



European Resuscitation Council Guidelines for Resuscitation 2005

Section 7. Cardiac arrest in special circumstances

Jasmeet Soar, Charles D. Deakin, Jerry P. Nolan, Gamal Abbas, Annette Alfonzo, Anthony J. Handley, David Lockey, Gavin D. Perkins, Karl Thies

7a. Life-threatening electrolyte disorders

Overview

Electrolyte abnormalities can cause cardiac arrhythmias or cardiopulmonary arrest. Life-threatening arrhythmias are associated 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 the 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 the rate of change of the electrolyte values.

There is little or no evidence base 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 the International Guidelines 2000.¹

Prevention of electrolyte disorders

- Treat life-threatening electrolyte abnormalities before cardiac arrest occurs.
- After initial treatment, remove any precipitating factors (e.g., drugs) and monitor electrolyte levels to prevent recurrence of the abnormality.
- Monitor renal function in patients at risk of electrolyte disorders.
- 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 3.5–5.0 mmol l⁻¹. A large concentration gradient normally exists between the intracellular and extracellular fluid compartments. This potassium gradient across the 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, serum potassium increases because

potassium shifts from the cellular to the vascular space. When serum pH increases, serum potassium decreases because potassium shifts intracellularly. We therefore anticipate the effects of pH changes on serum potassium during the 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 the cells or impaired excretion by the kidneys.

Definition. There is no universal definition, although 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 (ACEI), angiotensin II receptor blockers (ARB), 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, which may be the sole cause in patients with established renal failure. Abnormal erythrocytes or thrombocytosis may cause a spuriously high potassium concentration. The risk of hyperkalaemia is even greater when there is a combination of factors, such as the concomitant use of ACEI and NSAIDs or potassium-sparing diuretics.

Recognition of hyperkalaemia. Exclude hyperkalaemia in patients with an arrhythmia or cardiac arrest.² Patients may present with weakness progressing to flaccid paralysis, paraesthesia or depressed deep tendon reflexes. 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} .³ The ECG manifestations of hyperkalaemia are usually progressive and include:

- first-degree heart block (prolonged PR interval) $>0.2 \text{ s}$;
- flattened or absent *P* waves;
- tall, peaked (tenting) *T* waves, larger than *R* wave in more than one lead;
- ST segment depression;
- *S* and *T* waves merging;
- widened QRS $>0.12 \text{ s}$;
- ventricular tachycardia (VT);
- bradycardia;
- cardiac arrest, i.e., pulseless electrical activity (PEA), ventricular fibrillation (VF), asystole.

Treatment of hyperkalaemia. The five key steps in treating hyperkalaemia are:

1. cardiac protection by antagonising the effects of hyperkalaemia;
2. shifting potassium into cells;
3. removing potassium from the body;
4. monitoring serum potassium for rebound hyperkalaemia;
5. prevention of 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 management of hyperkalaemia is the subject of a recent Cochrane review.⁴

Patient not in cardiac arrest. If the patient is not in cardiac arrest, assess fluid status; if hypovolaemic, give fluid to enhance urinary potassium excretion. The values for classification are an approximate guide. For mild elevation ($5.5\text{--}6 \text{ mmol l}^{-1}$), remove potassium from the body with:

- potassium exchange resins, i.e., calcium resonium $15\text{--}30 \text{ g}$ or sodium polystyrene sulfonate (Kayexalate[®]) $15\text{--}30 \text{ g}$ in $50\text{--}100 \text{ ml}$ of 20% sorbitol, given either orally or by retention enema (onset in $1\text{--}3 \text{ h}$, maximal effect at 6 h);
- diuretics, i.e., furosemide 1 mg kg^{-1} IV slowly (onset with the diuresis);
- dialysis; haemodialysis is more efficient than peritoneal dialysis at removing potassium (immediate onset, $25\text{--}30 \text{ mmol potassium h}^{-1}$ removed with haemodialysis).

For moderate elevation ($6\text{--}6.5 \text{ mmol l}^{-1}$) without ECG changes, shift potassium into cells with:

- dextrose/insulin: 10 units short-acting insulin and 50 g glucose IV over $15\text{--}30 \text{ min}$ (onset in $15\text{--}30 \text{ min}$, maximal effect at $30\text{--}60 \text{ min}$; monitor blood glucose). Use in addition to removal strategies above.

For severe elevation ($\geq 6.5 \text{ mmol l}^{-1}$) without ECG changes, shift potassium into cells with:

- salbutamol, 5 mg nebulised. Several doses 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;^{5,6}
- use multiple shifting agents in addition to removal strategies above.

For severe elevation ($\geq 6.5 \text{ mmol l}^{-1}$) with toxic ECG changes, protect the heart *first* with:

- calcium chloride, i.e., 10 ml 10% calcium chloride IV over 2–5 min to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane. This protects the heart by reducing the risk of VF, but does not lower serum potassium (onset in 1–3 min). Use in addition to potassium removal and shifting strategies stated above.

Patient in cardiac arrest. If the patient is in cardiac arrest, there are no modifications to BLS in the presence of electrolyte abnormalities. For ALS, follow the universal algorithm. The general approach to treatment depends on the degree of hyperkalaemia, rate of rise of serum potassium and the patient's clinical condition.

In cardiopulmonary arrest, protect the heart first, then apply shifting and removal strategies using:

- calcium chloride: 10 ml of 10% calcium chloride IV by rapid bolus injection to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane;
- sodium bicarbonate: 50 mmol IV by rapid injection (if severe acidosis or renal failure);
- dextrose/insulin: 10 units short-acting insulin and 50 g glucose IV by rapid injection;
- haemodialysis: consider this for cardiac arrest induced by hyperkalaemia, which is resistant to medical treatment.

Indications for dialysis. Haemodialysis is the most effective method of removal of potassium from the body. The principal mechanism of action is the diffusion of potassium ions across the transmembrane 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. Consider haemodialysis early for hyperkalaemia associated with established renal failure, oliguric acute renal failure ($<400 \text{ ml day}^{-1}$ urine output) or when there is marked tissue

breakdown. Dialysis is also indicated when hyperkalaemia is resistant to medical management. Serum potassium frequently rebounds after initial treatment. In unstable patients, continuous veno-venous haemofiltration (CVVH) is less likely to compromise cardiac output than intermittent haemodialysis.

Hypokalaemia

Hypokalaemia is common in hospital patients.⁷ 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 $<3.5 \text{ mmol l}^{-1}$. Severe hypokalaemia is defined as a $\text{K}^+ < 2.5 \text{ mmol l}^{-1}$ 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 continuous ambulatory peritoneal dialysis (CAPD).

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

ECG features of hypokalaemia comprise:

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

Treatment. Treatment depends on the severity of hypokalaemia and the presence of symptoms and ECG abnormalities. Gradual replacement of potassium is preferable but in 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.⁸

Calcium and magnesium disorders

The recognition and management of calcium and magnesium disorders is summarised in [Table 7.1](#).

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 immediate treatment of the underlying electrolyte abnormalities can prevent many patients from progressing to cardiac arrest.

7b. Poisoning

General considerations

Poisoning is an infrequent cause of cardiac arrest, but remains a leading cause in victims younger than 40 years.^{9–12} Most research on this topic consists primarily of small case series, animal studies and case reports.

Self-poisoning with therapeutic or recreational drugs is the main reason for hospital admission. Drug toxicity can also be caused by inappropriate dosing and drug interactions. Accidental poisoning is commonest in children. Homicidal poisoning is uncommon. Industrial accidents, warfare or terrorism may cause extensive chemical or radiation exposure. Decontamination and safe management for mass casualty incidents is not part of these guidelines.

Resuscitation

Treatment of the self-poisoning ('overdose') patient is based on an ABCDE approach to prevent cardiorespiratory arrest whilst awaiting drug elimination.¹³ Airway obstruction and respiratory

arrest secondary to a decreased conscious level is a common cause of death. Alcohol excess is often associated with self-poisoning.

- After opening and clearing the airway, check for breathing and a pulse. Avoid mouth-to-mouth resuscitation in the presence of toxins, such as cyanide, hydrogen sulphide, corrosives and organophosphates. Ventilate the patient's lungs using a pocket- or bag-mask and the highest possible concentration of oxygen. Be careful in paraquat poisoning as pulmonary injury may be exacerbated by high concentrations of oxygen.¹⁴
- There is a high incidence of pulmonary aspiration of gastric contents after poisoning. Intubate unconscious patients who cannot protect their airway early, using a rapid-sequence induction with cricoid pressure to decrease the risk of aspiration (see section 4d). This must be undertaken by persons trained in the technique.
- In the event of cardiac arrest, provide standard basic and advanced life support.
- With the exception of torsades de pointes (see below), cardioversion is indicated for life-threatening tachyarrhythmias (see section 4f).
- Drug-induced hypotension is common after self-poisoning. This usually responds to fluid therapy, but occasionally inotropic support is required.
- Once resuscitation is under way, try to identify the poison(s). Relatives, friends and ambulance crews can usually provide useful information. Examination of the patient may reveal diagnostic clues, such as odours, needle puncture marks, pinpoint pupils, tablet residues, signs of corrosion in the mouth or blisters associated with prolonged coma.
- Measure the patient's temperature; hypo- or hyperthermia may occur after drug overdose (see sections 7d and 7e).
- Consult regional or national poisons centres for information on treatment of the poisoned patient.^{15,16} The World Health Organization lists poison centres on its website: <http://www.who.int/ipcs/poisons/centre/en/>.

Specific therapeutic measures

There are few specific therapeutic measures for poisons that are useful immediately. The emphasis is on intensive supportive therapy, with correction of hypoxia, hypotension and acid/base and electrolyte disorders.

Therapeutic measures include limiting absorption of ingested poisons, enhancing elimination, or the use of specific antidotes. For up-to-date guidance in severe or uncommon poisonings, seek

Table 7.1 Calcium (Ca²⁺) and magnesium (Mg²⁺) disorders with associated clinical presentation, ECG manifestations and recommended treatment

Disorder	Causes	Presentation	ECG	Treatment
Hypercalcaemia (Ca ²⁺ >2.6 mmol l ⁻¹)	Primary or tertiary hyperparathyroidism Malignancy Sarcoidosis Drugs	Confusion Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Short QT interval Prolonged QRS interval Flat T waves AV-block Cardiac arrest	Fluid replacement IV Furosemide, 1 mg kg ⁻¹ IV Hydrocortisone, 200–300 mg IV Pamidronate, 60–90 mg IV Calcitonin, 4–8 units kg ⁻¹ 8 h ⁻¹ IM Review medication Haemodialysis
Hypocalcaemia (Ca ²⁺ <2.1 mmol l ⁻¹)	Chronic renal failure Acute pancreatitis Calcium channel blocker overdose Toxic shock syndrome Rhabdomyolysis Tumour lysis syndrome	Paraesthesia Tetany Seizures AV-block Cardiac arrest	Prolonged QT interval T-wave inversion Heart block Cardiac arrest	Calcium chloride 10%, 10–40 ml Magnesium sulphate 50%, 4–8 mmol (if necessary)
Hypermagnesaemia (Mg ²⁺ > 1.1 mmol l ⁻¹)	Renal failure Iatrogenic	Confusion Weakness Respiratory depression AV-block Cardiac arrest	Prolonged PR and QT intervals T-wave peaking AV-block Cardiac arrest	Calcium chloride 10%, 5–10 ml, repeated if necessary Ventilatory support if necessary Saline diuresis: 0.9% saline with furosemide 1 mg kg ⁻¹ IV Haemodialysis
Hypomagnesaemia (Mg ²⁺ <0.6 mmol l ⁻¹)	Gastrointestinal loss Polyuria Starvation Alcoholism Malabsorption	Tremor Ataxia Nystagmus Seizures Arrhythmias: torsades de pointes Cardiac arrest	Prolonged PR and QT intervals ST-segment depression Torsades de pointes T-wave inversion Flattened P waves Increased QRS duration	Severe or symptomatic: 2 g 50% magnesium sulphate (4 ml = 8 mmol) IV over 15 min Torsade de pointes: 2 g 50% magnesium sulphate (4 ml = 8 mmol) IV over 1–2 min Seizure: 2 g 50% magnesium sulphate (4 ml = 8 mmol) IV over 10 min

advice from a poisons centre.

- Activated charcoal is known to adsorb certain drugs. Its value decreases over time after ingestion. There is no evidence that ingestion of charcoal improves clinical outcome. According to evidence from volunteer studies, 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.¹⁷ Give it only to patients with an intact or protected airway. Multiple doses of activated charcoal can be beneficial in life-threatening poisoning with carbamazepine, dapsone, phenobarbital, quinine and theophylline.
- Gastric lavage followed by activated charcoal therapy is useful only within 1 h of ingesting the poison.¹⁷ Generally this should be carried out after tracheal intubation. Delayed gastric lavage has very little effect on drug absorption and may propel drugs further along the gastrointestinal tract.¹⁸ Do not give ipecacuanha syrup to induce vomiting; there is little evidence of benefit.¹⁹
- There is little evidence for the use of laxatives, e.g., lactulose or magnesium citrate, to enhance drug elimination from the gut.²⁰
- Whole-bowel irrigation by enteral administration of a polyethylene glycol solution can reduce drug absorption by cleansing the gastrointestinal tract. It can be useful in cases of potentially toxic ingestion of sustained release or enteric-coated drugs, oral iron poisoning and the removal of ingested packets of illicit drugs.²¹
- Urine alkalinisation (pH 7.5) by giving IV sodium bicarbonate can be useful in moderate-to-severe salicylate poisoning in patients who do not need haemodialysis.²² Urine alkalinisation can also be useful in tricyclic overdose (see below).
- Haemodialysis or haemoperfusion can be useful for elimination of specific life-threatening toxins. Haemodialysis removes drugs or metabolites that are water soluble, have a low volume of distribution and low plasma protein binding.²³ It may be considered for poisoning with methanol, ethylene glycol, salicylates and lithium. Haemoperfusion involves passing blood through an absorptive-containing cartridge (usually charcoal). This technique removes substances that have a high degree of plasma protein binding. Charcoal haemoperfusion may be indicated for intoxications with carbamazepine, phenobarbital, phenytoin and theophylline.
- Specific antidotes (see below) which may be effective include: *N*-acetylcysteine for paracetamol; high-dose atropine for organophosphate insecticides; sodium nitrite, sodium thiosulphate

or dicobalt edetate for cyanides; digoxin-specific Fab antibodies for digoxin; flumazenil for benzodiazepines; and naloxone for opioids. Reversal of benzodiazepine intoxication with flumazenil is associated with significant toxicity in patients with benzodiazepine dependence or co-ingestion of proconvulsant medications, such as tricyclic antidepressants.²⁴ The routine use of flumazenil in the comatose patient with an overdose is not recommended.

Specific antidotes

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

Opioid poisoning

Opioid poisoning commonly causes respiratory depression followed by respiratory insufficiency or respiratory arrest. The respiratory effects of opioids are reversed rapidly by the opiate antagonist naloxone. In severe respiratory depression, the evidence shows fewer adverse events when airway opening, oxygen administration and ventilation are carried out before giving naloxone in cases of opioid-induced respiratory depression;^{25–30} however, 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), endotracheal (ET) 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 mcg IV,²⁷ 800 mcg IM, 800 mcg SC,²⁷ 2 mg IN³¹ or 1–2 mg ET. Large opioid overdoses may require titration to a total naloxone 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 opiate intoxication with caution in patients suspected of opioid dependence.

There is no good evidence that naloxone improves outcome 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.²⁶

Giving naloxone is unlikely to be harmful. Once cardiac arrest has occurred, follow the standard resuscitation protocols.

Tricyclic antidepressants

Self-poisoning with tricyclic antidepressants is common and can cause hypotension, seizures and arrhythmias. 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. A widening QRS complex indicates a greater risk of arrhythmias. There is evidence to support the use of sodium bicarbonate to treat arrhythmias induced by tricyclic antidepressants and/or hypotension.^{32–47} The exact threshold for starting treatment based on QRS duration has not been established. No study has investigated the optimal target arterial or urinary pH with bicarbonate therapy, but an arterial pH of 7.45–7.55 has been commonly accepted and seems reasonable. Hypertonic saline may also be effective in treating cardiac toxicity.⁴⁸

Cocaine toxicity

Sympathetic overstimulation associated with cocaine toxicity may cause agitation, symptomatic tachycardia, hypertensive crisis, hyperthermia and myocardial ischaemia with angina. Glyceryl trinitrate and phentolamine reverse cocaine-induced coronary vasoconstriction, labetalol has no significant effect, and propranolol makes it worse.^{49–52} Small doses of IV benzodiazepines (midazolam, diazepam, lorazepam) are effective first-line drugs. Use nitrates only as second-line therapy for myocardial ischaemia. Labetalol (alpha- and beta-blocker) is useful for the treatment of tachycardia and hypertensive emergencies due to cocaine toxicity.

Drug-induced severe bradycardia

Severe bradycardia from poisoning or drug overdose may be refractory to standard ALS protocols because of prolonged receptor binding or direct cellular toxicity. Atropine may be life saving in organophosphate, carbamate or nerve agent poisoning. Give atropine for bradycardia caused by acetylcholinesterase-inhibiting substances. Large (2–4 mg) and repeated doses may be required to achieve a clinical effect. Isoprenaline may be useful at high doses in refractory bradycardia induced by beta-antagonist receptor blockade. Heart block and ventricular arrhythmias associated with digoxin or digitalis glycoside poisoning may be treated effec-

tively with digoxin-specific antibody fragments.⁵³ Antibody-specific therapy may also be effective in poisoning from plants as well as Chinese herbal medications containing digitalis glycosides.^{53–55}

Vasopressors, inotropes, calcium, glucagon, phosphodiesterase inhibitors and insulin-glucose may all be useful in beta-blocker and calcium channel blocker overdose.^{56–58} Transcutaneous pacing may be effective for severe bradycardia caused by poisoning and overdose (see section 3).

Further treatment and prognosis

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 the overdose of some drugs. Retain samples of blood and urine for analysis.

Be prepared to continue resuscitation for a prolonged period, particularly in young patients as the poison may be metabolised 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.

7c. Drowning

Overview

Drowning is a common cause of accidental death in Europe. The most important and detrimental consequence of drowning is hypoxia. The duration of hypoxia is the 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 drowning. This will require bystander provision of CPR plus immediate activation of the EMS system. Victims who have spontaneous circulation and breathing when they reach hospital usually recover with good outcomes.

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;⁵⁹ 97% of deaths from drowning occur in low- and middle-income countries.⁵⁹ In 2002, there were 427 deaths from drowning in the United Kingdom (Royal Society for the Prevention of Accidents 2002) and 4073 in the United States (National Center for Injury Prevention 2002), yielding an annual incidence of drowning of 0.8 and 1.45 per 100,000 population, respectively. Death from drowning is more common in young males and is the leading cause of accidental death in Europe in this group.⁵⁹ Alcohol consumption is a contributory factor in up to 70% of drownings.⁶⁰

The guidelines in this chapter focus on the treatment of the individual drowning victim rather than the management of mass casualty aquatic incidents.

Definitions, classifications and reporting

Over 30 different terms have been used to describe the process and outcome from submersion- and immersion-related incidents.⁶¹ To improve clarity and to help comparability of future scientific and epidemiological reports, the International Liaison Committee on Resuscitation (ILCOR) has proposed new definitions related to drowning.⁶² Drowning itself is defined 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. 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.⁶²

Basic life support

Aquatic rescue and recovery from the water

Always be aware of personal safety and minimise the danger to yourself and the victim at all times. Whenever possible, attempt to save the drowning victim without entry into water. Talking to the vic-

tim, 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.

Remove all the 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 low (approximately 0.5%).⁶³ 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.⁶⁴ Despite potential spinal injury, victims who are pulseless and apnoeic should be removed from water as quickly as possible (even if a back support device is not available), while attempting to limit neck flexion and extension. Cervical spine immobilisation is not indicated unless signs of severe injury are apparent or the history is consistent with the possibility of severe injury.⁶⁵ These circumstances include a history of diving, water-slide use, signs of trauma or signs of alcohol intoxication. Whenever possible, remove the victim from the water in a horizontal position to minimise the risks of post-immersion hypotension and cardiovascular collapse.⁶⁶

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 the survival.^{67,68} In the apnoeic victim, start rescue breathing as soon as the victim's airway is opened and the rescuer's safety ensured. This can sometimes be achieved when the victim is still in shallow water. 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, start in-water rescue breathing if trained to do so, ideally with the support of a buoyant rescue aid,⁶⁹ although in-water, unsupported resuscitation may also be possible.⁷⁰ Untrained rescuers should not attempt to perform any form of resuscitation with a victim in deep water.

If there is no spontaneous breathing after opening the airway, give rescue breaths for approximately 1 min.⁶⁹ If the victim does not start breathing spontaneously, further management depends on the distance from land. If the victim can be brought to land in <5 min of rescue time, 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.⁶⁹

There is no need to clear the airway of aspirated water. The majority of drowning victims aspirate only a modest amount of water, and this is absorbed rapidly into the central circulation. An attempt to remove water from the air passages by any means other than suction is unnecessary and dangerous. Abdominal thrusts cause regurgitation of gastric contents and subsequent aspiration. They have been associated with other life-threatening injuries and should not be performed unless there are clear signs of foreign-body airway obstruction.⁷¹

Chest compression

As soon as the victim is removed from water, check for breathing. A healthcare professional who is trained in pulse checking may also check for pulse, but this may be even more difficult to find in a drowning victim, particularly if cold. If the victim is not breathing, start chest compressions immediately. Chest compression is ineffective in water.^{72,73}

Defibrillation

If the victim is unresponsive and not breathing and an AED is available, attach it to the victim and turn it on. Before attaching the AED pads, dry the victim's chest to enable adherence. Deliver shocks according to the AED prompts. If the victim is hypothermic with a core body temperature $\leq 30^{\circ}\text{C}$ (86°F), limit defibrillation to a total of three attempts until the core body temperature rises above 30°C (86°F).⁷⁴

Regurgitation during resuscitation

Regurgitation of stomach contents is common following resuscitation from drowning and will complicate efforts to maintain a patent airway. In one study, regurgitation occurred in two-thirds of victims who received rescue breathing and 86% of victims who required compression and ventilation.⁷⁵ If regurgitation occurs, turn the victim's mouth to the side and remove the regurgitated material using directed suction if possible. If spinal cord injury is suspected, log-roll the victim, keeping the head, neck and torso aligned, before aspirating the regurgitated material. Log-rolling will require several rescuers.

Advanced life support

Airway and breathing

Give high-flow oxygen during the initial assessment of the spontaneously breathing drowning victim. Consider non-invasive ventilation or continuous positive airway pressure if the victim fails to respond to treatment with high-flow oxygen.⁷⁶ Use pulse oximetry and arterial blood gas analysis to titrate the concentration of inspired oxygen and to provide an indicator of the adequacy of ventilation. Consider early 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 high risk of aspiration.⁷⁷ Protect the airway of the victim in cardiopulmonary arrest early in the resuscitation attempt, ideally with a tracheal tube. Reduced pulmonary compliance requiring high inflation pressures may limit the use of adjuncts, such as the laryngeal mask airway. Initiate ventilation with a high-inspired oxygen concentration as soon as possible, to treat the severe hypoxaemia that is likely to be present.

Circulation and defibrillation

Follow standard advanced life support protocols. If severe hypothermia is present (core body temperature $\leq 30^{\circ}\text{C}$ or 86°F), limit defibrillation attempts to three, and withhold IV drugs until the core body temperature increases above these levels. If moderate hypothermia is present, give IV drugs at longer than standard intervals (see section 7d).

During prolonged immersion, victims may become hypovolaemic from the hydrostatic pressure of water on the body. Give IV fluid to correct the hypovolaemia but avoid excessive volumes, which may cause pulmonary oedema. 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.⁷⁸ Continue resuscitation unless there is clear evidence that resuscitation 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.^{79,80}

Post-resuscitation care

Salt versus fresh water

Much attention has been focused in the past on differences between salt- 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 high risk of developing acute respiratory distress syndrome (ARDS) for up to 72 h after submersion. Protective ventilation strategies improve survival in patients with ARDS.⁸¹ The propensity towards alveolar collapse may require the use of PEEP or other alveolar recruitment manoeuvres to reverse severe hypoxaemia.⁸² Extracorporeal membrane oxygenation and nitric oxide administration have been used in some centres for refractory hypoxaemia in drowning victims but the efficacy of these treatments is unproven.⁶⁵

Pneumonia is common after drowning. Prophylactic antibiotics have not been shown to be of benefit, although they may be considered after submersion in grossly contaminated water such as sewage. Give broad-spectrum antibiotics if signs of infection develop subsequently.⁶⁵

Hypothermia

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 icy water.⁵⁹ Hypothermia may also develop as a secondary complication of the submersion, and subsequent heat loss through evaporation during attempted resuscitation. In these victims the hypothermia is not protective (see section 7d).

Several small clinical studies in patients with accidental hypothermia have shown that survival may be improved by either passive or active warming out of hospital or in the emergency room.⁶⁵

In contrast, there is evidence of benefit from induced hypothermia for comatose victims resuscitated from prehospital cardiac arrests.⁸³ To date, there is no convincing evidence to guide therapy in this patient group. A pragmatic approach might be to consider instituting active rewarming until a core temperature of 32–34 °C is achieved, and then actively 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 the outcome. In fact, signs of high ICP serve as a symptom of significant neurological hypoxic injury, and no evidence that attempts to alter the ICP will affect the outcome.⁶⁵

7d. Hypothermia

Definition

Hypothermia exists when the body core temperature is below 35 °C and is classified arbitrarily as mild (35–32 °C), moderate (32–30 °C) or severe (less than 30 °C). Hypothermia can occur in people with normal thermoregulation who are exposed to cold environments, particularly wet or windy conditions, or following immersion in cold water. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia may follow a mild cold insult. The risk of hypothermia is also increased by drug or alcohol ingestion, illness, injury or neglect. 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 diagnosis.

In some cases, hypothermia may exert a protective effect on the brain after cardiac arrest.^{84,85} Intact neurological recovery may be possible after hypothermic cardiac arrest, although those with non-asphyxial arrest have a better prognosis than those with asphyxial hypothermic arrest.^{86–88} Life-saving procedures should not be withheld on the basis of clinical presentation alone.⁸⁷

Decision to resuscitate

Beware of pronouncing death in a hypothermic patient, as cold alone may produce a very slow,

small-volume, irregular pulse and an unrecordable blood pressure. Hypothermia protects the brain and vital organs, and associated arrhythmias are potentially reversible either before or during rewarming. At 18°C the brain can tolerate periods of circulatory arrest for 10 times longer than at 37°C. Dilated pupils can be caused by a variety of insults and must not be taken as a sign of death.

On discovering a hypothermic cardiac arrest victim in cold environment, it is not always easy to distinguish between primary and secondary hypothermia. Cardiac arrest could be caused primarily by hypothermia, or hypothermia could follow a normothermic cardiac arrest (e.g., cardiac arrest caused by myocardial ischaemia in a person in cold environment).

Do not confirm death until the patient has been rewarmed or until attempts to raise the core temperature have failed; prolonged resuscitation may be necessary. In the prehospital setting, resuscitation should be withheld only if the patient has obvious lethal injuries or if the body is completely frozen making resuscitation attempts impossible.⁸⁹ In the hospital setting, 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. Do not delay the urgent procedures, such as intubation and insertion of vascular catheters. Intubation can provoke VF in a patient with severe hypothermia.^{87,90}

- Clear the airway and, if there is no spontaneous respiratory effort, ventilate the patient's lungs with high concentrations of oxygen. If possible, use warmed (40–46°C) and humidified oxygen. Consider careful tracheal intubation when indicated according to the ALS algorithm.
- Palpate a major artery and, if available, look at the ECG for up to 1 min and look for signs of life before concluding that there is no cardiac output. If a Doppler ultrasound probe is available, use it to establish whether there is peripheral blood flow. If the victim is pulseless, start chest compressions immediately. If there is any doubt about whether a pulse is present, start CPR.
- Once resuscitation is under way, confirm hypothermia with a low-reading thermometer. The method of temperature measurement should be the same throughout resuscitation and rewarming. Use oesophageal, bladder, rectal or tympanic temperature measurements.^{91,92}

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 compression difficult.

The hypothermic heart may be unresponsive to cardioactive drugs, attempted electrical pacing and attempted defibrillation. Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drug given repeatedly.⁹⁰ The evidence for the efficacy of drugs in severe hypothermia is limited and based mainly on animal studies. Adrenaline may be effective in increasing coronary perfusion pressure, but not survival, in severe hypothermic cardiac arrest.^{93,94} The efficacy of amiodarone is also reduced.⁹⁵ For these reasons, withhold adrenaline and other drugs until the patient has been warmed to a temperature greater than 30°C. Once 30°C has been reached, the intervals between doses should be doubled. As the patient's temperature returns towards normal, the standard drug protocols should be used.

Remember to rule out other primary causes of cardiorespiratory arrest using the four Hs and four Ts approach (e.g., drug overdose, hypothyroidism, trauma).

Arrhythmias

As the body core temperature decreases, sinus bradycardia tends to give way to atrial fibrillation (AF) followed by ventricular fibrillation (VF) and finally asystole.⁹⁶ Follow the standard treatment protocols.

Severely hypothermic victims (core temperature <30°C) in cardiac arrest in hospital must be rapidly rewarmed using 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 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; if VF/VT persists after three shocks, delay further defibrillation attempts until the core temperature is above 30°C.^{97,98} If an AED is used, follow the AED prompts while rewarming the patient.

Rewarming

General measures for all casualties include removal from the cold environment, prevention of further

heat loss and rapid transfer to the hospital. Remove cold or wet clothing as soon as possible. Cover the dry casualties with blankets and keep them out of the wind.

Rewarming may be passive external, active external, or active internal. Passive warming is achieved with blankets and a warm room, and is suitable for conscious victims with mild hypothermia. In severe hypothermia or cardiac arrest, active warming is required, but this must not delay transport to a hospital where more advanced rewarming techniques are available. Several techniques have been described, although there are no clinical trials of outcome to determine the best rewarming method. Studies show that forced air rewarming and warm IV fluids are effective in patients with severe hypothermia and a perfusing rhythm.^{99,100} Other warming techniques include the use of warm humidified gases, gastric, peritoneal, pleural or bladder lavage with warm fluids (at 40 °C), and extracorporeal blood warming with partial bypass.^{87,90,101–103}

In the patient with cardiac arrest and hypothermia, cardiopulmonary bypass is the preferred method of active internal rewarming because it also provides circulation, oxygenation and ventilation, while the core body temperature is increased gradually.^{104,105} Survivors in one case series had an average of 65 min of conventional CPR before cardiopulmonary bypass.¹⁰⁵ Unfortunately, facilities for cardiopulmonary bypass are not always available and a combination of methods may have to be used.

During rewarming, patients will require large volumes of fluids as their vascular space expands with vasodilation. Warm all the IV fluids. Use continuous haemodynamic monitoring and, if possible, treat the patient in a critical care unit. Avoid hyperthermia during and after the warming period. 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). There is no evidence for the routine use of steroids, barbiturates or antibiotics.^{106,107}

7e. Hyperthermia

Definition

Hyperthermia occurs when the body's ability to thermoregulate fails, and core temperature exceeds the one that is normally maintained by homeostatic mechanisms. Hyperthermia may be exogenous, caused by environmental condi-

tions, 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 that 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.¹⁰⁸

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 depolarising muscle relaxants.^{109,110}

The key features and treatment of heat stress and heat exhaustion are included in [Table 7.2](#).

Heat stroke (HS)

HS 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-exertion heat stroke (CHS) occurring during high environmental temperatures and often effecting the elderly during heat waves;¹¹¹ exertion heat stroke (EHS) occurring during strenuous physical exercise in high environmental temperatures and/or high humidity usually effecting healthy young adults.¹¹² Mortality from HS ranges between 10 and 50%.¹¹³

Predisposing factors

The elderly are at an increased risk for heat-related illness because of underlying illness, medication use, declining thermoregulatory mechanisms and limited social support. There are several risk factors: lack of acclimatisation, dehydration, obesity, alcohol, cardiovascular disease, skin conditions (psoriasis, eczema, scleroderma, burn, cystic fibrosis), hyperthyroidism, pheochromocytoma 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.¹¹⁴ Features include:

- core temperature 40.6 °C or more;
- hot, dry skin (sweating is present in about 50% of cases of exertional heat stroke);

Table 7.2 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

- early signs and symptoms, e.g., extreme fatigue, headache, fainting, facial flushing, vomiting and diarrhoea;
- cardiovascular dysfunction including arrhythmias¹¹⁵ and hypotension;
- respiratory dysfunction including ARDS;¹¹⁶
- central nervous system dysfunction including seizures and coma;¹¹⁷
- liver and renal failure;¹¹⁸
- coagulopathy;¹¹⁶
- rhabdomyolysis.¹¹⁹

Other clinical conditions need to be considered, including:

- drug toxicity;^{120,121}
- drug withdrawal syndrome;
- serotonin syndrome;¹²²
- neuroleptic malignant syndrome¹²³
- sepsis;¹²⁴
- central nervous system infection;
- endocrine disorders, e.g., thyroid storm, pheochromocytoma.¹²⁵

Management

The mainstay of treatment is supportive therapy based on optimising the ABCDEs and cooling the patient.^{126,127} Start cooling before the patient reaches the hospital. Aim to 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 the electrolyte abnormalities as described in Section 7a.

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;¹²⁸ however, this may cause peripheral vasoconstriction, shunt blood away from the periphery and reduce heat dissipation. Immersion is also not practical in the most sick patients.

Further techniques to cool patients with hyperthermia are similar to those used for therapeutic hypothermia after cardiac arrest (see section 4g). Gastric, peritoneal,¹²⁹ pleural or bladder lavage with cold water lowers the core temperature. Intravascular cooling techniques include the use of cold IV fluids,¹³⁰ intravascular cooling catheters^{131,132} and extracorporeal circuits,¹³³ e.g., continuous veno-venous haemofiltration or cardiopulmonary bypass.

Drug therapy in heat stroke

There are no specific drug therapies in heat stroke to lower the core temperature. There is no good evidence that antipyretics (e.g., non-steroidal anti-inflammatory drugs or paracetamol) are effective in heat stroke. Dantrolene (see below) has not been shown to be beneficial.¹³⁴

Malignant hyperthermia (MH)

MH is a life-threatening genetic sensitivity of skeletal muscles to volatile anaesthetics and depolarising neuromuscular blocking drugs, occurring during or after anaesthesia. Stop triggering agents immediately; give oxygen, correct acidosis and electrolyte abnormalities. Start active cooling and give dantrolene.¹³⁵

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. 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 that the prognosis is poor compared with normothermic cardiac arrest.^{136,137} The risk of unfavourable neurological outcome increases for each degree of body temperature $>37^{\circ}\text{C}$.¹³⁸ Provide post-resuscitation care according to the normal guidelines.

7f. Asthma

Introduction

Approximately 300 million people of all ages and ethnic backgrounds suffer from asthma worldwide.¹³⁹ Asthma still causes many deaths in young adults, mostly among those with chronic severe asthma, adverse psychosocial circumstances and poor medical management. National and international guidance for the management of asthma already exists.^{139,140} The following guidelines focus on the treatment of patients with near-fatal asthma and cardiac arrest.

Causes of cardiac arrest

Cardiac arrest in the asthmatic person is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden. Cardiac arrest in asthmatics 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 common cause of asthma-related arrhythmia. Arrhythmias can be caused by stimulant drugs

(e.g., beta-adrenergic agonists, aminophylline) or electrolyte abnormalities;

- dynamic hyperinflation, i.e., autospontaneous end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (breathed air entering and being unable to escape). Gradual build-up of pressure occurs and reduces blood flow and blood pressure;
- tension pneumothorax (often bilateral).

The four Hs and four Ts approach to reversible causes helps identify these causes in cardiac arrest.

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 the therapy because beta-agonists cause both bronchodilation and vasodilation and may increase intrapulmonary shunting initially.

Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis,¹⁴¹ pneumonia, foreign bodies, pulmonary embolism, bronchiectasis and subglottic mass.¹⁴²

The severity of an asthma attack is defined in Table 7.3.

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. 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 $\text{SaO}_2 \geq 92\%$. High-flow oxygen by mask is sometimes necessary. Consider rapid-sequence induction and tracheal intubation if, despite efforts to optimise drug therapy, the patient has:

- decreased conscious level, coma;
- profuse sweating;
- reduced muscle tone (clinical signs of hypercarbia);

Table 7.3 The severity of asthma¹⁴⁰

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 Heart rate >110/min 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.

- findings of severe agitation, confusion and fighting against the oxygen mask (clinical signs of hypoxemia).

Elevation of the PaCO₂ alone does not indicate the need for tracheal intubation. Treat the patient, not the numbers.

Nebulised beta₂-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.

Intravenous corticosteroids

Oxygen and beta-agonists are the most important therapies initially, but give corticosteroids (hydrocortisone, 200 mg IV,) early. Although there is no difference in clinical effects between oral and IV formulations of corticosteroids,¹⁴³ 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 h) may produce additional bronchodilation in severe asthma or in those who do not respond to beta-agonists.^{144,145}

Intravenous salbutamol

Several studies have shown intravenous salbutamol (250 mcg IV slowly) to provide additional benefit in severe asthmatics who are already receiving nebulised salbutamol.¹⁴⁶ Give an infusion of 3–20 mcg min⁻¹.

Intravenous magnesium sulphate

Magnesium sulphate (2 g, IV slowly) may be useful as a bronchodilator in severe or near-fatal asthma. A Cochrane meta-analysis of seven studies concluded that magnesium is beneficial, particularly for those with the most severe exacerbations.¹⁴⁷ Magnesium causes bronchial smooth muscle relaxation independent of the serum magnesium level and has only minor side effects (flushing, light-headedness).

Intravenous theophylline

Theophylline is given IV as aminophylline, a mixture of theophylline with ethylenediamine,

which is 20 times more soluble than theophylline alone. Aminophylline should only be considered in severe or near-fatal asthma. A loading dose of 5 mg kg^{-1} is given over 20–30 min (unless on maintenance therapy), followed by an infusion of $500\text{--}700 \text{ mcg kg}^{-1} \text{ h}^{-1}$. Addition of this drug to high doses of beta-agonists increases side effects more than it increases bronchodilation. Check levels to avoid toxicity.

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 mcg up to a total of three 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.¹⁴⁸ Terbutaline is given in a dose of 250 mcg 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,¹⁴⁹ one study concluded that terbutaline was superior.¹⁵⁰ These alternative routes may need to be considered when IV access is impossible.

Intravenous fluids

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.

Heliox

Heliox is a mixture of helium and oxygen (usually 80:20 or 70:30). A recent meta-analysis of four clinical trials did not support the use of heliox in the initial treatment of patients with acute asthma.¹⁵¹

Ketamine

Ketamine is a parenteral dissociative anaesthetic with bronchodilatory properties. One case series suggested substantial effectiveness,¹⁵² but the single randomised trial published to date showed no benefit to ketamine compared with standard care.¹⁵³

Non-invasive ventilation

Non-invasive ventilation decreases the intubation rate and mortality in COPD;¹⁵⁴ however, its role in patients with severe acute asthma is uncertain. Although promising, a recent Cochrane review suggests that more studies are needed.¹⁵⁵

Management of cardiac arrest

Basic life support

Give basic life support according to the standard guidelines. Ventilation will be difficult because of increased airway resistance; try to prevent gastric inflation.

Advanced life support

Modifications to standard ALS guidelines. Consider the need for intubation early. The peak airway pressures recorded during the ventilation of patients with severe asthma (mean $67.8 \pm 11.1 \text{ cmH}_2\text{O}$ in 12 patients) are significantly higher than the normal lower oesophageal sphincter pressure (approximately $20 \text{ cmH}_2\text{O}$).¹⁵⁶ 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.¹⁵⁷

The new recommended respiratory rate ($10 \text{ breaths min}^{-1}$) 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 the inspiratory time and inspiratory flow, while lung emptying depends on the 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.¹⁵⁶

There is limited evidence from the case reports of unexpected ROSC in patients with suspected gas trapping when the tracheal tube is disconnected.^{158–161} 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.

Dynamic hyperinflation increases transthoracic impedance.¹⁶² Consider the higher shock energies for defibrillation if initial defibrillation attempts fail.

There is no good evidence for the use of open-chest cardiac compressions in patients with asthma-associated cardiac arrest. Working through the four Hs and four Ts will identify potentially reversible causes of asthma related cardiac arrest. Tension pneumothorax can be difficult to diagnose in cardiac arrest; it may be indicated by unilateral expansion of the chest wall, shifting of the trachea and subcutaneous emphysema. Release air from the pleural space with needle decompression. Insert a large-gauge cannula in the second intercostal space 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.

Post-resuscitation care

The following should be added to usual management after ROSC:

- Optimise the medical management of bronchospasm.
- Use permissive hypercapnia; it may not be possible to achieve normal oxygenation and ventilation in a patient with severe bronchospasm. Efforts to achieve normal arterial blood gas values may worsen lung injury. Mild hypoventilation reduces the risk of barotraumas, and hypercapnoea is typically well-tolerated.¹⁶³ Target lower arterial blood oxygen saturations (e.g., 90%).
- Provide sedation (neuromuscular paralysis if needed) and controlled ventilation. Despite the absence of formal studies, ketamine and inhalational anaesthetics have bronchodilator properties that may be useful in the asthmatic patient who is difficult to ventilate.
- Involve a senior critical care doctor early.

7g. Anaphylaxis

Introduction

Anaphylaxis is a rare, but potentially reversible, cause of cardiac arrest. Although the management of cardiac arrest secondary to anaphylaxis follows the general principles described elsewhere in these guidelines, the pathophysiological processes occurring during anaphylaxis may require additional specific therapy.

Anaphylaxis is a severe life-threatening, generalised or systemic hypersensitivity reaction. Investigations will show whether the reaction is allergic (immunoglobulin E (IgE) or non IgE mediated) or non-allergic anaphylaxis. The term anaphylactoid reaction is no longer used. An anaphylactic reaction is generally defined as a severe, systemic allergic reaction characterized by multi-system involvement, including the airway, vascular system, gastrointestinal tract and skin. Severe cases may cause complete airway obstruction secondary to laryngeal oedema, bronchospasm, hypotension, cardiovascular collapse and death. Other symptoms include rhinitis, conjunctivitis, abdominal pain, vomiting, diarrhoea and a sense of impending doom. There is also usually a colour change; the patient may appear either flushed or pale. Anaphylactic reactions vary in severity, and progress may be rapid, slow or (unusually) biphasic. Rarely, manifestations may be delayed (this may occur with latex allergy), or persist for more than 24 h.

Pathophysiology

Initial exposure to an allergen may trigger an immune response that sensitises the body to subsequent exposure. This sensitisation results in antigen-specific IgE bound to the cell membrane of basophils and mast cells. On repeat exposure, the antigen is bound by the IgE, triggering release of a series of inflammatory mediators including histamines, leukotrienes, prostaglandins, thromboxanes and bradykinins. These mediators act systemically to cause increased mucous membrane secretion, increased capillary permeability and markedly reduced vascular smooth muscle tone. This causes the clinical symptoms of angioedema and airway swelling, bronchospasm, hypotension and cardiovascular collapse.

Anaphylaxis is caused by a hypersensitivity reaction in which histamine, serotonin and other vasoactive substances are released from basophils and mast cells in response to an IgE-mediated reaction. Antigen-specific immunoglobulins are produced after initial exposure to an allergen. Subsequent re-exposure to this allergen provokes an anaphylactic reaction, although many anaphylactic reactions occur without known previous exposure.

Aetiology

Although anaphylaxis is relatively common, progression to a severe life-threatening reaction is rare. Any antigen capable of activating IgE can theoretically be a trigger for anaphylaxis. The com-

most causes of life-threatening reactions are drugs, stinging insects and food. In as many as 5% of the cases, the antigen triggering the anaphylaxis cannot be identified.

Drugs

Neuromuscular blocking drugs (particularly suxamethonium) and antibiotics are the most common triggers for drug-induced anaphylaxis.¹⁶⁴ Aspirin, non-steroidal anti-inflammatory drugs and IV contrast agents are also common causes of life-threatening anaphylaxis.

Latex

Latex, or natural rubber, is a significant trigger of anaphylaxis among hospitalised patients because of frequent instrumentation and operations in which latex products are used. Avoidance is the only effective therapy, and the availability of latex-free clinic and hospital environments, including patient and operating rooms, is now a priority.¹⁶⁵ Life-threatening anaphylactic reactions to latex are very rare^{166,167} with a decade-long registry of anaphylactic deaths in England not registering any latex-associated deaths.^{168,169}

Stinging insects

The prevalence of IgE-mediated systemic reactions to insect stings is 2.8% in temperate climates, although higher in countries, such as Australia where exposure to insect stings is more common.¹⁷⁰ The stinging insects belong to the *Hymenoptera* order and include hornets, wasps, honeybees and fire ants. Most stings cause local reactions with pain and swelling at the site but progress to anaphylaxis in susceptible persons. Fatal anaphylaxis occurs in people who are re-stung after a previous sting has induced IgE antibodies. Fatal reactions occur within 10–15 min, with cardiovascular collapse being the commonest cause of death.^{168,169,171}

Foods

Life-threatening allergic reactions to food are increasing. Peanuts, seafood (in particular prawns and shellfish) and wheat are the foods associated most frequently with life-threatening anaphylaxis.¹⁷² Bronchospasm, angioedema, airway obstruction and asphyxia comprise the most common fatal mechanism.^{168,169,171}

Signs and symptoms

Anaphylaxis should be considered when two or more body systems are affected (cutaneous, respiratory, cardiovascular, neurological or gastrointestinal), with or without cardiovascular or airway involvement. Symptoms may be particularly severe in patients with asthma, those taking beta-adrenoceptor blockers and during neuraxial anaesthesia: states associated with reduced endogenous catecholamine response. The speed of the onset of signs and symptoms is related to the likely severity of the ensuing anaphylaxis.

Early signs and symptoms include urticaria, rhinitis, conjunctivitis, abdominal pain, vomiting and diarrhoea. Flushing is common but pallor may also occur. Marked upper airway (laryngeal) oedema and bronchospasm may develop, causing stridor and wheezing (or high airway pressures in ventilated patients). In asthmatics, this may be particularly severe and difficult to treat. Cardiovascular collapse is the most common peri-arrest manifestation. Vasodilation causes relative hypovolaemia, exacerbated by true volume loss as increased capillary permeability results in extravasation of intravascular fluid. Additional cardiac dysfunction may follow from underlying disease or from the development of myocardial ischaemia from adrenaline administration.^{168,169,171}

Differential diagnosis

The lack of any consistent clinical manifestation and a wide range of possible presentations may cause diagnostic difficulty. In each case, take as full a history and examination as possible. The history of previous allergic reactions, as well as that of the recent incident is important. Pay particular attention to the condition of the skin, the pulse rate, the blood pressure and the upper airways, and auscultate the chest. Measure and record the peak flow where possible. Consider the diagnosis of other conditions only after anaphylaxis has been excluded; failure to identify and treat anaphylaxis can be fatal.^{173,174}

- ACE inhibitors may cause angioedema with marked swelling of the upper airway. This reaction may occur at any time and is not related to an initial exposure to the drug. The best treatment for this form of angioedema is unclear, but early recognition and appropriate airway management are critical.¹⁷⁵
- Hereditary angioedema is familial and indistinguishable from the early angioedema of anaphylaxis or drug-related angioedema. An important

distinguishing feature is the absence of urticaria with hereditary angioedema. This is treated with C1 esterase inhibitor, either as a specific concentrate or contained within fresh frozen plasma.

- Severe asthma may present with bronchospasm and stridor, which are also common features of severe anaphylaxis. However, asthma attacks do not usually present with urticaria or angioedema.
- Rarely, panic attacks may be associated with functional stridor as a result of forced adduction of the vocal cords. As with asthma, there is no urticaria, angioedema, hypoxia or hypotension.
- Vasovagal reactions cause sudden collapse and extreme bradycardia that may be mistaken for absence of a pulse. Recovery is usually relatively rapid, and is not associated with urticaria, angioedema or bronchospasm.

Considerations in relation to treatment

Wide variations in aetiology, severity and organ involvement preclude standardised treatment recommendations. The lack of clinical trials necessitates guidelines based on consensus opinion.

Adrenaline is generally agreed to be the most important drug for any severe anaphylactic reaction. As an alpha-agonist, it reverses peripheral vasodilation and reduces oedema. Its beta-agonist properties dilate the airways, increase the force of myocardial contraction and suppress histamine and leukotriene release.

Adrenaline is most effective when given early after the onset of the reaction, but it is not without risk, particularly when given IV. When given intramuscularly, adrenaline is very safe. Adverse effects are extremely rare, and the only patient reported to have had a myocardial infarction after intramuscular injection had numerous risk factors for coronary disease. Sometimes there has been uncertainty as to whether complications (e.g., myocardial ischaemia) have been due to the effects of the allergen itself or to adrenaline given as treatment for it.^{168,176}

Rarely, adrenaline may fail to reverse the clinical manifestations of anaphylaxis, particularly in late reactions or in patients treated with beta-blockers. Other measures then assume greater importance, particularly volume replacement.

General resuscitation measures

All victims should recline in a position of comfort. Remove the likely allergen (i.e., stop drug infusion or blood transfusion). Lying flat, with or without leg elevation, may be helpful for hypotension but not helpful for breathing difficulties. Airway obstruction

can develop rapidly due to soft tissue swelling. Consider early tracheal intubation; delay may make intubation extremely difficult.

Oxygen

Give high-flow oxygen (10–15 l min⁻¹).

Adrenaline

Give adrenaline intramuscularly to all patients with clinical signs of shock, airway swelling or definite breathing difficulty; adrenaline will be absorbed rapidly. Inspiratory stridor, wheeze, cyanosis, pronounced tachycardia and decreased capillary filling indicate a severe reaction. For adults, give an IM dose of adrenaline, 0.5 ml of 1:1000 solution (500 mcg). If the patient's condition fails to improve, repeat the dose after about 5 min. In some cases several doses may be needed, particularly if improvement is transient. The IM route is preferable to SC administration because absorption is more rapid in shock.^{177,178}

IV adrenaline (in a dilution of at least 1:10,000; never 1:1000) is hazardous and must be reserved for patients with profound shock that is immediately life threatening and for special indications, for example during anaesthesia. A further 10-fold dilution to 1:100,000 adrenaline enables finer titration of the dose and increases its safety by reducing the risk of unwanted adverse effects. This should be carried out with a minimum of electrocardiographic monitoring. Doctors experienced in the use of IV adrenaline may prefer to use the IV route in any patient with signs of severe anaphylaxis.

Antihistamine

Give an H₁-antihistamine (e.g., chlorphenamine 10–20 mg) by slow IV injection. Consider also an H₂-blocker, e.g., ranitidine, 50 mg IV.¹⁷⁹

Hydrocortisone

Give hydrocortisone by slow IV injection after severe attacks to help avert late sequelae. This is particularly important for asthmatics (who are at an increased risk of severe or fatal anaphylaxis) if they have been treated with corticosteroids previously. Corticosteroids are considered as slow-acting drugs and may take up to 4–6 h to have an effect, even if given IV. However, they may help in the emergency treatment of an acute attack, and they also have a role in preventing or shortening the protracted reactions.

Inhaled bronchodilators

An inhaled beta₂ agonist, such as salbutamol (5 mg, repeated if necessary), may help reverse the refractory bronchospasm. Inhaled ipratropium (500 mcg, repeated as necessary) may be particularly useful for the treatment of bronchospasm in patients on beta-blockers. Some cases of near-fatal asthma may really be anaphylaxis, resulting in mistaken overtreatment with conventional bronchodilators rather than more specific treatment with adrenaline.¹⁴¹

Intravenous fluids

If severe hypotension does not respond rapidly to drug treatment, give fluid; a rapid infusion of 1–2 l may be required. Further fluid is likely to be necessary.

Potential therapies

Vasopressin. There are case reports that vasopressin may benefit severely hypotensive patients.^{180,181}

Atropine. Case reports also suggest that, when relative or severe bradycardia is present, there may be a role for atropine.¹⁷⁴

Glucagon. For patients unresponsive to adrenaline, especially those receiving beta-blockers, glucagon may be effective. This agent is short-acting (1–2 mg every 5 min IM, or IV). Nausea, vomiting and hyperglycaemia are common side effects.

Envenomation

Rarely, insect envenomation by bees, but not wasps, leaves a venom sac. Immediately scrape away any insect parts at the site of the sting.¹⁸² Squeezing may increase envenomation.

Cardiac arrest

In addition to the ALS drugs, consider the following therapies.

Rapid fluid resuscitation

Near-fatal anaphylaxis produces profound vasodilation and a relative hypovolaemia. Massive volume replacement is essential. Use at least two large-bore cannulae with pressure bags to give large volumes (as much as 4–8 l IV fluid may be necessary in the immediate resuscitation period).

Antihistamines

Give an antihistamine IV if antihistamine has not already been given before the arrest.¹⁷⁹

Steroids

Steroids given during a cardiac arrest will have little immediate effect but, if ROSC is restored, they may be effective in the post-resuscitation period.

Prolonged CPR

Patients with anaphylaxis are often young, with healthy hearts and cardiovascular systems. Effective CPR may maintain sufficient oxygen delivery until the catastrophic effects of the anaphylactic reaction resolve.

Airway obstruction

Airway obstruction may occur rapidly in severe anaphylaxis, particularly in patients with angioedema. Warning signs are lingual and labial swelling, hoarseness and oropharyngeal swelling. Consider early, elective intubation. As airway obstruction progresses, both LMAs and Combitubes are likely to be difficult to insert. Tracheal intubation and cricothyroidotomy will also become increasingly difficult. Attempts at tracheal intubation may exacerbate laryngeal oedema. Early involvement of a senior anaesthetist is mandatory when managing these patients.

Observation

Warn patients with even moderate attacks of the possibility of an early recurrence of symptoms and, in some circumstances, keep them under observation for 8–24 h. This caution is particularly applicable to:

- severe reactions with slow onset due to idiopathic anaphylaxis;
- reactions in severe asthmatics or with a severe asthmatic component;
- reactions with the possibility of continuing absorption of allergen;
- patients with a previous history of biphasic reactions.^{179,183–187}

A patient who remains symptom-free for 4 h after treatment may be discharged.¹⁸⁸

Investigations and further management

Measurement of mast cell tryptase may help with retrospective diagnosis of anaphylaxis.^{189,190} Take three 10-ml clotted blood samples:

- immediately after the reaction has been treated;
- about 1 h after reaction;
- about 6 h and up to 24 h after reaction.

It is important to identify the allergen after successful resuscitation from anaphylaxis, to prevent recurrence. Refer the patient to a specialist clinic. Patients at very high risk of anaphylaxis may carry their own adrenaline syringe for self-administration and wear a 'MediAlert' type bracelet. Report reactions to drugs to the appropriate monitoring agency.

7h. Cardiac arrest following cardiac surgery

Cardiac arrest following major cardiac surgery (both on and off bypass) is relatively common in the immediate postoperative phase, with a reported incidence of 0.7% in the first 24h¹⁹¹ and 1.4% within the first 8 days.¹⁹² Cardiac arrest is usually caused by specific pathology that is reversible if treated promptly and appropriately, and therefore, has a relatively high survival rate. Cardiac arrest is usually preceded by physiological deterioration,¹⁹³ although it may occur suddenly in stable patients.¹⁹¹ Continuous monitoring on the intensive care unit (ICU) enables immediate intervention at the time of arrest. Survival to hospital discharge of patients suffering from cardiac arrest during the first 24h after adult cardiac surgery is reported as 54%¹⁹²–79%^{191,194} and 41% in children.¹⁹³

Aetiology

Perioperative myocardial infarction is the commonest cause of sudden cardiac arrest and is often secondary to graft occlusion.^{191,192}

The main causes of cardiac arrest in the initial postoperative period include:

- myocardial ischaemia;
- tension pneumothorax;
- haemorrhage causing hypovolaemic shock;
- cardiac tamponade;
- disconnection of pacing system in pacing-dependent patient;
- electrolyte disturbances (particularly hypo/hyperkalaemia).

Diagnosis

An immediate decision on the likely cause of cardiac arrest must be made to enable rapid intervention and successful resuscitation. Auscultation of the chest, examination of the ECG and chest radiograph, transoesophageal/transthoracic echocardiography and measurement of blood loss from chest drains will aid in identifying the cause of the arrest. Actively seek and exclude reversible causes of cardiac arrest: the four Hs and four Ts. Myocardial ischaemia often causes myocardial irritability and progressive hypotension before an arrest. A tension pneumothorax and cardiac tamponade will cause progressive hypotension and an increasing central venous pressure. Increasing airway pressures and poor air entry in the affected lung will differentiate between the two conditions. Lack of drainage of blood from the chest drains does not exclude haemorrhage or tamponade, because drains may block with clot.

Treatment

Treatment of cardiac arrest following cardiac surgery follows the same principles of BLS and ALS that have already been described in these guidelines. Seek assistance from experienced clinicians without delay. Exclude immediately correctable causes, such as pacing-lead disconnection and tension pneumothorax. Extreme bradycardia or asystole may respond to pacing via internal pacing-wires (if present) connected to an external pacemaker. Ensure correction of hypo/hyperkalaemia and hypomagnesaemia. Rapid restoration of an adequate blood volume is important, ensuring that haemoglobin levels are maintained no lower than 8.0 g dL⁻¹. Be careful when giving IV adrenaline, as the resulting hypertension may cause catastrophic failure of anastomoses.

External chest compressions

External chest compressions may be necessary but may cause sternal subluxation, fractured ribs and damage to grafts. Continuous observation of the invasive blood pressure will enable the force of compression to be optimised. Effective external chest compressions should take precedence over the concerns of damage to grafts.

Internal cardiac massage

Mechanical factors (e.g., haemorrhage, tamponade, graft occlusion) account for a substantial proportion of causes of sudden cardiac arrest

occurring in haemodynamically stable patients during the immediate postoperative period.¹⁹¹ Correction of this pathology may require chest reopening and therefore internal cardiac massage. Up to 10% of patients may need chest reopening following cardiac surgery.¹⁹⁵ Overall survival to discharge the following internal cardiac massage is 17%¹⁹⁶–25%.¹⁹⁵ Cardiac arrest on the ICU, arrest within 24 h of surgery, and reopening within 10 min of arrest are independent predictors of survival.¹⁹⁵

The high incidence of potentially correctable mechanical causes of arrest, in conjunction with the high survival rate achieved by open CPR, supports an early approach to open-chest CPR in these patients.^{191,197} Reopen the patient's chest immediately if there is no output with external chest compressions or if there is a shockable rhythm refractory to cardioversion. Management of asystole usually requires prompt chest opening. Opening of the chest is relatively straightforward and, if indicated, should be undertaken within 10 min of cardiac arrest. Consider training the non-surgical medical staff to open the wound and remove sternal wires, while a surgeon is summoned. Make sure that a chest opening kit is immediately available on the ICU. The invasive blood pressure will guide the effectiveness of internal cardiac massage. Remove the blood clot carefully, either manually or by suctioning, to avoid damaging the grafts. Early identification and treatment of underlying pathology is challenging under these circumstances and requires an experienced surgeon.

Reinstitution of emergency cardiopulmonary bypass

The need for emergency cardiopulmonary bypass (CPB) may occur in approximately 0.8% patients, occurring at a mean of 7 h postoperatively,¹⁹⁸ and is usually indicated to correct surgical bleeding or graft occlusion and rest an exhausted myocardium. Emergency institution of CPB should be available on all units undertaking cardiac surgery. Survival to discharge the rates of 32%,¹⁹⁵ 42%¹⁹⁸ and 56.3%¹⁹⁹ have been reported when CPB is reinstated 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.¹⁹⁵ Ensuring adequate re-anticoagulation before commencing CPB, or the use of a heparin-bonded CPB circuit, is important. The need for a further period of aortic cross-clamping does not preclude a favourable outcome.¹⁹⁸

Internal defibrillation

Internal defibrillation using paddles applied directly across the ventricles requires considerably less energy than that used for external defibrillation. Biphasic shocks are substantially more effective than the monophasic shocks for direct defibrillation. For biphasic shocks, starting at 5 J creates the optimum conditions for lowest threshold and cumulative energy, whereas 10 or 20 J offers optimum conditions for more rapid defibrillation and fewer shocks.²⁰⁰ Monophasic shocks require approximately double these energy levels.²⁰⁰

7i. Traumatic cardiorespiratory arrest

Introduction

Cardiac arrest secondary to traumatic injury has a very high mortality, with an overall survival of just 2.2% (range, 0–3.7%) (Table 7.4).^{201–207} In those who survive, neurological disability is common, being absent in only 0.8% of those suffering from 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.

Comotio cordis

Comotio cordis is actual or near cardiac arrest caused by a blunt impact to the chest wall over the heart.^{208–211} A blow on the chest during the vulnerable phase of the cardiac cycle may cause malignant arrhythmias (usually VF). Syncope after chest wall impact may be caused by non-sustained arrhythmic events. Comotio cordis occurs mostly during sports (most commonly baseball) and recreational activities, and victims are usually young males (mean age 14 years). The Comotio Cordis Registry in Minneapolis is accruing 5–15 cases of comotio cordis each year. The overall survival rate from comotio cordis is 15%, but reaches 25% if resuscitation is started within 3 min.²¹¹

Trauma secondary to medical events

A cardiorespiratory arrest caused by a medical pathology (e.g., cardiac arrhythmia, hypoglycaemia, seizure) may precipitate a secondary

traumatic event (e.g., fall, road traffic accident, etc.). Traumatic injuries may not be the primary cause of a cardiorespiratory arrest.

Mechanism of injury

Blunt trauma

Of 1242 patients with cardiac arrest after blunt trauma, 19 (1.5%) survived, but only 2 (0.16%) had a good neurological outcome (Table 7.4).

Penetrating trauma

Of 839 patients with cardiac arrest after penetrating injury, there were 16 (1.9%) survivors, of whom 12 (1.4%) had a good neurological outcome (Table 7.4).

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 correlated significantly with survival.²¹⁷ In a study of penetrating trauma, pupil reactivity, respiratory activity and sinus rhythm were correlated with survival but were unreliable.²⁰⁷ Three studies reported no survivors among patients presenting with asystole or agonal rhythms.^{202,207,218} Another reported no survivors in PEA after blunt trauma.²¹⁹ Based on these studies, the American College of Surgeons and the National Association of EMS Physicians produced prehospital guidelines on withholding resuscitation.²²⁰ They recommend withholding resuscitation in:

- blunt trauma victims presenting apnoeic and pulseless, and without organised ECG activity;
- penetrating trauma victims found apnoeic and pulseless after rapid assessment for signs of life, such as pupillary reflexes, spontaneous movement or organised ECG activity.

A recent retrospective study questions these recommendations: in a series of 184 TCRA victims, several survivors met the criteria for non-resuscitation.²²¹

Treatment

Survival from TCRA is correlated with duration of CPR and prehospital time.^{205,222–226} Prolonged CPR is associated with a poor outcome; the maximum CPR time associated with a favourable outcome is 16 min.^{205,222–224} The level of prehospital 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 the essential lifesaving interventions on scene and, if the patient has signs of life, transfer rapidly to the nearest appropriate hospital. Consider on-scene thoracotomy for appropriate patients.^{227,228} Do not delay for unproven interventions, such as spinal immobilisation.²²⁹

Resuscitative thoracotomy

Prehospital. Resuscitative thoracotomy has been reported as futile if out-of-hospital time has exceeded 30 min;²²⁵ others consider thoracotomy to be futile in patients with blunt trauma requiring more than 5 min of prehospital CPR and in patients with penetrating trauma requiring more than 15 min of CPR.²²⁶ 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.²²⁷ Following this approach, of 39 patients who underwent thoracotomy at scene, 4 patients survived and 3 of these made a good neurological recovery.

Hospital. A relatively simple technique for resuscitative thoracotomy has been described recently.^{228,230} 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 EDTs.²³¹ The overall survival rate was 7.8%, and of 226 survivors (5%), only 34 (15%) exhibited a neurological deficit. The investigators concluded the following:

- After blunt trauma, EDT should be limited to those with vital signs on arrival and a witnessed cardiac arrest (estimated survival rate 1.6%).
- Emergency department thoracotomy is best applied to patients with penetrating cardiac injuries, who arrive at the trauma centre after short on-scene and transport times, with witnessed signs of life or ECG activity (estimated survival rate 31%).
- Emergency department thoracotomy should be undertaken in penetrating non-cardiac thoracic injuries even though survival rates are low.
- Emergency department thoracotomy should be undertaken in patients with exsanguinating abdominal vascular injury even though survival rates are low. This procedure should be used as

Table 7.4 Survival after traumatic cardiac arrest

Source	Entry criteria	Number of survivors neurologically intact	Number of survivors of penetrating trauma neurologically intact	Number of survivors of blunt trauma neurologically intact
Bouillon ²¹²	Pulseless, requiring CPR at scene	224 4 3		
Battistella ²⁰²	Pulseless, requiring CPR at scene, en route or in ED	604 16 9	300 12 9	304 4 0
Pasquale ²⁰⁶	CPR before or on hospital admission	106 3	21 1	85 2
Fisher ²¹³	Children requiring CPR before or on admission after blunt trauma	65 1 0		38 1 0
Hazinski ²¹⁴	Children requiring CPR or being severely hypotensive on admission after blunt trauma	38 1 0		65 1 0
Shimazu ²⁰³	TCRA on admission	267 7 4		
Calkins ²¹⁵	Children requiring CPR after blunt trauma	25 2 2		25 2 2
Yanagawa ²¹⁶	OHCA in blunt trauma	332 6 0		332 6 0
Rosemurgy ²⁰¹	CPR before admission	138 0 0	42 0 0	96 0 0
Stratton ²⁰⁷	Unconscious, pulseless at scene	879 9 3	497 4 3	382 5 0
Cera ²¹⁷	CPR on admission	161 15 ?		

For each study, the first number indicates the number of patients in cardiac arrest, the second indicates the numbers of survivors and the third indicates the number of survivors with a good neurological outcome. CPR = cardiopulmonary resuscitation; ED = emergency department; TCRA = traumatic cardiorespiratory arrest; OHCA = out-of-hospital cardiac arrest.

an adjunct to definitive repair of abdominal vascular injury.

Airway management

Effective airway management is essential to maintain oxygenation of the severely compromised trauma victim. In one study, tracheal intubation on-scene of patients with TCRA doubled the tolerated period of CPR, i.e., the mean time of CPR for sur-

vivors who were intubated in the field was 9.1 min versus 4.2 min for those who were not intubated.²²⁴

Tracheal intubation of trauma victims is a difficult procedure with a high failure rate if carried out by less experienced care providers.^{232–235} Use the 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 states positive pressure ventilation causes further circulatory depression, or even cardiac arrest, by impeding venous return to the heart.²³⁶ Monitor ventilation with capnometry 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 thoracostomy, which is likely to be more effective than needle thoracostomy and quicker than inserting a chest tube.²³⁷

Effectiveness of chest compressions in TCRA

In hypovolaemic cardiac arrest or cardiac tamponade, chest compressions are unlikely to be as effective as in cardiac arrest from other causes;²³⁸ nonetheless, return of spontaneous circulation with ALS in patients with TCRA is well described. 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.

Pericardiocentesis

In patients with suspected trauma-related cardiac tamponade, needle pericardiocentesis is probably not a useful procedure.²³⁹ There is no evidence of benefit in the literature. It may increase scene time, cause myocardial injury and delay effective therapeutic measures, such as emergency thoracotomy.

Fluids and blood transfusion on scene

Fluid resuscitation of trauma victims before haemorrhage is controlled is controversial, and there is no clear consensus on when it should be started and what fluids should be given.²⁴⁰ Limited evidence and general consensus support a more con-

servative approach to IV fluid infusion, with permissive hypotension until surgical haemostasis is achieved.^{241,242} In the UK, the National Institute for Clinical Excellence (NICE) has published guidelines on prehospital fluid replacement in trauma.²⁴³ 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. Prehospital fluid therapy may have a role in prolonged entrapments, but there is no reliable evidence for this.^{244,245}

Ultrasound

Ultrasound is a valuable tool in the evaluation of the compromised trauma victim. Haemoperitoneum, haemo- or pneumothorax and cardiac tamponade can be diagnosed reliably in minutes even in the prehospital phase.²⁴⁶ Diagnostic peritoneal lavage and needle pericardiocentesis have virtually disappeared from clinical practice since the introduction of sonography in trauma care. Prehospital ultrasound is now available, although its benefits are yet to be proven.

Vasopressors

The possible role of vasopressors (e.g., vasopressin) in trauma resuscitation is unclear and is based mainly on case reports.²⁴⁷

7j. Cardiac arrest associated with pregnancy

Overview

Mortality related to pregnancy in developed countries is rare, occurring in an estimated 1:30,000 deliveries.²⁴⁸ The fetus must always be considered when an adverse cardiovascular event occurs in a pregnant woman. Resuscitation guidelines for pregnancy are based largely on case series and scientific rationale. Most reports address the causes in developed countries, whereas the majority of pregnancy-related deaths occur in the developing world.

Significant physiological changes occur during pregnancy, e.g., cardiac output, blood volume, minute ventilation and oxygen consumption all increase. Furthermore, the gravid uterus may 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 UK²⁴⁸ showed that maternal death was associated with:

- pre-existing cardiac disease;
- thromboembolism;
- suicide;
- hypertensive disorders of pregnancy;
- sepsis;
- ectopic pregnancy;
- haemorrhage;
- amniotic fluid embolism;

Pregnant women can also suffer the same causes of cardiac arrest 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 caval 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 100% oxygen.
- Give a fluid bolus.
- Immediately re-evaluate the need for any drugs being given.
- Seek expert help early.

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.^{249,250} After cardiac arrest, the compromise in venous return and cardiac output by the gravid uterus limit the effectiveness of chest compressions. Non-cardiac arrest data show that the gravid uterus can be shifted away from the cava in most cases by placing the patient in 15 degrees of left lateral decubitus position.²⁵¹ Tilt may be accomplished by manual or mechanical means. There is no evidence to guide the hand position for optimum chest compressions in the pregnant patient. A hand position higher than the normal position for chest compression may be needed to adjust for the elevation

of the diaphragm and abdominal contents caused by the gravid uterus. Attempt defibrillation using standard energy doses.²⁵² 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.

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.²⁵³ Tracheal intubation may be more difficult in the pregnant patient.²⁵⁴ Expert help, a failed intubation drill and the use of alternative airway devices may be needed (see section 4d).²⁵⁵

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 to 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. Associations include ectopic pregnancy, placental abruption, placenta praevia and uterine rupture.²⁴⁸ 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;²⁵⁶
- correction of coagulopathy. There may be a role for recombinant Factor VIIa;²⁵⁷
- oxytocin and prostaglandins to correct uterine atony;²⁵⁸
- uterine compression sutures;²⁵⁹
- radiological embolisation;²⁶⁰
- hysterectomy;
- aortic cross-clamping in catastrophic haemorrhage.²⁶¹

Drugs

Iatrogenic overdose is possible in eclamptic women receiving magnesium sulphate, particularly if the woman becomes oliguric. Give calcium to treat magnesium toxicity (see life-threatening electrolyte abnormalities).

Central neural blockade for analgesia or anaesthesia may cause problems due to sympathetic blockade (hypotension, bradycardia) or local anaesthetic toxicity.²⁶²

Cardiovascular disease

Pulmonary hypertension causes most deaths from congenital heart disease. Peripartum cardiomyopathy, myocardial infarction and aneurysm or dissection of the aorta or its branches cause most deaths from acquired cardiac disease.^{263,264} Patients with known cardiac disease need to be managed in a specialist unit. Pregnant women with coronary artery disease may suffer an acute coronary syndrome. Percutaneous coronary intervention is the reperfusion strategy of choice for ST-elevation myocardial infarction in pregnancy because fibrinolytics are relatively contraindicated.²⁶⁵

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.^{266,267} Magnesium sulphate is effective in preventing approximately half of the cases of eclampsia developing in labour or immediately postpartum in women with pre-eclampsia.

Life-threatening pulmonary embolism

Successful use of fibrinolytics for massive, life-threatening pulmonary embolism in pregnant women has been reported.^{268–271}

Amniotic fluid embolism

Amniotic fluid embolism may present with breathlessness, cyanosis, arrhythmias, hypotension and haemorrhage associated with disseminated intravascular coagulopathy.²⁷² Presentation is variable and may be similar to anaphylaxis. Treatment is supportive, as there is no specific therapy. Successful use of cardiopulmonary bypass for women suffering life-threatening amniotic fluid embolism during labour and delivery is reported.²⁷³

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.^{274–276} 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.^{274,277–279} This requires that the provider commence the hysterotomy at about 4 min after cardiac arrest. Delivery will relieve caval compression and 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

Consider gestational age. 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. Portable ultrasounds are available in some emergency departments and may aid in determination of gestational age (in experienced hands) and positioning, provided their use does not delay the decision to perform emergency hysterotomy.²⁸⁰

- At gestational age <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.

Planning for emergencies. Advanced life support in pregnancy requires coordination of maternal resuscitation, Caesarean delivery of the fetus and newborn resuscitation within 5 min. To achieve this, units likely to deal with cardiac arrest in pregnancy should:

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

7k. 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).²⁸¹ Electrocution from lightning strikes is rare, but worldwide it causes 1000 deaths each year.²⁸²

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 ventricular fibrillation (VF) if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon).²⁸³ 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 kilovolts over a few ms. 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 industry 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.²⁸⁴ Explosive force may cause blunt trauma.²⁸⁵ The pattern and severity of injury from a lightning strike varies considerably, even among affected individuals from a single group.^{286–288} As with industrial and domestic electric shock, death is caused by cardiac^{287–291} or respiratory arrest.^{284,292} 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.^{293–295}

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.²⁸⁴

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.²⁸⁴

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;²⁹⁴ 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.²⁹¹
- Consider early surgical intervention in patients with severe thermal injuries.
- Maintain spinal immobilisation if there is a likelihood of head or neck trauma.^{296,297}
- Conduct a thorough secondary survey to exclude traumatic injuries caused by tetanic muscular contraction or by the person being thrown.^{297,298}
- 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 suffer 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.²⁹² Dilated or non-reactive pupils should never be used as a prognostic sign, particularly in patients suffering a lightning strike.²⁸⁴

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.²⁹⁹

Further treatment and prognosis

Immediate resuscitation in young victims of cardiac arrest due to electrocution can result in 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 suffered:

- 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 minimise the prevalence and severity of electrical injury.

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