising: (1) compression fraction, (2) compression rate, (3) compression depth, (4) leaning, and (5) by avoiding of excessive ventilation.[440]

Post-resuscitation care. Depending on the circumstances, patients successfully resuscitated after a very brief period of cardiac arrest, e.g. asystole from excessive vagal stimulation may not require anything more than standard post-operative care. All those resuscitated successfully after longer periods of cardiac arrest will require admission to an ICU – unless further active treatment is deemed inappropriate. In most circumstances, anything but immediately life-saving surgery should be abandoned to enable admission to ICU for post-resuscitation care. Patients resuscitated after a prolonged period of cardiac arrest may develop a marked systemic inflammatory response syndrome (SIRS) with the risk of multiple organ failure. They will require optimisation of mean arterial pressure, oxygenation and ventilation. These patients may have sustained a significant cerebral insult. Some may be suitable for targeted temperature management, but this requires careful consideration, given the lack of data on this therapy in the setting of perioperative cardiac arrest. Active bleeding would certainly be a contraindication to induced mild hypothermia but, at the very least, prevent fever in all cases. Avoidance of hyperthermia, from overwarming or a post-cardiac arrest syndrome[444] is important to optimise neurological recovery.

Do not attempt resuscitation decisions. Patients with DNAR decisions presenting for surgery present a dilemma for the anaesthetist. The anaesthetic will induce cardiovascular instability, many of the routine interventions undertaken could be considered as resuscitative, and the chances of surviving a perioperative cardiac arrest are better than those from in-hospital cardiac arrest in general. Consider each case on its individual merits and discuss with the patient and/or relatives. Some patients may wish a DNAR decision to remain valid despite the increased risk of a cardiac arrest and the

presence of potentially reversible causes; others will request that the DNAR decision is suspended temporarily. Discuss and agree on the time at which the DNAR decision is reinstated.[445]

Cardiac arrest following cardiac surgery

Introduction. Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase, with a reported incidence of 0.7–8%.[446–455] It is usually preceded by physiological deterioration,[456] although it can occur suddenly in stable patients.[452] There are usually specific causes of cardiac arrest, such as tamponade, hypovolaemia, myocardial ischaemia, tension pneumothorax, or pacing failure. These are all potentially reversible and if treated promptly cardiac arrest after cardiac surgery has a relatively high survival rate. Key to the successful resuscitation of cardiac arrest in these patients is recognition of the need to perform emergency resternotomy early, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective.

Starting CPR. If VF or asystole is diagnosed, immediately administer external defibrillation or emergency temporary pacing at maximum amplitude. Otherwise start external chest compressions immediately in patients who arrest with monitoring indicating no output. Verify the effectiveness of compressions by looking at the arterial trace, aiming to achieve a systolic blood pressure >60 mmHg [Society of Thoracic Surgeons (STS) Clinical Practice guidelines in preparation – personal communication from Joel Dunning] and a diastolic blood pressure >25 mmHg[440] at a rate of 100–120 min^{-1}. Inability to obtain this goal with external chest compressions indicates that cardiac tamponade or extreme hypovolaemia is likely and emergency resternotomy should be performed.

Consider other reversible causes:

- Hypoxia – check tracheal tube position, ventilate with 100% oxygen.
- Tension pneumothorax – check tracheal position, listen for air entry.
- Pacing failure – check pacing box output and pacing wire integrity. In asystole, secondary to a loss of cardiac pacing, chest compressions may be delayed momentarily as long as the surgically inserted temporary pacing wires can be connected rapidly and pacing re-established (DDD at 100 min^{-1} at maximum amplitude).

Defibrillation. There is concern that external chest compressions can cause sternal disruption or cardiac damage.[457–460] In the post-cardiac surgery ICU, a witnessed and monitored VF/pVT cardiac arrest should be treated immediately with up to three quick successive (stacked) defibrillation attempts. Three failed shocks in the post-cardiac surgery setting should trigger the need for emergency resternotomy. Further defibrillation is attempted as indicated in the universal algorithm and should be performed with internal paddles at 20 J if resternotomy has been performed.[461,462]

Emergency drugs. Use adrenaline very cautiously and titrate to effect (IV doses of up to 100 mcg in adults). Consider amiodarone 300 mg in patients with refractory shockable rhythms (VF/pVT), but do not delay resternotomy. Atropine is not recommended for asystole and temporary or external pacing should be employed.

Emergency resternotomy. This is an integral part of resuscitation after cardiac surgery, once all other reversible causes have been excluded. Once adequate airway and ventilation has been established, and if three attempts at defibrillation have failed in VF/pVT, undertake resternotomy without delay. Emergency resternotomy is also indicated in asystole or PEA, when other treatments have failed, and should be performed within 5 min of the cardiac arrest by anyone with appropriate training.

These guidelines are also appropriate for patients following non-sternotomy cardiac surgery, but surgeons performing these operations should have already made clear their instructions for chest reopening in an arrest.

Special considerations regarding treatment of patients with ventricular assist devices (VADs) are addressed in the section on special patients (see patients with ventricular assist devices).

Cardiac arrest in a cardiac catheterisation laboratory

Cardiac arrest may occur during percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI) or non-STEMI, but it may also be a complication of an angiography such as catheter wedging, air or thrombus embolism in the coronary artery, coronary artery intima dissection from the tip of the angiography catheter or caused by pericardial tamponade from a perforated coronary artery during the procedure. Most complications will result in VF with immediate need for defibrillation. For this reason, patients must be continuously monitored and a defibrillator must be available in the angiography room. Self-adhesive radiolucent defibrillation pads may already be placed at the beginning of the procedure in high-risk patients.

In this special setting with immediate response to monitored VF, defibrillation without preceding chest compressions is recommended. As the patient is early in the electrical phase of a cardiac arrest, in contrast to the guidelines for unmonitored and OHCAs, the result of defibrillation (VF termination and ROSC) can be determined before chest compressions are started. If needed for failed defibrillation or immediately recurring VF, immediate defibrillation may be repeated up to two times.

If VF persists after the initial three shocks or ROSC not immediately established with certainty, chest compressions and ventilations must be initiated without further delay and a cause for the unresolved problem sought with further coronary angiography. It is of extreme importance that chest compressions are not interrupted for angiography. On an angiography table with the image intensifier above the patient, delivering chest compressions with adequate depth and rate is almost impossible and exposes the rescuers to dangerous radiation. Therefore, early transition to the use of a mechanical chest compression device is strongly recommended.[247,463] If the problem is not rapidly resolved, very low quality evidence suggests that the use of extracorporeal life support (ECLS) can be considered as a rescue strategy if the infrastructure is available, and probably to be preferred over intra-aortic balloon pump (IABP).[464] There is no evidence to recommend circulatory support with the Impella pump only during cardiac arrest.

If the cardiac arrest is caused by a non-shockable rhythm, immediate transthoracic echocardiography should identify pericardial tamponade or other conditions.

Cardiac arrest in a dialysis unit

Introduction. Sudden cardiac death is the most common cause of death in haemodialysis patients and is usually preceded by ventricular arrhythmias.[465] Hyperkalemia contributes to 2–5% of deaths amongst haemodialysis patients[466] and accounts for up to 24% of emergency haemodialysis session in haemodialysis patients.[467] The frequency of cardiac arrest is highest on the first session of haemodialysis of the week (i.e. Monday or Tuesday) as fluid and electrolyte disturbances peak after the weekend interval.[468] Primary prevention of cardiac arrest in dialysis patients include the avoidance of low potassium dialysate solutions and proper use of medication, e.g. beta blockers or angiotensin-converting enzyme inhibitors.[465] There is little evidence to guide the treatment of cardiac arrest during haemodialysis, although some special considerations have been suggested.[469]

Initial steps.

- Call the resuscitation team and seek expert help immediately.
- Follow the universal ALS algorithm.
- Assign a trained dialysis nurse to operate the dialysis machine.
- Stop ultrafiltration (i.e. fluid removal) and give a fluid bolus.
- Return the patient's blood volume and disconnect from the dialysis machine.
- Leave dialysis access open and use for drug administration.
- Beware of wet surfaces (i.e. dialysis machines may leak).
- Minimise delay in delivering defibrillation.

Modifications to cardiopulmonary resuscitation.

Defibrillation. A shockable rhythm (VF/pVT) is more common in patients undergoing haemodialysis[465,470,471] than in the general population.[472,473] The safest method to deliver a shock during dialysis requires further study. Most haemodialysis machine manufacturers recommend disconnection from the dialysis equipment prior to defibrillation.[474] Ensure familiarity with local dialysis equipment and check if equipment has defibrillator-proof label in accordance with the International Electrotechnical Committee (IEC) standards. Automated external defibrillators in nurse-led dialysis centres can facilitate early defibrillation by first responders with appropriate training.[475]

Vascular access. Use dialysis access in life-threatening situations and cardiac arrest.[469]

Potentially reversible causes. All of the standard reversible causes (4 Hs and 4 Ts) apply to dialysis patients. Electrolyte disorders, particularly hyperkalaemia (see hypo-/hyperkalaemia and other electrolyte disorders), and fluid overload (e.g. pulmonary oedema) are most common causes.

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.

Cardiac arrest in the dental surgery

Introduction. Dental surgery emergencies include a variety of situations ranging from psychosomatic disorders precipitated by fear and anxiety to life-threatening situations requiring immediate life-saving procedures. Cardiac arrest in primary dental practice is rare with an incidence of 0.002–0.011 cases reported per dentist per year.[476–478]

The most frequent medical emergencies include vasovagal (pre-) syncope, orthostatic hypotension, hypertensive crisis, hyperventilation, seizures, moderate allergic reactions, hypoglycaemia, and angina.[476,479] The majority of dentists responded that they would be capable of performing initial treatment of common emergencies, while many felt unable to treat anaphylaxis, myocardial infarction, or cardiac arrest.[476,477]

A cardiac arrest occurring in a dental surgery is an event witnessed by medical professionals who have a duty of care and are required to be competent in the delivery of CPR.

Causes of cardiac arrest. Causes of cardiac arrest usually relate to pre-existing comorbidities or complications of the procedure. The life-threatening emergencies commonly arise from myocardial infarction, grand mal seizures or exacerbation of asthma. Dental procedures may cause loss of airway patency related to the primary pathology or complications of the procedure (e.g. bleeding, secretions, tissue swelling). Choking is rare, with a reported incidence of 0.07–0.09 cases per dentist per year.[476,477] 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.[480,481]

Although life-threatening anaphylaxis is rare, it is a documented cause of death during dental procedures. In addition to chlorhexidine mouthwash, other common causes may include penicillin and latex. Anaphylaxis to local anaesthetics is very rare and a reaction to this class of drug is usually due to a direct intravascular injection of adrenaline contained in the solution. True anaphylaxis (all causes) occurs in only 0.004–0.013 cases per dentist per year, compared with coronary symptoms (angina or myocardial infarction) occurring in 0.15–0.18 cases per year.[476,477]

Treatment of cardiac arrest. The following modifications to the initial sequence of actions are recommended if cardiac arrest occurs in a dental chair:

- In case of sudden loss of consciousness, immediately call for help.
- Look into the victim's mouth. Check and remove all solid materials from the oral cavity (e.g. retractor, suction tube, tampons, etc.). Prevention of airway obstruction should preceed positioning the patient on his back.
- Recline the dental chair into a fully horizontal position. Cardiac output can be restored if reduced venous return or vasodilation has caused loss of consciousness, e.g. vasovagal syncope, orthostatic hypotension. In these patients, raising the legs and/or placing the patient in a head-down position may also help.
- Simultaneously open the airway and check breathing (look, listen, feel). If breathing is not normal or absent, assume a cardiac arrest until proven otherwise. Send someone to get an AED if available.
- Some case reports describe successful CPR in a patient left on a dental chair.[482,483] Small simulation studies comparing the effectiveness of CPR on a dental chair and on the floor reported either lower or equivalent CPR quality.[484–487] However, the patient should not be moved from the dental chair because of the risk of injury to the patient and rescuers and the limited space that is likely to be available on the floor next to the patient.[482,483] Ensure that the dental chair is fully reclined into the horizontal position, support its head with a stool to increase stability, and start chest compressions immediately.[482,484]
- If feedback devices are used to monitor CPR quality, those using accelerometers may overestimate depth of compressions if used on a dental chair.[488]
- Follow the standard compression:ventilation ratios for adults and children. Consider the over-the-head technique of CPR if access to either side of the chest is limited.[489–492]
- Maintain the airway and ventilate the patient with a bag-valve-mask device, using the two-hand technique if necessary. Supraglottic airways may be inserted if the operator is skilled in their use, but tracheal intubation is not a recommended intervention required of dental practitioners and should be avoided.
- Switch on the AED and follow the instructions. Deliver the first shock as soon as possible if indicated.
- Continue with CPR until signs of life return, or the patient's handover to the professional resuscitation team (see adult basic life support and automated external defibrillation).[493]

Equipment and training. Follow national guidelines for recommended equipment to treat medical emergencies in a dental practice.[478] Basic resuscitation equipment should be available immediately in all primary care dental premises, including suction, self-inflating bag with face masks, oxygen, and emergency drug kit.[494,495] The role of early defibrillation should be emphasised to increase the availability of AEDs in dental surgeries,[482,496] which is still unsatisfactory, ranging from a reported 0.5–2.6% in Europe[497,498] to 11% in the United States.[499] We recommend that all dental practices delivering clinical care have immediate access to an AED, with all staff trained in its use. Advanced equipment

and special training is needed if analgesia or sedation is used in dental surgeries.[478,500] In patients with pacemakers, ECG monitoring and immediate availability of a defibrillator is recommended if electrical devices are used (e.g. diathermy, electric pulp tester, etc.).[482]

There is rightly 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 experience in treating a patient in cardiac arrest,[476,479,501] and their training in CPR varies significantly between countries.[476,477,501–503] Maintaining knowledge and competence to deal with medical emergencies must be an important part of the training of dentists. All dental care professionals should undergo annual practical training in the recognition and management of medical emergencies, and the delivery of CPR, including basic airway management and the use of an AED.[478]

Cardiac arrest in transportation vehicles

In-flight emergencies aboard airplanes

Introduction. Worldwide, 3.2 billion passengers fly on commercial airlines annually. The incidence of in-flight medical emergencies has been reported to be one event per 10,000–40,000 passengers.[504,505] The probability of at least one medical incident reaches 95% after 24 intercontinental flights.[505] Most of the cases involve middle-aged people.[506] Two large studies recently reviewed more than 22,000 in-flight emergencies from five American and two European airlines. The most common medical problems were syncope or presyncope (37.4–53.5%), respiratory symptoms (12.1%), gastrointestinal problems (8.9–9.5%), and cardiac conditions (4.9–7.7%) with some variations across airlines.[504,507] Surgical problems (e.g. deep venous thrombosis, appendicitis, gastrointestinal bleeding) were seen rarely (<0.5%).[504] In-flight incapacitation of the flight crew is very rare, the most common cause being acute myocardial ischaemia.[508]

The in-flight medical emergencies have very limited access to medical care, but the majority can be managed conservatively with fluids, oxygen and other treatment available from first aid kits on board. However, a quarter of these patients subsequently require additional evaluation in a hospital.[507] Immediate diversion of an aircraft is requested in 2.4–7.3% of all incidents, most commonly due to chest pain, suspected stroke, and seizures.[504,507,509,510]

Cardiac arrest on board has an incidence of 1 per 5–10 million passenger flights. An initial shockable rhythm is present in 25–31% patients,[505,511–513] and the in-flight use of an AED can result in 33–50% survival to hospital discharge.[511,513,514] Factors contributing to a high survival rate include a witnessed event, cabin crew trained in BLS and in 73–86% of cases, travelling medical professionals also providing immediate assistance.[504,507,509] However, approximately 1000 lives are lost per year in International Airlines Transport Association (IATA) carriers. Some studies have shown that 41–59% of cardiac arrests on board are unwitnessed, occurring during sleep. There were no survivors if the initial rhythm was asystole or an idioventricular rhythm.[511,513]

Cardiopulmonary resuscitation on the airplane. In case of cardiac arrest, follow the universal algorithm for BLS (see adult basic life support and automated external defibrillation).[493] Immediately request an AED and a first aid kit from cabin crew. Physicians and trained medical providers, e.g. nurses or EMS personnel, should also ask for advanced medical equipment. According to competencies and equipment available, provide the patient with advanced treatment, assuring that there is high quality CPR ongoing, and an AED was deployed appropriately (see adult advanced life support).[168]

Consider the following modifications to CPR:

- Introduce yourself to the cabin crew and state your professional qualifications.
- In case of cardiac arrest, performance of CPR is limited in an aircraft aisle due to space restrictions. Immediately transfer the patient to a suitable location, e.g. galley or exit area. Consider an over-the-head technique of CPR if access precludes conventional CPR.[489–492]
- During CPR, attach oxygen to the facemask or self-inflating bag.
- Request immediate flight diversion to the nearest appropriate airport. In other non-critical medical emergencies, coordinate an optimal course of action with the flight crew. Considerations for flight diversion will depend on the patient's condition and on the need for immediate treatment in a hospital: e.g. acute coronary syndrome, stroke, persistently altered mental status; but also technical and operational factors.
- Ask cabin crew whether medical consultation is provided by the airline, e.g. radiotelephony or satellite communication.[506,510]
- An AED with a monitor can be safely attached to a non-arrested patient for monitoring heart rhythm, e.g. syncope, chest pain, or arrhythmia.[507,512,513]
- Concerns about legal responsibility may arise when travelling physicians are asked for help. Based on ethical duties, every physician is required to offer help within his or her scope of practice, but the legal duty is only applicable for certain countries. However, the so-called Good Samaritan Act and other regulations, depending on the origin of an aircraft, always protect healthcare providers helping on board from possible legal consequences.[504,515]
- Death on board can legally be confirmed only by a physician. If a dead person is found, or CPR has been terminated (see ethics of resuscitation and end-of-life decisions),[243] flight diversion is not recommended.

Education and equipment.

Flight crew training. Both pilots and cabin crew must receive initial and recurrent training on emergency medical event procedures and operation of emergency medical equipment, including AEDs and first aid kits, but local operational procedures may also apply.[516]

Although civil aviation is regulated by a variety of national and international laws, some studies imply that the majority of in-flight emergencies stay unreported or are reported inconsistently.[504,517] Documentation of in-flight emergencies needs standardisation in order to improve cabin crew training and pre-flight assessments of selected groups of passengers.

On-board emergency equipment. The Federal Aviation Administration (FAA) requires every US registered commercial aircraft with a maximum payload capacity of more than 7500 pounds and with at least one flight attendant to carry an AED, intravenous drugs, and advanced emergency equipment,[518] while related regulations in Europe are less precise.[519] On every commercial aircraft registered in Europe, there must be a first-aid kit that all cabin crewmembers are trained to use. Aircraft with at least 30 seats must also carry an advanced medical kit, which can be used by competent personnel, although the contents vary significantly and may be inadequate for all but the most basic of emergencies.[504,517,520] Although most large European airlines carry AEDs, some of them only do so for intercontinental flights, but some do not even provide any equipment for CPR.[517]

Based on the outcome data from survivors of cardiac arrest and in the absence of any alternative treatment for shockable rhythms on board, we strongly recommend mandatory AEDs in all commercial European aircraft, including regional and low-cost carriers.

Healthcare professionals should be aware of the on-board medical equipment and commonly encountered medical

conditions in order to provide appropriate emergency treatment on request.[505] Distribution of supportive information to travelling physicians should be encouraged, e.g. 'Doctor on Board' programme introduced by Lufthansa and Austrian Airlines in 2006.

Cardiac arrest in HEMS and air ambulances

Introduction. Air ambulance services operate either a helicopter emergency medical service (HEMS) or fixed-wing air ambulances that routinely transport critically ill patients directly to specialty centres and perform secondary transfers between hospitals. Cardiac arrest may occur in flight, both in patients being transported from an accident site and also critically ill patients being transported between hospital.[521,522] In a retrospective analysis of 12,140 aeromedical journies, the incidence of cardiac arrest in flight was low (1.1%). Fourty-three percent were medical patients and 57% were patients with traumatic injuries. In the medical cohort, the rate of ROSC was 75%.[523]

The extent of treatment available on board of an air ambulance varies and depends on medical and technical factors, e.g. crew competences and configuration, cabin size and equipment. Ideally, all interventions should be performed before flight so that the need for unplanned treatment during flight is avoided.

Pre-flight preparation. When preparing transport of a critically ill patient, ensure that all necessary monitoring is attached and functioning. Check that IV access is secured and easily accessible and that all necessary drugs and medical equipment are available during flight.

Diagnosis. In monitored patients, asystole and shockable rhythms (VF/pVT) can be immediately identified, but recognition of PEA may be challenging, especially under sedation or general anaesthesia. Unexpected loss of consciousness (in alert patients), change of ECG pattern, and loss of the pulse oximeter signal should provoke a 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 also indicators of cardiac arrest.

Treatment. Cardiac arrest in the air ambulance services should be treated according to the universal ALS algorithm. Start chest compressions and ventilation immediately after confirmation of cardiac arrest, attach monitoring (if not already), and follow universal ALS algorithm.[168] If a shockable rhythm (VF/pVT) is recognised in a monitored patient and defibrillation can be accomplished rapidly, immediately give up to three-stacked shocks before starting chest compressions. In a US study, 33% of patients achieving ROSC following defibrillation did not require any chest compressions.[523]

In smaller helicopters, there may be insufficient room to perform effective resuscitation and an emergency landing may be necessary to allow better patient access.

Mechanical chest compression devices enable delivery of high quality chest compressions in the confined space of an air ambulance and their use should be considered.[248,524] If a cardiac arrest during flight is thought to be a possibility, consider fitting the patient within a mechanical chest compression device during packaging before flight.[50,525]

Cardiac arrest during sports activities

Resuscitation on the field of play

Introduction. The sudden and unexpected collapse, not associated with contact or trauma, of an athlete on the field of play is probably cardiac in origin and requires rapid recognition and effective treatment if the victim is to survive. Sudden cardiac death (SCD) is the most common cause of death of athletes during competition and training. Estimates of the incidence of SCD vary according to the methodology but recently the incidence has been quoted as 1:11,394 in basketball players, 1:21,293 for swimmers and 1:41,695 for cross-county athletes with a wide variation between male and female athletes (incidence expressed as number of athletes per year).[526] Hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC) are the most common causes in under 35 year olds whilst atherosclerotic coronary artery disease accounts for 80% of sudden cardiac arrests in over 35 year olds.[527] Congenital coronary artery abnormalities have been reported in 12–33% of athletes.[528]

Commotio cordis, the disruption of cardiac rhythm by a blow to the precordium, has a quoted incidence of 3%.[529] The striking object must strike the chest within the cardiac silhouette within a 20 ms window of the upstroke of the T-wave.[530] The overall survival rate from commotio cordis is reported to have improved with survival rates of up to 58% reported in recent years.[531]

Whatever the cause the sudden collapse of an athlete there should be an immediate response from the officials or medical team. The standard resuscitation procedures must be followed but with certain additional considerations as described below.

Access. The medical team should gain immediate access to the field of play. It is important that the medical team do observe access rules to the field of play but it would be hoped that the field of play officials will recognise or be alerted to the collapsed athlete and halt play so that it is safe to approach the competitor.

Where there is no medical team, during informal competition or in practice it is the responsibility of the referee, the coach or of the athletes' colleagues to recognise the collapse and to initiate a call for help and resuscitation.

Calling for help. The call for help is essential to providing the collapsed athlete with the best chance of survival. It is essential that sports officials, coaches and sports organisers have a plan for medical collapse or trauma. In its simplest form this could include ensuring the availability of a mobile telephone and knowledge of the site/address of the sport arena (field of play, club house) to provide best access for the ambulance. It would be hoped that more officials and coaches will be trained in BLS and AED usage.

Resuscitation. If the athlete is unresponsive and not breathing normally, commence BLS. If available attach an AED and follow the instructions; if this is SCD then the rhythm will probably be ventricular fibrillation and will respond to defibrillation.

The sports field of play is often an open arena and in major competition may be on view to many thousands of spectators and a television audience. Although treatment must not be compromised moving the collapsed athlete to a quieter and more private site for continued treatment may be considered. Where there is not an immediate response to treatment and there is an organised medical team, this move could be accomplished after three defibrillation attempts on the rationale of providing the highest efficacy of defibrillation in the first three shocks. The move, if decided, should be agreed and may need to be accomplished in stages to allow for near continuous chest compressions. Where there is no medical team or a defibrillator is not immediately available then BLS must continue until advanced care arrives.

If the athlete responds to resuscitation then they must be transported immediately to the nearest cardiac centre for further evaluation and treatment. As there is a possibility of the rhythm reverting this transportation must be under the supervision of a healthcare professional who is equipped and capable of administering resuscitation and further defibrillation.

Prevention. In an effort to predict and prevent SCD, the International Olympic Committee Medical Commission (IOC Medical Commission 2014) and many International Sport Federations have recommended cardiac screening for athletes. However, there is much debate about the effectiveness of the techniques being used and the population that should be screened.[532]

Water rescue and drowning

Introduction

Drowning is a common cause of accidental death.[533] Prompt and effective actions by bystanders, trained rescuers and emergency medical personnel can make the difference between life and death.[534–536] These guidelines provide advice about the initial rescue and resuscitation of victims involved in drowning incidents. They are intended for healthcare professionals and certain groups of lay responders that have a special responsibility in the care of the drowning victim, e.g. lifeguards, lifeboat crews, swimming pool instructors and water rescue teams.

Epidemiology

The World Health Organization (WHO) reports that every hour of every day, more than 40 people lose their lives to drowning; 372,000 deaths each year.[537] The WHO acknowledges that the true number of drownings worldwide is much higher. More than 90% of these deaths occur in low and middle-income countries. The incidence of drowning varies between countries, with eastern Europe having the highest rates in Europe.[533] Although risk groups vary between countries, in general males are much more likely to drown than females. Most accidental drownings are children who are unable to swim. In countries where aquatic leisure in combination with alcohol and drug use is common, young adults are a second group of risk.[538,539] Many countries also report a slight increase of drowning in the age groups above 70 years related to accidents and physical activities close to water. Drowning is commonest in inland waters (e.g. lakes, rivers) and during summer months.[538–540]

Definitions, classifications and reporting

The International Liaison Committee on Resuscitation (ILCOR) defines drowning as a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium. Implicit in this definition is that a liquid/air interface is present at the entrance of the victim's airway, preventing the victim from breathing air. The victim may live or die after this process, but whatever the outcome, he or she has been involved in a drowning incident.[541] Submersion occurs when the face is underwater or covered in water. Asphyxia and cardiac arrest occurs within a matter of minutes of submersion. Immersion, by contrast, is when the head remains above water, in most cases by means of the support of a lifejacket. In most situations of immersion, the victim remains immersed with an open airway and becomes hypothermic, although aspiration of water may occur if water splashes over the face or if the victim becomes unconscious with their face in the water. The difference between submersion and immersion is important in understanding the difference in epidemiology, pathophysiology, clinical course and prognostic parameters between the two drowning processes.

If the casualty is rescued, the process of drowning is interrupted, which is termed a non-fatal drowning. If the person dies at any time as a result of drowning, the term is fatal drowning. Avoid terms such as dry and wet drowning, active and passive drowning, silent drowning, secondary drowning and near-drowning.[541] To improve consistency in information between studies use the Utstein-style registration template for drowning when reporting outcomes from drowning incidents.[542]

Pathophysiology

Detailed summaries of the pathophysiology of drowning have been published.[536,541,543,544] In brief, following submersion, the victim initially breath holds by reflex. During this time the victim frequently swallows water. As breath holding continues, hypoxia and hypercapnia develop. A reflex laryngospasm may temporarily prevent the entrance of water into the lungs. Eventually these reflexes abate and the victim aspirates water. The key feature to note in the pathophysiology of drowning is that bradycardia as a consequence of hypoxia occurs before sustaining a cardiac arrest. Correction of hypoxaemia by ventilation-only resuscitation is critical and in itself may lead to return of spontaneous ventilation or circulation (ROSC) in some cases, probably because the presence of a circulation had not been detected.[545–549]

Drowning chain of survival

The Drowning Chain of Survival describes five critical links for improving survival from drowning (Fig. 4.5).[535] The first two links cover prevention of drowning and recognition of distress.[550,551] This chapter provides guidance on removal from water, initial and post resuscitation care.

Water rescue

Bystander response. Bystanders play a critical role in initial attempts at rescue and resuscitation.[534,548,552–555] At the same time, bystanders who attempt a rescue have died during the rescue attempt, mostly when drowning occurs in surf or fast moving water.[556] Whenever possible, bystanders should attempt to save the drowning victim without entry into the water. Talking to the victim, reaching with a rescue aid (e.g. stick or clothing), or throwing a rope or buoyant rescue aid may be effective if the victim is close to dry land. If entry into the water is essential, take a buoyant rescue aid, flotation device or boat.[535] It is safer to enter the water with two rescuers than alone. Never dive head first in the water when attempting a rescue. You may lose visual contact with the victim and run the risk of a spinal injury.

Trained rescuer response. Trained rescuers are often professionals who work in teams with specialist equipment to assist with search and rescue. Where the rescue takes time, the teams often seek guidance on the likelihood of survival. For this reason, ILCOR reviewed specific prognostic indicators and noted that submersion durations of less than 10 min were associated with a very high chance of favourable outcome; submersion durations longer than 25 min were associated with a low chance of favourable outcomes.[557] Age, emergency medical services (EMS) response time, fresh or salt water, water temperature, and witness status were not useful for predicting survival. Submersion in ice-cold water may prolong the window of survival and justify extended search and rescue activities.[558–560]

In-water resuscitation. Trained individuals may undertake in water ventilation ideally with the support of a buoyant rescue aid.[545,561,562] If a rescuer, in general a surf-lifeguard, finds a non-responding drowning victim in deep open water, the rescuer may start ventilation when trained to do so before moving the victim to dry land or rescue craft. Some victims may respond to this. If not responding, and depending on the local situation, such as sea conditions, distance to shore, availability of rescue boat or rescue helicopter, the rescuer should then decide to bring the victim to shore as quickly as possible without further ventilation while rescue-swimming with the victim or continue on the spot with in-water ventilation until support by crews of a rescue boat or rescue helicopter arrives to take over the resuscitation. One study suggests that the second option has a higher survival rate.[545]

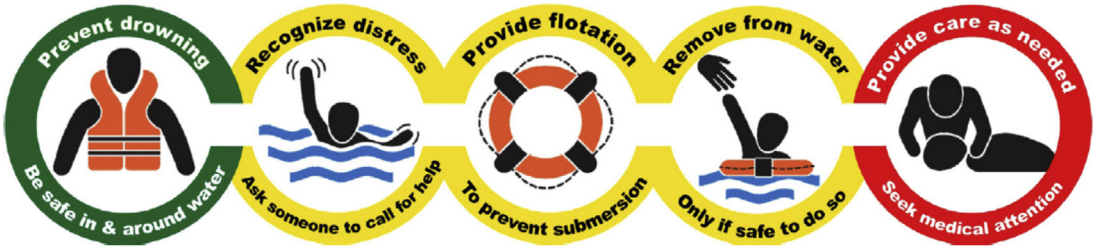

Fig. 4.5. Drowning chain of survival.[535]
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Removal from water. Remove the victim from the water promptly. The chances of a drowning victim sustaining a spinal injury are very low.[563] Spinal precautions are unnecessary unless there is a history of diving in shallow water, or signs of severe injury after water-slide use, waterskiing, kite-surfing, or watercraft racing. If the victim is pulseless and apnoeic, remove them from the water as quickly as possible while attempting to limit neck flexion and extension. Hypovolaemia after prolonged immersion may cause a circum-rescue collapse/arrest. Keep the victim in a horizontal position during and after retrieval from the water.

Initial resuscitation once retrieved from water

Follow the standard BLS sequence, initially by checking for response, opening the airway and checking for signs of life. The drowning victim rescued from the water within a few minutes of submersion is likely to exhibit abnormal (agonal) breathing. Do not confuse this with normal breathing.

Rescue breaths/ventilations. The BLS sequence in drowning (Fig. 4.6) reflects the critical importance of rapid alleviation of hypoxia. Inflation should take about 1 s and be sufficient to see the chest rise. However it often takes more time to insuflate air than under normal conditions due to reduced compliance and high airway resistance. The higher inflation pressure may precipitate inflation of the stomach with regurgitation and also reduce cardiac output. Expert opinion suggests that cricoid pressure applied by trained and skilled personnel in casualties without a secured airway may reduce gastric inflation and enhance ventilation in drowning.

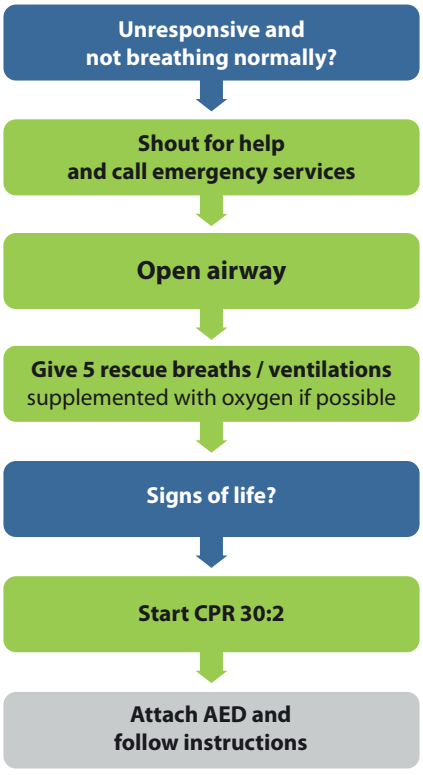


Fig. 4.6. Drowning treatment algorithm for rescuers with a duty to respond.

Chest compressions. If the victim has not responded to initial ventilations, they should be placed on a firm surface before starting chest compressions, as compressions are ineffective in the water.[564,565] Provide CPR in a ratio of 30 compressions to 2 ventilations. Most drowning victims will have sustained cardiac arrest secondary to hypoxia. In these patients, compression-only CPR is likely to be ineffective and should be avoided.

If sufficient rescuers are present, the person performing the aquatic rescue should be relieved of continuing CPR once on land as they are likely to be fatigued, which may impair the quality of CPR.[566,567]

Automated external defibrillation. Defer using an AED until after CPR has commenced. Dry the victim's chest, attach the AED pads and turn the AED on. Deliver shocks according to the AED prompts.

Fluid in the airway. In some situations, massive amounts of foam caused by admixing moving air with water are seen coming out of the mouth of the victim. Do not try and attempt to remove the foam as it will keep coming. Continue rescue breaths/ventilation until an ALS provider arrives and is able to intubate the victim. Regurgitation of stomach contents and swallowed water is common during resuscitation from drowning.[568] If this prevents ventilation completely, turn the victim on their side and remove the regurgitated material using directed suction if possible.

Modifications to advanced life support

Airway and breathing. During the initial assessment of the spontaneously breathing drowning victim, give high-flow oxygen (10–15 L min^{-1}), ideally through an oxygen mask with reservoir bag.[127] For victims who fail to respond to these initial measures, who have a reduced level of consciousness or are in cardiac arrest, consider early tracheal intubation and controlled ventilation by skilled personnel. Reduced pulmonary compliance requiring high inflation pressures may limit the use of a supraglottic airway device.[569] Take care to ensure optimal preoxygenation before attempting tracheal intubation. Pulmonary oedema fluid may pour from the airway and may need continuous suctioning to enable a view of the larynx. After the position of the tracheal tube is confirmed, titrate the inspired oxygen concentration to achieve a SpO_2 of 94–98%.[127] Pulse oximetry can give spurious readings following rescue from drowning.[570] Confirm adequate oxygenation and ventilation with arterial blood gases once available. Set positive end expiratory pressure (PEEP) to at least 5–10 cm H_2O. However, PEEP

levels of 15–20 cm H_2O may be required if the patient is severely hypoxaemic.[571] Decompress the stomach with a gastric tube.

Circulation and defibrillation. Palpation of the pulse as the sole indicator of the presence or absence of cardiac arrest is not always reliable. As soon as possible, use information from monitoring modalities such as the ECG trace, $ETCO_2$ and echocardiography to confirm the diagnosis of cardiac arrest.

If the victim is in cardiac arrest, follow standard ALS protocols. If the victim is hypothermic, modify the approach in accordance with the guidance for treatment of hypothermia (see hypo-/hyperthermia).

After prolonged immersion, most victims will have become hypovolaemic due to the cessation of the hydrostatic pressure of water on the body. Give rapid IV fluid to correct hypovolaemia. This should commence out-of-hospital if transfer time is prolonged.

Discontinuing resuscitation efforts

Making a decision to discontinue resuscitation efforts on a victim of drowning is notoriously difficult. No single factor can accurately predict good or poor survival with certainty. Frequently, decisions made in the field later prove to have been incorrect.[572] Continue resuscitation unless there is clear evidence that such attempts are futile (e.g. massive traumatic injuries, rigour mortis, putrefaction, etc.), or timely evacuation to a medical facility is not possible. Neurologically intact survival has been reported in several victims submerged for longer than 25 min, however these rare case reports almost invariably occur in children submerged in ice-cold water, when immersion hypothermia has preceeded hypoxia or in submersion of car occupants.[558,559,573,574] A retrospective study of 160 children who drowned in the Netherlands found that outcomes were extremely poor if ALS took longer than 30 min to achieve ROSC even if hypothermia was present.[560]

Post resuscitation care

Salt versus fresh water. Small differences in electrolyte disturbance are rarely of any clinical relevance and do not usually require treatment.[575,576]

Lung injury. The predominant pathophysiological process in the lungs is driven by surfactant wash-out and dysfunction, alveolar collapse, atelectasis, and intrapulmonary shunting. The severity of lung injury varies from a mild self-limiting illness to refractory hypoxaemia. Many victims of drowning are at risk of developing acute respiratory distress syndrome (ARDS).[577] Although there are no randomised controlled trials undertaken specifically in this population of patients, it seems reasonable to include strategies such as protective ventilation that have been shown to improve survival in patients with ARDS.[578,579] Extracorporeal membrane oxygenation (ECMO) has been used for those in refractory cardiac arrest, those with refactory hypoxaemia and in selected cases of submersion in ice cold water, although success rates remain low.[580–583]Pneumonia is common after drowning. Prophylactic antibiotics have not been shown to be of benefit[584] but they may be considered after submersion in grossly contaminated water such as sewage. Give broad-spectrum antibiotics if signs of infection develop subsequently.[585–587]

Neurological outcome. Neurological outcome, notably severe permanent neurological damage, is primarily determined by the duration of hypoxia. Attempts have been made to improve neurological outcome following drowning with the use of barbiturates, intracranial pressure (ICP) monitoring, and steroids. None of these interventions has altered outcome.[588]

Wilderness and environmental emergencies

Difficult terrain and remote areas

Geographical and meteorological considerations. Compared to urban areas some terrains will be more difficult to access and are remote from organised medical care. Exposed and steep terrain may render extrication dangerous and challenging. The chances of a good outcome from cardiac arrest may be reduced because of delayed access and prolonged transport. Furthermore, some environments are harsher than urban areas (e.g. cold, windy, wet, very bright due to light-reflection on ice and snow). Human and material resources may be greatly restricted.[589,590]

Compared with the partial pressure of oxygen at sea level (PO_2 about 21 kPa/159 mmHg), the PO_2 at high (>1500 m above sea level), very high (3500–5500 m) and extreme altitude (>5500 m) will be progressively lower, constraining the physical activity of rescuers. There is a physiological limit to acclimatisation (e.g. short term–hyperventilation and increased cardiac output; long-term–haemoglobin increase). The highest permanent settlement is at 5100 m (PO_2 about 11 kPa/84 mmHg). Above 7500 m the risk of lethal acute altitude illness is very high.

There are no epidemiological data on the causes of cardiac arrest at high altitude. However, it is conceivable that primary cardiac arrest is the major (60–70%) cause of sudden cardiac arrest. Thus, public access defibrillator (PAD) programmes in populated areas at altitude seem reasonable. For instance, public access defibrillators (PADs) should be placed in popular ski areas, busy mountain huts and restaurants, at mass-participation events, and in remote but often-visited locations that are not medically covered.[591] In areas where physicians are regularly involved in mountain rescue operations, the provided on-site treatment is more in line with resuscitation guidelines.[592]

Decision making. Continuous monitoring and treatment may be difficult during transport because the patient will be insulated from the harsh environment within a rescue bag, being well wrapped and secured on a stretcher. During transport, CPR may be limited in quality and nearly impossible in some circumstances (e.g. while carrying the patient, during abseiling or winching). In dangerous and difficult terrain where continuous CPR is impossible, delayed and intermittent CPR has been proposed for hypothermic patients.[45] Mechanical resuscitation devices may help to improve CPR quality during difficult extrication and prolonged transport.[50]

Transportation

Effective and safe immobilisation and splinting will reduce morbidity and mortality.[593] Whenever possible, transport the patient with air rescue.[593,594] The organisation of the helicopter emergency medical service (HEMS) affects the outcome.[595–597]

High altitude illness

Given the increasing popularity of travel at altitude, an increasing number of tourists at altitude have cardiovascular and metabolic risk factors for cardiac arrest. The pO_2 falls with increasing altitude and this oxygen deficiency may lead to acute manifestations of mountain sickness.

Persons travelling to an altitude of >3500 m are at risk of developing:

- acute mountain sickness (AMS) with headache, nausea, fatigue and dizziness;
- high altitude pulmonary oedema (HAPO) with severe dyspneoa and cyanosis;
- high altitude cerebral oedema (HACO) with gate disorder, disorientation and confusion.

Risk factors include a fast rate of ascent and a previous history of mountain sickness. If not treated promptly, HAPO and HACO may progress rapidly to loss of consciousness, severe respiratory distress, circulatory instability and cardiac arrest. The most important actions are immediate descent or transport to lower levels of altitude, administration of oxygen (2–6 L min^{-1}, target > 90% SpO_2), treatment in a portable hyperbaric chamber, in cases of HACO administration of dexamethasone 4–8 mg every 8 h, and in cases of HAPO, nifedipine 30 mg every 12 h.

Resuscitation at high altitude does not differ from standard CPR. With the lower pO_2, CPR is more exhausting for the rescuer than at sea level, and the average number of effective chest compressions may decrease within the first minute.[598–600] Use mechanical chest compression devices whenever possible.

Commonly, no physician will be present to give guidance to nurses or paramedics on when to stop CPR. Guidelines have therefore been proposed for these situations.[46]

CPR may be withheld or terminated in a patient with absent vital signs when:

- the risk is unacceptable to the rescuer
- the rescuer is exhausted
- extreme environments prevent CPR
- any of the following apply:
  - decapitation
  - truncal transection
  - whole body incinerated
  - decomposed
  - frozen solid
  - avalanche victim in asystole with obstructed airway and burial time > 60 min (see avalanche burial below).

CPR may be also terminated when all of the following criteria apply:

- unwitnessed loss of vital signs;
- no ROSC during 20 min of CPR;
- no shock advised at any time by AED or only asystole on ECG;
- no hypothermia or other reversible causes warranting extended CPR.

In situations where transport is not possible, and correction of reversible causes is not possible, further resuscitation is futile and CPR should be terminated. These recommendations should be interpreted in the context of local conditions and legislation.

Avalanche burial

Introduction. In Europe and North America together, there are about 150 snow avalanche deaths each year. Most are sports-related and involve skiers, snowboarders and snowmobilers. Fatalities are mainly due to asphyxia, sometimes associated with trauma and hypothermia. Prognostic factors are severity of injury, duration of complete burial, airway patency, core temperature and serum potassium.[601] Completely buried avalanche victims die from asphyxia within 35 min if the airway is obstructed. The average cooling rate is 3 °C h^{-1},[602] ranging from 0.6 °C h^{-1} to 9 °C h^{-1}[603,604]; moderate to severe hypothermia may become important after 60 min of burial if the airway is patent. The highest recorded potassium in an avalanche victim who was successfully resuscitated is 6.4 mmol L^{-1}.[601,605–607] The survival rate of avalanche victims presenting with cardiac arrest ranges from 7% to 17%.[605,606] Survival patterns differ across countries due to terrain, climate and prehospital medical care.[56,608–610]

Decision-making on scene. Avalanche victims are not likely to survive when they are:

- buried > 60 min (or if the initial core temperature is <30 °C) and in cardiac arrest with an obstructed airway on extrication;
- buried and in cardiac arrest on extrication with an initial serum potassium > 8 mmol L^{-1}.

Full resuscitative measures, including extracorporeal rewarming, are indicated for all other avalanche victims without evidence of an unsurvivable injury.

Avalanches occur in areas that are difficult to access by rescuers in a timely manner, and burials frequently involve multiple victims. The decision to initiate full resuscitative measures should be determined by the number of victims and the resources available, and should be informed by the likelihood of survival.[601] As adherence to present guidelines is poor,[611,612] the use of a standardised checklist is recommended.[613]

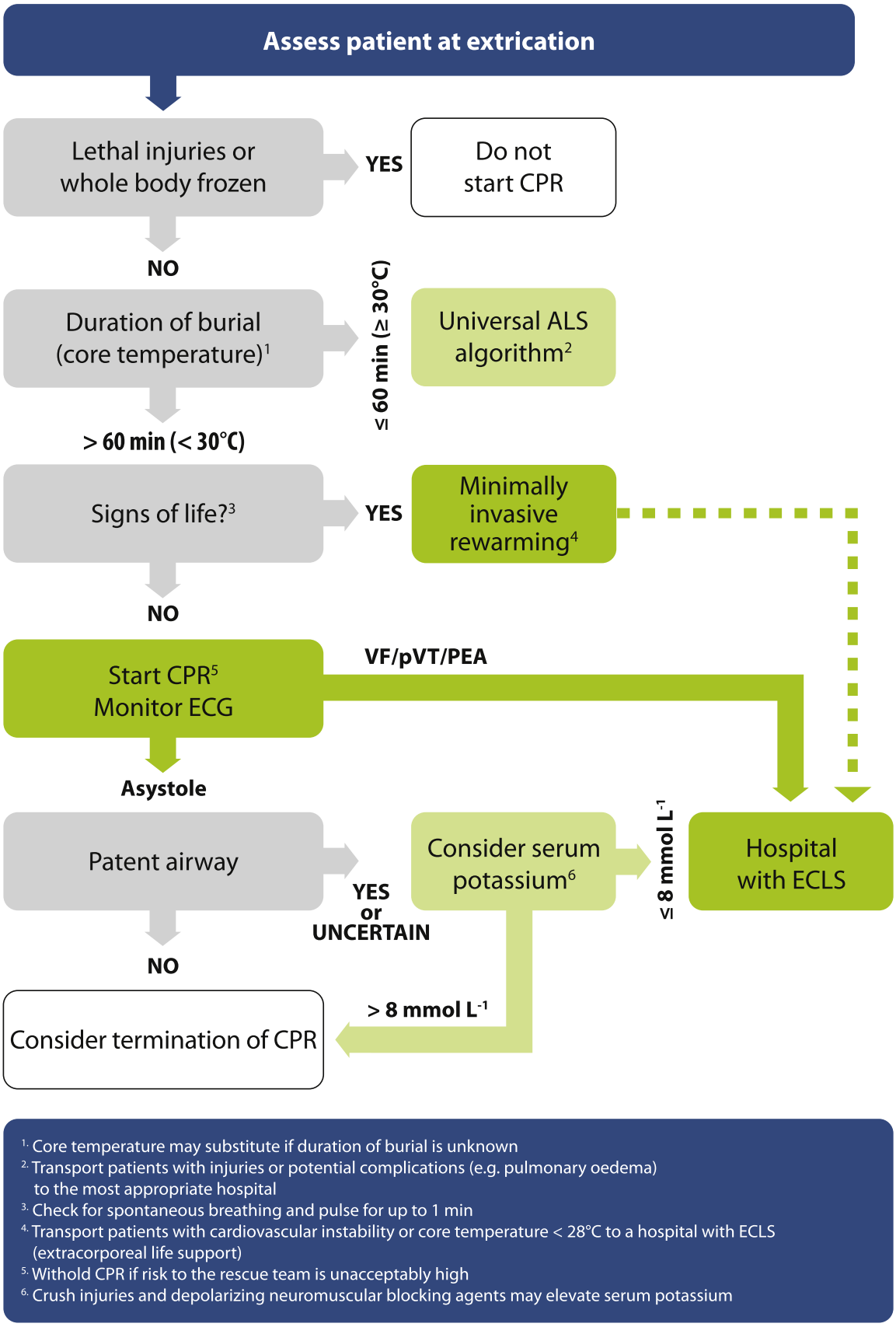
Management of completely buried avalanche victims. The algorithm for the management of buried avalanche victims is shown in Fig. 4.7:

- In all cases, extricate the body gently and use spinal precautions.
- Consider withholding resuscitation at the scene if it increases risk to the rescue team or if the victim is lethally injured or completely frozen.
- Determine the duration of burial. If unknown, core temperature may substitute for decision-making.
- If the duration of burial is ≤60 min (or initial core temperature is ≥30 °C) and cardiac arrest is confirmed, follow standard ALS guidelines (see adult advanced life support).[168] During CPR, measure core temperature, monitor ECG, give oxygen and apply insulation and heat packs to the trunk. Give drugs and fluids only if IV or IO access can be established within a few minutes. Resuscitation may be terminated in a normothermic asystolic patient if ALS is not successful after 20 min, in an absence of reversible cause (see ethics of resuscitation and end-of-life decisions).[243]
- Transport survivors with concern of respiratory (e.g. pulmonary oedema) or other-system critical illness or injury to the most appropriate medical centre. Provide specific trauma care as indicated. The admitting hospital must be capable of advanced active external or core internal rewarming.
- If the duration of burial is > 60 min (or initial core temperature is < 30 °C) and cardiac arrest is confirmed, start CPR and attach monitor. If there is any electrical activity or a patent airway in an asystolic patient, continue CPR. Defibrillation beyond three attempts may be delayed until core temperature ≥30 °C.
- Transport all patients who present with cardiovascular instability (i.e. ventricular arrhythmias, systolic blood pressure < 90 mmHg) or core temperature < 28 °C to an ECLS rewarming centre. Follow regional hypothermia protocols if needed.
- If direct transport to an ECLS rewarming centre is not possible in a timely manner, e.g. by HEMS, check potassium level at the nearest hospital. If potassium exceeds > 8 mmol L^{-1}, consider terminating resuscitation (after excluding crush injuries and considering if depolarizing muscle relaxants were used).

Lightning strike and electrical injuries

Introduction. Electrical injury is a relatively infrequent but potentially devastating multisystem injury with high morbidity and mortality, causing 0.54 deaths per 100,000 people each year. Most electrical injuries occur indoors. In adults, electrical injuries are common in the workplace and are generally associated with high voltage, whereas children are at risk primarily at home, where the voltage is lower (220 V in Europe, Australia and Asia; 110 V in the United States and Canada).[614] Electrocution from lightning strikes is rare, but worldwide it causes 1000 deaths each year.[615]

Electric shock injuries are caused by the direct effects of current on cell membranes and on vascular smooth muscle. The thermal



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

Fig. 4.7. Avalanche accident algorithm. Management of completely buried victims. (ECLS, extracorporeal life support).

energy associated with high-voltage electrocution will also cause burns. Factors influencing the severity of electrical injury include whether the current is alternating (AC) or direct (DC), voltage, magnitude of energy delivered, resistance to current flow, pathway of current through the patient, and the area and duration of contact. Skin resistance is decreased by moisture, which increases the likelihood of injury. Electric current follows the path of least resistance; conductive neurovascular bundles within limbs are particularly prone to damage. Contact with AC may cause tetanic contraction of skeletal muscle, which may prevent release from the source of electricity. Myocardial or respiratory failure may cause immediate death:

- Respiratory arrest may be caused by paralysis of the central respiratory control system or the respiratory muscles.
- Current may precipitate ventricular fibrillation (VF) if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon).[616] Electrical current may also cause myocardial ischaemia because of coronary artery spasm. Asystole may be primary, or secondary to asphyxia following respiratory arrest.

Current that traverses the myocardium is more likely to be fatal. A transthoracic (hand-to-hand) pathway is more likely to be fatal than a vertical (hand-to-foot) or straddle (foot-to-foot) pathway. There may be extensive tissue destruction along the current pathway.

Associated injuries are common. Blast (hyperbaric) injuries, injuries from being thrown from the point of contact and tetanic contraction causing limb fractures have all been reported.

Lightning strike. Lightning strikes deliver as much as 300 kV over a few milliseconds. Most of the current from a lightning strike passes over the surface of the body in a process called 'external flashover'. Both industrial shocks and lightning strikes cause deep burns at the point of contact. For industrial shocks the points of contact are usually on the upper limbs, hands and wrists, whereas for lightning they are mostly on the head, neck and shoulders. Injury may also occur indirectly through ground current or current splashing from a tree or other object that is hit by lightning.[617] Explosive force may cause blunt trauma.[618] The pattern and severity of injury from a lightning strike varies considerably, even among affected individuals from a single group.[619–621] As with industrial and domestic electric shock, death is caused by cardiac[620–624] or respiratory arrest.[617,625] In those who survive the initial shock, extensive catecholamine release or autonomic stimulation may occur, causing hypertension, tachycardia, non-specific ECG changes (including prolongation of the QT interval and transient T-wave inversion), and myocardial necrosis. Creatine kinase may be released from myocardial and skeletal muscle. Lightning can also cause central and peripheral nerve damage; brain haemorrhage and oedema, and peripheral nerve injury are common. Mortality from lightning injuries is as high as 30%, with up to 70% of survivors sustaining significant morbidity.[626,627]

Diagnosis. The circumstances surrounding the incident are not always known. Unique pattern of skin lesions called feathering or Lichtenberg figure is a pathognomonic symptom that is seen only in patients struck by lightning.[628] Unconscious patients with linear or punctuate burns (feathering) should be treated as victims of lightning strike.[617]

Safety measures. Ensure that any power source is switched off and do not approach the casualty until it is safe. High-voltage (above domestic mains) electricity can arc and conduct through the ground for up to a few metres around the casualty. It is safe to approach and handle casualties after lightning strike, although it would be wise to move to a safer environment, particularly if lightning has been seen within 30 min.[617]

Resuscitation. Patients struck by lightning are most likely to die if they sustain immediate cardiac or respiratory arrest and are not treated rapidly. When multiple victims are struck simultaneously by lightning, rescuers should give highest priority to patients in respiratory or cardiac arrest. Victims with respiratory arrest may require only ventilation to avoid secondary hypoxic cardiac arrest. Resuscitative attempts may have higher success rates in lightning victims than in patients with cardiac arrest from other causes, and efforts may be effective even when the interval before the resuscitative attempt is prolonged.[625] Dilated or non-reactive pupils should never be used as a prognostic sign, particularly in patients suffering a lightning strike.[617]

Start standard BLS and ALS without delay:

- Airway management may be difficult if there are electrical burns around the face and neck. Early tracheal intubation is needed in these cases, as extensive soft-tissue oedema may develop causing airway obstruction. Head and spine trauma can occur after electrocution. Immobilise the spine until evaluation can be performed.
- Muscular paralysis, especially after high voltage, may persist for several hours[627]; ventilatory support is required during this period.
- VF is the commonest initial arrhythmia after high-voltage AC shock; treat with prompt attempted defibrillation. Asystole is more common after DC shock; use standard protocols for this and other arrhythmias.
- Remove smouldering clothing and shoes to prevent further thermal injury.
- Vigorous fluid therapy is required if there is significant tissue destruction. Maintain a good urine output to enhance the excretion of myoglobin, potassium and other products of tissue damage.[624]
- Consider early surgical intervention in patients with severe thermal injuries.
- Maintain spinal immobilisation if there is a likelihood of head or neck trauma.[629,630]
- Conduct a thorough secondary survey to exclude traumatic injuries caused by tetanic muscular contraction or by the person being thrown.[629,631]
- Electrocution can cause severe, deep soft-tissue injury with relatively minor skin wounds, because current tends to follow neurovascular bundles; look carefully for features of compartment syndrome, which will necessitate fasciotomy.
- Although rare, consider abdominal visceral injuries caused directly by electrical damage.

There are conflicting reports on the vulnerability of the fetus to electric shock. The clinical spectrum of electrical injury ranges from a transient unpleasant sensation for the mother with no effect on her fetus, to placental abruption, fetal burn or intrauterine fetal death either immediately or a few days later. Several factors, such as the magnitude of the current and the duration of contact, are thought to affect outcome.[632]

Further treatment and prognosis. Immediate resuscitation of young victims in cardiac arrest from electrocution can result in long-term survival. Successful resuscitation has been reported after prolonged life support.

All those who survive electrical injury should be monitored in hospital if they have a history of cardiorespiratory problems or have had:

- loss of consciousness
- cardiac arrest

- electrocardiographic abnormalities
- soft tissue damage and burns.

Severe burns (thermal or electrical), myocardial necrosis, the extent of central nervous system injury, and secondary multisystem organ failure determine the morbidity and long-term prognosis. Bone marrow embolism has also been reported in some cases.[633] There is no specific therapy for electrical injury, and the management is supportive. Prevention remains the best way to minimise the prevalence and severity of electrical injury.

Mass casualty incidents

Introduction

Mass casualty incidents (MCIs), characterised by greater demand for medical care than available resources, are rare events. Among the 19.8 million yearly emergency medical services (EMS) activations in the United States, 0.3% had an MCI code, but incidence of real disasters is much lower.[634] The International Federation of Red Cross and Red Crescent Societies (IFRC) reports about 90 disasters in Europe and 650 events worldwide annually.[635] The MCI or disaster 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.[636] Initial triage of casualties enables identification of patient care priorities. Unlike normal circumstances, CPR is not usually initiated in MCI, in order to avoid delaying potentially effective treatment for salvageable victims. This critical decision depends on available resources in relation to the number of casualties.

Triage and decision-making on scene

Safety.

- Safety at scene is paramount. Those first on scene must identify the actual and potential hazards and appropriate assistance must be requested immediately. The presence of multiple unconscious victims should always alert rescuers to the possibility of a CBRN incident. Unexpected danger may be present at crime scenes, or places polluted by noxious substances e.g. carbon monoxide, industrial cyanides or other chemicals. During sarin attacks in Japan, 10% of 1363 EMS technicians developed poisoning, mostly from primary victims in poorly ventilated ambulances.[637]
- Use adequate protection measures and consider potential risks before approaching casualties. Be aware that wearing some personnel protective equipment may adversely affect performance of treatment interventions and limit the care that can be given in contaminated zones. Simulation studies have shown reduced success rate of advanced airway techniques, prolonged time for securing IV and IO access, and difficulties with drug preparation.[638–640]

Triage.

- Use a triage system to prioritise treatment, e.g. START (Simple Triage and Rapid Transport), *Newport Beach Fire Department, CA, USA*,[641] SALT (Sort-Assess-Lifesaving Interventions-Treat/Transport).[642,643] Advanced prehospital teams involved in the initial scene triage must avoid overtriage. Repeated triage (re-triage) is needed on entering the hospital and responsible personnel at all stages of emergency care must be familiar with the triage system used.
- If the START triage sieve is used, everyone able to walk is directed to clear the scene, and respiratory status of non-walking patients is assessed. If the casualty does not breathe, open the airway using basic manoeuvers (head tilt and chin lift, or jaw thrust). Look, listen and feel for no more than 10 s. A patient who does not begin breathing is triaged as dead. If an unresponsive victim is breathing normally, turn them into the recovery position and label as immediate (highest priority) for treatment. Further assessment of casualties, e.g. respiratory rate, capillary refill time, etc., and depends on individual triage protocols.
- The decision to use an MCI triage sieve, and withhold CPR to those with imminent death (including victims without signs of life), is the responsibility of a medical commander who is usually the most experienced EMS clinician on scene.
- 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.[644] Modern technologies such as educational video games enhance learning and improve subsequent performance when compared to traditional educational methods, e.g. card-sort exercise.[645] Training allows fast and correct recognition of those requesting life-saving procedures, and reduces the risk of inappropriate care given to futile cases.
- Consider assigning a higher triage risk level to the elderly and to survivors of high-energy trauma in order to reduce preventable deaths. After an aeroplane crash in the Netherlands, 9% of the minor injuries (lowest priority), and 17% of all walking casualties were undertriaged while suffering serious injuries.[646] In the National Trauma Database (NTDB), patients in all triage levels were compared to mortality outcomes. There were 322,162 subjects assigned to the lowest priority triage level of which 2046 died before hospital discharge. Age was the primary predictor of undertriage.[641]
- Perform life-saving interventions in patients triaged as immediate (highest priority) to prevent cardiac arrest: control major haemorrhage, open airway using basic techniques, perform chest decompression for tension pneumothorax, use antidotes, and consider initial rescue breaths in a non-breathing child.[642]
- In children, use of a special triage tape or a paediatric-specific MCI triage system (e.g. JumpSTART, *Team Life Support, Inc., FL, USA*, http://www.jumpstarttriage.com) or a universal SALT system.[647] If it is not available, use any triage system for adults.

C – SPECIAL PATIENTS

Cardiac arrest associated with concomitant diseases

Asthma

Introduction. Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma.[648] The worldwide prevalence of asthma symptoms ranges from 1 to 18% of the population with a high prevalence in some European countries (United Kingdom, Scandinavia and Netherlands) and in Australia.[648,649] In recent years, the prevalence of asthma and its related morbidity and mortality appears to have plateaued and may even have decreased in some countries, especially in children and adolescents.[650–653] The World Health Organization (WHO) has estimated that 15 million disability-adjusted life years (DALYs) are lost annually from asthma, representing 1% of the global disease burden. Annual worldwide deaths from asthma have been estimated at 250,000. The death rate does not appear to be correlated with asthma prevalence.[648] National and international guidance for the management of severe asthma already exists.[654] This guidance focuses on the treatment of patients with near-fatal asthma and subsequent cardiac arrest.

Patients at risk of asthma-related cardiac arrest. The risk of near-fatal asthma attacks is not necessarily related to asthma severity.[655] Patients most at risk include those with:

- a history of near-fatal asthma requiring intubation and mechanical ventilation;[656]
- hospitalisation or emergency care for asthma in the past year;[657]
- low or no use of inhaled corticosteroids;[658]
- increasing use and dependence of beta-2 agonists;[659]
- anxiety, depressive disorders and/or poor compliance with therapy;[660,661]
- food allergy in a patient with asthma.[662]

A national confidential enquiry carried out in the UK in 2014 showed that the majority of asthma-related deaths occurred before admission to hospital.[663] Compared with younger adults, older adults have higher rates of near-fatal asthma-related events and higher comorbidity-adjusted risk of mortality.[664]

Causes of cardiac arrest. Cardiac arrest in a person with asthma is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden. Cardiac arrest in those with asthma has been linked to:

- severe bronchospasm and mucous plugging leading to asphyxia (this condition causes the vast majority of asthma-related deaths);
- cardiac arrhythmias caused by hypoxia, which is the commonest cause of asthma-related arrhythmia.[665] Arrhythmias can also be caused by stimulant drugs (e.g. beta-adrenergic agonists, aminophylline) or electrolyte abnormalities;
- dynamic hyperinflation, i.e. auto positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure;
- tension pneumothorax (often bilateral).

Diagnosis. Wheezing is a common physical finding, but severity does not correlate with the degree of airway obstruction. The absence of wheezing may indicate critical airway obstruction, whereas increased wheezing may indicate a positive response to bronchodilator therapy. SpO_2 may not reflect progressive alveolar hypoventilation, particularly if oxygen is being given. SpO_2 may initially decrease during therapy because beta-agonists cause both bronchodilation and vasodilation, initially increasing intrapulmonary shunting.

Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis,[101] pneumonia, foreign bodies, pulmonary embolism, and subglottic mass.[666]

The severity of an asthma attack is defined in Table 4.3.

Table 4.3
The severity of asthma (PEF, peak expiratory flow)

Near-fatal asthma	Raised $PaCO_2$ and/or mechanical ventilation with raised inflation pressures	
Life-threatening asthma	Any one of the following in a patient with severe asthma:	
	Clinical signs	Measurements
	Altered conscious level	PEF < 33% best or predicted
	Exhaustion	SpO_2 < 92%
	Arrhythmia	PaO_2 < 8 kPa (60 mmHg)
	Hypotension	'Normal' $PaCO_2$ (4.6–6.0 kPa; 35-45 mmHg)
	Cyanosis	
	Silent chest	
	Poor expiratory effort	
Acute severe asthma	Any one of: - PEF 33–50% best or predicted - respiratory rate ≥ 25 min^{-1} - heart rate ≥ 110 min^{-1} - inability to complete sentences in one breath	

Prevention of cardiac arrest. A patient with severe asthma requires immediate and aggressive medical management to prevent deterioration. Base the assessment and treatment on a systematic ABCDE approach. Patients with SpO_2 < 92% or with features of life-threatening asthma are at risk of hypercapnia and require arterial blood gas measurement. Experienced clinicians should treat these high-risk patients in a critical-care area. The specific drugs and the treatment sequence will vary according to local practice.

Oxygen. Use a concentration of inspired oxygen that will achieve an SpO_2 94–98%.[127] High-flow oxygen by mask is sometimes necessary. Lack of pulse oximetry should not prevent the use of oxygen.

Nebulised beta-2 agonists. Inhaled beta-2 agonists are first line drugs in patients with an acute asthma attack and should be administered as early as possible. Intravenous beta-2 agonists should be reserved for those patients in who inhaled therapy cannot be used reliably. Salbutamol, 5 mg nebulised, is the cornerstone of therapy for acute asthma in most of the world. Repeated doses every 15–20 min are often needed. Severe asthma may necessitate continuous nebulised salbutamol. Nebuliser units that can be driven by high-flow oxygen (at least 6 L min^{-1}) should be available. The hypoventilation associated with severe or near-fatal asthma may prevent effective delivery of nebulised drugs. If a nebuliser is not immediately available, beta-2 agonists can be temporarily administered by repeating activations of a metered dose inhaler via a large volume spacer device.[667,668] Nebulised adrenaline does not provide additional benefit over and above nebulised beta-2 agonists in acute asthma.[669]

Nebulised anticholinergics. Nebulised anticholinergics (ipratropium, 0.5 mg 4–6 hourly) may produce additional bronchodilation in severe asthma or in those who do not respond to beta-agonists.[670,671]

Nebulised magnesium sulphate. Although limited evidence suggests that magnesium sulphate has some bronchodilator effects[672] a review of 16 randomised or pseudo-randomised controlled trials in adults and children with acute asthma showed that inhaled magnesium alone or in combination with inhaled beta(2)-agonists (with or without inhaled ipratropium) was not associated with significant benefit in terms of improved pulmonary function or reduced hospital admissions.[673] Results of small studies in adults with severe exacerbations of asthma showed improvements in pulmonary function with additional inhaled magnesium, however evidence was too limited to come to a definite conclusion. Inhaled magnesium sulphate is currently not recommended for the treatment of acute asthma.

Intravenous magnesium sulphate. Studies of IV magnesium sulphate in acute severe and life-threatening asthma have produced conflicting results.[672,674,675] A systematic review assessing 14 studies (three of which were multicentre trials) including a total of 2313 adult or mostly adult patients treated for acute asthma in the emergency department showed that a single infusion of 1.2 or 2 g IV $MgSO_4$ over 15–30 min significantly reduced hospital admissions compared with placebo (odds ratio [OR] 0.75, 95% confidence interval [CI] 0.60–0.92) and improved lung function.[676] Participants in almost all of the studies had already been given at least oxygen, nebulised short-acting beta-2-agonists and IV corticosteroids in the emergency department. No difference was observed for other

outcomes such as intensive care admissions and length of hospital stay.

Give a single dose of IV magnesium sulphate to patients with acute severe asthma (PEF < 50% best or predicted) who have not had a good initial response to inhaled bronchodilator therapy. The most commonly reported adverse effects of IV magnesium sulphate are flushing, fatigue, nausea, headache and hypotension.

Intravenous corticosteroids. Early use of systemic corticosteroids for acute asthma in the emergency department significantly reduces hospital admission rates, especially for those patients not receiving concomitant corticosteroid therapy.[677] Although there is no difference in clinical effects between oral and IV formulations of corticosteroids,[678] the IV route is preferable because patients with near-fatal asthma may vomit or be unable to swallow.

Intravenous bronchodilators. There is a lack of definitive evidence for or against the use of IV bronchodilators in this setting. Trials have primarily included spontaneously breathing patients with moderate- to life-threatening exacerbations of asthma; evidence in ventilated patients with life-threatening asthma or cardiac arrest is sparse. The use of IV bronchodilators should generally be restricted to patients unresponsive to nebulised therapy or where nebulised/inhaled therapy is not possible (e.g. a patient receiving bag-mask ventilation). A Cochrane review of intravenous beta-2 agonists compared with nebulised beta-2 agonists found no evidence of benefit and some evidence of increased side effects compared with inhaled treatment.[679] Salbutamol may be given as either a slow IV injection (250 mcg IV slowly) or continuous infusion of 3–20 mcg min^{-1}.

Aminophylline. A Cochrane review of intravenous aminophylline found no evidence of benefit and a higher incidence of adverse effects (tachycardia, vomiting) compared with standard care alone.[680,681] Whether aminophylline has a place as an additional therapy after treatment with established medications such as inhaled beta-agonists and systemic corticosteroids remains uncertain. If, after obtaining senior advice, the decision is taken to administer IV aminophylline, give a loading dose of 5 mg kg^{-1} over 20–30 min (unless on maintenance therapy), followed by an infusion of 500–700 mcg kg^{-1} h^{-1}. Maintain serum theophylline concentrations below 20 mcg mL^{-1} to avoid toxicity.

Leukotriene receptor antagonists. There are few data on the use of intravenous leukotriene receptor antagonists.[682] Limited evidence suggests improvement of lung function and a non-significant trend towards reduced hospital admission when the intravenous leukotriene receptor antagonist montelukast was used as a rescue therapy in adults with acute asthma.[683,684] Further studies are required to confirm the usefulness of leukotriene receptor antagonists in this setting.

Intravenous fluids and electrolytes. Severe or near-fatal asthma is associated with dehydration and hypovolaemia, and this will further compromise the circulation in patients with dynamic hyperinflation of the lungs. If there is evidence of hypovolaemia or dehydration, give IV crystalloids. Beta-2 agonists and steroids may induce hypokalaemia, which should be monitored and corrected with electrolyte supplements as required.

Heliox. Heliox is a mixture of helium and oxygen (usually 80:20 or 70:30). A meta-analysis of four clinical trials did not support the use of heliox in the initial treatment of patients with acute asthma.[685]

Intramuscular adrenaline. Sometimes it may be difficult to distinguish severe life-threatening asthma from anaphylaxis. Treat patients presenting with severe 'asthma-like' symptoms, but without pre-existing pulmonary disease (asthma, COPD), as if the cause was anaphylaxis. In these circumstances, administration of adrenaline 0.5 mg IM according to the anaphylaxis guidelines may be appropriate (see anaphylaxis).

Referral to intensive care. An intensive care specialist should assess patients that fail to respond to initial treatment, or develop signs of life-threatening asthma. Intensive care admission after asthma-related cardiac arrest is associated with significantly poorer outcomes compared with those in who a cardiac arrest does not occur.[686]

Consider rapid sequence induction and tracheal intubation if, despite efforts to optimise drug therapy, the patient has:

- a decreasing conscious level, or coma;
- persisting or worsening hypoxaemia;
- deteriorating respiratory acidosis, despite intensive therapy;
- severe agitation, confusion and fighting against the oxygen mask (clinical signs of hypoxaemia);
- progressive exhaustion;
- respiratory or cardiac arrest.

Elevation of the PCO_2 alone does not indicate the need for tracheal intubation.[687] Treat the patient, not the numbers. All patients transferred to intensive care units should be accompanied by a doctor suitably equipped and skilled to intubate.

Non-invasive ventilation. Non-invasive ventilation (NIV) decreases the intubation rate and mortality in COPD[688]; however, its role in patients with severe acute asthma is uncertain. There is insufficient evidence to recommend its routine use in asthma.[689]

Treatment of cardiac arrest.

Basic life support. Give BLS according to standard guidelines. Ventilation will be difficult because of increased airway resistance; try to avoid gastric inflation.

Advanced life support. Modifications to standard ALS guidelines include considering the need for early tracheal intubation. The peak airway pressures recorded during ventilation of patients with severe asthma (mean 67.8 ± 11.1 cmH_2O in 12 patients) are significantly higher than the normal lower oesophageal sphincter pressure (approximately 20 cmH_2O).[690] 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 less than normal.[691]

Respiratory rates of 8–10 breaths per minute and a tidal volume required for a normal chest rise during CPR should minimise dynamic hyperinflation of the lungs (air trapping). 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.[690]

Some case reports have reported ROSC in patients with air trapping when the tracheal tube was disconnected.[692–696] If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest while disconnecting tracheal tube may relieve air trapping. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.

Dynamic hyperinflation increases transthoracic impedance,[697] but modern impedance-compensated biphasic defibrillation waveforms are no less effective in patients with a higher impedance. As with standard ALS defibrillation protocols, consider increasing defibrillation energy if the first shock is unsuccessful and a manual defibrillator is available.

There is no good evidence for the use of open-chest cardiac compressions in patients with asthma-associated cardiac arrest. Working through the four Hs and four Ts will identify potentially

reversible causes of asthma-related cardiac arrest. Tension pneumothorax can be difficult to diagnose in cardiac arrest; it may be indicated by unilateral expansion of the chest wall, shifting of the trachea and subcutaneous emphysema. Pleural ultrasound in skilled hands is faster and more sensitive than chest X-ray for the detection of pneumothorax.[698] If a pneumothorax is suspected, perform needle decompression using a large gauge cannula and being careful to avoid direct puncture of the lung. Any attempt at needle decompression should be followed by insertion of a chest tube. Always consider bilateral pneumothoraces in asthma-related cardiac arrest (see tension pneumothorax).

ECLS can ensure both organ perfusion and gas exchange in cases of otherwise refractory respiratory and circulatory failure. Cases of successful treatment of asthma-related cardiac arrest in adults using ECLS have been reported[699,700]; however, the role of ECLS in cardiac arrest caused by asthma has never been investigated in controlled studies. The use of ECLS requires appropriate skills and equipment that may not be available in all hospitals.

Patients with ventricular assist devices

Introduction. All clinicians caring for patients with ventricular assist devices (VADs) should have received full training in the procedures for equipment failure and the cardiac arrest situation. The management of patients with VADs is more complex, in that a cardiac arrest may be due to mechanical failure and in this situation there may be actions specific to the device that are required. The use of external chest compression in patients with ventricular assist devices has been reviewed.[701] There are isolated case reports of successful external chest compression without damage to the VAD. External chest compression may be particularly useful to decompress a non-functional right ventricle in cardiac arrests and often the right ventricle may be the cause of the loss of output.

Diagnosis of cardiac arrest. Confirming cardiac arrest in these patients may be difficult. A patient with invasive monitoring should be considered to have arrested if the arterial line reads the same as the central venous pressure (CVP) line. In patients without invasive monitoring, if the patient has no signs of life and is not breathing, then they should be considered to have suffered a cardiac arrest. Transthoracic/transoesophageal echocardiography (TTE/TOE), capnography or Doppler flow readings in a major artery may assist in the diagnosis of whether there is meaningful perfusion. These devices also display pump flow and this should be used to assist in a diagnosis of whether there has been a genuine loss of blood flow, or whether there is just a low flow situation with reduced conscious level.

Management of cardiac arrest. Patients with an implantable left ventricular assist devices (LVAD) such as a HeartMate (Thoratec, Pleasanton, CA, USA) or HeartWare (HeartWare, Framingham, MA, USA) device should have the same algorithm followed as the algorithm for arrest after cardiac surgery (see cardiac arrest following cardiac surgery). Check the rhythm; perform defibrillation for shockable rhythms (VF/pVT), start pacing for asystole. In pulseless electrical activity (PEA), turn the pacing off and verify there is no underlying VF, which must be treated by defibrillation. External chest compressions should be performed if immediate resuscitative efforts fail. Importantly, the airway and breathing checks should always be performed.

It is possible for a patient to have asystole or VF, but still have adequate cerebral blood flow due to adequate and continued pump flow. If the patient is conscious and responding then you will have more time in which to resolve this arrhythmia and external chest compressions will not be needed.

Resternotomy should be performed in an established cardiac arrest within 10 days of surgery and after this time, either resternotomy or extracorporeal membrane oxygenation (ECMO) is a reasonable option.

Cardiac arrest associated with neurological disease

Causes of cardiac arrest. Cardiac arrest associated with acute neurological disease is relatively uncommon and can occur with subarachnoid haemorrhage, intracerebral haemorrhage, epileptic seizures, and ischaemic stroke.[702] In addition brain injury associated with trauma can cause cardiac arrest.

Cardiac arrest associated with neurological disease can be due to:

- Loss of consciousness, causing airway obstruction, hypoxaemia and respiratory arrest followed by cardiac arrest. Loss of consciousness is also associated with an increased risk of aspiration of gastric contents into the lungs.
- Respiratory and cardiac depression caused by compression of the brain stem.
- Arrhythmias and myocardial dysfunction associated with acute neurological injury and in particular sub-arachnoid haemorrhage.
- Sudden unexpected death in epilepsy (SUDEP) effects about 1 in every 1000 people with epilepsy.[703]

Neurological symptoms. Patients can have prodromal signs suggesting a neurological cause before cardiac arrest such as headache, seizures, impaired consciousness, and focal signs,[704] but these are often non-specific and can include syncope, shortness of breath and chest pain. Cardiac or respiratory arrest occurs in between 3 and 11% of patents with subarachnoid haemorrhage,[705] and the initial rhythm is usually non-shockable.

Treatment. Preventive measures for cardiac or respiratory arrest should be aimed at treating the underlying cause. Once cardiac arrest occurs, follow standard BLS and ALS guidelines. If ROSC is achieved, address the underlying cause in addition to standard post resuscitation care.

Patients with subarachnoid haemorrhage may have ECG changes that suggest an acute coronary syndrome.[704,706] Certain features such as a young age, female gender, non-shockable initial rhythm and neurological antecedents (e.g. headache, seizures, neurological deficits) are common but non-specific for neurological cause.[707] Individuals with neurological prodromal symptoms who achieve ROSC may be considered for CT brain scan. Whether this is done before or after coronary angiography will depend on clinical judgement regarding the likelihood of a subarachnoid haemorrhage versus acute coronary syndrome.

Outcome. Survival depends on the underlying cause and traditional factors (e.g. witnessed, bystander CPR) associated with survival.[702] Prognosis is poor in those with ROSC after a subarachnoid haemorrhage.[704,706,708] Individuals who achieve ROSC after a primary neurological cause of cardiac arrest will often fulfil neurological criteria for death and should be considered as potential organ donors.[709]

Obesity

Introduction. Worldwide obesity has more than doubled since 1980. In 2014, more than 1.9 billion (39%) adults were overweight, and of these over 600 million (13%) were obese.

The World Health Organization (WHO) uses body mass index (BMI; weight in kg divided by height in m^2) to define obesity in adults as[710–712]:

- overweight (25.0–29.9 $kg\,m^{-2}$);
- obese (30.0–34.9 $kg\,m^{-2}$);
- very obese ($\geq$35.0 $kg\,m^{-2}$).

Many clinical studies have linked BMI to outcomes for a wide variety of cardiovascular and non-cardiovascular conditions.[713–715] Traditional cardiovascular risk factors (hypertension, diabetes, lipid profile, prevalent coronary heart disease, heart failure, and left ventricular hypertrophy) are common in obese patient. Obesity is associated with increased risk of sudden cardiac death.[715] Leading causes of death are dilated cardiomyopathy and severe coronary atherosclerosis.[716]

Modifications to cardiopulmonary resuscitation. No changes to 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 standardised to a patient's BMI or weight.[710] More rescuers than usual may be required to assist in moving the patient and rescuer fatigue, particularly in relation to the delivery of chest compressions, may necessitate more regular changes of the rescuer than normal.

Chest compressions. As with all cardiac arrests, chest compressions are most effective when performed with the patient lying on a firm surface, but it may be unsafe for the patient and rescuers to attempt to move the patient down onto the floor. However, it is not always necessary in obese patients because the heavier torso sinks into the mattress, leaving less potential for mattress displacement during chest compression.[717]

In order to maintain sufficient depth of chest compresisons (approximately 5 cm but no more than 6 cm), rescuer fatigue may necessitate the need to change rescuers more frequently than the standard 2 min interval. Use of mechanical resuscitation devices is limited by the slope of the anterior chest wall, thoracic dimensions (sternum height up to 303 mm, and maximal width of 449 mm for piston devices (LUCAS); chest circumference up to 130 cm and maximal chest width of 380 mm for devices with a load-distributing band), and patient weight (up to 136 kg) (AutoPulse).

Defibrillation. Optimal defibrillation energy levels in obese patients are unknown. Unlike monophasic defibrillators, modern biphasic defibrillators are impedance-compensated and adjust their output according to the patient's impedance. Two small retrospective studies have demonstrated no apparent weight-based influence on defibrillation efficacy,[718] with a biphasic waveform of 150 J achieving high shock success rates without need for energy escalation.[719] Defibrillation protocols for obese patients should therefore follow those recommended for patients with a normal BMI. Consider higher shock energies for defibrillation if initial defibrillation attempts fail.

Ventilation. Higher inspiration pressure is needed for positive pressure ventilation due to increased intraabdominal pressure.[720] Early tracheal intubation by an experienced provider removes the need for prolonged bag-valve-mask ventilation, and may reduce any risk of aspiration. In all patients with extreme obesity, difficult intubation must be anticipated, with a clear failed intubation drill if necessary.[721] If intubation fails, use of a supraglottic airway device (SAD) with oesophaegeal drainage tube is a suitable option.

Logistical considerations. A patient's BMI should be considered when organising prehospital resuscitation, especially with regard to technical support and number of ambulance crew members.[722] Special response vehicles modified to carry extremely obese patients, equipped with extra-wide interiors, reinforced stretchers and specialised lifting gear, should be used if possible. Weight limits of both stretchers and hospital beds must be checked prior to use.[723] Underestimation of the technical aspects of rescue operations may cause secondary transportation trauma, or even prohibit safe transfer of obese patient to the hospital.[722]

Outcome. The relation between obesity and outcome from cardiac arrest is unclear. One large registry study has shown that survival from cardiac arrests caused by shockable rhythms (VF/pVT) was highest in overweight patients but was significantly lower in those who were very obese.[710] In contrast, survival to discharge of non-shockable rhythms was similar across all BMI groups. Evidence from clinical cohort studies has suggested that overweight and obese patients may actually have a more favourable short-term and long-term prognosis than leaner patients once they are successfully resuscitated from cardiac arrest.[711,724]

Cardiac arrest associated with pregnancy

Introduction

Mortality related to pregnancy is relatively rare in Europe (estimate 16 per 100,000 live births) although there is a large variation between countries.[725] The fetus must always be considered when an adverse cardiovascular event occurs in a pregnant woman. Fetal survival usually depends on maternal survival and initial resuscitation efforts should focus on the pregnant mother. Resuscitation guidelines for pregnancy are based largely on case series, extrapolation from non-pregnant arrests, manikin studies and expert opinion based on the physiology of pregnancy and changes that occur in normal labour.[726,727]

Significant physiological changes occur during pregnancy, e.g. cardiac output, blood volume, minute ventilation and oxygen consumption all increase. Furthermore, the gravid uterus can cause significant compression of iliac and abdominal vessels when the mother is in the supine position, resulting in reduced cardiac output and hypotension.

Causes of cardiac arrest

In developed regions, haemorrhage, embolism (thromboembolic and amniotic fluid), hypertensive disorders of pregnancy, abortion and genital tract sepsis account for most deaths directly associated with pregnancy, and pre-existing medical conditions for those indirectly related to pregnancy.[728] A review of over 2 million pregnancies in the UK showed that maternal deaths (death during pregnancy, childbirth, or within 42 days after delivery) were associated with cardiac disease, neurological conditions, psychiatric conditions, and malignancies.[729] A quarter of pregnant women who died in the UK had sepsis, and 1 in 11 had influenza. Pregnant women can also sustain cardiac arrest from the same causes as women of the same age group.

Prevention of cardiac arrest in pregnancy

In an emergency, use a systematic ABCDE approach. Many cardiovascular problems associated with pregnancy are caused by aorto-caval compression.

Treat a pregnant patient as follows:

- Place the patient in the left lateral position or manually and gently displace the uterus to the left.
- Give oxygen, guided by pulse oximetry to correct any 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 and neonatal specialists should be involved early in the resuscitation.
- Identify and treat the underlying cause, e.g. rapid recognition and treatment of sepsis, including early intravenous antibiotics.

Modifications to basic life support

From 20 weeks' gestation, the uterus can compress both the inferior vena cava (IVC) and aorta, impeding venous return and cardiac output. Uterine obstruction of venous return can cause pre-arrest hypotension or shock and, in the critically ill patient, may precipitate cardiac arrest.[730,731] During cardiac arrest, the compromise in venous return and cardiac output by the gravid uterus limits the effectiveness of chest compressions.

Non-arrest studies show that left lateral tilt improves maternal blood pressure, cardiac output and stroke volume[732–734] and improves fetal oxygenation and heart rate.[735–737] Non cardiac arrest data show that the gravid uterus can be shifted away from the IVC in most cases, by placing the patient in 15° of left lateral decubitus position.[738] The value of relieving aortic or IVC compression during CPR is, however, unknown. 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 rescuers knees,[739] pillows or blankets, or the Cardiff wedge[740] although their efficacy in actual cardiac arrests is unknown. Even when a tilting table is used, the angle of tilt is often overestimated.[741] In a manikin study, the ability to provide effective chest compressions decreased as the angle of left lateral tilt increased and at an angle of greater than 30° the manikin tended to roll.[740]

The key steps for BLS in a pregnant patient are:

- Call for expert help early (including an obstetrician and a neonatologist).
- Start BLS according to standard guidelines.
- Ensure high-quality chest compressions with minimal interruptions.
- The hand position for chest compressions may need to be slightly higher on the sternum for patients with advanced pregnancy e.g. third trimester.[726]
- Manually displace the uterus to the left to reduce IVC compression.
- Add left lateral tilt if this is feasible and ensure the chest remains supported on a firm surface (e.g. in the operating room) – the optimal angle of tilt is unknown. Aim for between 15 and 30°. 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.
- Start preparing for emergency Caesarean section (see below) – the fetus will need to be delivered if initial resuscitation efforts fail.

Modifications to advanced life support

Defibrillation. For cardiac arrest with a shockable rhythm (VF/pVT) 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.[742] There is no evidence that shocks from a direct current defibrillator have adverse effects on the fetal heart.

Airway management. During pregnancy, there is a greater potential for gastro-oesophageal sphincter insufficiency and risk of pulmonary aspiration of gastric contents.[743,744] Although pregnant patients are at risk of aspiration, oxygenation and ventilation is the priority over aspiration prevention. Early tracheal intubation will however make ventilation of the lungs easier in the presence of increased intra-abdominal pressure.

A tracheal tube 0.5–1 mm internal diameter (ID) smaller than that used for a non-pregnant woman of similar size may be necessary because of maternal airway narrowing from oedema and swelling.[745] One study documented that the upper airways in the third trimester of pregnancy are narrower compared with their post partum state and to non-pregnant controls.[746] Tracheal intubation may be more difficult in the pregnant patient.[747] Expert help, a failed intubation drill and the use of alternative airway devices may be needed.[748]

Intravascular access. Early intravenous or intraosseous access will enable drug and fluid administration. Aiming for access above the diaphragm may address any theoretical concerns associated with delayed circulation caused by IVC compression if drugs are infused through sites below the IVC.

Reversible causes

Rescuers should attempt to identify common and reversible causes of cardiac arrest in pregnancy during resuscitation (see special causes). The 4 Hs and 4 Ts approach helps identify all the common causes of cardiac arrest in pregnancy. Pregnant patients are also 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 possible causes during cardiac arrest; however, do not delay other treatments and minimise interruptions to chest compressions.

Specific causes of cardiac arrest in pregnancy include the following:

Haemorrhage. Life-threatening haemorrhage can occur both antenatally and postnatally.[728] Postpartum haemorrhage is the commonest single cause of maternal death worldwide and is estimated to cause one maternal death every 7 min.[749] Associations include ectopic pregnancy, placental abruption, placenta praevia, placenta accreta, and uterine rupture.[750] A massive haemorrhage protocol must be used in all units and should be updated 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. The key step is to stop the bleeding.

Consider the following[751,752]:

- Fluid resuscitation, including use of rapid transfusion system and cell salvage.[753]
- Oxytocin and prostaglandin analogues to correct uterine atony.[754]
- Massaging the uterus.[755]
- Correction of coagulopathy including use of tranexamic acid and/or recombinant activated factor VII.[756–758]
- Uterine balloon tamponade or packing.[759,760]
- Uterine compression sutures.[761]
- Angiography and endovascular embolisation.[762]
- Hysterectomy.[763,764]
- Aortic cross-clamping in catastrophic haemorrhage.[765]

Cardiovascular disease. Myocardial infarction and aneurysm or dissection of the aorta or its branches, and peripartum cardiomyopathy cause most deaths from acquired cardiac disease.[766–768] 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.[750,769] 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. 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.[770]

Increasing numbers of women with congenital heart disease are becoming pregnant.[771] Heart failure and arrhythmias are the commonest problems, especially in those with cyanotic heart disease. Pregnant women with known congenital heart disease should be managed in specialist centres.

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.[772,773] Magnesium sulphate is effective in preventing approximately half of the cases of eclampsia developing in labour or immediately postpartum in women with pre-eclampsia.[774–777] Use magnesium sulphate infusion for the treatment of eclampsia.[778–781]

Pulmonary embolism. The estimated incidence of pulmonary embolism is 1–1.5 per 10,000 pregnancies, with a case fatality of 3.5% (95% CI 1.1–8.0%).[782] Risk factors include obesity, increased age, and immobility. Successful use of fibrinolytics for massive, life-threatening pulmonary embolism in pregnant women has been reported.[770,783–786]

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.[787] Patients may have warning signs preceding collapse including breathlessness, chest pain, feeling cold, lightheadedness, 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.[788]

Treatment is supportive, as there is no specific therapy based on an ABCDE approach and correction of coagulopathy. Successful use of extracorporeal life support techniques for women suffering life-threatening amniotic fluid embolism during labour and delivery is reported.[789]

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. Three observational studies of 154 subjects collectively[790–792] provide very low quality evidence regarding the use of peri-mortem Caesarean section. Based on expert opinion, when initial resuscitation attempts fail, delivery of the fetus may improve the chances of successful resuscitation of the mother and fetus.[793–795] One systematic review documented 38 cases of Caesarean section during CPR, with 34 surviving infants and 13 maternal survivors at discharge, suggesting that Caesarean section may have improved maternal and neonatal outcomes.[796] The best survival rate for infants over 24–25 weeks' gestation occurs when delivery of the infant is achieved within 5 min after the mother's cardiac arrest.[793,797–799] This requires that the provider commence the hysterotomy at about 4 min after cardiac arrest. At older gestational ages (30–38 weeks), infant survival is possible even when delivery was after 5 min from the onset of maternal cardiac arrest[796] A case series suggests increased use of Caesarean section during CPR with team training[791]; in this series no deliveries were achieved within 5 min after starting resuscitation. Eight of the twelve women had ROSC after delivery, with two maternal and five newborn survivors. Maternal case fatality rate was 83%. Neonatal case fatality rate was 58%.[791]

Delivery will relieve IVC compression and may improve chances of maternal resuscitation. The Caesarean delivery also enables access to the infant so that newborn resuscitation can begin.

Decision-making for emergency hysterotomy (Caesarean section). The gravid uterus reaches a size that will begin to compromise aorto-caval blood flow at approximately 20 weeks gestation; however, fetal viability begins at approximately 24–25 weeks.[800] Portable ultrasound is available in some emergency departments and may aid in determination of gestational age (in experienced hands) and positioning, provided its use does not delay the decision to perform emergency hysterotomy.[801]

- At gestational age less than 20 weeks, urgent Caesarean delivery need not be considered, because a gravid uterus of this size is unlikely to significantly compromise maternal cardiac output.
- At gestational age approximately 20–23 weeks, initiate emergency hysterotomy to enable successful resuscitation of the mother, not survival of the delivered infant, which is unlikely at this gestational age.
- At gestational age approximately ≥24–25 weeks, initiate emergency hysterotomy to save the life of both the mother and the infant.

Post resuscitation care

Post resuscitation care should follow standard guidelines. Targeted temperature management (TTM) 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.[802] Implantable cardioverter defibrillators (ICDs) have been used in patients during pregnancy.[803]

Preparation for cardiac arrest in pregnancy

ALS in pregnancy requires coordination of maternal resuscitation, Caesarean delivery of the fetus and newborn resuscitation ideally within 5 min.

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 and neonatal teams
- ensure regular training in obstetric emergencies.[804,805]

Elderly people

Epidemiology

More than 50% of people resuscitated from OHCAs in the United States are aged 65 years or older.[806] The incidence of cardiac arrests in elderly people is likely to increase as the world population ages. The incidence of cardiac arrest increases with age. In males, the incidence of OHCA at 80 years of age is about seven times greater than at 40 years of age.[807] In females above 70 years of age it is more than 40 times greater than in women below 45 years of age. In an observational study on in-hospital cardiac arrest patients above 65 years of age accounted for 46% of the total hospital admissions in the study period and for 65% of the ward cardiac arrests.[808] In this study, the incidence of arrests was more than twice that in the younger patient population (2.2 versus 1.0 per 1000 patient admissions).

Causes of cardiac arrest

The incidence of both coronary heart disease and chronic heart failure increases with age. As a consequence, elderly people have

an increased incidence of cardiac causes of arrest.[809] However, the proportion of deaths that are sudden (i.e. due to a primary ventricular arrhythmia) decreases with age, due to a parallel increase in the proportion of deaths due to other cardiovascular causes.[810] The incidence of PEA as the first recorded rhythm increases significantly with age[809,811]; with a parallel decrease of the incidence of shockable rhythms (VF/pVT).[812]

Prevention

Deterioration of vital signs leading to cardiac arrest is detected less accurately in elderly patients, compared with younger patients.[813] Clinical signs of acute life-threatening conditions such as sepsis,[814] acute myocardial infarction[815] or heart failure[816] are often blunted or non-specific in elderly patients, resulting in less physiological aberration and a lower Modified Early Warning Score (MEWS) in the 4 h preceding cardiac arrest.[808]

Treatment

Management of periarrest conditions. Ageing is associated with several pathophysiological changes that should be taken into account when managing peri-arrest conditions. Increasing age is associated with autonomic and baroreflex dysfunction and with myocardial stiffening which impairs early diastolic filling.[817] In addition, elderly critically ill patients are often hypovolaemic due to a reduction of both fluid intake and urine-concentrating ability.[818] These changes compromise the cardiovascular response to fluid loss or postural changes and increase the hypotensive effect of sedatives and other vasoactive drugs. Elderly patients are at increased risk of severe hypotension during emergency airway management.[819]

Atrial fibrillation is the most common supraventricular arrhythmia in the elderly. It often causes cardiovascular compromise due to loss of the atrial contribution for diastolic filling, particularly in the elderly who have reduced ventricular compliance. Hypotension and an increased heart rate may reduce coronary perfusion and precipitate cardiac ischaemia, which is more likely in an elderly population with a greater incidence of coronary artery disease.

Older patients are more likely to develop apnea or respiratory depression following the administration of opioid or benzodiazepines.[818] Their lower baseline oxygen tension also increases the risk of developing hypoxia. Advancing age is associated with an increased rate of comorbidities. Elderly patients often take several medications, which may interfere with drugs administered in peri-arrest conditions. The incidence of adverse drug reactions in the elderly is 2–3 times higher than in younger patients.[820]

Management of cardiac arrest. No modifications of standard resuscitation protocols are needed when managing aged patients in cardiac arrest. Rescuers, however, should be aware that the risk of both sternal and rib fractures is higher in elderly.[821–823] The incidence of CPR-related injuries increases with duration of CPR.[823]

Outcome

Older age is associated with an increasingly lower short-term survival rate after cardiac arrest.[824–829] In a large registry of OHCAs, survival to discharge was 8% for those aged 65–79 years, 4% for octogenarians and 2% for nonagenarians.[826] In another study, the adjusted risk for 30-day mortality in elderly resuscitated comatose patients was 1.04 (95% CI 1.03–1.06) per year of age.[812]

Increasing age is also associated with lower long-term survival after resuscitation. In a retrospective cohort study on elderly patients discharged alive after CPR from in-hospital cardiac arrest the risk-adjusted rate of 1-year survival was 63.7%, 58.6%, and 49.7% among patients 65–74, 75–84, and ≥85 years of age, respectively ($P < 0.001$).[827] In another study patients ≥65 years of age discharged alive after resuscitation from VF/pVT cardiac arrest showed a significantly lower long-term survival than an age- and gender-matched controls, while this was not observed in younger resuscitated patients.[830]

In those who do survive, neurological outcome is good in elderly survivors of cardiac arrest, with 95% having a cerebral performance category (CPC) score of 1–2 on discharge from ICU[824] and 72% at hospital discharge.[827]

Decision to resuscitate

Elderly patients with cardiac arrest are significantly less likely to receive resuscitation than younger patients.[831,832] When deciding to resuscitate elderly patients, age alone should not be the only criterion to consider and other more established criteria, i.e. witnessed status, resuscitation times, and first recorded rhythm, are important factors.[833] In addition, we suggest that pre-arrest factors, such as the degree of autonomy, quality of life, mental status and the presence of major comorbidities, should also be considered. Whenever possible, a decision to resuscitate or not, should be discussed in advance with the patient and his/her family (see ethics of resuscitation and end-of-life decisions).[243]

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Conflicts of interest

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