

Learning outcomes

To understand how resuscitation techniques are modified in the special circumstances of:

- ▶ **Life-threatening electrolyte disorders**
- ▶ **Poisoning**
- ▶ **Accidental hypothermia**
- ▶ **Hyperthermia**
- ▶ **Drowning**
- ▶ **Asthma**
- ▶ **Anaphylaxis**
- ▶ **Cardiac arrest following cardiac surgery**
- ▶ **Trauma**
- ▶ **Pregnancy**
- ▶ **Electrocution**

Introduction

Resuscitation needs to be modified in specific circumstances. Early recognition of signs and symptoms and effective treatment will often prevent cardiac arrest. These conditions account for a large proportion of cardiac arrests in younger patients with no co-existing disease. It is essential to ask for appropriate expert help early for most of these conditions as they will require specialist interventions.

Survival in all these conditions still relies on using the ABCDE approach to help prevent cardiac arrest. If cardiac arrest does occur, high quality CPR with minimal interruption and treatment of reversible causes are still the most important interventions.

Life-threatening electrolyte disorders

Electrolyte abnormalities can cause cardiac arrhythmias or cardiorespiratory arrest. Potassium disorders pose the greatest risk. Consider starting treatment in life-threatening electrolyte disorders before laboratory results are available. Electrolyte values for definitions are quoted as a guide to clinical decision-making. The precise values that trigger treatment decisions will depend on the patient's clinical condition and rate of change of electrolyte values.

Prevention of electrolyte disorders

- Treat life-threatening electrolyte abnormalities before cardiac arrest occurs.
- Remove precipitating factors (e.g. drugs) and monitor electrolyte concentrations to prevent recurrence of the abnormality.
- Monitor renal function in patients at risk of electrolyte disorders (e.g. patients with chronic kidney disease, heart failure).
- Review renal replacement therapy (e.g. haemodialysis) 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 intracellular and extracellular fluid compartments. Evaluation of serum potassium must take into consideration the effects of changes in serum pH. When serum pH decreases (acidaemia), serum potassium concentration increases, because potassium shifts from the cellular to the vascular space. When serum pH increases (alkalaemia), serum potassium concentration decreases because potassium shifts into cells. Anticipate the effects of pH changes on serum potassium during therapy for hyperkalaemia or hypokalaemia.

Hyperkalaemia

Hyperkalaemia is usually caused by increased potassium release from cells or impaired excretion by the kidneys.

Definition

There is no universal definition. We have defined hyperkalaemia as a serum potassium concentration > 5.5 mmol l⁻¹; in practice, hyperkalaemia is a continuum. As the potassium concentration increases, the risk of adverse events increases and the need for urgent treatment increases. Severe hyperkalaemia has been defined as a serum potassium concentration > 6.5 mmol l⁻¹.

Causes

The causes of hyperkalaemia include:

- 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 (skeletal muscle (rhabdomyolysis), tumour lysis, haemolysis);
- metabolic acidosis;
- endocrine disorders (Addison's disease);
- hyperkalaemic periodic paralysis;
- diet (may be the principal cause in patients receiving chronic renal replacement therapy).

Abnormal erythrocytes or thrombocytosis may cause a spuriously high potassium concentration. The risk of hyperkalaemia increases when there is a combination of causative factors such as the concomitant use of ACEI and NSAIDs or potassium sparing diuretics.

Recognition of hyperkalaemia

Exclude hyperkalaemia in all patients with an arrhythmia or cardiac arrest. Patients can present with weakness progressing to flaccid paralysis, paraesthesia, or depressed deep tendon reflexes. The effect of hyperkalaemia on the ECG depends on the absolute serum potassium concentration as well as the rate of increase (Figure 12.1).

ECG changes with hyperkalaemia are usually progressive and include:

- first degree heart block (prolonged PR interval) (> 0.2 s);
- flattened or absent P waves;
- tall, peaked (tented) T waves (T wave larger than R wave in more than one lead);
- ST-segment depression;
- S and T wave merging (sine wave pattern);
- widened QRS (> 0.12 s);
- bradycardia (sinus bradycardia or AV block);
- ventricular tachycardia;
- cardiac arrest (PEA, VF/VT, asystole).

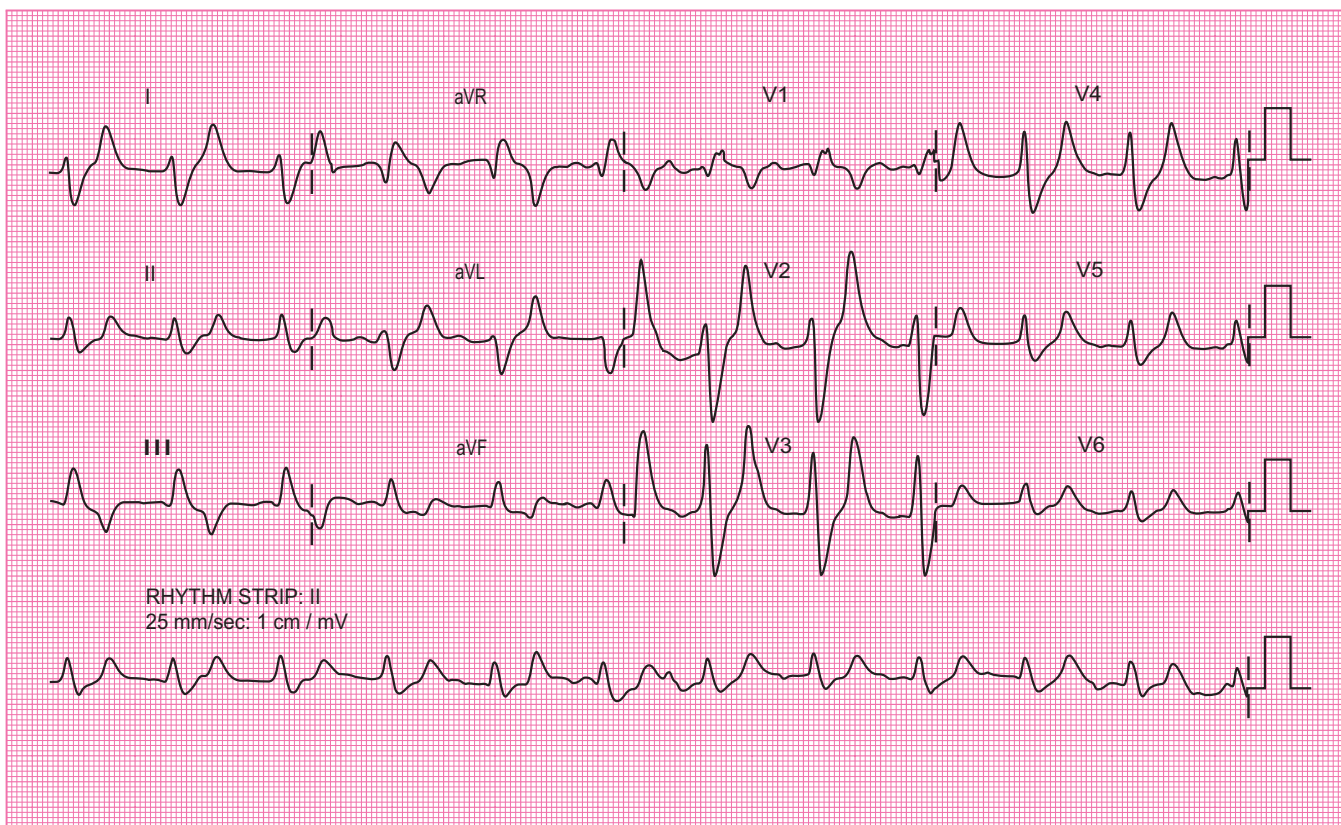


Figure 12.1 12-lead ECG showing features of hyperkalaemia

Most patients will have ECG abnormalities at a serum potassium concentration $> 6.7 \text{ mmol l}^{-1}$. The use of a blood gas analyser that measures potassium helps reduce delays in recognition.

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 concentration 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. Involve expert help from renal or intensive care teams at an early stage especially for those patients who might require renal replacement therapies (e.g. haemodialysis).

Patient not in cardiac arrest

Assess ABCDE (Airway, Breathing, Circulation, Disability, Exposure) and correct any abnormalities. If hypovolaemic, give fluid to enhance urinary potassium excretion. Obtain intravenous access, check serum potassium and record an ECG. Treatment is determined according to severity of hyperkalaemia. Approximate values are provided to guide treatment.

Mild elevation ($5.5 - 5.9 \text{ mmol l}^{-1}$): remove potassium from the body with:

- Potassium exchange resins - calcium resonium 15 - 30 g OR sodium polystyrene sulfonate (Kayexalate) 15 - 30 g in 50 - 100 ml of 20 % sorbitol, given either orally or by retention enema (onset in 1 - 3 h; maximal effect at 6 h), or
- Diuretics: furosemide 1 mg kg^{-1} IV slowly (onset with the diuresis).

Address the cause of hyperkalaemia to correct and avoid further rise in serum potassium (e.g. drugs, diet)

Moderate elevation ($6 - 6.4 \text{ mmol l}^{-1}$) without ECG changes: use strategies above plus:

- Shift potassium into cells with glucose/insulin: 10 units short-acting Insulin and 25 g glucose IV over 15 - 30 min (onset in 15 - 30 min; maximal effect at 30 - 60 min; monitor blood glucose).

- Consider renal replacement therapy (e.g. haemodialysis) to remove potassium from the body if patient is oliguric or potassium level increasing or not improving. Haemodialysis is more efficient than peritoneal dialysis at removing potassium (immediate onset; 25 - 30 mmol potassium h^{-1} removed with haemodialysis). Seek expert help.

Severe elevation ($\geq 6.5 \text{ mmol l}^{-1}$) without ECG changes: seek expert help and shift potassium into cells with:

- Glucose/insulin (see above)
- Salbutamol 5 mg nebulised. Several doses (10 - 20 mg) may be required (onset in 15 - 30 min).
- Sodium bicarbonate: 50 mmol IV over 5 to 15 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.

Remove potassium from the body with multiple strategies above.

Severe elevation ($\geq 6.5 \text{ mmol l}^{-1}$) WITH toxic ECG changes (Figure 12.1): SEEK EXPERT HELP and protect the heart first with:

- Calcium chloride: 10 ml 10% calcium chloride IV over 2 - 5 min to antagonise the toxic effects of hyperkalaemia at the myocardial cell membrane. This protects the heart by reducing the risk of VF, but does not lower serum potassium (onset in 1 - 3 min).
- Use potassium removal and shifting strategies stated above.
- Prompt specialist referral is essential. In hospitals without a dedicated renal unit, intensive care units can often provide emergency renal replacement therapies.

Patient in cardiac arrest

Modifications to BLS

There are no modifications to basic life support in the presence of electrolyte abnormalities.

Modifications to ALS

Follow the ALS algorithm. Hyperkalaemia can be confirmed rapidly using a blood gas analyser if available.

Cardiac arrest: protect the heart first; then use shifting and removal strategies

- Calcium chloride: 10 ml 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).
- Glucose/insulin: 10 units short-acting insulin and 25 g glucose IV by rapid injection.
- Haemodialysis: consider this for cardiac arrest induced by hyperkalaemia which is resistant to medical treatment. Several dialysis modes have been used safely and effectively in cardiac arrest, but this may only be available in specialist centres that offer acute renal replacement therapy in critically ill patients.

Indications for haemodialysis

Haemodialysis is the most effective method for removal of potassium from the body. The principle mechanism of action is the diffusion of potassium ions across the membrane down the potassium ion gradient. The typical decline in serum potassium is 1 mmol l⁻¹ in the first 60 min, followed by 1 mmol l⁻¹ over the next 2 h.

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

Cardiac arrest during haemodialysis

- Primary cardiac arrest is common in patients on long-term haemodialysis
- Call the resuscitation team and seek expert help immediately.
- Start resuscitation according to standard protocols ensuring high quality CPR and minimising interruptions.
- A trained dialysis nurse should be assigned to the dialysis machine.
- VF/VT is more common in patients undergoing haemodialysis than in the general population.
- All of the standard reversible causes (4 Hs and 4 Ts) apply to dialysis patients. Electrolyte disorders, particularly hyperkalaemia, and fluid overload (e.g. pulmonary oedema) are most common causes.
- Some haemodialysis machine manufacturers

recommend disconnection from dialysis equipment for defibrillation. Renal units should have a protocol based on their equipment for disconnection for defibrillation and a number of methods have been described. In clinical practice, following standard safety protocols for defibrillation will be safe for the patient and resuscitation team.

- In life-threatening circumstances and cardiac arrest, vascular access used for dialysis can be used to give drugs.

Hypokalaemia

Hypokalaemia is common in hospital patients. 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 serum potassium < 3.5 mmol l⁻¹. Severe hypokalaemia is defined as potassium < 2.5 mmol l⁻¹ and may be associated with symptoms.

Causes

Causes of hypokalaemia include:

- gastrointestinal losses (diarrhoea);
- drugs (diuretics, laxatives, steroids, adrenaline, isoprenaline, etc);
- renal losses (renal tubular disorders, diabetes insipidus, dialysis);
- endocrine disorders (Cushing's Syndrome, hyperaldosteronism);
- metabolic alkalosis;
- magnesium depletion;
- poor dietary intake.

Treatment for hyperkalaemia can 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 or constipation. In severe cases (K⁺ < 2.5 mmol l⁻¹), rhabdomyolysis, ascending paralysis and respiratory difficulties may occur.

ECG features of hypokalaemia are:

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

Treatment of hypokalaemia

This depends on the severity of hypokalaemia and the presence of symptoms and ECG abnormalities. Gradual replacement of potassium is preferable, but in an emergency intravenous potassium is required. Seek

expert help. The maximum recommended IV infusion rate 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 or has occurred. Continuous ECG monitoring is essential during IV infusion. Adjust the dose after repeated sampling of serum potassium levels.

Patients who are potassium deficient can also be deficient in magnesium. Repletion of magnesium stores will facilitate more rapid correction of hypokalaemia and is recommended in severe cases of hypokalaemia.

Calcium and magnesium disorders

The recognition and management of calcium and magnesium disorders is summarised in Table 12.1.

Disorder	Causes	Presentation	ECG	Treatment
Hypercalcaemia Total Calcium* > 2.6 mmol l ⁻¹	Primary or tertiary hyperparathyroidism Malignancy Sarcoidosis Drugs	Confusion Weakness Abdominal pain Hypotension Arrhythmias Cardiac arrest	Short QT interval Prolonged QRS Interval Flat T waves AV block Cardiac arrest	Fluid replacement IV Furosemide 1mg kg ⁻¹ IV Hydrocortisone 200 - 300mg IV Pamidronate 30 - 90mg IV Treat underlying cause
Hypocalcaemia Total Calcium* < 2.1 mmol l ⁻¹	Chronic renal failure Acute pancreatitis Calcium channel blocker overdose Toxic shock syndrome Rhabdomyolysis Tumour lysis syndrome	Paraesthesia Tetany Seizures AV - block Cardiac arrest	Prolonged QT interval T wave inversion Heart block Cardiac arrest	Calcium chloride 10% 10 - 40ml IV Magnesium sulphate 50% 4 - 8 mmol (if necessary) IV
Hypermagnesaemia [Magnesium] > 1.1 mmol l ⁻¹	Renal failure Iatrogenic	Confusion Weakness Respiratory depression AV - block Cardiac arrest	Prolonged PR and QT intervals T wave peaking AV - block Cardiac arrest	Consider treatment when [Magnesium] > 1.75 mmol l ⁻¹ Calcium chloride 10% 5-10ml IV repeated if necessary Ventilatory support if necessary Saline diuresis - 0.9% saline
Hypomagnesaemia [Magnesium] < 0.6 mmol l ⁻¹	GI loss Polyuria Starvation Alcoholism Malabsorption	Tremor Ataxia Nystagmus Seizures Arrhythmias - torsade de pointes Cardiac arrest	Prolonged PR and QT Intervals ST-segment depression T-wave inversion Flattened P waves Increased QRS duration Torsade de pointes	Severe or symptomatic: 2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 15 min. Torsade de pointes: 2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 10 min. Seizure: 2 g 50% magnesium sulphate (4 ml; 8 mmol) IV over 10 min.

Table 12.1 Calcium and magnesium disorders

* A normal *total calcium* is about 2.2 to 2.6 mmol l⁻¹. A normal *ionized calcium* is about 1.1 to 1.3 mmol l⁻¹. Calcium values need to be interpreted with caution. Seek expert help if not sure. Total calcium depends on serum albumin values and will need to be corrected for low albumin values (corrected total calcium). Ionized calcium values are often measured by blood gas machines. It is important not to confuse ionized calcium, total calcium and corrected calcium values.

Poisoning

Poisoning is an infrequent cause of cardiac arrest, but remains a leading cause in victims younger than 40 years. It is also a common cause of non-traumatic coma in this age group.

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 chemical, biological, radiological or nuclear (CBRN) exposure. Decontamination and safe management for individual or mass casualty incidents is not part of this manual.

Initial treatment

Supportive care based on the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach to prevent cardiorespiratory arrest whilst awaiting drug elimination is the mainstay of treatment. Airway obstruction and respiratory arrest secondary to a decreased conscious level is common. Alcohol excess is often present with self-poisoning.

- After opening and clearing the airway, check for breathing and a pulse (if trained to do so). Avoid mouth-to-mouth ventilation in the presence of toxins such as cyanide, hydrogen sulphide, corrosives and organophosphates. Ventilate the patient's lungs using a pocket mask or bag-mask and the highest possible concentration of oxygen. In paraquat poisoning, lung injury may be exacerbated by high concentrations of oxygen; adjust the inspired oxygen concentration according to pulse oximetry or arterial blood gases.
- There is a high incidence of pulmonary aspiration of gastric contents after poisoning. In unconscious patients who cannot protect their airway, use a rapid sequence induction with cricoid pressure to intubate the trachea and decrease the risk of aspiration. This must be undertaken by persons trained and competent in the technique.
- Provide standard basic and advanced life support if cardiac arrest occurs.
- Cardioversion is indicated for life-threatening tachyarrhythmia. Use the guidelines for peri-arrest arrhythmias (Chapter 11). Try to correct reversible causes.
- Drug-induced hypotension is common after self-poisoning. This usually responds to fluid therapy, but

occasionally vasopressors (e.g. noradrenaline infusion) are required.

- Once resuscitation is under way, try to identify the poison(s). Relatives, friends and ambulance crews can usually provide useful information. Patient examination may give 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.
- Patients with life-threatening features or at risk of further deterioration should be cared for in critical care settings.
- Consult a regional or national poisons centre for information on treatment of the poisoned patient. In the UK, specialist advice about specific poisons can be obtained by accessing TOXBASE® (www.toxbase.org). Similar centres exist in other countries. The World Health Organization lists poison centres at: www.who.int/ipcs/poisons/centre/directory/en/

Specific treatments

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

Therapies include limiting absorption of ingested poisons, enhancing elimination, or the use of specific antidotes. Seek advice from a poisons centre for up-to-date guidance for severe or uncommon poisonings.

- Activated charcoal adsorbs certain drugs. Its value decreases over time after ingestion. There is little evidence that treatment with activated charcoal improves clinical outcome. Consider giving a single dose of activated charcoal to patients who have ingested a potentially toxic amount of a poison known to be adsorbed by activated charcoal up to one hour previously. Give only to patients with an intact or protected airway. Multiple doses may be beneficial in life-threatening poisoning with carbamazepine, dapsone, phenobarbital, quinine and theophylline.
- Gastric lavage followed by activated charcoal therapy is useful only within one hour of ingesting the poison. 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.

- Whole-bowel irrigation can reduce drug absorption by cleansing the gastro-intestinal tract by enteral administration of a polyethylene glycol solution. Consider in potentially toxic ingestion of sustained release or enteric-coated drugs, oral iron poisoning, and the removal of ingested packets of illicit drugs.
- Laxatives (cathartics) or emetics (e.g. ipecacuanha) have no role in the management of the acutely poisoned patient and are not recommended.
- Urine alkalinisation (urine pH > 7.5) by giving IV sodium bicarbonate can be useful in moderate to severe salicylate poisoning in patients who do not need haemodialysis).
- Consider haemodialysis for poisoning with methanol, ethylene glycol, salicylates, and lithium. Charcoal haemoperfusion may be indicated for intoxication with carbamazepine, phenobarbital, phenytoin, or theophylline.
- Consider the use of lipid emulsion (Intralipid) for cardiac arrest caused by local anaesthetic toxicity (see below).
- Specific antidotes include: acetylcysteine for paracetamol; high-dose atropine for organophosphate insecticides; sodium nitrite, sodium thiosulfate, hydroxocobalamin, and amyl nitrite for cyanides; digoxin-specific Fab antibodies for digoxin; flumazenil for benzodiazepines; naloxone for opioids.

Specific antidotes

This section addresses only some causes of cardiac arrest from poisoning.

Opioid poisoning

Opioid poisoning causes respiratory depression, pinpoint pupils and coma followed by respiratory arrest. The opioid antagonist naloxone rapidly reverses these effects. There are fewer adverse events when the airway is opened and patients receive oxygen and ventilation (e.g. with pocket mask or bag-mask) before naloxone in opioid-induced respiratory depression; however, the use of naloxone may prevent the need for intubation.

The route for giving naloxone depends on the skills of the rescuer: intravenous (IV), intramuscular (IM), subcutaneous (SC), and intranasal (IN) routes can be used. The non-IV routes may 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, 800 mcg IM, 800 mcg SC or

2 mg IN. Large opioid overdoses require titration to a total naloxone dose of 6 - 10 mg. The duration of action of naloxone is 45 - 70 min, but respiratory depression may 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. Give increments of naloxone until the victim is breathing adequately and has protective airway reflexes.

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

Cardiac arrest is usually secondary to a respiratory arrest and associated with severe brain hypoxia. Prognosis is poor. Giving naloxone is unlikely to be harmful. Once cardiac arrest has occurred, follow standard resuscitation guidelines.

Benzodiazepines

Overdose of benzodiazepines can cause loss of consciousness, respiratory depression and hypotension. Flumazenil, a competitive antagonist of benzodiazepines, should be used only for reversal of sedation caused by a single ingestion of any of the benzodiazepines and when there is no history or risk of seizures. Reversal of benzodiazepine intoxication with flumazenil can cause significant toxicity (seizure, arrhythmia, hypotension, and withdrawal syndrome) in patients with benzodiazepine dependence or co-ingestion of proconvulsant medications such as tricyclic antidepressants. Do not use flumazenil routinely in the comatose overdose patient. There are no specific modifications required for cardiac arrest caused by benzodiazepines.

Tricyclic antidepressants

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

A widening QRS complex and right axis deviation indicates a greater risk of arrhythmias (Figure 12.2). Sodium bicarbonate should be considered for the treatment of tricyclic-induced ventricular conduction abnormalities. While no study has investigated the optimal target arterial pH with bicarbonate therapy, a pH of 7.45 - 7.55 has been commonly accepted.

Local anaesthetic toxicity

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

Patients with both cardiovascular collapse and cardiac arrest attributable to local anaesthetic toxicity may benefit from treatment with intravenous 20% lipid emulsion in addition to standard advanced life support. Give an initial intravenous bolus of 1.5 ml kg⁻¹ 20% lipid emulsion followed by an infusion at 15 ml kg⁻¹ h⁻¹. Give up to three bolus doses of lipid at 5-minute intervals and continue the infusion until the patient is stable or has received up to a maximum of 12 ml kg⁻¹ of lipid emulsion (Figure 12.3).

Cocaine toxicity

Sympathetic overstimulation associated with cocaine toxicity may cause agitation, symptomatic tachycardia, hypertensive crisis, hyperthermia and myocardial ischaemia with angina. Small doses of intravenous benzodiazepines (midazolam, diazepam, lorazepam) are effective first-line drugs. Glyceryl trinitrate and phentolamine can reverse cocaine-induced coronary vasoconstriction. Use nitrates only as second-line therapy for myocardial ischaemia. Possible myocardial necrosis should be assessed using the ECG and cardiac markers (e.g. troponin) in patients with cocaine-related chest pain. If cardiac arrest occurs, follow standard resuscitation guidelines.

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 can 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-receptor blockade. Heart block and ventricular arrhythmias associated with digoxin or digitalis glycoside poisoning may be treated effectively with digoxin-specific antibody fragments.

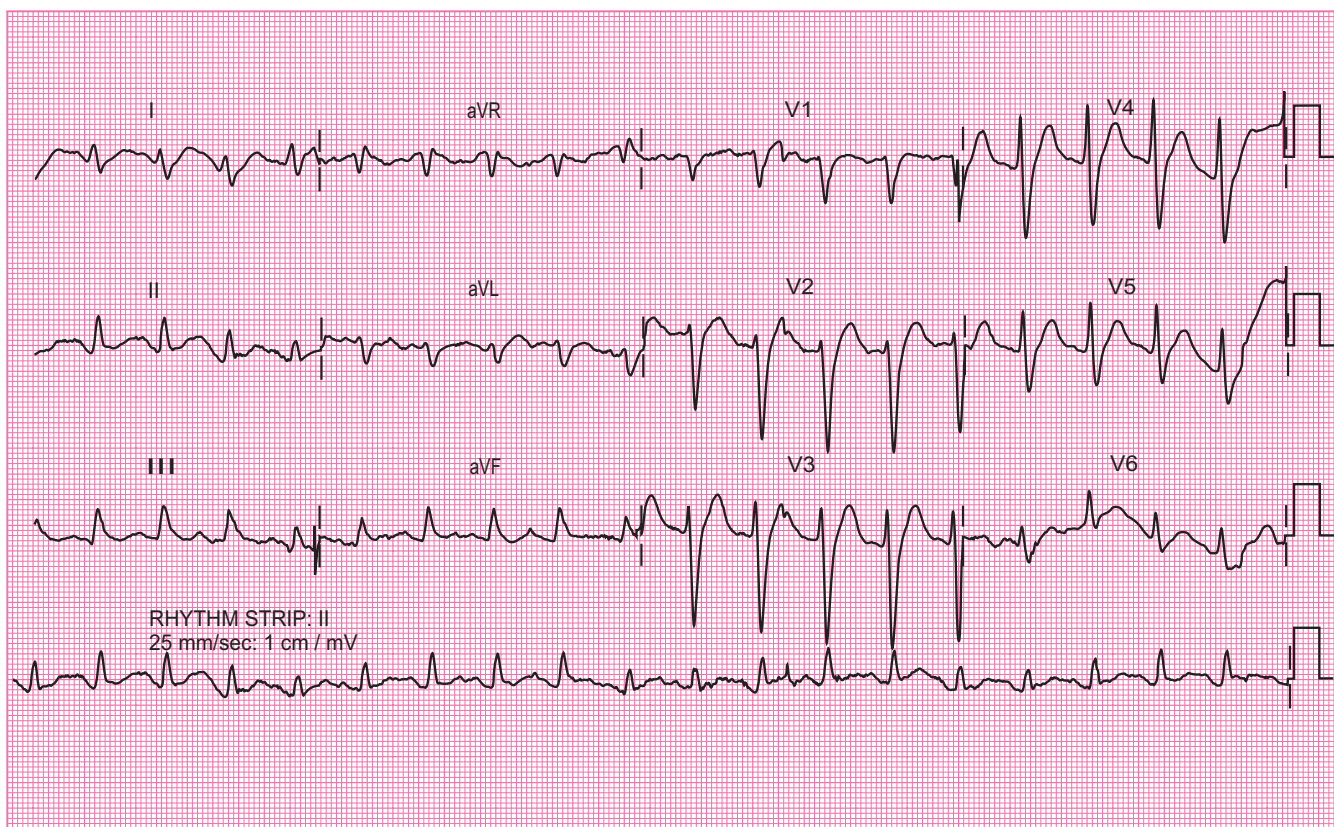


Figure 12.2 12-lead ECG showing features of severe tricyclic antidepressant toxicity

AAGBI Safety Guideline

Management of Severe Local Anaesthetic Toxicity



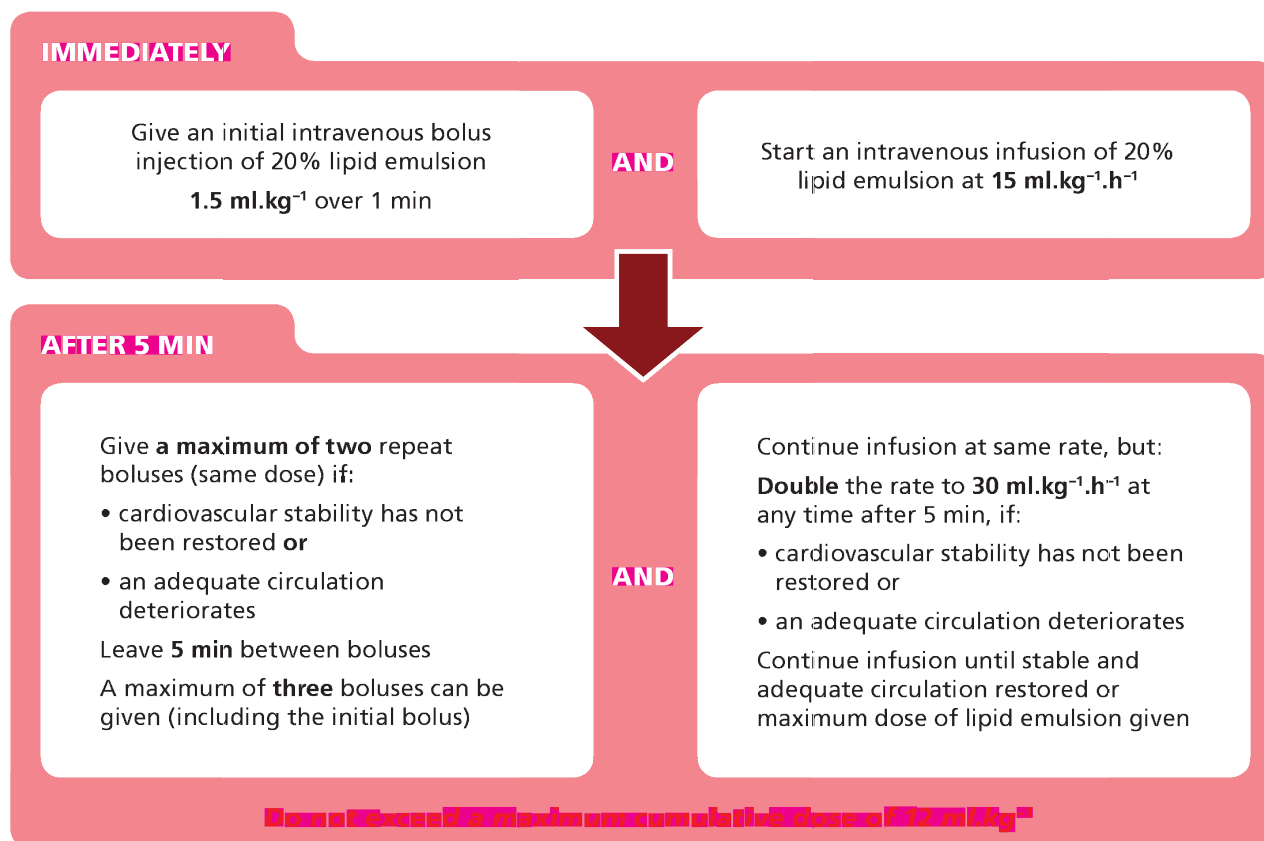
1 Recognition	Signs of severe toxicity: <ul style="list-style-type: none"> • Sudden alteration in mental status, severe agitation or loss of consciousness, with or without tonic-clonic convulsions • Cardiovascular collapse: sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias may all occur • Local anaesthetic (LA) toxicity may occur some time after an initial injection 		
2 Immediate management	<ul style="list-style-type: none"> • Stop injecting the LA • Call for help • Maintain the airway and, if necessary, secure it with a tracheal tube • Give 100% oxygen and ensure adequate lung ventilation (hyperventilation may help by increasing plasma pH in the presence of metabolic acidosis) • Confirm or establish intravenous access • Control seizures: give a benzodiazepine, thiopental or propofol in small incremental doses • Assess cardiovascular status throughout • Consider drawing blood for analysis, but do not delay definitive treatment to do this 		
3 Treatment	<table border="1"> <tr> <td data-bbox="507 913 986 1547"> IN CIRCULATORY ARREST <ul style="list-style-type: none"> • Start cardiopulmonary resuscitation (CPR) using standard protocols • Manage arrhythmias using the same protocols, recognising that arrhythmias may be very refractory to treatment • Consider the use of cardiopulmonary bypass if available GIVE INTRAVENOUS LIPID EMULSION (following the regimen overleaf) <ul style="list-style-type: none"> • Continue CPR throughout treatment with lipid emulsion • Recovery from LA-induced cardiac arrest may take >1 h • Propofol is not a suitable substitute for lipid emulsion • Lidocaine should not be used as an anti-arrhythmic therapy </td><td data-bbox="986 913 1452 1547"> WITHOUT CIRCULATORY ARREST Use conventional therapies to treat: <ul style="list-style-type: none"> • hypotension, • bradycardia, • tachyarrhythmia CONSIDER INTRAVENOUS LIPID EMULSION (following the regimen overleaf) <ul style="list-style-type: none"> • Propofol is not a suitable substitute for lipid emulsion • Lidocaine should not be used as an anti-arrhythmic therapy </td></tr> </table>	IN CIRCULATORY ARREST <ul style="list-style-type: none"> • Start cardiopulmonary resuscitation (CPR) using standard protocols • Manage arrhythmias using the same protocols, recognising that arrhythmias may be very refractory to treatment • Consider the use of cardiopulmonary bypass if available GIVE INTRAVENOUS LIPID EMULSION (following the regimen overleaf) <ul style="list-style-type: none"> • Continue CPR throughout treatment with lipid emulsion • Recovery from LA-induced cardiac arrest may take >1 h • Propofol is not a suitable substitute for lipid emulsion • Lidocaine should not be used as an anti-arrhythmic therapy 	WITHOUT CIRCULATORY ARREST Use conventional therapies to treat: <ul style="list-style-type: none"> • hypotension, • bradycardia, • tachyarrhythmia CONSIDER INTRAVENOUS LIPID EMULSION (following the regimen overleaf) <ul style="list-style-type: none"> • Propofol is not a suitable substitute for lipid emulsion • Lidocaine should not be used as an anti-arrhythmic therapy
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4 Follow-up	<ul style="list-style-type: none"> • Arrange safe transfer to a clinical area with appropriate equipment and suitable staff until sustained recovery is achieved • Exclude pancreatitis by regular clinical review, including daily amylase or lipase assays for two days • Report cases as follows: <ul style="list-style-type: none"> in the United Kingdom to the National Patient Safety Agency (via www.npsa.nhs.uk) in the Republic of Ireland to the Irish Medicines Board (via www.imb.ie) <p>If Lipid has been given, please also report its use to the international registry at www.lipidregistry.org. Details may also be posted at www.lipidrescue.org</p>		

Your nearest bag of Lipid Emulsion is kept

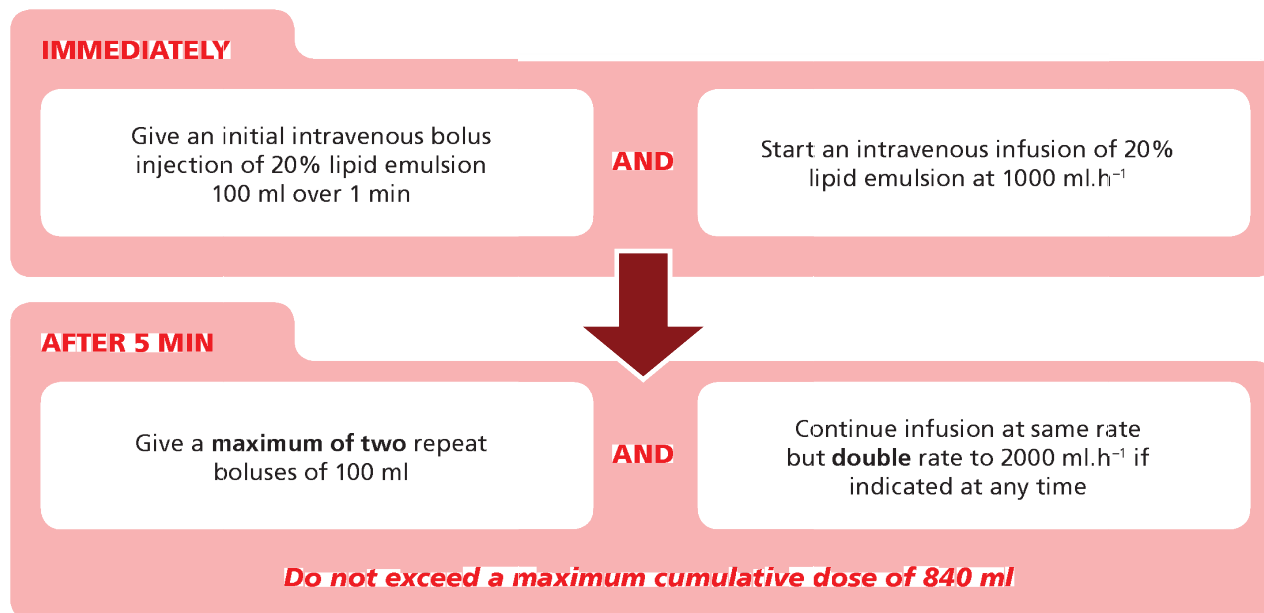
This guideline is not a standard of medical care. The ultimate judgement with regard to a particular clinical procedure or treatment plan must be made by the clinician in the light of the clinical data presented and the diagnostic and treatment options available.

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Figure 12.3 Management of severe local anaesthetic toxicity



An approximate dose regimen for a 70-kg patient would be as follows:



This AAGBI Safety Guideline was produced by a Working Party that comprised:
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This Safety Guideline is endorsed by the Australian and New Zealand College of Anaesthetists (ANZCA).

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Figure 12.3 Management of severe local anaesthetic toxicity

Vasopressors, inotropes, calcium, glucagon, phosphodiesterase inhibitors and insulin-glucose may all be useful in beta-blocker and calcium channel blocker overdose. Transcutaneous pacing may be effective for severe bradycardia caused by poisoning and overdose (Chapters 10 and 11).

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 gas values. Monitor temperature because thermoregulation is impaired. Both hypothermia and hyperthermia (hyperpyrexia) can occur after overdose of some drugs. Retain samples of blood and urine for analysis. 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.

Hypothermia

Definition

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

Diagnosis

Accidental hypothermia may be under-diagnosed in countries with a temperate climate. In people with normal thermoregulation, hypothermia can develop during exposure to cold environments, particularly wet or windy conditions, and in people who have been immobilised, or following immersion in cold water. When thermoregulation is impaired, for example, in the elderly and very young, hypothermia can follow a mild insult. The risk of hypothermia is also increased by drug or alcohol ingestion, exhaustion, illness, injury or neglect especially when there is a decrease in the level of consciousness. Hypothermia may be suspected from the clinical history or a brief external examination of a collapsed patient. A low-reading thermometer is needed to measure the core temperature and confirm the diagnosis. The core temperature measured in the lower third of the oesophagus correlates well with the temperature of the heart. 'Tympanic' measurement - using a thermistor technique - is a reliable alternative but may be lower than the oesophageal temperature if the environmental temperature is very cold, the probe is not well insulated, the external auditory canal is blocked or during cardiac

arrest when there is no flow in the carotid artery. Widely available 'tympanic' thermometers based on infrared technique do not seal the ear canal and are often not suitable for low temperature readings.

Decision to resuscitate

Cooling of the human body decreases cellular oxygen consumption by about 6% per 1°C decrease in core temperature. In some cases, hypothermia can exert a protective effect on the brain and vital organs and intact neurological recovery is possible even after prolonged cardiac arrest if deep hypothermia develops before asphyxia.

Beware of diagnosing death in a hypothermic patient because cold alone may produce a very slow, small-volume, irregular pulse and unrecordable blood pressure. In a hypothermic patient, no signs of life (Swiss hypothermia stage IV) alone are unreliable for declaring death. At 18°C the brain can tolerate periods of circulatory arrest for ten times longer than at 37°C. Dilated pupils can be caused by a variety of insults and must not be regarded as a sign of death. Good quality survival has been reported after cardiac arrest and a core temperature of 13.7°C after immersion in cold water with prolonged CPR.

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

Treatment of hypothermia

The standard principles of prevention and life support apply to the hypothermic patient. Do not delay urgent procedures, such as tracheal intubation and insertion of vascular catheters.

- Open 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. Procedures can precipitate VF. The advantages of adequate oxygenation and protection from aspiration outweigh the minimal risk of triggering VF by performing tracheal intubation.
- 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. Both the respiratory rate and pulse may be very slow in

severe hypothermia so more assessment time is necessary. Echocardiography or Doppler ultrasound can be used to establish if there is a cardiac output or peripheral blood flow.

- If the victim is pulseless, start chest compressions immediately. Use the same ventilation and chest compression rates as for a normothermic patient. Hypothermia can cause stiffness of the chest wall, making ventilation and chest compressions more difficult. If you are not experienced in patient assessment or if there is any doubt about whether a pulse is present, start chest compressions until more experienced help is available.
- Once resuscitation is under way, confirm hypothermia with a low reading thermometer. Use oesophageal, bladder, rectal, or tympanic temperature measurements. Try to use a consistent method to allow serial comparisons of temperature.
- The hypothermic heart may be unresponsive to cardio-active drugs, attempted electrical pacing, and attempted defibrillation. Drug metabolism is slowed, leading to potentially toxic plasma concentrations of any drugs given repeatedly. Withhold adrenaline and other drugs until the patient has been warmed to a temperature greater than about 30°C. Once 30°C has been reached, double the intervals between doses (twice as long as normal). As the patient's temperature returns towards normal (above 35°C), use the standard drug protocols.
- Give drugs via a central or large proximal vein if possible.
- Remember to rule out other primary causes of cardiorespiratory arrest (e.g. drug overdose, hypothyroidism or trauma) or reversible causes using the four Hs and four Ts approach.
- Monitor electrolytes, glucose and blood gases regularly during resuscitation and post-resuscitation care as rapid changes can occur.
- Blood gas analysers will give blood gas values for a temperature of 37°C unless the patient's temperature is entered in to the analyser. Oxygen and carbon dioxide partial pressures are lower in hypothermia because gases become more soluble as blood temperature decreases. In clinical practice it is much easier to make all the measurements at 37°C i.e. temperature uncorrected values. It is then only necessary to compare them with the well-known normal values for 37°C. This also enables comparison of serial results from blood gas samples taken during rewarming.

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. Follow standard treatment protocols.

- Arrhythmias other than VF tend to revert spontaneously as the core temperature increases and usually do not require immediate treatment. Bradycardia can be physiological in severe hypothermia. Cardiac pacing is not indicated unless the bradycardia persists after rewarming.
- If VF/VT is detected, give a shock; if VF/VT persists after three shocks, delay further defibrillation attempts until the core temperature is above 30°C. If an AED is used, follow the AED prompts while rewarming the patient.

Rewarming

General measures for all victims include removal from the cold environment, prevention of further heat loss and rapid transfer to hospital. Rewarming may be passive, active external, or active internal.

- In the field, a patient with moderate or severe hypothermia should be immobilised and handled carefully, oxygenated adequately, monitored (including ECG and core temperature), and the whole body dried and insulated. Wet clothes should be cut off rather than stripped off; this will avoid excessive movement of the victim.
- Conscious victims can mobilise as exercise re-warms a person more rapidly than shivering. Exercise can increase any after-drop, i.e. further cooling after removal from a cold environment. Somnolent or comatose victims have a low threshold for developing VF or pulseless VT and should be immobilised and kept horizontal to avoid an after-drop or cardiovascular collapse.
- Passive rewarming is appropriate in conscious victims with mild hypothermia who are still able to shiver. This is best achieved by full body insulation with wool blankets, aluminium foil, a hat and warm environment. The application of chemical heat packs to the trunk is particularly helpful in moderate and severe hypothermia to prevent further heat loss in the prehospital setting.
- Rewarming in the field with heated intravenous fluids and warm humidified gases is not efficient. Intensive active rewarming must not delay transport to a hospital where advanced rewarming techniques, continuous monitoring and observation are available.

In general, alert hypothermic and shivering victims without an arrhythmia can be transported to the nearest hospital for passive rewarming and observation. Hypothermic victims with an altered consciousness should be taken to a hospital capable of active external and internal rewarming.

- Active external rewarming techniques include forced air rewarming and warmed (up to 42°C) intravenous fluids. These techniques are effective (rewarming rate 1 - 1.5°C h⁻¹) in patients with severe hypothermia and a perfusing rhythm.
- Active internal rewarming techniques include warm humidified gases; gastric, peritoneal, pleural or bladder lavage with warmed fluids (at 40°C), and extracorporeal rewarming.
- In a hypothermic patient with apnoea and cardiac arrest, extracorporeal rewarming is the preferred method of active internal rewarming because it provides sufficient circulation and oxygenation while the core body temperature is increased by 8 - 12°C h⁻¹. Survivors in one case series had an average of 65 min of conventional CPR before cardiopulmonary bypass. Unfortunately, facilities for extracorporeal rewarming are not always available and a combination of rewarming techniques may have to be used.
- During rewarming, patients will require large volumes of fluids as vasodilation causes expansion of the intravascular space. Continuous haemodynamic monitoring and warm IV fluids are essential.

Avalanche burial

In Europe and North America, there are about 150 snow avalanche deaths each year. Most are sports-related and involve skiers, snowboarders and snowmobilers. Death from avalanches is due to asphyxia, trauma and hypothermia. Avalanches occur in areas that are difficult to access by rescuers in a timely manner, and burials frequently involve multiple victims. Avalanche victims are not likely to survive when they are:

- buried > 35 min and in cardiac arrest with an obstructed airway on extrication;
- buried initially and in cardiac arrest with an obstructed airway on extrication, and an initial core temperature of < 32°C;
- buried initially and in cardiac arrest on extrication with an initial serum potassium of > 12 mmol l⁻¹.

Post-resuscitation care

Avoid hyperthermia during and after the warming period. Once ROSC has been achieved, use standard strategies

for post-resuscitation care, including mild hypothermia if appropriate. There is no evidence for the routine use of steroids, barbiturates, or antibiotics.

Hyperthermia

Definition

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

Environment-related hyperthermia occurs where heat, usually in the form of radiant energy, is absorbed by the body at a rate faster than can be lost by thermoregulatory mechanisms. Hyperthermia occurs along a continuum of heat-related conditions starting with heat stress, progressing to heat exhaustion, heat stroke and culminating in multi-organ dysfunction and cardiac arrest in some instances.

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.

Heat stroke

Heat stroke is a systemic inflammatory response with a core temperature > 40.6°C accompanied by mental state change and varying levels of organ dysfunction. There are two forms of heat stroke: classic non-exertional heat stroke occurs during high environmental temperatures and often affects the elderly during heat waves; exertional heat stroke occurs during strenuous physical exercise in high environmental temperatures and/or high humidity and usually affects healthy young adults. Mortality from heat stroke ranges between 10 - 50%.

Predisposing factors

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

- core temp 40.6°C or more;
- hot, dry skin (sweating is present in half cases of exertional heat stroke);
- early signs and symptoms include: extreme fatigue, headache, fainting, facial flushing, vomiting and diarrhoea;
- cardiovascular dysfunction including arrhythmias and hypotension;
- respiratory dysfunction including ARDS;
- central nervous system dysfunction including seizures and coma;
- liver and renal failure;
- coagulopathy;
- rhabdomyolysis.

Other clinical conditions need to be considered, including:

- drug toxicity;
- drug withdrawal syndrome;
- serotonin syndrome;
- neuroleptic malignant syndrome;
- sepsis;
- central nervous system infection;
- endocrine disorders e.g. thyroid storm, pheochromocytoma.

Treatment

The mainstay of treatment is supportive therapy based on optimising the ABCDEs and rapidly cooling the patient.

- Start cooling before the patient reaches hospital. Aim to rapidly reduce the core temperature to approximately 39°C. Patients with severe heat stroke need to be managed in a critical care setting.
- Use haemodynamic monitoring to guide fluid therapy. Large volumes of fluid may be required. Correct electrolyte abnormalities.

- If cardiac arrest occurs, follow standard procedures for basic and advanced life support and cool the patient. Attempt defibrillation, if appropriate, according to current guidelines, while continuing to cool the patient. Animal studies suggest the prognosis is poor compared with normothermic cardiac arrest. The risk of unfavourable neurological outcome increases for each degree of body temperature > 37°C.
- Provide post-resuscitation care according to normal guidelines (Chapter 13).

Cooling techniques

Several cooling methods have been described but there are few formal trials on which method is best.

- Simple techniques include cool drinks, fanning the undressed patient and spraying tepid water on the patient. Ice packs over areas where there are large superficial blood vessels (axillae, groins, neck) are also useful. Surface cooling may cause shivering.
- In cooperative stable patients immersion in cold water is effective; however, this can cause peripheral vasoconstriction and reduce heat dissipation. Immersion is not practical in very sick patients.
- Use the same advanced cooling techniques as used for therapeutic hypothermia after cardiac arrest (Chapter 13). Consider the use of cold IV fluids, intravascular cooling catheters, surface cooling devices and extra corporeal circuits, e.g. continuous veno-veno haemofiltration or cardiopulmonary bypass.
- No specific drugs lower core temperature in heat stroke. There is no good evidence that antipyretics (e.g. non-steroidal anti-inflammatory drugs or paracetamol) are effective in heat stroke. Diazepam may be useful to treat seizures and facilitate cooling. Dantrolene (see below) has not been shown to be beneficial.

Malignant hyperthermia

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

Drowning

Drowning is a common cause of accidental death. The most important detrimental consequence of drowning is hypoxia. Cardiac arrest is usually a secondary event. The duration of hypoxia is a 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. Patients who have spontaneous circulation and breathing when they reach hospital usually recover with good outcomes. Remember, some patients may have had a primary cardiac arrest (e.g. caused by myocardial infarction whilst swimming). Death from drowning is more common in young males, and is the leading cause of accidental death in Europe in this group.

Definition

Drowning 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. 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.

Decision to resuscitate

Deciding whether to start or stop resuscitation of a drowning victim is notoriously difficult. No single factor predicts prognosis accurately.

- Start and 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 > 60 min.

Treatment

Treatment of a drowning victim involves four phases. These comprise (i) aquatic rescue (ii) basic life support (iii) advanced life support (iv) post-resuscitation care.

Aquatic rescue and basic life support

- Ensure personal safety and minimise the danger to yourself at all times. If possible, attempt to save the drowning victim without entering the water. Talk to the victim, use a rescue aid (e.g. stick or clothing), or throw a rope or buoyant rescue aid if the victim is close to dry land. Alternatively, use a boat or other water vehicle to

help 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. It is safer to enter the water with two rescuers than alone.

- Remove the victim from the water and start resuscitation as quickly and safely as possible. Cervical spine injury is uncommon in drowning victims (approximately 0.5%). Spinal immobilisation is difficult in the water and delays removal from the water and adequate resuscitation of the victim. Consider cervical spine immobilisation if there is a history of diving, water slide use, signs of severe injury, or signs of alcohol intoxication. Despite potential spinal injury, if the victim is pulseless and apnoeic remove them from the water as quickly as possible (even if a back support device is not available) whilst attempting to limit neck flexion and extension.
- Try to remove the victim from the water in a horizontal position to minimise the risks of post-immersion hypotension and cardiovascular collapse.

Ventilation

- Prompt initiation of rescue breathing or positive pressure ventilation increases survival. If possible supplement ventilation with oxygen. Give five initial ventilations as soon as possible.
- Rescue breathing can be initiated whilst the victim is still in shallow water provided the safety of the rescuer is not compromised. It is likely to be difficult to pinch the victim's nose, so mouth-to-nose ventilation may be used as an alternative to mouth-to-mouth ventilation.
- If the victim is in deep water, open their airway and if there is no spontaneous breathing start in-water rescue breathing if trained to do so. In-water resuscitation is possible, but should ideally be performed with the support of a buoyant rescue aid. Give 10 - 15 rescue breaths over approximately 1 min. If normal breathing does not start spontaneously, and the victim is < 5 min from land, continue rescue breaths while towing. If more than an estimated 5 min from land, give rescue breaths over 1 min, then bring the victim to land as quickly as possible without further attempts at ventilation.

Chest compressions

As soon as the victim is removed from the water, check for breathing. If the victim is not breathing (or is making occasional gasps), start chest compressions immediately. Continue CPR in a ratio of 30 compressions to 2 ventilations. Most drowning victims will have sustained cardiac arrest secondary to hypoxia. In these patients, compression-only CPR is likely to be less effective and standard CPR should be used.

Defibrillation

Dry the victim's chest before placing defibrillation electrodes. Standard procedures for defibrillation using an AED or manual defibrillator should be followed.

Regurgitation during resuscitation

- Regurgitation of stomach contents is common following resuscitation from drowning and makes airway management more difficult. 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. Log rolling requires several rescuers. There is no need to clear the airway of aspirated water. Remove debris manually or when dry land has been reached with suction if available. Most drowning victims aspirate small amounts of water, and this is absorbed rapidly into the central circulation. Do not use abdominal thrusts or tip the victim head down to remove water from the lungs or stomach.

Advanced life support

Airway and breathing

- Give high-flow oxygen, ideally through an oxygen mask with reservoir bag, during the initial assessment of the spontaneously breathing drowning victim. Consider non-invasive ventilation or continuous positive airway pressure if the victim fails to respond to treatment with high-flow oxygen. Use pulse oximetry and arterial blood gas analysis to titrate the concentration of inspired oxygen.
- Consider early tracheal intubation and controlled ventilation for victims who fail to respond to these initial measures or who have a reduced level of consciousness. Take care to ensure optimal pre-oxygenation before intubation. Use a rapid-sequence induction with cricoid pressure to reduce the risk of aspiration. Pulmonary oedema fluid may pour from the airway and may need suctioning to enable a view of the larynx.
- After the tracheal tube is confirmed in position, titrate the inspired oxygen concentration to achieve a SaO_2 of 94 - 98%. High positive end-expiratory pressure (PEEP) levels may be required if the patient is severely hypoxaemic.
- In the event of cardiac arrest protect the airway of the victim early in the resuscitation attempt, ideally with a cuffed tracheal tube - reduced pulmonary compliance requiring high inflation pressures may limit the use of a supraglottic airway device.

Circulation and defibrillation

- Differentiating respiratory from cardiac arrest is

particularly important in the drowning victim. Delaying the initiation of chest compressions if the victim is in cardiac arrest will reduce survival.

- The typical post-arrest gasping is very difficult to distinguish from the initial respiratory efforts of a spontaneous recovering drowning victim. Palpation of the pulse as the sole indicator of the presence or absence of cardiac arrest is unreliable. When available additional diagnostic information should be obtained from other monitoring modalities such as ECG trace, ETCO_2 , echocardiography to confirm the diagnosis of cardiac arrest.
- If the victim is in cardiac arrest, follow standard advanced life support protocols. If the victim's core body temperature is less than 30°C , limit defibrillation attempts to three, and withhold IV drugs until the core body temperature increases above 30°C .
- During prolonged immersion, victims may become hypovolaemic from the hydrostatic pressure of the water on the body. Give IV fluid to correct hypovolaemia. After ROSC, use haemodynamic monitoring to guide fluid resuscitation.

Post-resuscitation care

- Victims of drowning are at risk of developing acute respiratory distress syndrome (ARDS) after submersion and standard ventilation strategies should be used.
- 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.
- There are no differences in the treatment of victims of fresh or sea water drowning.
- If submersion occurs in icy water ($< 5^\circ\text{C}$), hypothermia may develop rapidly and provide some protection against hypoxia. Once the victim is resuscitated it is unclear whether further therapeutic hypothermia is beneficial. A pragmatic approach might be to consider rewarming until a core temperature of $32 - 34^\circ\text{C}$ is achieved, taking care to avoid hyperthermia ($> 37^\circ\text{C}$) during the subsequent period of intensive care.
- Attempts have been made to improve neurological outcome following drowning with the use of barbiturates, intracranial pressure (ICP) monitoring, and steroids. None of these interventions has been shown to alter outcome.
- Cardiac arrhythmias may cause rapid loss of consciousness leading to drowning if the victim is in water at the time. Take a careful history in survivors of

a drowning incident to identify features suggestive of arrhythmic syncope. Symptoms may include syncope (whilst supine position, during exercise, with brief prodromal symptoms, repetitive episodes or associated with palpitations), seizures or a family history of sudden death. The absence of structural heart disease at post mortem does not rule the possibility of sudden cardiac death. Post mortem genetic analysis has proved helpful in these situations and should be considered if there is uncertainty over the cause of a drowning death.

Asthma

Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma with a high prevalence in some European countries (United Kingdom, Ireland and Scandinavia). Annual worldwide deaths from asthma have been estimated at 250,000. Most deaths in the UK occur before hospital admission. Good asthma control and prevention of acute asthma is therefore important. The British Thoracic Society (BTS) and Scottish Intercollegiate Guidelines Network (SIGN) have published guidelines for the management of asthma available at www.brit-thoracic.org.uk

This guidance focuses on the treatment of patients with near-fatal asthma and cardiac arrest

Patients at risk of asthma-related cardiac arrest

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

- a history of near-fatal asthma requiring intubation and mechanical ventilation;
- a hospitalisation or emergency care for asthma in the past year;
- an increasing use and dependence of beta-2 agonists;
- anxiety, depressive disorders and/or poor compliance with therapy.

Causes of cardiac arrest

Cardiac arrest in the asthmatic 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;
- cardiac arrhythmias due to hypoxia, stimulant drugs (e.g. β -adrenergic agonists, aminophylline) or electrolyte abnormalities;
- dynamic hyperinflation, i.e. auto-positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape).

Gradual build-up of pressure occurs and reduces venous return and blood pressure;

- tension pneumothorax (often bilateral).

The 4 Hs and 4 Ts approach to reversible causes will help identify these causes in cardiac arrest.

Assessment and treatment

Use the ABCDE approach to assess severity and guide treatment. The severity of acute asthma is summarised in Table 12.2.

Asthma severity		
Near-fatal asthma	Raised PaCO ₂ and/or mechanical ventilation with raised inflation pressures	
Life-threatening asthma	Any one of the following in a patient with severe asthma:	
	Clinical signs	Measurements
	Altered conscious level	PEF < 33% best or predicted
	Exhaustion	SpO ₂ < 92%
	Arrhythmia	PaO ₂ < 8 kPa
	Hypotension	'normal' PaCO ₂ (4.6 - 6.0 kPa)
	Cyanosis	
	Silent chest	
	Poor expiratory effort	
Acute severe asthma	Any one of: <ul style="list-style-type: none"> - PEF 33 - 50% best or predicted - respiratory rate \geq 25 min⁻¹ - heart rate \geq 110 min⁻¹ - inability to complete sentences in one breath 	
Moderate asthma exacerbation	<ul style="list-style-type: none"> - increasing symptoms - PEF > 50 - 75% best or predicted - no features of acute severe asthma 	
Brittle asthma	<ul style="list-style-type: none"> - Type 1: wide PEF variability (> 40% diurnal variability for > 50% of time over a period of > 150 days) despite intensive therapy - Type 2: sudden severe attacks on a background of apparently well controlled asthma 	

PEF = peak expiratory flow

Table 12.2 Severity of acute asthma exacerbations

(From British Thoracic Society/Scottish Intercollegiate Guidelines Network Guideline on the Management of Asthma - www.brit-thoracic.org.uk)

- Wheezing is a common physical finding, but severity does not correlate with the degree of airway obstruction. Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis, pneumonia, foreign bodies, pulmonary embolism, bronchiectasis, subglottic mass.
- The patient with acute severe asthma requires aggressive medical management to prevent deterioration. Experienced clinicians should treat these patients in a critical care area.
- Use a concentration of inspired oxygen that will achieve an SpO₂ 94 - 98%. High-flow oxygen by mask is sometimes necessary.
- Salbutamol (5 mg nebulised) is the main therapy for acute asthma. Repeated doses every 15 - 20 min, or continuous doses, may be needed. Nebuliser units that can be driven by high-flow oxygen should be used. Remember that nebulised drugs will not be delivered to the lungs effectively if the patient is tired and hypoventilating. If a nebuliser is not immediately available beta-2 agonists can be temporarily administered by repeating activations of a metered dose inhaler via a large volume spacer device.
- Give corticosteroids (prednisolone 30 - 40 mg orally or hydrocortisone 100 mg IV 6 - hourly) early. Oral formulations have a longer half-life but the IV route is easier to give in near fatal asthma.
- Nebulised anticholinergics (ipratropium 0.5 mg 4 - 6 hourly) produce additional bronchodilation in severe asthma and in those who do not respond to beta-agonists.
- Magnesium sulphate (2 g IV slowly = 8 mmol) is a bronchodilator and may be useful in severe or near-fatal asthma. Nebulised magnesium sulphate (250 mmol l⁻¹) in a volume of 2.5 - 5 ml is also safe and can be beneficial.
- Consider intravenous salbutamol (250 mcg IV slowly) in patients unresponsive to nebulised therapy or where nebulised / inhaled therapy is not possible (e.g. a patient receiving bag-mask ventilation). Use an infusion of 3 - 20 mcg min⁻¹ if necessary.
- Aminophylline should be considered only in severe or near-fatal asthma. If after obtaining senior advice the decision is taken to administer IV aminophylline a loading dose of 5 mg kg⁻¹ is given over 20 - 30 min (unless on maintenance therapy), followed by an infusion of 500 - 700 mcg kg⁻¹ h⁻¹. Serum theophylline concentrations should be maintained below 20 mcg ml⁻¹ to avoid toxicity.

- These patients are often dehydrated or hypovolaemic and will benefit from fluid replacement. Beta-2 agonists and steroids may induce hypokalaemia, which should be corrected with electrolyte supplements.
- Patients that fail to respond to initial treatment, or develop signs of life-threatening asthma, must be assessed by an intensive care specialist. These patients may benefit from tracheal intubation and ventilatory support.

Cardiac arrest

- Follow standard BLS and ALS protocols. Ventilation will be difficult because of increased airway resistance; try to avoid gastric inflation.
- Intubate the trachea early. There is a significant risk of gastric inflation and hypoventilation of the lungs when attempting to ventilate a severe asthmatic without a tracheal tube.
- The recommended respiratory rate (10 breaths min⁻¹) and tidal volume required for a normal chest rise during CPR should not cause dynamic hyperinflation of the lungs (gas trapping).
- 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. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.
- Dynamic hyperinflation increases transthoracic impedance. In VF, consider higher shock energies for defibrillation if initial defibrillation attempts fail.
- Look for reversible causes using the 4 Hs and 4 Ts approach.
- Tension pneumothorax can be difficult to diagnose in cardiac arrest; it may be indicated by unilateral expansion of the chest wall, shifting of the trachea, and subcutaneous emphysema. Pleural ultrasound in skilled hands is faster and more sensitive than chest X-ray for the detection of pneumothorax. Early needle decompression (thoracocentesis) followed by chest drain insertion is needed. Needle decompression may fail due to inadequate needle length. In the ventilated patient, thoracostomy (a surgical hole in the chest wall and pleura) may be quicker to do and more effective for decompressing the chest (see trauma section).
- Always consider bilateral pneumothoraces in asthma-related cardiac arrest.
- Follow standard guidelines for post-resuscitation care.

Anaphylaxis

Definition

Anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction.

This is characterised by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.

This guidance is based on Emergency Treatment of Anaphylactic Reactions, Resuscitation Council UK, 2008 (For more details see www.resus.org.uk).

Aetiology

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

Anaphylaxis is not always recognised, so studies may underestimate the incidence. Anaphylaxis is triggered by a broad range of triggers, with food, drugs and venom being the most common. Food triggers are commonest in children and drugs much more common in older people. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. In many cases, no cause can be identified. A significant number of cases of anaphylaxis are idiopathic (non-IgE mediated).

The risk of death is increased in those with pre-existing asthma, particularly if the asthma is poorly controlled or in those asthmatics who fail to use, or delay treatment with, adrenaline. There are approximately 20 anaphylaxis deaths reported each year in the UK, although this may be a substantial under-estimate.

When anaphylaxis is fatal, death usually occurs very soon after contact with the trigger. Fatal food reactions cause respiratory arrest typically after 30 - 35 min; insect stings cause collapse from shock after 10 - 15 min; and deaths caused by intravenous medication occurred most commonly within 5 min. Death rarely occurs more than six hours after contact with the trigger.

Recognition

- Anaphylaxis is likely if a patient who is exposed to a trigger (allergen) develops a sudden illness (usually within minutes of exposure) with rapidly progressing skin changes and life-threatening airway and/or breathing and/or circulation problems. The reaction is usually unexpected.

- The lack of any consistent clinical manifestation and a range of possible presentations cause diagnostic difficulty. Patients have been given injections of adrenaline inappropriately for allergic reactions just involving the skin, or for vasovagal reactions or panic attacks. Guidelines for the treatment of an anaphylactic reaction must therefore take into account some inevitable diagnostic errors, with an emphasis on the need for safety.

Anaphylaxis is likely when all of the following three criteria are met:

- Sudden onset and rapid progression of symptoms
- Life-threatening Airway and/or Breathing and/or Circulation problems
- Skin and/or mucosal changes (flushing, urticaria, angioedema)

The following supports the diagnosis:

- Exposure to a known allergen for the patient

Remember:

- Skin or mucosal changes alone are not a sign of anaphylaxis
- Skin and mucosal changes can be subtle or absent in up to 20% of reactions (some patients can have only a decrease in blood pressure, i.e. a Circulation problem)
- There can also be gastrointestinal symptoms (e.g. vomiting, abdominal pain, incontinence)

Sudden onset and rapid progression of symptoms:

- The patient will feel and look unwell.
- Most reactions occur over several minutes. Rarely, reactions may be slower in onset.
- An intravenous trigger will cause a more rapid onset of reaction than stings which, in turn, tend to cause a more rapid onset than orally ingested triggers.
- The patient is usually anxious and can experience a "sense of impending doom".

Life-threatening Airway, Breathing and Circulation problems:

Use the ABCDE approach to recognise these.

Airway problems:

- Airway swelling, e.g. throat and tongue swelling (pharyngeal/laryngeal oedema). The patient has difficulty in breathing and swallowing and feels that the throat is closing up.
- Hoarse voice.
- Stridor - this is a high-pitched inspiratory noise caused by upper airway obstruction.

Breathing problems:

- Shortness of breath - increased respiratory rate.
- Wheeze.
- Patient becoming tired.
- Confusion caused by hypoxia.
- Cyanosis - this is usually a late sign.
- Respiratory arrest.

Circulation problems:

- Signs of shock - pale, clammy.
- Tachycardia.
- Hypotension - feeling faint, collapse.
- Decreased conscious level or loss of consciousness.
- Anaphylaxis can cause myocardial ischaemia and electrocardiograph (ECG) changes even in individuals with normal coronary arteries.
- Cardiac arrest.

Circulation problems (often referred to as anaphylactic shock) can be caused by direct myocardial depression, vasodilation and capillary leak, and loss of fluid from the circulation.

The above Airway, Breathing and Circulation problems can all alter the patient's neurological status (**Disability problems**) because of decreased brain perfusion. There may be confusion, agitation and loss of consciousness.

Skin and, or mucosal changes

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

- They are often the first feature and present in over 80% of anaphylactic reactions.
- They can be subtle or dramatic.
- There may be just skin, just mucosal, or both skin and mucosal changes.
- There may be erythema - a patchy, or generalised, red rash.
- There may be urticaria (also called hives, nettle rash, weals or welts), which can appear anywhere on the body. The weals may be pale, pink or red, and may look like nettle stings. They can be different shapes and sizes, and are often surrounded by a red flare. They are usually itchy.
- Angioedema is similar to urticaria but involves swelling of deeper tissues, most commonly in the eyelids and lips, and sometimes in the mouth and throat.

Although skin changes can be worrying or distressing for patients and those treating them, skin changes without life-threatening airway, breathing or circulation problems do not signify anaphylaxis.

Differential diagnosis

Life-threatening conditions:

- Sometimes an anaphylactic reaction can present with symptoms and signs that are very similar to life-threatening asthma.
- A low blood pressure (or normal in children) with a petechial or purpuric rash can be a sign of septic shock. Seek help early if there are any doubts about the diagnosis and treatment.
- Following the ABCDE approach will help with treating the differential diagnoses.

Non life-threatening conditions (these usually respond to simple measures):

- Faint (vasovagal episode).
- Panic attack.
- Breath-holding episode in child.
- Idiopathic (non-allergic) urticaria or angioedema.

There can be confusion between an anaphylactic reaction and a panic attack. Victims of previous anaphylaxis may be particularly prone to panic attacks if they think they have been re-exposed to the allergen that caused a previous problem. The sense of impending doom and

breathlessness leading to hyperventilation are symptoms that resemble anaphylaxis in some ways. While there is no hypotension, pallor, wheeze, or urticarial rash or swelling, there may sometimes be flushing or blotchy skin associated with anxiety adding to the diagnostic difficulty. Diagnostic difficulty may also occur with vasovagal attacks after immunisation procedures, but the absence of rash, breathing difficulties, and swelling are useful distinguishing features, as is the slow pulse of a vasovagal attack compared with the rapid pulse of a severe anaphylactic episode. Fainting will usually respond to lying the patient down and raising the legs.

Treatment

As the diagnosis of anaphylaxis is not always obvious, all those who treat anaphylaxis must use the systematic ABCDE approach to the sick patient. Treat life-threatening problems as you find them. The key steps are described in the anaphylaxis algorithm (Figure 12.4).

- All patients should be placed in a comfortable position. Patients with airway and breathing problems may prefer to sit up as this will make breathing easier. Lying flat with or without leg elevation is helpful for patients with a low blood pressure (Circulation problem). If the patient feels faint, do not sit or stand them up - this can cause cardiac arrest. Patients who are breathing and unconscious should be placed on their side (recovery position).
- Removing the trigger for an anaphylactic reaction is not always possible. Stop any drug suspected of causing an anaphylactic reaction (e.g. stop intravenous infusion of a gelatin solution or antibiotic). Do not delay definitive treatment if removing the trigger is not feasible.
- Give the highest concentration of oxygen possible during resuscitation.
- Adrenaline is the most important drug for the treatment of an anaphylactic reaction. As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. Its beta-receptor activity dilates the bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release. Adrenaline works best when given early after the onset of the reaction but it is not without risk, particularly when given intravenously. Adverse effects are extremely rare with correct doses injected intramuscularly (IM). Sometimes there has been uncertainty about whether complications (e.g. myocardial ischaemia) have been caused by the allergen itself or by the adrenaline given to treat it.
- The intramuscular (IM) route is the best for most individuals who have to give adrenaline to treat an anaphylactic reaction. Monitor the patient as soon as possible (pulse, blood pressure, ECG, pulse oximetry). This will help monitor the response to adrenaline.
- For adults give an initial IM adrenaline dose of 0.5 mg (0.5 ml of 1:1000 adrenaline = 0.5 mg = 500 mcg). Further doses can be given at about 5-min intervals according to the patient's response.
- The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The needle used for injection needs to be sufficiently long to ensure that the adrenaline is injected into muscle.
- The use of IV adrenaline applies only to those experienced in the use and titration of vasopressors in their normal clinical practice (e.g. anaesthetists, emergency physicians, intensive care doctors). In patients with a spontaneous circulation, intravenous adrenaline can cause life-threatening hypertension, tachycardia, arrhythmias, and myocardial ischaemia. Patients who are given IV adrenaline must be monitored - continuous ECG and pulse oximetry and frequent non-invasive blood pressure measurements as a minimum.
- Titrate IV adrenaline using 50 mcg boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion. The pre-filled 10 ml syringe of 1:10,000 adrenaline contains 100 mcg ml⁻¹. A dose of 50 mcg is 0.5 ml, which is the smallest dose that can be given accurately. Do not give the undiluted 1:1000 adrenaline concentration IV.
- Auto-injectors are often given to patients at risk of anaphylaxis for their own use. Healthcare professionals should be familiar with the use of the most commonly available auto-injector devices. If an adrenaline auto-injector is the only available adrenaline preparation when treating anaphylaxis, healthcare providers should use it.
- Give a rapid IV fluid challenge (500 - 1000 ml in an adult) and monitor the response; give further doses as necessary. There is no evidence to support the use of colloids over crystalloids in this setting. Consider colloid infusion as a cause in a patient receiving a colloid at the time of onset of an anaphylactic reaction and stop the infusion. Hartmann's solution or 0.9% saline are suitable fluids for initial resuscitation. A large volume of fluid may be needed.
- Antihistamines are a second line treatment for an anaphylactic reaction. Antihistamines (H1-antihistamine) may help counter histamine-mediated vasodilation and bronchoconstriction. They may not help in reactions depending in part on other mediators but they have the virtue of safety. Used alone, they are unlikely to be life-saving in a true anaphylactic reaction. Give chlorphenamine 10 mg IM or IV slowly.
- Corticosteroids may help prevent or shorten protracted reactions. Inject hydrocortisone 200 mg IM or IV slowly.

Anaphylaxis algorithm

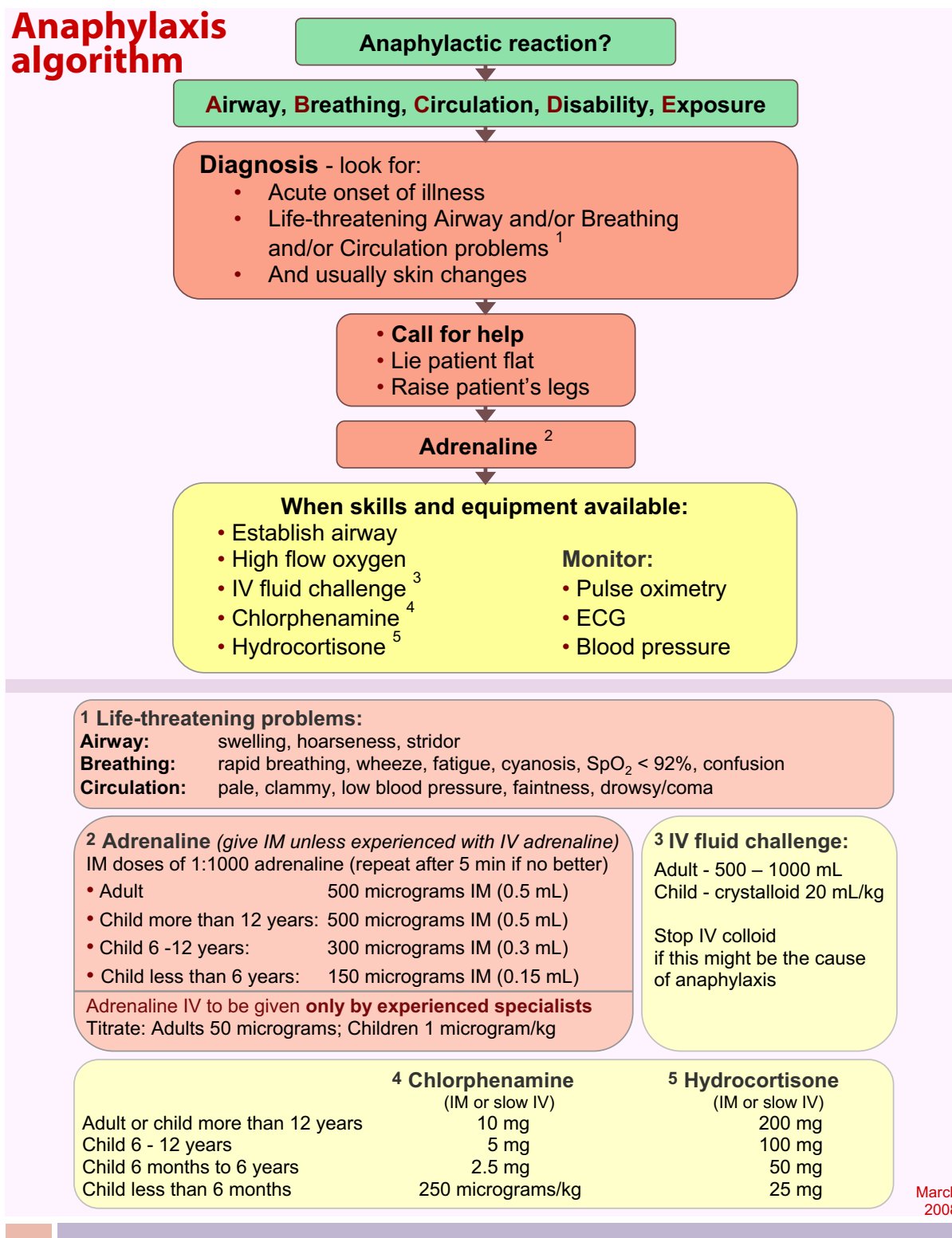


Figure 12.4 Anaphylaxis algorithm

- If the patient has asthma-like features alone, treat as for asthma. As well as the drugs listed above, consider further bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), aminophylline (IV) or magnesium (IV). Remember that intravenous magnesium is a vasodilator and can cause hot flushes and make hypotension worse.
- Adrenaline remains the first line vasopressor for the treatment of anaphylactic reactions. Consider other vasopressors and inotropes (noradrenaline, vasopressin, metaraminol and glucagon) when initial resuscitation with adrenaline and fluids has not been successful. Only use these drugs in specialist settings (e.g. intensive care units) where there is experience in their use. Glucagon can be useful to treat an anaphylactic reaction in a patient taking a beta-blocker.
- If cardiorespiratory arrest occurs, as well as standard ALS, consider the use of steroids, antihistamines (if not given already) and large volumes of intravenous fluids. Prolonged resuscitation may be necessary.
- Airway obstruction may occur rapidly in severe anaphylaxis, particularly in patients with angioedema. Warning signs are swelling of the tongue and lips, hoarseness and oropharyngeal swelling.
- Consider early tracheal intubation; delay may make intubation extremely difficult. As airway obstruction progresses, supraglottic airway devices (e.g. LMA) are likely to be difficult to insert. Attempts at tracheal intubation may exacerbate laryngeal oedema. Early involvement of a senior anaesthetist is mandatory when managing these patients. A surgical airway may be required if tracheal intubation is not possible.

Investigations

The specific test to help confirm a diagnosis of an anaphylactic reaction is measurement of mast cell tryptase. In anaphylaxis, mast cell degranulation leads to markedly increased blood tryptase concentrations.

Mast cell tryptase sample timing

The time of onset of the anaphylactic reaction is the time when symptoms were first noticed.

- Minimum: one sample at 1 - 2 h after the start of symptoms.
 - Ideally: Three **timed** samples:
 - Initial sample as soon as feasible after resuscitation has started - do not delay resuscitation to take sample.
 - Second sample at 1 - 2 h after the start of symptoms
 - Third sample either at 24 h or in convalescence. This provides baseline tryptase levels - some individuals have an elevated baseline level.
- Use a serum or clotted blood ('liver function test' bottle) sample.
 - Record the timing of each sample accurately* on the sample bottle and request form.
 - Consult your local laboratory if you have any queries.

Discharge and follow-up

Patients who have had a suspected anaphylactic reaction should be treated and then observed for at least 6 h in a clinical area with facilities for treating life-threatening ABC problems. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 h. This caution is particularly applicable to:

- Severe reactions with slow onset caused by idiopathic anaphylaxis.
- Reactions in individuals with severe asthma or with a severe asthmatic component.
- Reactions with the possibility of continuing absorption of allergen.
- Patients with a previous history of biphasic reactions.
- Patients presenting in the evening or at night, or those who may not be able to respond to any deterioration.
- Patients in areas where access to emergency care is difficult.

The exact incidence of biphasic reactions is unknown. There is no reliable way of predicting who will have a biphasic reaction. It is therefore important that decisions about discharge are made for each patient by an experienced clinician.

Before discharge from hospital all patients must be:

- Given clear instructions to return to hospital if symptoms return.
- Considered for anti-histamines and oral steroid therapy for up to 3 days. This is helpful for treatment of urticaria and may decrease the chance of further reaction.
- Have a plan for follow-up, including contact with the patient's general practitioner.

- *All patients* presenting with anaphylaxis should be referred to an allergy clinic to identify the cause, and thereby reduce the risk of future reactions and prepare the patient to manage future episodes themselves.

Cardiac arrest following cardiac surgery

After major cardiac surgery, cardiac arrest is relatively common in the immediate post-operative phase, with a reported incidence of 0.7% - 2.9%. Cardiac arrest is usually preceded by physiological deterioration, although it may occur suddenly in stable patients. Continuous monitoring on the intensive care unit (ICU) enables immediate intervention at the time of arrest. Survival to hospital discharge of patients having a cardiac arrest during the first 24 h after cardiac surgery is reported as 54 - 79% in adults and 41% in children.

Aetiology

There are usually specific causes of cardiac arrest that are all potentially reversible. The main causes of cardiac arrest in the initial post-operative period include:

- cardiac tamponade;
- myocardial ischaemia;
- haemorrhage causing hypovolaemic shock;
- disconnection of the pacing system in a pacing-dependent patient;
- tension pneumothorax;
- 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. Patients in the ICU are highly monitored and an arrest is most likely to be signalled by monitoring alarms where absence of pulsation or perfusing pressure on the arterial line, loss of pulse oximeter trace, pulmonary artery (PA) trace, or end-tidal CO₂ trace and rapid assessment of the patient can be sufficient to indicate cardiac arrest without the need to palpate a central pulse. Call for senior help early including a cardiothoracic surgeon and cardiac anaesthetist.

Treatment

- Start external chest compressions immediately in all patients who collapse without an output. Consider reversible causes using the 4 Hs and 4 Ts approach: hypoxia - check tube position, ventilate with 100%

oxygen; tension pneumothorax - clinical examination, thoracic ultrasound; hypovolaemia, pacing failure.

- In asystole, secondary to a loss of cardiac pacing, chest compression can be delayed momentarily as long as the surgically inserted temporary pacing wires can be connected rapidly and pacing re-established (DDD [Dual chamber pacing, Dual chamber sensing and Dual chamber response] at 100 min⁻¹ at maximum amplitude).
- The effectiveness of compressions may be verified by looking at the arterial trace. Inability to attain a perfusing blood pressure during compressions (e.g. systolic pressure of 80 mmHg) may indicate tamponade, tension pneumothorax, or severe haemorrhage and should precipitate emergency resternotomy.
- Intra-aortic balloon pumps should be changed to pressure triggering during CPR.
- In PEA, switch off the pacemaker as it may potentially hide underlying VF.
- External chest compressions can cause sternal disruption or cardiac damage. In the post cardiac surgery ICU, a witnessed and monitored VF/VT cardiac arrest should be treated immediately with up to three quick successive (stacked) defibrillation attempts.
- Three failed shocks in the post cardiac surgery setting should trigger the need for emergency resternotomy. Further defibrillation is attempted as indicated in the ALS algorithm and should be performed with internal paddles at 20 J if resternotomy has been performed.
- Use adrenaline very cautiously and titrate to effect (intravenous doses of 100 mcg or less in adults).
- Emergency resternotomy is an integral part of resuscitation after cardiac surgery, once all other reversible causes have been excluded. Once an adequate airway and ventilation has been established, and if three attempts at defibrillation have failed in VF/VT, undertake resternotomy without delay. Emergency resternotomy is also indicated in asystole or PEA, when other treatments have failed. Resuscitation teams should be well rehearsed in this technique so that it can be performed safely within 5 min of the onset of cardiac arrest. Resternotomy equipment should be prepared as soon as an arrest is identified.
- Consider re-instituting cardiopulmonary bypass if necessary.

Traumatic cardiorespiratory arrest

Cardiac arrest secondary to traumatic injury has a very high mortality, with an overall survival of 5.6% (range 0 - 17%). In survivors, neurological disability is common.

Cardiac arrest from a primary medical problem (e.g. cardiac arrhythmia, hypoglycaemia, seizure) can cause a secondary traumatic event (e.g. fall, road traffic accident). Despite the initial reported mechanism, traumatic injuries may not be the primary cause of a cardiorespiratory arrest and standard advanced life support, including chest compressions, are appropriate. Survival usually depends on early resuscitation by experienced rescuers.

Causes of cardiac arrest in trauma patients include: severe traumatic brain injury, hypovolaemia from massive blood loss, hypoxia from respiratory arrest, direct injury to vital organs and major vessels, tension pneumothorax, and cardiac tamponade.

Commotio cordis is actual or near cardiac arrest caused by a blunt impact to the chest wall over the heart. A blow to the chest can cause VF. Commotio cordis occurs mostly during sports (most commonly baseball) and recreational activities and victims are usually teenage males. Follow standard CPR guidelines. Early defibrillation is important for survival.

Treatment

- Survival from traumatic cardiac arrests is correlated with duration of CPR and pre-hospital time. Prolonged CPR is associated with a poor outcome. Treatment on scene should focus on high-quality CPR, advanced life support and exclusion of reversible causes using the 4 Hs and 4 Ts.
- Undertake only essential life-saving interventions on scene and, if the patient has signs of life, transfer rapidly to the nearest appropriate hospital. Do not delay for spinal immobilisation.
- Effective airway management is essential to maintain oxygenation of the severely compromised trauma patient. Early tracheal intubation by experienced rescuers can be beneficial. Use basic airway management manoeuvres and alternative airways to maintain oxygenation if tracheal intubation cannot be accomplished immediately. If these measures fail, a surgical airway is indicated.
- In low cardiac output conditions, positive pressure ventilation causes further circulatory depression, or even cardiac arrest, by impeding venous return to the heart. Monitor ventilation with continuous waveform capnography and adjust to achieve normocapnia. This may enable slow respiratory rates and low tidal volumes and the corresponding decrease in transpulmonary pressure may increase venous return and cardiac output.

- Treatment of reversible causes:
 - Hypoxaemia - (oxygenation, ventilation).
 - Hypovolaemia - compressible haemorrhage (pressure, pressure dressings, tourniquets, novel haemostatic agents) or non-compressible haemorrhage (splints, intravenous fluid).
 - Tension pneumothorax - decompress quickly by lateral or anterior thoracostomy (incision in chest wall through to the pleural cavity). This is likely to be more effective than needle thoracostomy and quicker than inserting a chest tube.
 - Cardiac tamponade - immediate thoracotomy.
- Chest compressions may not be effective in hypovolaemic cardiac arrest but most survivors of traumatic cardiac arrest do not have hypovolaemia and standard advanced life support may be lifesaving.
- If available, ultrasound will help diagnose rapidly haemoperitoneum, haemopneumothorax, tension pneumothorax and cardiac tamponade. This requires a trained operator and should not delay treatment.
- Give intravenous fluids conservatively until bleeding is controlled. In the presence of uncontrolled bleeding, excessive fluid will increase the bleeding. The choice of fluid and blood products will depend on local practice. In the UK, the National Institute for Health and Clinical Excellence (NICE) has published guidelines on pre-hospital fluid replacement in trauma. 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.

Emergency thoracotomy

- Consider on-scene resuscitative thoracotomy in cardiac arrest caused by penetrating chest trauma if it can be accomplished within 10 min after the loss of the pulse. This requires a trained rescuer.
- Consider emergency department thoracotomy (EDT) in the following circumstances:
 - After blunt trauma EDT should be limited to those with vital signs on arrival and a witnessed cardiac arrest (estimated survival rate 1.6%).
 - Penetrating cardiac injuries who arrive at hospital after a short on scene and transport time with witnessed signs of life or ECG activity are candidates for EDT (estimated survival rate 31%).
 - Penetrating non-cardiac thoracic injuries even though survival rates are low.

- EDT should be undertaken in patients with exsanguinating abdominal vascular injury even though survival rates are low. This procedure should be used as an adjunct to definitive repair of abdominal vascular injury.

Pregnancy

Mortality related to pregnancy in developed countries is rare, occurring in an estimated 1:30,000 deliveries. The mother and fetus must be considered in emergencies during pregnancy. Effective resuscitation of the mother is often the best way to optimise fetal outcome. Significant physiological changes occur during pregnancy; for example, cardiac output, circulatory volume, minute ventilation, and oxygen consumption all increase. The gravid uterus can cause compression of iliac and abdominal vessels when the mother is in the supine position, resulting in reduced cardiac output and hypotension. Resuscitation guidelines for pregnancy are based largely on case series, extrapolation from non-pregnant arrests, manikin studies and expert opinion based on the physiology of pregnancy and changes that occur in normal labour.

Causes of cardiac arrest in pregnancy

Cardiac arrest in pregnancy is most commonly caused by:

- Cardiac disease
- Pulmonary embolism
- Psychiatric disorders
- Hypertensive disorders of pregnancy
- Sepsis
- Haemorrhage
- Amniotic fluid embolism
- Ectopic pregnancy.

Pregnant women can also have the same causes of cardiac arrest as females of the same age group (e.g. anaphylaxis, drug overdose, trauma).

Treatment

Key interventions to prevent cardiac arrest

In an emergency, use the ABCDE approach. Many cardiovascular problems associated with pregnancy are caused by compression of the inferior vena cava. Treat a distressed or compromised pregnant patient as follows.

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

- Immediately re-evaluate the need for any drugs being given.
- Seek expert help early. Obstetric and neonatal specialists should be involved early in the resuscitation.
- Identify and treat the underlying cause.

Modifications for cardiac arrest

- In cardiac arrest, all the principles of basic and advanced life support apply.
- Summon help immediately. For effective resuscitation of mother and fetus, expert help must be obtained; this should include an obstetrician and neonatologist.
- Start CPR according to standard guidelines. Ensure good quality chest compressions with minimal interruptions.
- After 20 weeks gestation the pregnant woman's uterus can press down against the inferior vena cava and the aorta, impeding venous return, cardiac output and uterine perfusion. Caval compression limits the effectiveness of chest compressions.
- Manually displace the uterus to the left to remove caval compression. Add left lateral tilt if this is feasible - the optimal angle of tilt is unknown. Aim for between 15 and 30 degrees. Even a small amount of tilt may be better than no tilt. The angle of tilt used needs to allow high quality chest compressions and if needed permit Caesarean delivery of the fetus (see below).
- The method used for tilting will depend on where the patient is and what is available. Improvisation will be needed. The patient's body will need to be supported on a firm surface to allow effective chest compressions. Methods for tilting include:
 - if the patient is already on a spinal board or operating table the board or table can be tilted to provide a left lateral tilt;
 - sand bags, firm pillows, or a purpose made wedge (e.g. Cardiff Wedge);
 - using the thighs of kneeling rescuers to tilt the torso.
- Start preparing for emergency Caesarean section (see below) - the fetus will need to be delivered if initial resuscitation efforts fail.
- There is an increased risk of pulmonary aspiration of gastric contents in pregnancy. Early tracheal intubation decreases this risk. Tracheal intubation can be more difficult in the pregnant patient. Expert help, a failed intubation drill, and the use of alternative airway devices may be needed.

- Attempt defibrillation using standard energy doses. Left lateral tilt and large breasts can make it difficult to place an apical defibrillator pad.

Reversible causes

Look for reversible causes using the 4 Hs and 4 Ts approach. Abdominal ultrasound by a skilled operator to detect possible causes during cardiac arrest can be useful. It can also permit an evaluation of fetal viability, multiple pregnancy and placental localisation. It should not however delay treatments. Specific reversible causes of cardiac arrest in pregnancy are:

- **Haemorrhage:** This can occur both antenatally and postnatally. Causes include ectopic pregnancy, placental abruption, placenta praevia and uterine rupture. Maternity units should have a massive haemorrhage protocol. Treatment is based on the ABCDE approach. The key step is to stop the bleeding. Consider the following: fluid resuscitation including use of a rapid transfusion system and cell salvage, correction of coagulopathy, oxytocin, ergometrine and prostaglandins to correct uterine atony, uterine compression sutures, intrauterine balloon devices, radiological embolisation of a bleeding vessel, and surgical control including aortic cross clamping/compression and hysterectomy. Placenta percreta may require extensive intra-pelvic surgery.
- **Drugs:** Overdose can occur in women with eclampsia receiving magnesium sulphate, particularly if the patient becomes oliguric. Give calcium to treat magnesium toxicity (see life-threatening electrolyte abnormalities). Central neural blockade for analgesia or anaesthesia can cause problems due to sympathetic blockade (hypotension, bradycardia) or local anaesthetic toxicity (see poisoning section).
- **Cardiovascular disease:** Myocardial infarction and aneurysm or dissection of the aorta or its branches, and peripartum cardiomyopathy cause most deaths from acquired cardiac disease. Patients with known cardiac disease need to be managed in a specialist unit. Pregnant women may develop an acute coronary syndrome, typically in association with risk factors such as obesity, older age, higher parity, smoking, diabetes, pre-existing hypertension and a family history of ischaemic heart disease. Pregnant patients can have atypical features such as epigastric pain and vomiting. Percutaneous coronary intervention (PCI) is the reperfusion strategy of choice for ST-elevation myocardial infarction in pregnancy. Thrombolysis should be considered if urgent PCI is unavailable. Increasing numbers of women with congenital heart disease are becoming pregnant. Pregnant women with known congenital heart disease should be managed in specialist centres.
- **Pre-eclampsia and eclampsia:** Eclampsia is defined as the development of convulsions and/or

unexplained coma during pregnancy or postpartum in patients with signs and symptoms of pre-eclampsia. Magnesium sulphate treatment may prevent eclampsia developing in labour or immediately postpartum in women with pre-eclampsia.

- **Amniotic fluid embolism** usually presents around the time of delivery often in the labouring mother with sudden cardiovascular collapse, breathlessness, cyanosis, arrhythmias, hypotension and haemorrhage associated with disseminated intravascular coagulopathy. Treatment is supportive based on the ABCDE approach and correction of coagulopathy. There is no specific therapy.
- **Pulmonary embolus** causing cardiopulmonary collapse can present throughout pregnancy. CPR should be started with modifications as necessary. The use of fibrinolysis (thrombolysis) needs considerable thought, particularly if a peri-mortem Caesarean section is being considered. If the diagnosis is suspected and maternal cardiac output has not returned it should be given.

Peri-mortem Caesarean section

When initial resuscitation attempts fail, delivery of the fetus may improve the chances of successful resuscitation of both the mother and fetus. 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. This requires that Caesarean section starts at about 4 min after cardiac arrest. Delivery relieves caval compression and may improve the likelihood of resuscitating the mother by permitting an increase in venous return during the CPR attempt. Delivery also enables access to the abdominal cavity so that aortic clamping or compression is possible. Internal cardiac massage is also possible. Once the fetus has been delivered resuscitation of the newborn child can also begin.

In the supine position, the gravid uterus begins to compromise blood flow in the inferior vena cava and abdominal aorta at approximately 20 weeks' gestation; however, fetal viability currently begins at approximately 24 weeks.

- **Gestational age < 20 weeks.** Urgent Caesarean delivery need not be considered, because a gravid uterus of this size is unlikely to compromise maternal cardiac output and fetal viability is not an issue.
- **Gestational age approximately 20 - 23 weeks.** Initiate emergency delivery of the fetus to permit successful resuscitation of the mother, not survival of the delivered infant, which is unlikely at this gestational age.
- **Gestational age approximately > 24 weeks.** Initiate emergency delivery to help save the life of both the mother and the infant.

Planning for resuscitation in pregnancy

Advanced life support in pregnancy requires co-ordination 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 in place plans and equipment for resuscitation of both the pregnant patient and the newborn child;
- ensure early involvement of obstetric and neonatal teams;
- ensure regular training of staff in obstetric emergencies.

Electrocution

Electrical injury is a relatively infrequent but potentially devastating multi-system injury with high morbidity and mortality. 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, Asia; 110 V in the USA and Canada). Electrocution from lightning strikes is rare, but causes about 1000 deaths worldwide each year.

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 central respiratory depression or paralysis of the respiratory muscles.
- Current may precipitate **VF** if it traverses the myocardium during the vulnerable period (analogous to an R-on-T phenomenon). 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 strikes deliver as much as 300 kV over a few milliseconds. Most of the current from a lightning strike passes over the surface of the body in a process called external flashover. Both industrial shocks and lightning strikes cause deep burns at the point of contact - in industry the points of contact are usually on the upper limbs, hands and wrists, whilst with 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. Explosive force generated by a lightning strike may cause blunt trauma.

The pattern and severity of injury from a lightning strike varies considerably. As with industrial and domestic electric shock, death is caused by cardiac or respiratory arrest. In those who survive the initial shock, extensive catecholamine release or autonomic stimulation may occur, causing hypertension, tachycardia, nonspecific 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 also causes various central and peripheral neurological problems.

Treatment

Ensure that any power source is switched off and do not approach the victim 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 victim. It is safe to approach and handle casualties after lightning strike, although it would be wise to move to a safer environment. Follow standard resuscitation guidelines.

- Airway management can be difficult if there are electrical burns around the face and neck. Intubate the trachea early in these cases as soft tissue oedema can cause subsequent airway obstruction. Consider cervical spine immobilisation. This should not delay airway management.
- Muscular paralysis, especially after high voltage, may persist for several hours; ventilatory support is required during this period.
- Ventricular fibrillation is the commonest initial arrhythmia after high voltage AC shock; treat with prompt attempted defibrillation. Asystole is more common after DC shock; use standard guidelines for treatment of this and of other arrhythmias.
- Remove smouldering clothing and shoes to prevent further thermal injury.
- Give fluids if there is significant tissue destruction. Maintain a good urine output to increase excretion of myoglobin, potassium and other products of tissue damage.
- Consider early surgical intervention in patients with severe thermal injuries.

- Conduct a thorough secondary survey to exclude injuries caused by tetanic muscular contraction or from the person being thrown by the force of the shock.
- 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.

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 multiple system 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.

Key learning points

- The conditions described in this chapter account for a large proportion of cardiac arrests in younger patients.
- Use the ABCDE approach for early recognition and treatment to prevent cardiac arrest
- High quality CPR and treatment of reversible causes is the mainstay of treatment of cardiac arrest from any cause.
- Call for expert help early when specialist procedures are needed - e.g. delivery of fetus for cardiac arrest in pregnancy.

Further reading

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