European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: Electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution

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8a. Life-threatening electrolyte disorders

Overview

Electrolyte abnormalities can cause cardiac arrhythmias or cardiopulmonary arrest. Life-threatening arrhythmias are associated most commonly with potassium disorders, particularly hyperkalaemia, and less commonly with disorders of serum calcium and magnesium. In some cases therapy for life-threatening electrolyte disorders should start before laboratory results become available.

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. There are no major changes in the treatment of these disorders since Guidelines 2005.[1]

Prevention of electrolyte disorders

Identify and treat life-threatening electrolyte abnormalities before cardiac arrest occurs. Remove any precipitating factors (e.g., drugs) and monitor electrolyte values to prevent recurrence of the abnormality. Monitor renal function in patients at risk of electrolyte disorders (e.g., chronic kidney disease, cardiac failure). In haemodialysis patients, review the dialysis prescription regularly to avoid inappropriate electrolyte shifts during treatment.

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. When serum pH increases (alkalaemia), serum potassium decreases because potassium shifts intracellularly. Anticipate the effects of pH changes on serum potassium during therapy for hyperkalaemia or hypokalaemia.

Hyperkalaemia

This is the most common electrolyte disorder associated with cardiopulmonary arrest. It is usually caused by increased potassium release from cells, impaired excretion by the kidneys or accidental potassium chloride administration.

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

There are several potential causes of hyperkalaemia, including renal failure, drugs (angiotensin converting enzyme inhibitors (ACE-I), angiotensin II receptor antagonists, potassium sparing diuretics, non-steroidal anti-inflammatory drugs (NSAIDs), beta-blockers, trimethoprim), tissue breakdown (rhabdomyolysis, tumour lysis, haemolysis), metabolic acidosis, endocrine disorders (Addison's disease), hyperkalaemic periodic paralysis, or diet (may be sole cause in patients with advanced chronic kidney disease). Abnormal erythrocytes or thrombocytosis may cause a spuriously high potassium concentration.[2] The risk of hyperkalaemia is even greater when there is a combination of factors such as the concomitant use of ACE-I and NSAIDs or potassium sparing diuretics.

Recognition of hyperkalaemia

Exclude hyperkalaemia in patients with an arrhythmia or cardiac arrest.[3] 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, cardiopulmonary arrest or sudden death. The effect of hyperkalaemia on the ECG depends on the absolute serum potassium as well as the rate of increase. Most patients will have ECG abnormalities at a serum potassium concentration higher than $6.7\,mmol\,l^{-1}$.[4] The use of a blood gas analyser that measures potassium can reduce delay in recognition.

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 [T wave larger than R wave in more than 1 lead];
- ST-segment depression;
- S and T wave merging (sine wave pattern);
- widened QRS [>0.12 s];
- ventricular tachycardia;
- bradycardia;

- cardiac arrest (pulseless electrical activity [PEA], ventricular fibrillation/pulseless ventricular tachycardia [VF/VT], asystole).

Treatment of hyperkalaemia

There are three key treatments for hyperkalaemia[5]:

1. cardiac protection;
2. shifting potassium into cells;
3. removing potassium from the body.

Intravenous calcium salts are not generally indicated in the absence of ECG changes. Monitor effectiveness of treatment, be alert to rebound hyperkalaemia and take steps to prevent recurrence of hyperkalaemia. 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 of hyperkalaemia has been the subject of a Cochrane review.[6]

Patient not in cardiac arrest.   Assess ABCDE (Airway, Breathing, Circulation, Disability, Exposure) and correct any abnormalities. Obtain intravenous access, check serum potassium and record an ECG. Treatment is determined according to severity of hyperkalaemia.

Approximate values are provided to guide treatment.

Mild elevation ($5.5–5.9\,mmol\,l^{-1}$):

- Remove potassium from body: potassium exchange resins – calcium resonium 15–30 g OR sodium polystyrene sulfonate (Kayexalate) 15–30 g in 50–100 ml of 20% sorbitol, given either orally or by retention enema (onset in 1–3 h; maximal effect at 6 h).
- Address cause of hyperkalaemia to correct and avoid further rise in serum potassium (e.g., drugs, diet).

Moderate elevation ($6–6.4\,mmol\,l^{-1}$) 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; monitor blood glucose).
- Remove potassium from the body as described above.
- Haemodialysis: consider if oliguric; haemodialysis is more efficient than peritoneal dialysis at removing potassium.

Severe elevation ($\geq6.5\,mmol\,l^{-1}$) without ECG changes. Seek expert help and:

- Use multiple shifting agents.
- Glucose/insulin (see above).
- Salbutamol 5 mg nebulised. Several doses (10–20 mg) may be required (onset in 15–30 min).
- Sodium bicarbonate: 50 mmol IV over 5 min if metabolic acidosis present (onset in 15–30 min). Bicarbonate alone is less effective than glucose plus insulin or nebulised salbutamol; it is best used in conjunction with these medications.[7,8]
- Use removal strategies above.

Severe elevation ($\geq6.5\,mmol\,l^{-1}$) WITH toxic ECG changes. Seek expert help and:

- Protect the heart first 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 pulseless VT/VF but does not lower serum potassium (onset in 1–3 min).

- Use multiple shifting agents (see above).
- Use removal strategies.
- Prompt specialist referral is essential.

Patient in cardiac arrest. Modifications to BLS There are no modifications to basic life support in the presence of electrolyte abnormalities.

Modifications to ALS

- Follow the universal algorithm. Hyperkalaemia can be confirmed rapidly using a blood gas analyser if available. Protect the heart first: give 10 ml 10% calcium chloride IV by rapid bolus injection.
- Shift potassium into cells:
  ○ Glucose/insulin: 10 units short-acting insulin and 25 g glucose IV by rapid injection.
  ○ Sodium bicarbonate: 50 mmol IV by rapid injection (if severe acidosis or renal failure).
- Remove potassium from body: dialysis: consider this for cardiac arrest induced by hyperkalaemia that is resistant to medical treatment. Several dialysis modes have been used safely and effectively in cardiac arrest, but this may only be available in specialist centres.

Indications for dialysis

Haemodialysis (HD) is the most effective method for removal of potassium from the body. The principle mechanism of action is the diffusion of potassium ions across the membrane down the potassium ion gradient. The typical decline in serum potassium is 1 mmol l^{-1} in the first 60 min, followed by 1 mmol l^{-1} over the next 2 h. The efficacy of HD in decreasing serum potassium concentration can be improved by performing dialysis with a low potassium concentration in the dialysate,[9] a high blood flow rate[10] or a high dialysate bicarbonate concentration.[11]

Consider haemodialysis early for hyperkalaemia associated with established renal failure, oliguric acute kidney injury (<400 ml day^{-1} urine output) or when there is marked tissue breakdown. Dialysis is also indicated when hyperkalaemia is resistant to medical treatment. Serum potassium frequently rebounds after initial treatment. In unstable patients continuous renal replacement therapy (CRRT) (e.g., continuous veno-veno haemofiltration) is less likely to compromise cardiac output than intermittent haemodialysis. CRRT is now widely available in many intensive care units.

Cardiac arrest in haemodialysis patients

Cardiac arrest is the most common cause of death in haemodialysis patients.[12] Events occurring particularly during haemodialysis treatment, pose several novel considerations.

Initial steps. Call the resuscitation team and seek expert help immediately. Whilst BLS is underway, a trained dialysis nurse should be assigned to the dialysis machine. The conventional practice is to return the patient's blood volume and take off haemodialysis, although this approach is not the most time-efficient.[13]

Defibrillation. A shockable cardiac rhythm (VF/VT) is more common in patients undergoing haemodialysis[14,15] than in the general population.[16,17]

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.[18] An alternative and rapid disconnect technique for haemodialysis has been described. Disconnection during continuous venovenous haemofiltration is not required.[13] The use of automated external defibrillators in dialysis centres can facilitate early defibrillation.[19]

Vascular access. In life-threatening circumstances and cardiac arrest, vascular access used for haemodialysis can be used to give drugs.[13]

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

Hypokalaemia

Hypokalaemia is common in hospital patients.[20] Hypokalaemia increases the incidence of arrhythmias, particularly in patients with pre-existing heart disease and in those treated with digoxin.

Definition

Hypokalaemia is defined as a serum potassium < 3.5 mmol l^{-1}. Severe hypokalaemia is defined as a K$^+$ < 2.5 mmol l^{-1} and may be associated with symptoms.

Causes

Causes of hypokalaemia include gastrointestinal loss (diarrhoea), drugs (diuretics, laxatives, steroids), renal losses (renal tubular disorders, diabetes insipidus, dialysis), endocrine disorders (Cushing's Syndrome, hyperaldosteronism), metabolic alkalosis, magnesium depletion, and 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 occurs commonly 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 (K$^+$ < 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;
- cardiopulmonary arrest (PEA, pulseless VT/VF, 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 levels, particularly in the myocardium. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases of hypokalaemia.[21]

Table 8.1
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	Confusion	Short QT interval	Fluid replacement IV
	Malignancy	Weakness	Prolonged QRS Interval	Furosemide 1 mg kg⁻¹ IV
	Sarcoidosis	Abdominal pain	Flat T waves	Hydrocortisone 200–300 mg IV
	Drugs	Hypotension	AV-block	Pamidronate 30–90 mg IV
		Arrhythmias	Cardiac arrest	Treat underlying cause
		Cardiac arrest		
Hypocalcaemia [Calcium] < 2.1 mmol l⁻¹	Chronic renal failure	Paraesthesia	Prolonged QT interval	Calcium chloride 10% 10–40 ml
	Acute pancreatitis	Tetany	T wave inversion	Magnesium sulphate 50% 4–8 mmol (if necessary)
	Calcium channel blocker overdose	Seizures	Heart block	
	Toxic shock syndrome	AV-block	Cardiac arrest	
	Rhabdomyolysis	Cardiac arrest		
	Tumour lysis syndrome			
Hypermagnesaemia [Magnesium] > 1.1 mmol l⁻¹	Renal failure	Confusion	Prolonged PR and QT intervals	Consider treatment when [Magnesium] > 1.75 mmol l⁻¹
	Iatrogenic	Weakness	T wave peaking	
		Respiratory depression	AV-block	Calcium chloride 10% 5–10 ml repeated if necessary
		AV-block	Cardiac arrest	Ventilatory support if necessary
		Cardiac arrest		Saline diuresis – 0.9% saline with furosemide 1 mg kg⁻¹ IV Haemodialysis
Hypomagnesaemia [Magnesium] < 0.6 mmol l⁻¹	GI loss	Tremor	Prolonged PR and QT Intervals	Severe or symptomatic:
	Polyuria	Ataxia	ST-segment depression	2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 15 min.
	Starvation	Nystagmus	T-wave inversion	Torsade de pointes:
	Alcoholism	Seizures	Flattened P waves	2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 1–2 min.
	Malabsorption	Arrhythmias – torsade de pointes	Increased QRS duration	Seizure:
		Cardiac arrest	Torsade de pointes	2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 10 min.

Calcium and magnesium disorders

The recognition and management of calcium and magnesium disorders is summarised in Table 8.1.

Summary

Electrolyte abnormalities are among the most common causes of cardiac arrhythmias. Of all the electrolyte abnormalities, hyperkalaemia is most rapidly fatal. A high degree of clinical suspicion and aggressive treatment of underlying electrolyte abnormalities can prevent many patients from progressing to cardiac arrest.

8b. Poisoning

General considerations

Poisoning rarely causes cardiac arrest, but is a leading cause of death in victims younger than 40 years of age.[22] Evidence for treatment consists primarily of small case-series, animal studies and case reports. 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 harmful substances.

Prevention of cardiac arrest

Assess using the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach. Airway obstruction and respiratory arrest secondary to a decreased conscious level is a common cause of death after self-poisoning.[23] Pulmonary aspiration of gastric contents can occur after poisoning with central nervous system depressants. Early tracheal intubation of unconscious patients by a trained person decreases the risk of aspiration. Drug-induced hypotension usually responds to fluid infusion, but occasionally vasopressor support (e.g., noradrenaline infusion) is required. A long period of coma in a single position can cause pressure sores and rhabdomyolysis. Measure electrolytes (particularly potassium), blood glucose and arterial blood gases. Monitor temperature because thermoregulation is impaired. Both hypothermia and hyperthermia (hyperpyrexia) can occur after overdose of some drugs. Retain samples of blood and urine for analysis. Patients with severe poisoning should be cared for in a critical-care setting.

Interventions such as decontamination, enhanced elimination and antidotes may be indicated and are usually second line interventions.[24] Alcohol excess is often associated with self-poisoning.

Modifications to BLS/ALS

- Have a high index of personal safety where there is a suspicious cause or unexpected cardiac arrest. This is especially so when more than one casualty collapses simultaneously.

- Avoid mouth-to-mouth ventilation 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 Section 4, Advanced Life Support).[24a] This includes correction of electrolyte and acid-base abnormalities.
- 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 Sections 8d and 8e).
- Be prepared to continue resuscitation for a prolonged period, particularly in young patients, as the poison may be metabolized or excreted during extended life support measures.
- Alternative approaches which may be effective in severely poisoned patients include: higher doses of medication than in standard protocols; non-standard drug therapies; prolonged CPR.
- 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/.
- Online databases for information on toxicology and hazardous chemicals: (http://toxnet.nlm.nih.gov/).

Specific therapeutic measures

There are few specific therapeutic measures for poisoning that are useful immediately and improve outcomes.[25–29]

Therapeutic measures include decontamination, multiple-dose activated charcoal, enhancing elimination and the use of specific antidotes. 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.

Gastrointestinal decontamination

Activated charcoal adsorbs most drugs. Its benefit decreases over time after ingestion. There is no evidence that treatment with activated charcoal improves clinical outcome. Consider giving a single dose of activated charcoal to patients who have ingested a potentially toxic amount of poison (known to be adsorbed by activated charcoal) up to 1 h previously.[30] Give it only to patients with an intact or protected airway.

Multiple-dose activated charcoal significantly increases drug elimination, but no controlled study in poisoned patients has shown a reduction in morbidity and mortality and should only be used following expert advice. There is little evidence to support the use of gastric lavage. It should only be considered within 1 h of ingestion of a potentially life-threatening amount of a poison. Even then, clinical benefit has not been confirmed in controlled studies. Gastric lavage is contraindicated if the airway is not protected and if a hydrocarbon with high aspiration potential or a corrosive substance has been ingested.[27,28]

Volunteer studies show substantial decreases in the bioavailability of ingested drugs but no controlled clinical trials show that whole-bowel irrigation improves the outcome of the poisoned patient. Based on volunteer studies, whole-bowel irrigation may be considered for potentially toxic ingestions of sustained-release or enteric-coated drugs. Its use for the removal of iron, lead, zinc, or packets of illicit drugs is a theoretical option. Whole-bowel irrigation is contraindicated in patients with bowel obstruction, perforation, ileus, and haemodynamic instability.[31]

Laxatives (cathartics) or emetics (e.g., ipecacuanha) have no role in the management of the acutely poisoned patient and are not recommended.[26,32,33]

Enhancing elimination

Urine alkalinisation (urine pH of 7.5 or higher) by intravenous sodium bicarbonate infusion is a first line treatment for moderate-to-severe salicylate poisoning in patients who do not need haemodialysis.[25] Urine alkalinisation with high urine flow (approximately 600 ml h^{-1}) should also be considered in patients with severe poisoning by the herbicides 2,4-dichlorophenoxyacetic acid and methylchlorophenoxypropionic acid (mecoprop). Hypokalaemia is the most common complication of alkalaemia.

Haemodialysis or haemoperfusion should be considered in specific life-threatening poisonings only. Haemodialysis removes drugs or metabolites that are water soluble, have a low volume of distribution and low plasma protein binding. Haemoperfusion can remove substances that have a high degree of plasma protein binding

Specific poisonings

These guidelines address only some causes of cardiorespiratory arrest due to acute poisoning.

Benzodiazepines

Patients at risk of cardiac arrest
Overdose of benzodiazepines can cause loss of consciousness, respiratory depression and hypotension. Flumazenil, a competitive antagonist of benzodiazepines, should only be used only for reversal of sedation caused by a single ingestion of any of the benzodiazepines and 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.[34–36] The routine use of flumazenil in the comatose overdose patient is not recommended.

Modifications to BLS/ALS
There are no specific modifications required for cardiac arrest caused by benzodiazepines.[36–40]

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.

Patients at risk of cardiac arrest
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.[41–47] The use of naloxone can prevent the need for intubation. The preferred route for giving naloxone depends on the skills of the rescuer: IV, intramuscular (IM), subcutaneous (SC) and intranasal (IN) routes can be used. The non-IV routes can be quicker because time is saved in not having to establish IV access, which can be extremely difficult in an IV drug abuser. The initial doses of naloxone are 400 μg IV,[43] 800 μg IM, 800 μg SC,[43] or 2 mg IN.[48,49] Large opioid overdoses may require titration of naloxone to a total dose of 6–10 mg. The duration of action of naloxone is approximately 45–70 min, but respiratory depression can persist for 4–5 h after opioid overdose.

Thus, the clinical effects of naloxone may not last as long as those of a significant opioid overdose. Titrate the dose until the victim is breathing adequately and has protective airway reflexes.

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

Modifications for ALS

There are no studies supporting the use of naloxone once cardiac arrest associated with opioid toxicity has occurred. Cardiac arrest is usually secondary to a respiratory arrest and associated with severe brain hypoxia. Prognosis is poor.[42] Giving naloxone is unlikely to be harmful. Once cardiac arrest has occurred, follow standard resuscitation protocols.

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.[50–52]

Patient at risk of cardiac arrest

A widening QRS complex (>100 ms) and right axis deviation indicates a greater risk of arrhythmias.[53–55] Sodium bicarbonate should be considered for the treatment of tricyclic-induced ventricular conduction abnormalities.[56–63] While no study has investigated the optimal target arterial pH with bicarbonate therapy, a pH of 7.45–7.55 has been commonly accepted and seems reasonable.

Intravenous lipid infusions in experimental models of tricyclic toxicity have suggested benefit but there are few human data.[64,65] Anti-tricyclic antibodies have also been beneficial in experimental models of tricyclic cardiotoxicity.[66–71] One small human study[72] provided evidence of safety but clinical benefit has not been shown.

Modifications to BLS/ALS

There are no randomised controlled trials evaluating conventional versus alternative treatments for cardiac arrest caused by tricyclic toxicity. One small case-series of cardiac arrest patients, showed improvement with the use of sodium bicarbonate.[73]

Cocaine

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

Patients at risk of cardiac arrest

In patients with severe cardiovascular toxicity, alpha blockers (phentolamine),[74] benzodiazepines (lorazepam, diazepam),[75,76] calcium channel blockers (verapamil),[77] morphine,[78] and sublingual nitroglycerine[79,80] may be used as needed to control hypertension, tachycardia, myocardial ischaemia and agitation. The evidence for or against the use of beta-blocker drugs,[81–84] including those beta-blockers with alpha blocking properties (carvedilol

and labetolol).[85–87] is limited. The best choice of anti-arrhythmic drug for the treatment of cocaine-induced tachyarrhythmias is not known.

Modifications to BLS/ALS

If cardiac arrest occurs, follow standard resuscitation guidelines.[88]

Local anaesthetics

Systemic toxicity of local anaesthetics involves the central nervous system, the cardiovascular system. Severe agitation, loss of consciousness, with or without tonic–clonic convulsions, sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias can all occur. Toxicity can be potentiated in pregnancy, extremes of age, or hypoxaemia. Toxicity typically occurs in the setting of regional anaesthesia, when a bolus of local anaesthetic inadvertently enters an artery or vein.

Patients at risk of cardiac arrest

Evidence for specific treatment is limited to case reports involving cardiac arrest and severe cardiovascular toxicity and animal studies. 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 advanced life support.[89–103] Give an initial intravenous bolus of 20% lipid emulsion followed by an infusion at $15\,\mathrm{ml\,kg^{-1}\,h^{-1}}$. Give up to three bolus doses of lipid at 5-min intervals and continue the infusion until the patient is stable or has received up to a maximum of $12\,\mathrm{ml\,kg^{-1}}$ of lipid emulsion.[104]

Modifications to BLS/ALS

Standard cardiac arrests drugs (e.g., adrenaline) should be given according to standard guidelines, although animal studies provide inconsistent evidence for their role in local anaesthetic toxicity.[100,103,105–107]

Beta-blockers

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

Patients at risk of cardiac arrest

Evidence for treatment is based on case reports and animal studies. Improvement has been reported with glucagon ($50–150\,\mu\mathrm{g\,kg^{-1}}$),[108–121] high-dose insulin and glucose,[122–124] phosphodiesterase inhibitors,[125,126] calcium salts,[127] extracorporeal and intra-aortic balloon pump support,[128–130] and calcium salts.[131]

Calcium channel blockers

Calcium channel blocker overdose is emerging as a common cause of prescription drug poisoning deaths.[22,132] 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. Asymptomatic patients are unlikely to develop symptoms if the interval between the ingestion and the call is greater than 6 h for immediate-release products, 18 h for modified-release products other than verapamil, and 24 h for modified-release verapamil.

Patients at risk of cardiac arrest

Intensive cardiovascular support is needed for managing a massive calcium channel blocker overdose. While calcium chlo-

ride 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 given with glucose supplementation and electrolyte monitoring in addition to standard treatments including fluids and inotropic drugs.[133–148] Other potentially useful treatments include glucagon, vasopressin and phosphodiesterase inhibitors.[139,149]

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.

Patients at risk of cardiac arrest

Standard resuscitation measures and specific antidote therapy with digoxin-specific antibody fragments should be used if there are arrhythmias associated with haemodynamic instability.[150–163] Antibody-specific therapy may also be effective in poisoning from plants as well as Chinese herbal medications containing digitalis glycosides.[150,164,165] Digoxin-specific antibody fragments interfere with digoxin immunoassay measurements and can lead to overestimation of plasma digoxin concentrations.

Cyanide

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. 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 at risk of cardiac arrest

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, including oxygen. Initial therapy should include a cyanide scavenger (either intravenous hydroxocobalamin or a nitrite – i.e., intravenous sodium nitrite and/or inhaled amyl nitrite), followed as soon as possible by intravenous sodium thiosulphate.[166–175] Hydroxocobalamin and nitrites are equally effective but hydroxocobalamin may be safer because it does not cause methaemoglobin formation or hypotension.

Modifications to BLS/ALS

In case of cardiac arrest caused by cyanide, standard ALS treatment will fail 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 in the US in 2005.[176] Patients who develop cardiac arrest caused by carbon monoxide rarely survive to hospital discharge, even if

return of spontaneous circulation is achieved; however, hyperbaric oxygen therapy may be considered in these patients as it may reduce the risk of developing persistent or delayed neurological injury.[177–185] The risks inherent in transporting critically ill post-arrest patients to a hyperbaric facility may be significant, and must be weighed against the possibility of benefit on a case-by-case basis. Patients who develop myocardial injury caused by carbon monoxide have an increased risk of cardiac and all-cause mortality lasting at least 7 years after the event; it is reasonable to recommend cardiology follow-up for these patients.[186,187]

8c. Drowning

Overview

Drowning is a common cause of accidental death in Europe. After drowning the duration of hypoxia is the most critical factor in determining the victim's outcome; therefore, oxygenation, ventilation, and perfusion should be restored as rapidly as possible. Immediate resuscitation at the scene is essential for survival and neurological recovery after a drowning incident. This will require provision of CPR by a bystander and immediate activation of the EMS system. Victims who have spontaneous circulation and breathing when they reach hospital usually recover with good outcomes. Research into drowning is limited in comparison with primary cardiac arrest and there is a need for further research in this area.[188] These guidelines are intended for healthcare professionals and certain groups of lay responders that have a special interest in the care of the drowning victim, e.g., lifeguards.

Epidemiology

The World Health Organization (WHO) estimates that, worldwide, drowning accounts for approximately 450,000 deaths each year. A further 1.3 million disability-adjusted life-years are lost each year as a result of premature death or disability from drowning[189]; 97% of deaths from drowning occur in low- and middle-income countries.[189] In 2006 there were 312 accidental deaths from drowning in the United Kingdom[190] and 3582 in the United States,[191] yielding an annual incidence of drowning of 0.56 and 1.2 per 100,000 population, respectively.[192] Death from drowning is more common in young males, and is the leading cause of accidental death in Europe in this group.[189] Factors associated with drowning (e.g., suicide, traffic accidents, alcohol and drug abuse) varies between countries.[193]

Definitions, classifications and reporting

Over 30 different terms have been used to describe the process and outcome from submersion- and immersion-related incidents.[194] 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".[195] Immersion means to be covered in water or other fluid. For drowning to occur, usually at least the face and airway must be immersed. Submersion implies that the entire body, including the airway, is under the water or other fluid.

ILCOR recommends that the following terms, previously used, should no longer be used: dry and wet drowning, active and passive drowning, silent drowning, secondary drowning and drowned versus near-drowned.[195] The Utstein drowning style should be used

when reporting outcomes from drowning incidents to improve consistency in information between studies.[195]

Pathophysiology

The pathophysiology of drowning has been described in detail.[195,196] In brief, after submersion, the victim initially breath holds before developing laryngospasm. During this time the victim frequently swallows large quantities of water. As breath holding/laryngospasm continues, hypoxia and hypercapnia develops. Eventually these reflexes abate and the victim aspirates water into their lungs leading to worsening hypoxaemia. Without rescue and restoration of ventilation the victim will become bradycardic before sustaining a cardiac arrest. The key feature to note in the pathophysiology of drowning is that cardiac arrest occurs as a consequence of hypoxia and correction of hypoxaemia is critical to obtaining a return of spontaneous circulation.

Treatment

Treatment of a drowning victim involves four distinct but inter-related phases. These comprise (i) aquatic rescue, (ii) basic life support, (iii) advanced life support, and (iv) post-resuscitation care. Rescue and resuscitation of the drowning victim almost always involves a multi-professional team approach. The initial rescue from the water is usually undertaken either by bystanders or those with a duty to respond such as trained lifeguards or lifeboat operators. Basic life support is often provided by the initial responders before arrival of the emergency medical services. Resuscitation frequently continues into hospital where, if return of spontaneous circulation is achieved, transfer to critical care often follows. Drowning incidents vary in their complexity from an incident involving a single victim to one that involves several or multiple victims. The emergency response will vary according to the number of victims involved and available resources. If the number of victims outweighs the available resources then a system of triage to determine who to prioritise for treatment is likely to be necessary. The remainder of this section will focus on the management of the individual drowning victim where there are sufficient resources available.

Basic life support

Aquatic rescue and recovery from the water. Always be aware of personal safety and minimize the danger to yourself and the victim at all times. Whenever possible, 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. Alternatively, use a boat or other water vehicle to assist with the rescue. Avoid entry into the water whenever possible. If entry into the water is essential, take a buoyant rescue aid or flotation device.[197] 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.

Remove all drowning victims from the water by the fastest and safest means available and resuscitate as quickly as possible. The incidence of cervical spine injury in drowning victims is very low (approximately 0.5%).[198] Spinal immobilisation can be difficult to perform in the water and can delay removal from the water and adequate resuscitation of the victim. Poorly applied cervical collars can also cause airway obstruction in unconscious patients.[199] Cervical spine immobilisation is not indicated unless signs of severe injury are apparent or the history is consistent with the possibility of severe injury.[200] These circumstances include a history of diving,

water-slide use, signs of trauma or signs of alcohol intoxication. If the victim is pulseless and apnoeic remove them from the water as quickly as possible (even if a back support device is not available), while attempting to limit neck flexion and extension.

Rescue breathing. The first and most important treatment for the drowning victim is alleviation of hypoxaemia. Prompt initiation of rescue breathing or positive pressure ventilation increases survival.[201–204] If possible supplement rescue breaths/ventilations with oxygen.[205] Give five initial ventilations/rescue breaths as soon as possible.

Rescue breathing can be initiated whilst the victim is still in shallow water provided the safety of the rescuer is not compromised. It is likely to be difficult to pinch the victim's nose, so mouth-to-nose ventilation may be used as an alternative to mouth-to-mouth ventilation.

If the victim is in deep water, open their airway and if there is no spontaneous breathing start in-water rescue breathing if trained to do so. In-water resuscitation is possible,[206] but should ideally be performed with the support of a buoyant rescue aid.[207] Give 10–15 rescue breaths over approximately 1 min.[207] If normal breathing does not start spontaneously, and the victim is <5 min of from land, continue rescue breaths while towing. If more than an estimated 5 min from land, give further rescue breaths over 1 min, then bring the victim to land as quickly as possible without further attempts at ventilation.[207]

Chest compression. The victim should be placed on a firm surface before starting chest compressions as compressions are ineffective in the water.[208,209] Confirm the victim is unresponsive and not breathing normally and then give 30 chest compressions. Continue 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 less effective and should be avoided.

Automated external defibrillation. Once CPR is in progress, if an AED is available, dry the victim's chest, attach the AED pads and turn the AED on. Deliver shocks according to the AED prompts.

Regurgitation during resuscitation. Although rescue breathing is difficult to perform perfectly on a drowning victim because of the need for very high inflation pressures or the presence of fluid in the airway, every attempt should be made to continue ventilation until advanced life support providers arrive. Regurgitation of stomach contents and swallowed/inhaled water is common during resuscitation from drowning.[210] If this prevents ventilation completely, turn the victim on their side and remove the regurgitated material using directed suction if possible. Care should be taken if spinal injury is suspected but this should not prevent or delay life-saving interventions such as airway opening, ventilations and chest compressions. Abdominal thrusts can cause regurgitation of gastric contents and other life-threatening injuries and should not be used.[211]

Advanced life support

Airway and breathing. Give high-flow oxygen, ideally through an oxygen mask with reservoir bag, during the initial assessment of the spontaneously breathing drowning victim.[205] Consider non-invasive ventilation or continuous positive airway pressure if the victim fails to respond to treatment with high-flow oxygen.[212] Use pulse oximetry and arterial blood gas analysis to titrate the concentration of inspired oxygen. Consider early tracheal intubation and controlled ventilation for victims who fail to respond to these initial measures or who have a reduced level of consciousness. Take

care to ensure optimal preoxygenation before intubation. Use a rapid-sequence induction with cricoid pressure to reduce the risk of aspiration.[213] Pulmonary oedema fluid may pour from the airway and may need suctioning to enable a view of the larynx.

After the tracheal tube is confirmed in position, titrate the inspired oxygen concentration to achieve an SaO_2 of 94–98%.[205] Set positive end-expiratory pressure (PEEP) to at least 5–10 cm H_2O, however higher PEEP levels (15–20 cm H_2O) may be required if the patients is severely hypoxaemic.[214]

In the event of cardiopulmonary arrest protect the airway of the victim early in the resuscitation attempt, ideally with a cuffed tracheal tube – reduced pulmonary compliance requiring high inflation pressures may limit the use of a supraglottic airway device.

Circulation and defibrillation. Differentiating respiratory from cardiac arrest is particularly important in the drowning victim. Delaying the initiation of chest compressions if the victim is in cardiac arrest will reduce survival.

The typical post-arrest gasping is very difficult to distinguish from the initial respiratory efforts of a spontaneous recovering drowning victim. Palpation of the pulse as the sole indicator of the presence or absence of cardiac arrest is unreliable.[215] When available additional diagnostic information should be obtained from other monitoring modalities such as ECG trace, end-tidal CO_2, and echocardiography to confirm the diagnosis of cardiac arrest.

If the victim is in cardiac arrest, follow standard advanced life support protocols. If the victims core body temperature is less than 30 °C, limit defibrillation attempts to three, and withhold IV drugs until the core body temperature increases above 30 °C (see Section 8d).

During prolonged immersion, victims may become hypovolaemic from the hydrostatic pressure of the water on the body. Give IV fluid to correct hypovolaemia. After return of spontaneous circulation, use haemodynamic monitoring to guide fluid resuscitation.

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 100% certainty. Decisions made in the field frequently prove later to have been incorrect.[216] Continue resuscitation unless there is clear evidence that such attempts are futile (e.g., massive traumatic injuries, *rigor mortis*, putrefaction etc), or timely evacuation to a medical facility is not possible. Neurologically intact survival has been reported in several victims submerged for greater than 60 min however these rare case reports almost invariably occur in children submerged in ice-cold water.[217,218]

Post-resuscitation care

Salt versus fresh water. Much attention has focused in the past on differences between salt-water and fresh-water drowning. Extensive data from animal studies and human case-series have shown that, irrespective of the tonicity of the inhaled fluid, the predominant pathophysiological process is hypoxaemia, driven by surfactant wash-out and dysfunction, alveolar collapse, atelectasis, and intrapulmonary shunting. Small differences in electrolyte disturbance are rarely of any clinical relevance and do not usually require treatment.

Lung injury. Victims of drowning are at risk of developing acute respiratory distress syndrome (ARDS) after submersion.[219] 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.[220] The severity of lung injury varies from a mild self-limiting illness to refractory hypoxaemia. In severe cases extracorporeal membrane oxygenation has been used with some success.[221,222] The clinical and cost effectiveness of these interventions has not been formally tested in randomised controlled trials.

Pneumonia is common after drowning. Prophylactic antibiotics have not been shown to be of benefit,[223] although they may be considered after submersion in grossly contaminated water such as sewage. Give broad-spectrum antibiotics if signs of infection develop subsequently.[200,224]

Hypothermia after drowning. Victims of submersion may develop primary or secondary hypothermia. If the submersion occurs in icy water (<5 °C or 41 °F), hypothermia may develop rapidly and provide some protection against hypoxia. Such effects, however, have typically been reported after submersion of children in ice-cold water.[189] Hypothermia may also develop as a secondary complication of the submersion and subsequent heat loss through evaporation during attempted resuscitation (see Section 8d).

Case reports describing patients with severe accidental hypothermia have shown that survival is possible after either passive or active warming.[200] In contrast, there is evidence of benefit from induced hypothermia for comatose victims resuscitated from pre-hospital cardiac arrests.[225,226] To date, there is no convincing evidence to guide therapy in this patient group. A pragmatic approach might be to consider rewarming until a core temperature of 32–34 °C is achieved, taking care to avoid hyperthermia (>37 °C) during the subsequent period of intensive care (International Life Saving Federation, 2003).

Other supportive care. 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 been shown to alter outcome. In fact, signs of intracranial hypertension serve as a symptom of significant neurological hypoxic injury, and there is no evidence that attempts to alter the ICP will affect outcome.[200]

Follow-up. Cardiac arrhythmias may cause rapid loss of consciousness leading to drowning if the victim is in water at the time. Take a careful history in survivors of a drowning incident to identify features suggestive of arrhythmic syncope. Symptoms may include syncope (whilst supine position, during exercise, with brief prodromal symptoms, repetitive episodes or associated with palpitations), seizures or a family history of sudden death. The absence of structural heart disease at post-mortem examination does not rule the possibility of sudden cardiac death. Post-mortem genetic analysis has proved helpful in these situations and should be considered if there is uncertainty over the cause of a drowning death.[227–229]

8d. Accidental hypothermia

Definition

Accidental hypothermia exists when the body core temperature unintentionally drops below 35 °C. Hypothermia can be classified arbitrarily as mild (35–32 °C), moderate (32–28 °C) or severe (less than 28 °C).[230] The Swiss staging system[231] based on clinical signs can be used by rescuers at the scene to describe victims: stage I – clearly conscious and shivering; stage II – impaired consciousness without shivering; stage III – unconscious; stage IV – no breathing; and stage V – death due to irreversible hypothermia.

Diagnosis

Accidental hypothermia may be under-diagnosed in countries with a temperate climate. In persons with normal thermoregulation, hypothermia may develop during exposure to cold environments, particularly wet or windy conditions, and in people who have been immobilised, or following immersion in cold water. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia may follow a mild insult. The risk of hypothermia is also increased by drug or alcohol ingestion, exhaustion, illness, injury or neglect especially when there is a decrease in the level of consciousness. Hypothermia may be suspected from the clinical history or a brief external examination of a collapsed patient. A low-reading thermometer is needed to measure the core temperature and confirm the diagnosis. The core temperature measured in the lower third of the oesophagus correlates well with the temperature of the heart. Epitympanic ('tympanic') measurement – using a thermistor technique – is a reliable alternative but may be lower than the oesophageal temperature if the environmental temperature is very cold, the probe is not well insulated, the external auditory canal is blocked or during cardiac arrest when there is no flow in the carotid artery.[232] Widely available 'tympanic' thermometers based on infrared technique do not seal the ear canal and are not designed for low core temperature readings.[233] In the hospital setting, the method of temperature measurement should be the same throughout resuscitation and rewarming. Use oesophageal, bladder, rectal or tympanic temperature measurements.[234,235]

Decision to resuscitate

Cooling of the human body decreases cellular oxygen consumption by ∼6% per 1 °C decrease in core temperature.[236] At 28 °C oxygen consumption is reduced by ∼50% and at 22 °C by ∼75%. In some cases, hypothermia can exert a protective effect on the brain and vital organs[237] 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 cold alone may produce a very slow, small-volume, irregular pulse and unrecordable blood pressure. In a hypothermic patient, no signs of life (Swiss hypothermia stage IV) alone is unreliable for declaring death. At 18 °C the brain can tolerate periods of circulatory arrest for ten times longer than at 37 °C. Dilated pupils can be caused by a variety of insults and must not be regarded as a sign of death. Good quality survival has been reported after cardiac arrest and a core temperature of 13.7 °C after immersion in cold water with prolonged CPR.[238] In another case, a severely hypothermic patient was resuscitated successfully after six and a half hours of CPR.[239]

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

Resuscitation

All the principles of prevention, basic and advanced life support apply to the hypothermic patient. Use the same ventilation and chest compression rates as for a normothermic patient. Hypothermia can cause stiffness of the chest wall, making ventilation and chest compressions more difficult. Do not delay urgent procedures, such as inserting vascular catheters and tracheal intubation. The advantages of adequate oxygenation and protection from aspiration outweigh the minimal risk of triggering VF by performing tracheal intubation.[240]

Clear the airway and, if there is no spontaneous respiratory effort, ventilate the patient's lungs with high concentrations of oxygen. Consider careful tracheal intubation when indicated according to advanced life support guidelines. Palpate a central artery, look at the ECG (if available), and look for signs of life for up to 1 min before concluding that there is no cardiac output. Echocardiography or ultrasound with Doppler may be used to establish whether there is a cardiac output or peripheral blood flow. If there is any doubt about whether a pulse is present, start CPR immediately. 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 drugs given repeatedly.[241] 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, adrenaline may be effective in increasing coronary perfusion pressure, but not survival.[242,243] The efficacy of amiodarone is also reduced.[244] For these reasons, withhold adrenaline and other CPR drugs until the patient has been warmed to a temperature higher than approximately 30 °C. Once 30 °C has been reached, the intervals between drug doses should be doubled when compared with normothermia intervals. As normothermia is approached (over 35 °C), standard drug protocols should be used. Remember to rule out other primary causes of cardiorespiratory arrest using the 4 Hs and 4 Ts approach (e.g., drug overdose, hypothyroidism, trauma).

Arrhythmias

As the body core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation followed by VF and finally asystole.[245] Once in hospital, severely hypothermic victims in cardiac arrest should be rewarmed with active internal methods. Arrhythmias other than VF tend to revert spontaneously as the core temperature increases, and usually do not require immediate treatment. Bradycardia may be physiological in severe hypothermia, and cardiac pacing is not indicated unless bradycardia associated with haemodynamic compromise persists after rewarming.

The temperature at which defibrillation should first be attempted and how often it should be tried in the severely hypothermic patient has not been established. AEDs may be used on these patients. If VF is detected, give a shock at the maximum energy setting; if VF/VT persists after three shocks, delay further defibrillation attempts until the core temperature is above 30 °C.[246] If an AED is used, follow the AED prompts while rewarming the patient. CPR and rewarming may have to be continued for several hours to facilitate successful defibrillation.[246]

Rewarming

General measures for all victims include removal from the cold environment, prevention of further heat loss and rapid transfer to hospital. In the field, a patient with moderate or severe hypothermia (Swiss stages ≥ II) should be immobilised and handled carefully, oxygenated adequately, monitored (including ECG and core temperature), and the whole body dried and insulated.[241] Wet clothes should be cut off rather than stripped off; this will avoid excessive movement of the victim. Conscious victims can mobilise as exercise rewarms a person more rapidly than shivering. Exercise can increase any after-drop, i.e., further cooling after removal from a cold environment. Somnolent or comatose victims have a low threshold for developing VF or pulseless VT and should be

immobilised and kept horizontal to avoid an after-drop or cardio-vascular collapse. Adequate oxygenation is essential to stabilise the myocardium and all victims should receive supplemental oxygen. If the patient is unconscious, the airway should be protected. Pre hospital, prolonged investigation and treatments should be avoided, as further heat loss is difficult to prevent.

Rewarming may be passive, active external, or active internal. Passive rewarming is appropriate in conscious victims with mild hypothermia who are still able to shiver. This is best achieved by full body insulation with wool blankets, aluminium foil, cap and warm environment. The application of chemical heat packs to the trunk is particularly helpful in moderate and severe hypothermia to prevent further heat loss in the pre hospital setting. If the patient is unconscious and the airway is not secured, insulation should be arranged around the patient in the recovery (lateral decubitus) position. Rewarming in the field with heated intravenous fluids and warm humidified gases is not efficient. Infusing a litre of 40 °C warm fluid to a 70 kg patient at 28 °C elevates the core temperature by only about 0.3 °C.[241] Intensive active rewarming must not delay transport to a hospital where advanced rewarming techniques, continuous monitoring and observation are available. In general, alert hypothermic and shivering victims without an arrhythmia may be transported to the nearest hospital for passive rewarming and observation. Hypothermic victims with an altered consciousness should be taken to a hospital capable of active external and internal rewarming.

Several active in-hospital rewarming techniques have been described, although in a patient with stable circulation no technique has shown better survival over others. Active external rewarming techniques include forced air rewarming and warmed (up to 42 °C) intravenous fluids. These techniques are effective (rewarming rate 1–1.5 °C h^{-1}) in patients with severe hypothermia and a perfusing rhythm.[247,248] Even in severe hypothermia no significant after-drop or malignant arrhythmias have been reported. Rewarming with forced air and warm fluid has been widely implemented by clinicians because it is easy and effective. Active internal rewarming techniques include warm humidified gases; gastric, peritoneal, pleural or bladder lavage with warmed fluids (at 40 °C), and extracorporeal rewarming.[237,249–253]

In a hypothermic patient with apnoea and cardiac arrest, extracorporeal rewarming is the preferred method of active internal rewarming because it provides sufficient circulation and oxygenation while the core body temperature is increased by 8–12 °C h^{-1}.[253] Survivors in one case-series had an average of 65 min of conventional CPR before cardiopulmonary bypass,[254] which underlines that continuous CPR is essential. Unfortunately, facilities for extracorporeal rewarming are not always available and a combination of rewarming techniques may have to be used. It is advisable to contact the destination hospital well in advance of arrival to make sure that the unit can accept the patient for extracorporeal rewarming. Extracorporeal membrane oxygenation (ECMO) reduces the risk of intractable cardiorespiratory failure commonly observed after rewarming and may be a preferable extracorporeal rewarming procedure.[255]

During rewarming, patients will require large volumes of fluids as vasodilation causes expansion of the intravascular space. Continuous haemodynamic monitoring and warm IV fluids are essential. Avoid hyperthermia during and after rewarming. Although there are no formal studies, once ROSC has been achieved use standard strategies for post-resuscitation care, including mild hypothermia if appropriate (Section 4g).[24a]

Avalanche burial

In Europe and North America, there are about 150 snow avalanche deaths each year. Most are sports-related and involve skiers, snowboarders and snowmobilers. Death from avalanches is due to asphyxia, trauma and hypothermia. 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.[256] Avalanche victims are not likely to survive when they are:

- buried >35 min and in cardiac arrest with an obstructed airway on extrication;
- buried initially and in cardiac arrest with an obstructed airway on extrication, and an initial core temperature of <32°;
- buried initially and in cardiac arrest on extrication with an initial serum potassium of >12 mmol.

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

8e. Hyperthermia

Definition

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 occurs along a continuum of heat-related conditions, starting with heat stress, progressing to heat exhaustion, to heat stroke (HS) and finally multiorgan dysfunction and cardiac arrest in some instances.[257]

Malignant hyperthermia (MH) 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 depolarizing muscle relaxants.[258,259]

The key features and treatment of heat stress and heat exhaustion are included in Table 8.2.

Heat stroke

Heat stroke is a systemic inflammatory response with a core temperature above 40.6 °C, accompanied by mental state change and varying levels of organ dysfunction. There are two forms of HS: classic non-exertional heat stroke (CHS) occurs during high environmental temperatures and often effects the elderly during heat waves[260]; The 2003 heatwave in France was associated with an increased incidence of cardiac arrests in those over 60-years old.[261] Exertional heat stroke (EHS) occurs during strenuous physical exercise in high environmental temperatures and/or high humidity usually effects healthy young adults.[262] Mortality from heat stroke ranges between 10 and 50%.[263]

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 acclimatization, dehydration, obesity, alcohol, cardiovascular disease, skin conditions (psoriasis, eczema,

Table 8.2
Heat stress and heat exhaustion.

Condition	Features	Treatment
Heat stress	Normal or mild temperature elevation Heat oedema: swelling of feet and ankles Heat syncope: vasodilation causing hypotension Heat cramps: sodium depletion causing cramps	Rest Elevation of oedematous limbs Cooling Oral rehydration Salt replacement
Heat exhaustion	Systemic reaction to prolonged heat exposure (hours to days) Temperature > 37 °C and < 40 °C Headache, dizziness, nausea, vomiting, tachycardia, hypotension, sweating muscle pain, weakness and cramps Haemoconcentration Hyponatraemia or hypernatraemia May progress rapidly to heat stroke	As above Consider IV fluids and ice packs for severe cases

scleroderma, burn, cystic fibrosis), hyperthyroidism, phaeochromocytoma and drugs (anticholinergics, diamorphine, cocaine, amphetamine, phenothiazines, sympathomimetics, calcium channel blockers, beta-blockers).

Clinical presentation

Heat stroke can resemble septic shock and may be caused by similar mechanisms.[264] A single centre case-series reported 14 ICU deaths in 22 heat stroke patients admitted to ICU with multiple organ failure.[265] Features include:

- core temperature 40.6 °C or more;
- hot, dry skin (sweating is 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[266] and hypotension;
- respiratory dysfunction including ARDS[267];
- central nervous system dysfunction including seizures and coma[268];
- liver and renal failure[269];
- coagulopathy[267];
- rhabdomyolysis.[270]

Other clinical conditions need to be considered, including:

- drug toxicity[271,272];
- drug withdrawal syndrome;
- serotonin syndrome[273];
- neuroleptic malignant syndrome[274];
- sepsis[275];
- central nervous system infection;
- endocrine disorders, e.g., thyroid storm, phaeochromocytoma.[276]

Management

The mainstay of treatment is supportive therapy based on optimizing the ABCDEs and rapidly cooling the patient.[277–279] Start cooling before the patient reaches hospital. Aim to rapidly reduce the core temperature to approximately 39 °C. Patients with severe heat stroke need to be managed in a critical-care setting. Use haemodynamic monitoring to guide fluid therapy. Large volumes of fluid may be required. Correct electrolyte abnormalities as described in Section 8a.

Cooling techniques

Several cooling methods have been described, but there are few formal trials to determine which method is best. Simple cooling techniques include drinking cool 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[280]; 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 therapeutic hypothermia after cardiac arrest (see Section 4g).[24a] Cold intravenous fluids will decrease body temperature. Gastric, peritoneal,[281] pleural or bladder lavage with cold water will lower the core temperature. Intravascular cooling techniques include the use of cold IV fluids,[282] intravascular cooling catheters[283,284] and extracorporeal circuits,[285] e.g., continuous veno-veno haemofiltration or cardiopulmonary bypass.

Drug therapy in heat stroke

There are no specific drug therapies in heat stroke to 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.[286] Dantrolene (see below) has not been shown to be beneficial.[287–289]

Malignant hyperthermia

Malignant hyperthermia is a life-threatening genetic sensitivity of skeletal muscles to volatile anaesthetics and depolarizing neuromuscular blocking drugs, occurring during or after anaesthesia.[290] Stop triggering agents immediately; give oxygen, correct acidosis and electrolyte abnormalities. Start active cooling and give dantrolene.[291]

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.[292]

Modifications to cardiopulmonary resuscitation and post-resuscitation care

There are no specific studies on cardiac arrest in hyperthermia. If cardiac arrest occurs, follow standard procedures for basic

and advanced life support and cool the patient. Cooling techniques similar to those used to induce therapeutic hypothermia should be used (see Section 4g).[24a] There are no data on the effects of hyperthermia on defibrillation threshold; therefore, attempt defibrillation according to current guidelines, while continuing to cool the patient. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest.[293,294] The risk of unfavourable neurological outcome increases for each degree of body temperature >37 °C.[295] Provide post-resuscitation care according to normal guidelines.

8f. Asthma

Introduction

Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma.[296] The worldwide prevalence of asthma symptoms ranges from 1 to 18% of the population with a high prevalence in some European countries (United Kingdom, Ireland and Scandinavia).[296] International differences in asthma symptom prevalence appears to be decreasing in recent years, especially in adolescents.[297] The World Health Organisation 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.[296] National and international guidance for the management of asthma already exists.[296,298] This guidance focuses on the treatment of patients with near-fatal asthma and cardiac arrest.

Patients at risk of asthma-related cardiac arrest

The risk of near-fatal asthma attacks is not necessarily related to asthma severity.[299] Patients most at risk include those with:

- a history of near-fatal asthma requiring intubation and mechanical ventilation;
- a hospitalisation or emergency care for asthma in the past year[300];
- low or no use of inhaled corticosteroids[301];
- an increasing use and dependence of beta-2 agonists[302];
- anxiety, depressive disorders and/or poor compliance with therapy.[303]

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.[304] 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. SaO_2 may not reflect progressive alveolar hypoventilation, particularly if oxygen is being given. The SaO_2 may initially decrease during therapy because beta-agonists cause both bronchodilation and vasodilation and may initially increase intrapulmonary shunting.

Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis,[305] pneumonia, foreign bodies, pulmonary embolism, bronchiectasis and subglottic mass.[306]

The severity of an asthma attack is defined in Table 8.3.

Key interventions to prevent arrest

The patient with severe asthma requires aggressive medical management to prevent deterioration. Base assessment and treatment on an ABCDE approach. Patients with $SaO_2 < 92\%$ 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 SaO_2 94–98%.[205] High-flow oxygen by mask is sometimes necessary.

Table 8.3
The severity of asthma.

Asthma	Features
Near-fatal	Raised $PaCO_2$ and/or requiring mechanical ventilation with raised inflation pressures
Life-threatening	Any one of: PEF < 33% best or predicted bradycardia $SpO_2 < 92\%$, dysrhythmia $PaO_2 < 8$ kPa, hypotension Normal $PaCO_2$ (4.6–6.0 kPa (35–45 mmHg)), exhaustion Silent chest, confusion Cyanosis, coma Feeble respiratory effort
Acute severe	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
Moderate exacerbation	Increasing symptoms PEF > 50–75% best or predicted No features of acute severe asthma
Brittle	Type 1: wide PEF variability (>40% diurnal variation for >50% of the time over a period >150 days) despite intense therapy Type 2: sudden severe attacks on a background of apparently well controlled asthma

PEF, peak expiratory flow.

Nebulised beta-2 agonists

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 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.[298,307] Nebulised adrenaline does not provide additional benefit over and above nebulised beta-2 agonists in acute asthma.[308]

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.[309] Although there is no difference in clinical effects between oral and IV formulations of corticosteroids,[310] the IV route is preferable because patients with near-fatal asthma may vomit or be unable to swallow.

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.[311,312]

Nebulised magnesium sulphate

Results of small randomised controlled trials showed that a nebulised isotonic solution of magnesium sulphate ($250 \, \mathrm{mmol \, l^{-1}}$) in a volume of 2.5–5 ml in combination with beta-2 agonists is safe and it is associated with both an improvement of pulmonary function tests and a non-significant trend towards lower rates of hospital admission in patients with acute severe asthma.[313] Further studies are needed to confirm those findings.

Intravenous bronchodilators

Nebulised bronchodilators are the first line treatment for acute severe and life-threatening exacerbations of asthma. There is a lack of definitive evidence for or against the use of intravenous 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 intravenous 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).

Intravenous magnesium sulphate

Studies of intravenous magnesium sulphate in acute severe and life-threatening asthma have produced conflicting results.[314,315] Magnesium sulphate causes bronchial smooth muscle relaxation independent of the serum magnesium level and has only minor side effects (flushing, light-headedness). Given the low risk of serious side effects from magnesium sulphate it would seem reasonable to use intravenous magnesium sulphate (1.2–2 g IV slowly) in adults with life-threatening unresponsive to nebulised therapy. The multicentre randomised controlled trial 3Mg (ISRCTN04417063) is due to report in 2012 and should provide definitive evidence on the role of magnesium in acute severe asthma.

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.[316,317] 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 a loading dose of $5 \, \mathrm{mg \, kg^{-1}}$ is given over 20–30 min (unless on maintenance therapy), followed by an infusion of 500–$700 \, \mathrm{\mu g \, kg^{-1} \, h^{-1}}$. Serum theophylline concentrations should be maintained below $20 \, \mathrm{\mu g \, ml^{-1}}$ to avoid toxicity.

Beta-2 agonists

A Cochrane review on 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.[318] Salbutamol may be given as either a slow IV injection ($250 \, \mathrm{\mu g}$ IV slowly) or continuous infusion of 3–$20 \, \mathrm{\mu g \, min^{-1}}$.

Leukotriene receptor antagonists

There are few data on the use of intravenous leukotriene receptor antagonists.[319] Further studies are required to confirm the findings of a recent randomised controlled trial which demonstrated evidence of additional bronchodilation when intravenous LRTA montelukast was used as a rescue therapy.[320]

Subcutaneous or intramuscular adrenaline and terbutaline

Adrenaline and terbutaline are adrenergic agents that may be given subcutaneously to patients with acute severe asthma. The dose of subcutaneous adrenaline is $300 \, \mathrm{\mu g}$ up to a total of 3 doses at 20-min intervals. Adrenaline may cause an increase in heart rate, myocardial irritability and increased oxygen demand; however, its use (even in patients over 35-years old) is well tolerated.[321] Terbutaline is given in a dose of $250 \, \mathrm{\mu g}$ subcutaneously, which can be repeated in 30–60 min. These drugs are more commonly given to children with acute asthma and, although most studies have shown them to be equally effective,[322] one study concluded that terbutaline was superior.[323] These alternative routes may need to be considered when IV access is impossible. Patients with asthma are at increased risk of anaphylaxis. Sometimes it may be difficult to distinguish severe life-threatening asthma from anaphylaxis. In these circumstances intramuscular adrenaline given according to the anaphylaxis guidelines may be appropriate (Section 8g).

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 fluids. Beta-2 agonists and steroids may induce hypokalaemia, which should be corrected with electrolyte supplements.

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.[324]

Referral to intensive care

Patients that fail to respond to initial treatment, or develop signs of life-threatening asthma, should be assessed by an intensive care specialist. Admission to intensive care after asthma-related cardiac arrest is associated with significantly poorer outcomes than if cardiac arrest does not occur.[325]

Rapid sequence induction and tracheal intubation should be considered if, despite efforts to optimize drug therapy, the patient has:

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

Elevation of the $PaCO_2$ alone does not indicate the need for tracheal intubation.[326] Treat the patient, not the numbers.

Non-invasive ventilation

Non-invasive ventilation decreases the intubation rate and mortality in COPD[327]; however, its role in patients with severe acute asthma is uncertain. There is insufficient evidence to recommend its routine use in asthma.[328]

Treatment of cardiac arrest

Basic life support

Give basic life support 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 cm H_2O in 12 patients) are significantly higher than the normal lower oesophageal sphincter pressure (approximately 20 cm H_2O).[329] 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.[330]

Respiratory rates of 8–10 breaths min^{-1} and tidal volume required for a normal chest rise during CPR should not cause dynamic hyperinflation of the lungs (gas 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.[329]

There is limited evidence from case reports of unexpected ROSC in patients with suspected gas trapping when the tracheal tube is disconnected.[331–335] If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest wall and/or a period of apnoea (disconnection of tracheal tube) may relieve gas trapping if dynamic hyperinflation occurs. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.[336]

Dynamic hyperinflation increases transthoracic impedance.[337] Consider higher shock energies for defibrillation if initial defibrillation attempts fail.[338]

There is no good evidence for the use of open-chest cardiac compressions in patients with asthma-associated cardiac arrest. Working through the 4 Hs and 4 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.[339] If pneumothorax is suspected, release air from the pleural space with needle decompression. Insert a large-gauge cannula in the second intercostal space, above the rib, in the mid-clavicular line, being careful to avoid direct puncture of the lung. If air is emitted, insert a chest tube. Always consider bilateral pneumothoraces in asthma-related cardiac arrest.

Extracorporeal life support (ECLS) can ensure both organ perfusion and gas exchange in case of otherwise untreatable respiratory and circulatory failure. Cases of successful treatment of asthma-related cardiac arrest in adults using ECLS have been reported[340,341]; 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 which may not be available everywhere.

8g. Anaphylaxis

Definition of anaphylaxis

A precise definition of anaphylaxis is not important for its emergency treatment. There is no universally agreed definition. The European Academy of Allergology and Clinical Immunology Nomenclature Committee proposed the following broad definition[342]: 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.[305,343]

Anaphylaxis usually involves the release of inflammatory mediators from mast cells and, or basophils triggered by an allergen interacting with cell-bound immunoglobulin E (IgE). Non-IgE-mediated or non-immune release of mediators can also occur. Histamine and other inflammatory mediator release are responsible for the vasodilatation, oedema and increased capillary permeability.

Epidemiology

The overall frequency of episodes of anaphylaxis using current data lies between 30 and 950 cases per 100,000 persons per year and a lifetime prevalence of between 50 and 2000 episodes per 100,000 persons or 0.05–2.0%.[344] Anaphylaxis can be triggered by any of a very broad range of triggers including foods, drugs, stinging insects, and latex. Food is the commonest trigger in children and drugs the commonest in adults.[345] Virtually any food or drug can be implicated, but certain foods (nuts) and drugs (muscle relaxants, antibiotics, NSAIDs and aspirin) cause most reactions.[346] A significant number of cases of anaphylaxis are idiopathic.

The overall prognosis of anaphylaxis is good, with a case fatality ratio of less than 1% reported in most population-based studies. 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 with adrenaline.[347,348] 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 after 30–35 min; insect stings cause collapse from shock after 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.

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.

Many patients with anaphylaxis are not given the correct treatment.[349] Confusion arises because some patients have systemic allergic reactions that are less severe. For example, generalised urticaria, angioedema, and rhinitis would not be described as anaphylaxis, because the life-threatening features are not present. Anaphylaxis guidelines must therefore take into account some inevitable diagnostic errors, with an emphasis on the need for safety. Patients can have either an airway and/or breathing and/or circulation problem:

Airway problems

- Airway swelling, e.g., throat and tongue swelling (pharyngeal/laryngeal oedema).
- Hoarse voice.
- Stridor.

Breathing problems

- Shortness of breath.
- Wheeze.
- Confusion caused by hypoxia.
- Respiratory arrest.
- Life-threatening asthma with no features of anaphylaxis can be triggered by food allergy.[350]

Circulation problems

- Pale, clammy.
- Tachycardia.
- Hypotension.
- Decreased conscious level.
- Myocardial ischaemia and electrocardiograph (ECG) changes even in individuals with normal coronary arteries.[351]
- Cardiac arrest.

Circulation problems (often referred to as anaphylactic shock) can be caused by direct myocardial depression, vasodilation and capillary leak, and loss of fluid from the circulation. Bradycardia is usually a late feature, often preceding cardiac arrest.[352]

Skin and, or mucosal changes

These should be assessed as part of the exposure when using the ABCDE approach.

- They are often the first feature and present in over 80% of anaphylaxis cases.[353]
- They can be subtle or dramatic.
- There may be just skin, just mucosal, or both skin and mucosal changes any where on the body.

- There may be erythema, urticaria (also called hives, nettle rash, weals or welts), or angioedema (eyelids, lips, and sometimes in the mouth and throat).

Most patients who have skin changes caused by allergy do not go on to develop anaphylaxis.

Treatment of an anaphylaxis

Use an ABCDE approach to recognise and treat anaphylaxis. Treat life-threatening problems as you find them. The basic principles of treatment are the same for all age groups. All patients who have suspected anaphylaxis should be monitored (e.g., by ambulance crew, in the emergency department etc,) as soon as possible. Minimal monitoring includes pulse oximetry, non-invasive blood pressure and 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.[354] 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 (circulation problem).

Remove the trigger if possible

Stop any drug suspected of causing anaphylaxis. Remove the stinger after a bee sting. Early removal is more important than the method of removal.[355] Do not delay definitive treatment if removing the trigger is not feasible.

Cardiorespiratory arrest following an anaphylaxis

Start cardiopulmonary resuscitation (CPR) immediately and follow current guidelines. Prolonged CPR may be necessary. Rescuers should ensure that help is on its way as early advanced life support (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. Call for expert help early.

Drugs and their delivery

Adrenaline (epinephrine)

Adrenaline is the most important drug for the treatment of anaphylaxis.[356,357] Although there are no randomised controlled trials,[358] adrenaline is a logical treatment and there is consistent anecdotal evidence supporting its use to ease breathing and circulation problems associated with anaphylaxis. As an 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. There are beta-2 adrenergic receptors on mast cells that inhibit activation, and so early adrenaline attenuates the severity of IgE-mediated allergic reactions. Adrenaline seems to work best when given early after the onset of the reaction[359] but it is not without risk, particularly when given intravenously. Adverse effects are extremely rare with correct doses injected intramuscularly (IM).

Adrenaline should be given 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 (IM) 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, and 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.

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.[360] The subcutaneous or inhaled routes for adrenaline are not recommended for the treatment of anaphylaxis because they are less effective than the IM route.[361–363]

Adrenaline IM dose. The evidence for the recommended doses is weak. Doses are based on what is considered to be safe and practical to draw up and inject in an emergency.
(The equivalent volume of 1:1000 adrenaline is shown in brackets)

>12 years and adults:	500 µg IM (0.5 ml)
>6–12 years:	300 µg IM (0.3 ml)
>6 months–6 years:	150 µg IM (0.15 ml)
<6 months:	150 µg IM (0.15 ml)

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

Intravenous (IV) 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 IV adrenaline.[364] Intravenous adrenaline should be used only 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, intravenous 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 IV bolus dose – adult. Titrate IV adrenaline using 50 µg boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion.[352,365]

Adrenaline IV bolus dose – 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 µg kg^{-1}. This requires very careful dilution and checking to prevent dose errors.

Oxygen (give as soon as available)
Initially, give the highest concentration of oxygen possible using a mask with an oxygen reservoir.[205] Ensure high-flow oxygen (usually greater than 10 litres 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 there is intravenous access, infuse intravenous 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 intravenous access is delayed or impossible, the intra-osseous route can be used for fluids or drugs. Do not delay the administration of IM adrenaline attempting intra-osseous access.

Antihistamines (after initial resuscitation)
Antihistamines are a second line treatment for anaphylaxis. The evidence to support their use is weak, but there are logical reasons for them.[366] Antihistamines (H$_1$-antihistamine) help counter histamine-mediated vasodilation and bronchoconstriction. There is little evidence to support the routine use of an H$_2$-antihistamine (e.g., ranitidine, cimetidine) for the initial treatment of an anaphylaxis.

Steroids (give after initial resuscitation)
Corticosteroids may help prevent or shorten protracted reactions although the evidence is very limited.[367] 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 a severe anaphylaxis and life-threatening asthma can be the same. Consider further bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), aminophyline (IV) or magnesium (IV) (see Section 8f above). Intravenous 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.[368–380] Use these drugs only in specialist settings (e.g., intensive care units) where there is experience in their use. Glucagon can be useful to treat anaphylaxis in a patient taking a beta-blocker.[381] Some case reports of cardiac arrest suggest cardiopulmonary bypass [382,383] or mechanical support of circulation[384] may also be helpful.

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.[385] 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.[386]

Discharge and follow-up

Patients who have had suspected anaphylaxis (i.e., an airway, breathing or circulation (ABC) 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 an anaphylaxis and whether the initial treatment was appropriate.[387] 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 a senior clinician.
- Given clear instructions to return to hospital if symptoms return.
- Considered for antihistamines and oral steroids therapy for up to 3 days. This is helpful for treatment of urticaria and may decrease the chance of further reaction.
- Considered for an adrenaline auto-injector, or given a replacement.[388–390]
- Have a plan for follow-up, including contact with the patient's general practitioner.

An adrenaline auto-injector is an appropriate treatment for patients at increased risk of idiopathic anaphylaxis, or for anyone at continued high risk of reaction, e.g., to triggers such as venom stings and food-induced reactions (unless easy to avoid). An auto-injector is not usually necessary for patients who have suffered drug-induced anaphylaxis, unless it is difficult to avoid the drug. Ideally, all patients should be assessed by an allergy specialist and have a treatment plan based on their individual risk. Individuals provided with an auto-injector on discharge from hospital must be given instructions and training on when and how to use it. Ensure appropriate follow-up including contact with the patient's general practitioner. All patients presenting with anaphylaxis should be referred to an allergy clinic to identify the cause, and thereby reduce the risk of future reactions and prepare the patient to manage future episodes themselves. Patients need to know the allergen responsible 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.[391]

8h. Cardiac arrest following cardiac surgery

Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase, with a reported incidence of 0.7–2.9%.[392–400] It is usually preceded by physiological deterioration,[401] although it can occur suddenly in stable patients.[398] 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. If cardiac arrest occurs during the first 24 h after cardiac surgery, the rate of survival to hospital discharge is 54%[399] to 79%[398,402] in adults and 41% in children.[401]

Key to the successful resuscitation of cardiac arrest in these patients is the need to perform emergency resternotomy early, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective.

Identification of cardiac arrest

Patients in the ICU are highly monitored and an arrest is most likely to be signalled by monitoring alarms where absence of pulsation or perfusing pressure on the arterial line, loss of pulse oximeter, pulmonary artery (PA) trace, or end-tidal CO_2 trace can be sufficient to indicate cardiac arrest without the need to palpate a central pulse.

Starting CPR

Start external chest compressions immediately in all patients who collapse without an output. Consider reversible causes: hypoxia – check tube position, ventilate with 100% oxygen; tension pneumothorax – clinical examination, thoracic ultrasound; hypovolaemia, pacing failure. In asystole, secondary to a loss of cardiac pacing, external massage 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). The effectiveness of compressions may be verified by looking at the arterial trace, aiming to achieve a systolic blood pressure of at least 80 mmHg at a rate of $100\,min^{-1}$. Inability to attain this pressure may indicate tamponade, tension pneumothorax, or exanguinating haemorrhage and should precipitate emergency resternotomy. Intra-aortic balloon pumps should be changed to pressure triggering during CPR. In PEA, switch off the pacemaker – a temporary pacemaker may potentially hide underlying VF.

Defibrillation

There is concern that external chest compressions can cause sternal disruption or cardiac damage.[403–406] In the post-cardiac surgery ICU, a witnessed and monitored VF/VT 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.

Emergency drugs

Use adrenaline very cautiously and titrate to effect (intravenous doses of 100 or less micrograms in adults). In order to exclude a medication error as the cause of the arrest, stop all drug infusions and check if they are correct. If there is concern about patient awareness, restart the anaesthetic drugs. Atropine is no longer recommended for the treatment of cardiac arrest as there is little evidence to show it is effective in patients who have been given adrenaline. Individual clinicians may use atropine at their discretion in post-cardiac surgery cardiac arrest if they feel it is indicated. Treat bradycardia with atropine, according to the bradycardia algorithm (see Section 4 Advanced Life Support).[24a]

Give amiodarone 300 mg after the 3rd failed defibrillation attempt but do not delay resternotomy. An irritable myocardium following cardiac surgery is caused most commonly by myocardial ischaemia and correction of this, rather than giving amiodarone, is more likely to achieve myocardial stability.

Emergency resternotomy

This is an integral part of resuscitation after cardiac surgery, once all other reversible causes have been excluded. Once adequate an airway and ventilation has been established, and if three attempts at defibrillation have failed in VF/VT, undertake resternotomy without delay. Emergency resternotomy is also indicated in asystole or PEA, when other treatments have failed. Resuscitation teams should be well rehearsed in this technique so that it can be performed safely within 5 min of the onset of cardiac arrest. Resternotomy equipment should be prepared as soon as an arrest is identified. Simplification of the resternotomy tray and regular manikin rehearsals are key measures to ensure a prompt resternotomy.[407,408] All medical members of the patient care team should be trained to perform resternotomy if a surgeon is not available within 5 min. Improved survival and better quality of life is well documented with rapid resternotomy.[394,395,409]

Resternotomy should be a standard part of resuscitation within 10 days after cardiac surgery. The overall survival to discharge following internal cardiac massage is 17%[394] to 25%[395] although survival rates are much lower when chest opening is performed outside the specialised environment of the post-cardiac surgery ICU.[395]

Reinstitution of emergency cardiopulmonary bypass

The need for emergency cardiopulmonary bypass (CPB) occurs in approximately 0.8% patients at a mean of 7 h post-operatively[396] and is usually indicated to correct surgical bleeding or graft occlusion and rest the myocardium. Emergency institution of CPB should be available on all units undertaking cardiac surgery. Survival to discharge rates of 32%,[395] 42%[396] and 56.3%[410] have been reported when CPB is reinstituted on the ICU.

Survival rates decline rapidly when this procedure is undertaken more than 24 h after surgery and when performed on the ward rather than the ICU. Emergency CPB should probably be restricted to patients who arrest within 72 h of surgery, as surgically remediable problems are unlikely after this time.[395] Ensuring adequate anticoagulation before starting CPB, or the use of a heparin-bonded circuit, is important. The need for a further period of cross-clamping does not preclude a favourable outcome.[396]

Patients with non-sternotomy cardiac surgery

These guidelines are appropriate for patients following non-sternotomy cardiac surgery, but surgeons performing these operations should have already given clear instructions for chest reopening. Patients undergoing port-access mitral procedures or minimally invasive coronary bypass graft surgery are likely to require an emergency sternotomy, as very poor access is obtained by opening or extending a mini-thoracotomy incision. Equipment and guidelines should be kept close to the patient.

Children

The incidence of cardiac arrest after cardiac surgery in children is 4%[411] and survival rates are similar to those of adults. The causes are also similar, although one case-series documented primary respiratory arrest in 11%. The guidance given in this section is equally applicable to children, with appropriate modification of defibrillation energy and drug doses (see Section 6 Paediatric Life Support).[411a] Use extreme caution and check doses carefully when giving intravenous adrenaline doses to children in cardiac arrest after cardiac surgery. Use smaller doses of adrenaline in this setting (e.g., 1 μg kg^{-1}) under the guidance of experienced clinicians.

Internal defibrillation

Internal defibrillation using paddles applied directly across the ventricles requires considerably less energy than that used for external defibrillation. Biphasic shocks are more effective than monophasic shocks for direct defibrillation.[412] For biphasic shocks, starting at 5 J creates the optimum conditions for lowest threshold and cumulative energy, whereas 10–20 J offers optimum conditions for more rapid defibrillation and fewer shocks,[412] thus 20 J is the most applicable energy in an arrest situation, whereas 5 J would be adequate if the patient has been placed on cardiopulmonary bypass.

Continuing cardiac compressions using the internal paddles whilst charging the defibrillator and delivering the shock during the decompression phase of compressions may improve shock success.[413,414]

It is acceptable to perform external defibrillation after emergency resternotomy. Apply external pads preoperatively to all patients undergoing resternotomy surgery.[415] Use the defibrillation energy level indicated in the universal algorithm. If the sternum is widely open the impedence may be significantly increased – if external defibrillation is chosen over internal defibrillation close the sternal retractor before shock delivery.

8i. Traumatic cardiorespiratory arrest

Introduction

Cardiac arrest caused by trauma has a very high mortality, with an overall survival of just 5.6% (range 0–17%) (Table 8.4).[416–422] For reasons that are unclear, reported survival rates in the last 5 years are better than reported previously (Table 8.4) In those who survive (and where data are available) neurological outcome is good in only 1.6% of those sustaining traumatic cardiorespiratory arrest (TCRA).

Diagnosis of traumatic cardiorespiratory arrest

The diagnosis of TCRA is made clinically: the trauma patient is unresponsive, apnoeic and pulseless. Both asystole and organised cardiac activity without cardiac output are regarded as TCRA.

Table 8.4
Survival after out of hospital traumatic cardiac arrest.

Source	Entry criteria: children or adults requiring CPR before or on hospital admission	Number of patients/survivors/ good neurological outcome	Penetrating/ survivors/good neurological outcome	Blunt/survivors/ good neurological outcome
Shimazu and Shatney[417]	TCRA on admission	267 7 4		
Rosemurgy et al.[416]	CPR before admission	138 0 0	42 0 0	96 0 0
Bouillon et al.[429]	CPR on scene	224 4 3		
Battistella et al.[418]	CPR at scene, en route or in the ED	604 16 9	300 12 9	304 4 0
Fisher and Worthen[430]	Children requiring CPR before or on admission after blunt trauma	65 1 0		65 1 0
Hazinski et al.[431]	Children requiring CPR or being severely hypotensive on admission after blunt trauma	38 1 0		38 1 0
Stratton et al.[422]	Unconscious, pulseless at scene	879 9 3	497 4 3	382 5 0
Calkins et al.[432]	Children requiring CPR after blunt trauma	25 2 2		25 2 2
Yanagawa et al.[433]	OHCA in blunt trauma	332 6 0		332 6 0
Pickens et al.[434]	CPR on scene	184 14 9	94 9 5	90 5 4
Di Bartolomeo et al.[435]	CPR on scene	129 2 0		
Willis et al.[436]	CPR on scene	89 4 4	18 2 2	71 2 2
David et al.[437]	CPR on scene	268 5 1		
Crewdson et al.[438]	80 children who required CPR on scene after trauma	80 7 3	7 0 0	73 7 3
Huber-Wagner et al.[439]	CPR on scene or after arrival	757 130 28	43 ? ?	714 ? ?
Pasquale et al.[421]	CPR before or on hospital admission	106 3	21 1	85 2
Lockey et al.[440]	CPR on scene	871 68	114 9	757 59
Cera et al.[441]	CPR on admission	161 15		
Totals		5217 293 (5.6%)	1136 37 (3.3%)	3032 94 (3.1%)

Commotio cordis

Commotio cordis is actual or near cardiac arrest caused by a blunt impact to the chest wall over the heart.[423–427] A blow to the chest during the vulnerable phase of the cardiac cycle may cause malignant arrhythmias (usually ventricular fibrillation). Syncope after chest wall impact may be caused by non-sustained arrhythmic events. Commotio cordis occurs mostly during sports (most commonly baseball) and recreational activities and victims are usually young males (mean age 14 years). In a series of 1866 cardiac arrests

in athletes in Minneapolis, 65 (3%) were due to commotio cordis.[428] The registry is accruing 5–15 cases of commotio cordis each year. The overall survival rate from commotio cordis is 15%, but 25% if resuscitation is started within 3 min.[427]

Trauma secondary to medical events

A cardiorespiratory arrest due to a medical condition (e.g., cardiac arrhythmia, hypoglycaemia, seizure) can cause a secondary traumatic event (e.g., fall, road traffic accident etc). Despite the initial reported mechanism, traumatic injuries may not be the primary cause of a cardiorespiratory arrest and standard advanced life support, including chest compressions, may be appropriate.

Mechanism of injury

Blunt trauma

Of 3032 patients with cardiac arrest after blunt trauma, 94 (3.1%) survived. Only 15 out of 1476 patients (1%) were reported to have a good neurological outcome (Table 8.4).

Penetrating trauma

Of 1136 patients with cardiac arrest after penetrating injury, there were 37 (3.3%) survivors 19 of which (1.9%) had a good neurological outcome (Table 8.4).

A confounding factor in both blunt and penetrating trauma survival rates is that some studies report survival including those pronounced dead on scene and others do not.

Signs of life and initial ECG activity

There are no reliable predictors of survival for TCRA. One study reported that the presence of reactive pupils and sinus rhythm correlate significantly with survival.[441] In a study of penetrating trauma, pupil reactivity, respiratory activity and sinus rhythm were correlated with survival but were unreliable.[422] Three studies reported no survivors in patients presenting with asystole or agonal rhythms.[418,422,442] Another reported no survivors in PEA after blunt trauma.[443] Based on these studies, the American College of Surgeons and the National Association of EMS physicians produced pre-hospital guidelines on withholding resuscitation.[444] They recommend withholding resuscitation in:

(i) blunt trauma patients presenting with apnoea, pulselessness and without organised ECG activity;
(ii) penetrating trauma patients found apnoeic and pulseless after rapid assessment for signs of life such as pupillary reflexes, spontaneous movement, or organised ECG activity.

Three retrospective studies question these recommendations and report survivors who would have had resuscitation withheld if the guidelines had been followed.[434,436,440]

Treatment

Survival from TCRA is correlated with duration of CPR and pre-hospital time.[420,445–449] Prolonged CPR is associated with a poor outcome; the maximum CPR time associated with favourable outcome is 16 min.[420,445–447] The level of pre-hospital intervention will depend on the skills of local EMS providers, but treatment on scene should focus on good quality BLS and ALS and exclusion of reversible causes. Look for and treat any medical condition that may have precipitated the trauma event. Undertake only essential life-saving interventions on scene and, if the patient has signs of life, transfer rapidly to the nearest appropriate hospital. Consider on scene thoracostomy for appropriate patients.[450,451] Do not delay for unproven interventions such as spinal immobilisation.[452]

1. Treatment of reversible causes:

- Hypoxaemia (oxygenation, ventilation).
- Compressible haemorrhage (pressure, pressure dressings, tourniquets, novel haemostatic agents).
- Non-compressible haemorrhage (splints, intravenous fluid).
- Tension pneumothorax (chest decompression).
- Cardiac tamponade (immediate thoracotomy)

2. Chest compressions: although they may not be effective in hypovolaemic cardiac arrest most survivors do not have hypovolaemia and in this subgroup standard advanced life support may be life-saving.

3. Standard CPR should not delay the treatment of reversible causes (e.g., thoracotomy for cardiac tamponade).

Resuscitative thoracotomy

Pre-hospital

Resuscitative thoracotomy has been reported as futile if out of hospital time has exceeded 30 min[448]; others consider thoracotomy to be futile in patients with blunt trauma requiring more than 5 min of pre-hospital CPR and in patients with penetrating trauma requiring more than 15 min of CPR.[449] With these time limits in mind, 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 thoracotomy should be considered.[450] Based on this approach, of 71 patients who underwent thoracotomy at scene, thirteen patients survived and eleven of these made a good neurological recovery.[453] In contrast, pre-hospital thoracotomy for 34 patients with *blunt* trauma in Japan has not produced any survivors.[454]

Hospital

A relatively simple technique for resuscitative thoracotomy has been described recently.[451,455]

The American College of Surgeons has published practice guidelines for emergency department thoracotomy (EDT) based on a meta-analysis of 42 outcome studies including 7035 EDT's.[456] The overall survival rate was 7.8% and, of 226 survivors (5%), only 34 (15%) had a neurological deficit. The investigators concluded that EDT:

1. After blunt trauma, should be limited to those with vital signs on arrival and a witnessed cardiac arrest (estimated survival rate 1.6%).
2. Is best applied to patients with penetrating cardiac injuries who arrive at the trauma centre after a short on scene and transport time with witnessed signs of life or ECG activity (estimated survival rate 31%).
3. Should be undertaken in penetrating non-cardiac thoracic injuries even though survival rates are low.
4. Should be undertaken in patients with exsanguinating abdominal vascular injury even though survival rates are low. This procedure should be used as an adjunct to definitive repair of abdominal vascular injury.

One European study reports a survival rate of 10% in blunt trauma patients undergoing EDT within twenty min after witnessed cardiac arrest. Three of the four survivors had intrabdominal haemorrhage. They conclude that in moribund patients with blunt chest or abdominal trauma EDT should be performed as early as possible.[457]

Airway management

Effective airway management is essential to maintain oxygenation of the severely compromised trauma patient. In one study, tracheal intubation on scene of patients with TCRA doubled the tolerated period of CPR before emergency department thoracotomy – the mean duration of CPR for survivors who were intubated in the field was 9.1 versus 4.2 min for those were not intubated.[447]

Tracheal intubation in trauma patients is a difficult procedure with a high failure rate if carried out by less experienced care providers.[458–462] Use basic airway management manoeuvres and alternative airways to maintain oxygenation if tracheal intubation cannot be accomplished immediately. If these measures fail a surgical airway is indicated.

Ventilation

In low cardiac output conditions, positive pressure ventilation causes further circulatory depression, or even cardiac arrest, by impeding venous return to the heart.[463] Monitor ventilation with continuous waveform capnography and adjust to achieve normocapnia. This may enable slow respiratory rates and low tidal volumes and the corresponding decrease in transpulmonary pressure may increase venous return and cardiac output.

Chest decompression

Effective decompression of a tension pneumothorax can be achieved quickly by lateral or anterior thoracostomy, which, in the presence of positive pressure ventilation, is likely to be more effective than needle thoracostomy and quicker than inserting a chest tube.[464]

Effectiveness of chest compressions in TCRA

In hypovolaemic cardiac arrest, chest compressions are unlikely to be as effective as in cardiac arrest from other causes.[465] However most survivors of TCRA have reasons other than pure hypovolaemia for their arrest and these patients may benefit from standard advanced life support interventions.[436,438,440] Patients with cardiac tamponade are also less likely to benefit from chest compressions and should, where possible, have immediate surgical release of tamponade. Return of spontaneous circulation with advanced life support in patients with TCRA has been described and chest compressions are still the standard of care in patients with cardiac arrest irrespective of aetiology.

Haemorrhage control

Early haemorrhage control is vital. Handle the patient gently at all times to prevent clot disruption. Apply external compression, and pelvic and limb splints when appropriate. Delays in surgical haemostasis are disastrous for patients with exsanguinating trauma. Recent conflicts have seen a resurgence in the use of tourniquets to stop life-threatening limb haemorrhage.[466] It is unlikely that the same benefits will be seen in civilian trauma practice.

Pericardiocentesis

In patients with suspected trauma-related cardiac tamponade, needle pericardiocentesis is probably not a useful procedure.[467] There is no evidence of benefit in the literature. It may increase scene time, can cause myocardial injury and delays effective therapeutic measures such as emergency thoracotomy.

Fluids and blood transfusion on scene

Fluid resuscitation of trauma patients before haemorrhage is controlled is controversial and there is no clear consensus on when it should be started and what fluids should be given.[468,469] Limited evidence and general consensus support a more conservative approach to intravenous fluid infusion, with permissive hypotension until surgical haemostasis is achieved.[470,471] In the UK, the National Institute for Clinical Excellence (NICE) has published guidelines on pre-hospital fluid replacement in trauma.[472] The recommendations include giving 250 ml boluses of crystalloid solution until a radial pulse is achieved and not delaying rapid transport of trauma victims for fluid infusion in the field. Pre-hospital fluid therapy may have a role in prolonged entrapments but there is no reliable evidence for this.[473,474]

Ultrasound

Ultrasound is a valuable tool in the evaluation of the compromised trauma patient. Haemoperitoneum, haemo-or pneumothorax and cardiac tamponade can be diagnosed reliably in minutes even in the pre-hospital phase.[475] Diagnostic peritoneal lavage and needle pericardiocentesis have virtually disappeared from clinical practice since the introduction of sonography in trauma care. Pre-hospital ultrasound is now available, although its benefits are yet to be proven.[476]

Vasopressors

The possible role of vasopressors (e.g., vasopressin) in trauma resuscitation is unclear and is based mainly on case reports.[477]

8j. Cardiac arrest associated with pregnancy

Overview

Mortality related to pregnancy in developed countries is rare, occurring in an estimated 1:30,000 deliveries.[478] The fetus must always be considered when an adverse cardiovascular event occurs in a pregnant woman. Fetal survival usually depends on maternal survival. 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. Studies tend to address causes in developed countries, whereas the most pregnancy-related deaths occur in developing countries. There were an estimated 342,900 maternal deaths (death during pregnancy, childbirth, or in the 42 days after delivery) worldwide in 2008.[479]

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

There are many causes of cardiac arrest in pregnant women. A review of nearly 2 million pregnancies in the UK[480] showed that maternal deaths (death during pregnancy, childbirth, or in the 42 days after delivery) between 2003 and 2005 were associated with:

- cardiac disease;
- pulmonary embolism;

- psychiatric disorders;
- hypertensive disorders of pregnancy;
- sepsis;
- haemorrhage;
- amniotic-fluid embolism;
- ectopic pregnancy.

Pregnant women can also sustain cardiac arrest from the same causes as women of the same age group.

Key interventions to prevent cardiac arrest

In an emergency, use an ABCDE approach. Many cardiovascular problems associated with pregnancy are caused by aortocaval compression. Treat a distressed or compromised pregnant patient as follows:

- Place the patient in the left lateral position or manually and gently displace the uterus to the left.
- Give high-flow oxygen guided by pulse oximetry.
- Give a fluid bolus if there is hypotension or evidence of hyovolaemia.
- 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.

Modifications to BLS guidelines for cardiac arrest

After 20 weeks gestation, the pregnant woman's uterus can press down against the inferior vena cava and the 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 arrest.[481,482] After 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 [483–485] and improves fetal oxygenation and heart rate.[486–488] Two studies found no improvement in maternal or fetal variables with 10–20° left lateral tilt.[489,490] One study found more aortic compression at 15° left lateral tilt when compared with a full left lateral tilt.[484] Aortic compression has been found to persist at over 30° of tilt.[491] Two non-arrest studies show that manual left uterine displacement with the patient supine is as good as or better than left lateral tilt in relieving aortocaval compression, as assessed by the incidence of hypotension and ephedrine use.[492,493] Non-cardiac arrest data show that the gravid uterus can be shifted away from the cava in most cases by placing the patient in 15° of left lateral decubitus position.[494] The value of relieving aortic or caval 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 good quality chest compressions. A variety of methods to achieve a left lateral tilt have been described including placing the victim on the rescuers knees[495], pillows or blankets, and the Cardiff wedge[496] although their efficacy in actual cardiac arrests is unknown. Even when a tilting table is used, the angle of tilt is often overestimated.[497] In a manikin study, the ability to provide effective chest compressions decreased as the angle of left lateral tilt increased and that at an angle of greater than 30° the manikin tended to roll.[496]

The key steps for BLS in a pregnant patient are:

- Call for expert help early (including an obstetrician and neonatologist).

- Start basic life support according to standard guidelines. Ensure good quality chest compressions with minimal interruptions.
- Manually displace the uterus to the left to remove caval compression.
- Add left lateral tilt if this is feasible – 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 allow good 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

There is a greater potential for gastro-oesophageal sphincter insufficiency and risk of pulmonary aspiration of gastric contents. Early tracheal intubation with correctly applied cricoid pressure decreases this risk. Tracheal intubation will 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.[498] One study documented that the upper airways in the third trimester of pregnancy are narrower compared with their postpartum state and to non-pregnant controls.[499] Tracheal intubation may be more difficult in the pregnant patient.[500] Expert help, a failed intubation drill and the use of alternative airway devices may be needed (see Section 4).[24a,501]

There is no change in transthoracic impedance during pregnancy, suggesting that standard shock energies for defibrillation attempts should be used in pregnant patients.[502] There is no evidence that shocks from a direct current defibrillator have adverse effects on the fetal heart. Left lateral tilt and large breasts will make it difficult to place an apical defibrillator paddle. Adhesive defibrillator pads are preferable to paddles in pregnancy.

Reversible causes

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

Haemorrhage

Life-threatening haemorrhage can occur both antenatally and postnatally. Postpartum haemorrhage is the commonest single cause of maternal death worldwide and is estimated to cause one maternal death every 7 min.[503] Associations include ectopic pregnancy, placental abruption, placenta praevia, placenta accreta, and uterine rupture.[480] A massive haemorrhage protocol must be available in all units and should be updated and rehearsed regularly in conjunction with the blood bank. Women at high risk of bleeding should be delivered in centres with facilities for blood transfusion, intensive care and other interventions, and plans should be made in advance for their management. Treatment is based on an ABCDE approach. The key step is to stop the bleeding. Consider the following:

- fluid resuscitation including use of rapid transfusion system and cell salvage[504];
- oxytocin and prostaglandin analogues to correct uterine atony[505];
- massaging the uterus[506];
- correction of coagulopathy including use of tranexamic acid or recombinant activated factor VII[507–509];
- uterine balloon tamponade[510,511];
- uterine compression sutures[512];
- angiography and endovascular embolization[513];
- hysterectomy[514,515];
- aortic cross-clamping in catastrophic haemorrhage.[516]

Cardiovascular disease

Myocardial infarction and aneurysm or dissection of the aorta or its branches, and peripartum cardiomyopathy cause most deaths from acquired cardiac disease.[517,518] 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.[480,519] 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.[520]

Increasing numbers of women with congenital heart disease are becoming pregnant.[521] 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.[522,523] Magnesium sulphate is effective in preventing approximately half of the cases of eclampsia developing in labour or immediately postpartum in women with pre-eclampsia.[524–526]

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%).[527] Risk factors include obesity, increased age, and immobility. Successful use of fibrinolytics for massive, life-threatening pulmonary embolism in pregnant women has been reported.[520,528–531]

Amniotic-fluid embolism

Amniotic-fluid embolism usually presents around the time of delivery with sudden cardiovascular collapse, breathlessness, cyanosis, arrhythmias, hypotension and haemorrhage associated with disseminated intravascular coagulopathy.[532] Patients may have warning signs preceding collapse including breathlessness, chest pain, feeling cold, light-headedness, distress, panic, a feeling of pins and needles in the fingers, nausea, and vomiting.

The UK Obstetric Surveillance System identified 60 cases of amniotic-fluid embolism between 2005 and 2009. The reported incidence was 2.0 per 100,000 deliveries (95% CI 1.5–2.5%)[533] The case fatality is 13–30% and perinatal mortality is 9–44%.[532]

Amniotic-fluid embolism was associated with induction of labour, multiple pregnancy, older, and ethnic-minority women. Caesarean delivery was associated with postnatal amniotic-fluid embolism.

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.[534]

If immediate resuscitation attempts fail

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. When initial resuscitation attempts fail, delivery of the fetus may improve the chances of successful resuscitation of the mother and fetus.[535–537] 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.[538] 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.[535,539–541] 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.[538] A case-series suggests increased use of Caesarean section during CPR with team training[542]; 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%.[542]

Delivery will relieve caval 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 aortocaval blood flow at approximately 20 weeks gestation; however, fetal viability begins at approximately 24–25 weeks.[543] 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.[544] Aim for delivery within 5 min of onset of cardiac arrest. This will mean that Caesarean section needs to ideally take place where the cardiac arrest has occurred to avoid delays.

- 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. Therapeutic hypothermia 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.[544a] Implantable cardioverter defibrillators (ICDs) have been used in patients during pregnancy.[545]

Preparation for cardiac arrest in pregnancy

Advanced life support in pregnancy requires coordination of maternal resuscitation, Caesarean delivery of the fetus and newborn resuscitation ideally within 5 min. 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.[546]

8k. Electrocution

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 in adults occur in the workplace and are associated generally 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 USA and Canada).[547] Electrocution from lightning strikes is rare, but worldwide it causes 1000 deaths each year.[548]

Electric shock injuries are caused by the direct effects of current on cell membranes and vascular smooth muscle. The thermal 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 VF if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon).[549] 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.

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.[550] Explosive force may cause blunt trauma.[551] The pattern and severity of injury from a lightning strike varies considerably, even among affected individuals from a single group.[552–554] As with industrial and domestic electric shock, death is caused by cardiac[553–557] or respiratory arrest.[550,558] 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.[559–561]

Diagnosis

The circumstances surrounding the incident are not always known. Unconscious patients with linear or punctuate burns or feathering should be treated as a victims of lightning strike.[550]

Rescue

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.[550]

Resuscitation

Start standard basic and advanced life support 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[560]; 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.[557]
- Consider early surgical intervention in patients with severe thermal injuries.
- Maintain spinal immobilisation if there is a likelihood of head or neck trauma.[562,563]
- Conduct a thorough secondary survey to exclude traumatic injuries caused by tetanic muscular contraction or by the person being thrown.[563,564]

- 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.

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.[558] Dilated or non-reactive pupils should never be used as a prognostic sign, particularly in patients suffering a lightning strike.[550]

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 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.[565]

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. There is no specific therapy for electrical injury, and the management is symptomatic. Prevention remains the best way to minimize the prevalence and severity of electrical injury.

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