Massive blood transfusion

Loss of 50% of circulating blood volume within 3hr is perhaps the most relevant ED definition of massive blood loss. Resuscitation requires an interdisciplinary team and clear organization.

In the event of massive blood loss

- Protect the airway and give O_2 as required.
- Get help—two nurses and a senior doctor.
- Insert two large-bore cannulae.
- Activate the Massive Haemorrhage Protocol ahead of sending samples.
- Take blood for FBC, U&E, LFTs, coagulation, and cross-matching. Label the blood tubes and ensure they are sent directly to the laboratory. Do not leave them unlabelled or lying around in the resuscitation room.
- Accurate patient ID is essential, even if the patient is unknown. Ensure that the patient is wearing an identifying wristband.
- Call an appropriate senior surgeon—to stop the bleeding as soon as possible.
- Give tranexamic acid 1g IV if within 3hr of injury/bleed.
- Start the Massive Haemorrhage Protocol as per local guidelines. Usually give 1U of FFP per 1U of RBCs (starting with O –ve blood in ♀; O –ve or O +ve blood in ♂).
- Reverse anticoagulation (see ➲ Patients on anticoagulants, pp. 178–9).
- Repeat all bloods, including FBC, clotting, U&E, Ca^{2+}, Mg^{2+}, and fibrinogen levels, every 30min.
- Start platelet transfusion if platelet count falls below 75×10^9/L or if large volumes of blood and FFP have been given.
- Aim to maintain fibrinogen >1.0g/L and INR and APTT <1.5 times normal. Once fibrinogen begins dropping below 2.0g/L, give cryoprecipitate to maintain levels >1.5g/L.
- Recombinant factor VIIa might have been used as a 'last ditch attempt' to control bleeding in the past in some patients but is contraindicated due to thrombotic risk.

Massive transfusion complications

Rapid infusion of blood products may lead to:

Hypothermia Blood products are normally stored at 2–6°C. Rapid infusion can cause significant hypothermia. Use blood warmers routinely for rapid transfusions (eg >50mL/kg/hr or 15mL/kg/hr in children). Never warm a blood product by putting a pack into hot water, on a radiator, or any other heat source.

Electrolyte disturbances With massive transfusion, the citrate anticoagulant may cause significant toxicity, ↓ plasma Ca^{2+} (impairing cardiac function), and acid–base balance disturbance. This is aggravated in patients with underlying liver disease, hypotension, or hypothermia. Citrate may also bind Mg^{2+}, causing arrhythmias. Prophylactic or routine administration of IV Ca^{2+} salts is not recommended. Monitor ECG and measure ionized plasma Ca^{2+} levels during massive transfusion. K^+ levels ↑ in stored blood, and hyperkalaemia may follow massive infusion—check plasma K^+ levels regularly. Transient hypokalaemia may follow 24hr after a large transfusion.

Transfusion reactions

Perform a full set of observations at baseline, after 15min, and at the end of each unit transfused, monitoring to detect early clinical evidence of acute reactions. If the patient develops an ↑ T°, shortness of breath, chest or abdominal pain, or hypotension, suspect a transfusion reaction. Treat allergic reactions, including itching, urticaria, bronchospasm, and fever, conventionally (see ➲ Anaphylaxis, pp. 44–5).

Mismatched transfusion

By far, the most common cause is a clerical error when labelling, ordering, or administering blood. Transfusion of ABO-incompatible blood causes acute severe haemolysis and circulatory collapse. In a hypovolaemic, shocked, or anaesthetized patient, these features may be obscured and missed.

If a transfusion reaction is suspected

ABO incompatibility, haemolytic reaction, bacterial infection, severe allergic reaction, or transfusion-related acute lung injury:

- Stop the transfusion.
- Keep the IV line open with 0.9% saline.
- Record all observations, and give supplemental O_2 as required.
- Double-check the blood unit label with the patient's wrist identity band and other identifiers.
- Send the unit of blood product and the giving set to the blood bank.
- Take blood and send it as follows:
  - An anticoagulated sample for blood bank.
  - U&E, LFTs, immunoglobulin A (IgA) level.
  - Serial mast cell tryptase (at 3hr and 24hr) if severe anaphylaxis.
  - Coagulation screening.
  - Blood cultures if sepsis suspected.
- Contact the blood bank directly by phone.
- Contact the haematologist directly.
- Give broad-spectrum antibiotic if infection suspected.
- Monitor fluid balance and urinary output, and check for Hb in urine.

Transfusion-associated circulatory overload

This is defined as acute or worsening pulmonary oedema within 6hr of transfusion. It is currently the largest cause of mortality and morbidity from transfusion. Assess all patients for their risk of TACO—if at high risk, give units more slowly (over 3.5hr), together with a diuretic and more frequent observations.

The role of blood products

Blood transfusion is not a panacea. An improvement in O_2 delivery cannot be assumed. RBC function deteriorates during storage, and changes in O_2 affinity occur with ↓ 2,3-diphosphoglycerate (DPG) levels, whilst ↓ adenosine triphosphate (ATP) levels alter RBC membrane deformability, causing ↑ cell stiffness and micro-circulatory problems. UK donations are routinely screened for hepatitis B, HIV, human T-cell lymphotropic virus (HTLV), syphilis, and, where necessary, CMV. However, blood cannot be sterilized—small, but definite, risks of infection transmission exist.

Sickle-cell disease

Sickle-cell disease occurs in African, Indian, Middle Eastern, Caribbean, USA, and Mediterranean populations. It is caused by a genetic mutation in one of the chains of the Hb molecule. The normal adult Hb genotype AA produces HbA. In heterozygotes (sickle-cell trait), one gene is abnormal (HbAS) and about 40% of the patient's Hb will be HbS. In homozygotes (sickle-cell anaemia), both genes are abnormal (SS) and >80% of the Hb will be HbS. HbS molecules polymerize in deoxygenated or acidotic conditions, causing RBC sickling. Sickle cells are rigid and fragile. They may haemolyse or block small vessels, leading to tissue ischaemia, infarction, and further sickling (see Fig. 3.39). Sickling also occurs with genes coding for other analogous amino acid substitutions (eg HbSC and SD diseases).

Clinical features

Sickle-cell trait causes no disability, except during conditions of severe hypoxia (eg sudden depressurization in aircraft or during cardiac arrest).

Patients with *sickle-cell anaemia* have chronic anaemia (Hb 80–100g/L), with alternating good health and acute crises. Later, chronic ill health supervenes with renal failure, bone necrosis (evident in 50% of patients by age 35y), osteomyelitis, leg ulcers, and iron overload as a consequence of transfusions. There is predisposition to infection, especially *Staphylococcus, Pneumococcus*, and *Haemophilus*.

Sickle-cell crises can occur *de novo* or follow infection, cold, dehydration, or any situation where tissue hypoxia/ischaemia occurs. The crisis may involve thrombosis, haemolysis, marrow aplasia, or acute splenic/liver sequestration (especially in children aged <5y). Any acute medical or surgical emergency may be mimicked (eg acute abdomen, PE, stroke). Severe aching bony pain and low-grade fever (even in the absence of infection) are common. Cerebral sickling may present with bizarre behaviour, psychosis, fits, TIAs, stroke, or other focal neurological signs. Priapism, jaundice, and painful swelling of the hands and feet may occur.

Acute chest syndrome

The leading cause of death in sickle-cell anaemia. It presents as chest pain, hypoxia, and pulmonary infiltrates. There may be cough, tachypnoea, and wheezing. Poorly understood, but infection may be a precipitant.

Acute splenic sequestration

Sudden trapping of large numbers of RBCs in the spleen results in severe anaemia, an enlarging spleen, hypovolaemia, and thrombocytopenia. It occurs most commonly in young children—those with sickle-cell disease have a 30% chance of having acute splenic sequestration by the age of 5y. It may present with shock and splenomegaly, with a mortality of >15%.

Osteomyelitis and septic arthritis

Osteomyelitis and septic arthritis occur more commonly in sickle-cell disease. Be suspicious if a patient presents with high fever, soft tissue swelling, or pain in a different pattern to normal. *Salmonella* is frequently implicated.

Investigations

No specific tests can detect a sickle-cell crisis:

- All patients in the at-risk groups require a sickle test before any anaesthetic procedure (including regional anaesthesia and Bier's block).
- Sickle testing (using an oxidizing agent) will detect sickling in homo- and heterozygous forms. Hb electrophoresis can then distinguish between HbSS, HbAS, and other Hb variants.
- FBC typically reveals significant anaemia (Hb 60–80g/L, but Hb may be much lower if acute haemolysis, sequestration, or aplasia are present). Post-splenectomy features may be seen on blood film. WCC may be ↑ (20–60 × 10^9/L) in the absence of infection and platelet count is also usually ↑.
- Infection screen, including blood cultures, MSU, and CXR.
- Joint aspiration for culture if septic arthritis is suspected.
- U&E, ABG, ECG.
- Arrange CT brain scan if there are neurological symptoms or signs.

Management of crises

Provide supportive therapy, directed to the patient's symptoms:

- Get expert help!
- Keep the patient warm and rested, and give O_2 if any obvious symptoms or SpO_2 <94%.
- Opioids (given IV and titrated to response) are often required for pain. Consider morphine IVI or a patient analgesia pump.
- Commence rehydration with PO or IV fluids, but take care not to precipitate heart failure.
- Transfusion may be required if severe anaemia from acute haemolysis, sequestration, or aplasia occurs, or if there are CNS or lung complications.

Empirical antibiotic therapy may be required if infection is thought to be the trigger for the sickling crisis.

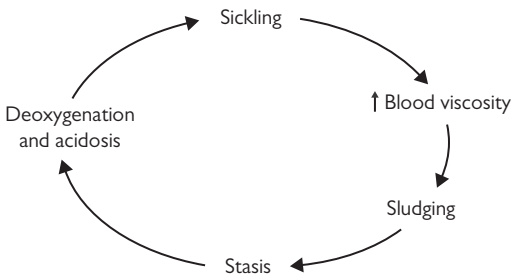

Fig. 3.39 The sickling cycle.

Chapter 4

Toxicology

Poisons: background

Types of poisoning

Unintentional poisoning Most common in inquisitive small children (1–4y) who eat tablets, household chemicals, and plants. Older children and adults may be poisoned by chemicals at school or work, or by drinking toxic fluids decanted into drink bottles. Poisoning by drugs may result from miscalculation or confusion of doses or by taking the same drug under different names. Drug packers and stuffers who swallow drugs wrapped in condoms or polythene or who stuff them in the rectum or vagina may suffer poisoning if the packages leak (see ➔ Body packers, p. 225).

Self-poisoning The most common form of poisoning in adults and may occur in children as young as 6y (usually with a family history of self-poisoning). Drugs or poisons are often taken impulsively, sometimes to manipulate friends or family. Suicidal intent may be relatively uncommon, but assess all patients for this (see ➔ Self-harm, pp. 628–9). Sometimes patients leave suicide notes and conceal drugs/poison to evade detection.

Non-accidental poisoning of children A form of fabricated or induced illness (see ➔ Fabricated or induced illness, p. 760), in which a parent deliberately poisons a child. Homicidal poisoning is rare and may involve acute or chronic poisoning with chemicals such as arsenic or thallium.

Chemical plant incidents and terrorism Potential threats to large numbers of people.

National Poisons Information Service

TOXBASE, the UK National Poisons Information Service (NPIS) database on clinical toxicology, is available at 🖱 https://www.toxbase.org and can be downloaded as an app for phones and tablets. It includes information about poisoning with drugs, household products, plants, and fungi, as well as industrial and agricultural chemicals and agents which might be deliberately released by terrorists. Access to TOXBASE is password-protected and is restricted to NHS staff in the UK and hospitals in Ireland.

When accessing TOXBASE for clinical guidance, print off the information and place it in the notes to guide ongoing care. TOXBASE was most frequently consulted in 2015/16 in relation to poisoning by the following: paracetamol, ibuprofen, codeine, diazepam, sertraline, citalopram, mirtazapine, zopiclone, quetiapine, and tramadol.

TOXBASE provides sufficient information for most cases of poisoning—refer to it 'routinely', as toxicology is a dynamic specialty and advice frequently changes and is updated. More detailed information/advice is available from Poisons Information Centres and is especially useful for complex cases or severe poisoning.

The UK NPIS has four centres, with a single telephone number 0344 892 0111 which directs the call to the nearest centre, or to the on-call centre out of hours. In Ireland, advice is available from the National Poisons Information Centre, Dublin, telephone (01)809 2566.

Enquiries to Poisons Information Centres are usually answered initially by an information officer using TOXBASE and other reference sources. Medical staff with specialist toxicology experience are available for advice about seriously poisoned patients. Poisons Information Centres can also advise about sources of supply of antidotes that are needed only occasionally and about laboratory analyses that may be helpful in managing some patients.

An additional useful source of information is the *BNF*, which contains a chapter on the emergency treatment of poisoning.

Advice for the public

NHS 111 in England, NHS 24 in Scotland, and NHS Direct in Wales will provide advice to members of the public who have concerns about poisoning.

Psychiatric assessment and admission after poisoning

Adults

Admit patients who are seriously poisoned to a medical ward or, if appropriate, to ICU. However, most patients who take overdoses suffer no serious ill effects and can be treated on an ED observation ward or in a Clinical Decisions Unit. Even if there is no risk of toxicity, admission overnight provides an opportunity for a 'cooling off' period for the patient to get away from the situation that precipitated the overdose and/or time to sober up. This should allow a more rational appraisal of the problems and may reduce the risk of further self-poisoning.

Look for the causes of every episode of self-harm. Observe carefully in the ED and on the ward any patient who appears suicidal (see ➔ Self-harm, pp. 628–9), because of the risk of further self-harm.

Children with poisoning

Serious poisoning is uncommon in children. Many children appear well but have been exposed to an unknown amount of a compound which could be toxic. Admit such children to a paediatric ward for observation—they can be discharged after a few hours if no toxic effects occur. A child may be discharged home directly from the ED if the substance taken is known to be non-toxic. The health visitor may usefully visit the home to advise about poisoning prevention. In children >6y, consider the possibility of intentional self-harm and the need for assessment by the child and adolescent mental health services (CAMHS).

Diagnosis of poisoning

The patient or relatives/friends may state what drugs or poison have been taken, but this information is not always accurate. Self-poisoning is often an impulsive act whilst under the influence of alcohol—the patient may not know which tablets (and how many) he/she took. There is often confusion around compound analgesics and the use of trade names. Check any bottles or packets for the names and quantities of drugs or poisons that were available. If a patient is unconscious or severely poisoned, check hospital records for details of previous overdoses and find out from the GP what drugs had been prescribed. Record the time of ingestion of the drug or poison. Examine for signs of poisoning, injection marks, or self-injury. Exclude other processes mimicking poisoning (eg head injury, meningitis). Traditional Chinese medicines or herbal preparations can cause significant toxicity. Drugs of abuse and 'legal highs' (new psychoactive substances) pose an ↑ burden on EDs.

Toxidromes: features suggesting a particular poison

- Coma with dilated pupils, divergent squint, tachycardia, ↑ muscle tone, and ↑ reflexes and extensor plantars suggest tricyclic antidepressant or orphenadrine poisoning (see ➲ Tricyclic antidepressant poisoning, pp. 202–3).
- Coma with hypotension, respiratory depression, and ↓ muscle tone suggest barbiturates, clomethiazole (see ➲ Clomethiazole poisoning, p. 204), benzodiazepines with alcohol, or severe tricyclic antidepressant poisoning (see ➲ Tricyclic antidepressant poisoning, pp. 202–3).
- Coma with slow respiration and pinpoint pupils is typical of opioid poisoning (give naloxone) (see ➲ Opioid poisoning, p. 196).
- Tinnitus, deafness, hyperventilation, sweating, nausea, and tachycardia are typical of salicylate poisoning (see ➲ Salicylate poisoning, p. 197).
- Agitation, tremor, dilated pupils, and tachycardia suggest amphetamines, ecstasy, cocaine, sympathomimetics (see ➲ Recreational drugs, pp. 222–3), tricyclic antidepressants (see ➲ Tricyclic antidepressant poisoning, pp. 202–3), or selective serotonin reuptake inhibitors (SSRIs) (see ➲ Serotonin syndrome, p. 224).

Assessment and monitoring

- Assess and record conscious level (see ➲ Glasgow Coma Score (adults), p. 369). Observe frequently.
- Check blood glucose in patients with confusion, coma, or fits.
- Monitor breathing and RR. Use a pulse oximeter—note that the SpO_2 may be misleading in CO poisoning (see ➲ Carbon monoxide poisoning, p. 216).
- Check ABG (or VBG) if the patient is deeply unconscious or breathing abnormally.
- Record and monitor the ECG if the patient is unconscious, has tachy- or bradycardia, or has taken drugs or poisons with a risk of arrhythmias.
- Record BP and T°.

Investigations in poisoned patients

Most useful are: paracetamol and salicylate levels, blood glucose, ABG/VBG, and U&E. Measure paracetamol if there is any possibility of paracetamol poisoning (this includes all unconscious patients). Record the time of the sample accurately. Many labs can measure salicylate, iron, and lithium and even check for paraquat, if necessary. Comprehensive drug screening is rarely needed and is only available in specialist centres (discuss with NPIS) (see ➲ National Poisons Information Service, pp. 188–9).

Poisons: supportive care

Protect airway, monitor breathing, and ventilate if necessary

Hypoxia and CO_2 retention are common in deep coma. In an unconscious patient, use a cuffed endotracheal (ET) tube if there is no gag reflex. If an oral or nasal airway is needed, nurse in the recovery position to minimize the risk of aspiration in case vomiting or regurgitation occurs.

Hypotension

This may result from relative hypovolaemia, arrhythmias, and cardiodepressive effects of drugs. Treat according to the cause. Elevate the foot of the trolley. If BP is <90mmHg, consider giving saline 500mL IV. Vasopressors, inotropes, glucagon, or high-dose insulin may be required, under expert guidance.

Cardiac arrhythmias

Generally rare in poisoned patients. Drugs most implicated are tricyclics, β-blockers, chloral hydrate, digoxin, K^+, bronchodilators, verapamil, and amphetamines. Correct hypoxia, respiratory depression, metabolic acidosis, and electrolyte abnormalities. Anti-arrhythmic drugs are rarely needed—get expert help.

Convulsions

Dangerous because they cause hypoxia and acidosis and may precipitate cardiac arrest. Drugs responsible include tricyclic antidepressants, mefenamic acid, and theophylline. Check for, and correct, hypoxia and hypoglycaemia. Do not give anticonvulsants if fits are single and brief, but if fits are repeated or prolonged, give lorazepam 4mg IV (or PR diazepam or buccal midazolam if venous access is not available).

Hypothermia

May occur with any drug causing coma, especially barbiturates, clomethiazole, and phenothiazines. Check rectal T° with a low-reading thermometer. Insulation and passive rewarming are usually adequate.

Hyperthermia

(See ➲ Heat illness, pp. 274–5.)

May occur with amphetamines, cocaine, ecstasy, monoamine oxidase inhibitors (MAOIs), sympathomimetics, and theophylline. Consider serotonin syndrome (see ➲ Serotonin syndrome, p. 224). Convulsions and rhabdomyolysis are common. Active cooling, cyproheptadine, chlorpromazine, and possibly dantrolene are needed. Get expert help.

Complications of immobility

Prolonged immobility (eg due to tricyclics and barbiturates) risks pressure areas. Treat blisters like minor burns. Immobility may cause rhabdomyolysis (leading to renal failure), nerve palsies, and compartment syndrome—if this is suspected, check CK; test urine for myoglobinuria, and get urgent orthopaedic advice about measuring compartment pressures.

Urinary retention

Common in coma, especially after tricyclic poisoning. Suprapubic pressure often stimulates reflex bladder emptying. Catheterization may be needed to empty the bladder or to measure urine output.

Reducing absorption of poison

Several methods aiming to reduce absorption of a poison have been described, but none can be recommended routinely.

Gastric lavage

Now almost only of historical interest, gastric lavage (for an adult) involves the insertion of a large orogastric tube (36 or 40FG), then after clinically confirming the position, pouring 300mL aliquots of tepid water down the tube, then siphoning it out until the effluent is clear.

This does not empty the stomach of solids and may force gastric contents through the pylorus into the small bowel. It may cause hypoxia, aspiration pneumonia, and occasionally oesophageal perforation. Gastric lavage >1hr after an overdose is ineffective in ↓ the absorption of poisons.

Induced emesis

Never use emetics. *Ipecacuanha* (ipecac) was once used frequently, but there is no indication for its use. *Salt solutions* may cause fatal hypernatraemia and must never be used as an emetic.

Activated charcoal

Given within 1hr, this ↓ the absorption of therapeutic doses of many drugs, but there is little evidence of clinical benefit when it is used after an overdose. Charcoal ↓ the half-life of some drugs (eg digoxin), which undergo entero-hepatic recycling. However, charcoal is messy and unpleasant to take, and often causes vomiting. Aspiration into the lungs can result in fatal pneumonitis. Various formulations of activated charcoal are available (eg *Charcodote*® and *Carbomix*®). *Carbomix*® may cause severe constipation, especially if given in repeated doses.

Do not give activated charcoal for substances which do not bind to it. These include: iron, lithium, boric acid, cyanide, ethanol, ethylene glycol, methanol, organophosphates, petroleum distillates, and strong acids and alkalis. Charcoal is most likely to be useful for poisons which are toxic in small quantities (eg tricyclic antidepressants and theophylline derivatives). If a dangerous overdose has been taken in the previous 1hr, give charcoal (PO or via an orogastric tube: adult 50g; child 1g/kg, max 50g). Charcoal may be effective for >1hr for sustained-release formulations or drugs that delay gastric emptying (eg tricyclic antidepressants and opioids). Obtain expert advice before giving charcoal in repeated doses, which are only helpful in life-threatening poisoning with a few drugs (eg carbamazepine, dapsone, digoxin, phenobarbital, quinine, theophylline, and salicylate, and a few other drugs rarely taken in overdose).

Whole-bowel irrigation

Whole-bowel irrigation is rarely needed and should only be used on expert advice. The aim of whole-bowel irrigation is to empty the bowel rapidly of solid contents by giving fluid PO or down an NG tube until the rectal effluent becomes clear. The value of this is uncertain. It may be useful for poisoning with sustained-release drug formulations or for poisons such as iron or lithium, which are not absorbed by activated charcoal. It has also been used to remove packets of cocaine from body packers and button batteries from children.

Bowel-cleansing solutions of polyethylene glycol and electrolytes (eg Klean-Prep®) are used in whole-bowel irrigation—2L/hr in adults (500mL/hr in small children) for up to 6hr, or occasionally longer (up to a maximum of 12hr). Do not use normal saline, since it may cause fluid overload and hypokalaemia. Nausea, vomiting, abdominal pain, and electrolyte disturbances may occur. Monitor ECG, U&E, and urine output.

Antidotes for poisons

Antidotes are available for only a few drugs and poisons (see Table 4.1) and are often not necessary. More information is available from TOXBASE and Poisons Information Centres (see ➲ National Poisons Information Centre, pp. 188–9).

Table 4.1 Antidotes for poisons

Poison	Antidote	Notes
Benzodiazepines	Flumazenil‡	See ➲ Benzodiazepine poisoning, p. 204
β-blockers	Glucagon, atropine	See ➲ Beta-blocker poisoning, p. 206
Calcium channel blockers	Calcium, glucagon, high-dose insulin euglycaemic therapy	See ➲ Beta-blocker poisoning, p. 206
CO	O_2	See ➲ Carbon monoxide poisoning, p. 216
Cyanide	Sodium nitrite, sodium thiosulfate, dicobalt edetate, hydroxocobalamin	See ➲ Cyanide poisoning, p. 215
Digoxin	Digoxin antibodies (DigiFab®)†	See ➲ Digoxin poisoning, p. 207
Ethylene glycol	Ethanol, fomepizole†	See ➲ Ethylene glycol poisoning, p. 212
Hydrofluoric acid	Calcium gluconate	See ➲ Chemical burns, p. 405
Iron salts	Desferrioxamine	See ➲ Iron poisoning, p. 209
Local anaesthetics	Lipid emulsion (Intralipid®)	See ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195; ➲ Local anaesthetic toxicity, p. 294
MDMA	Dantrolene†	See ➲ Recreational drugs, pp. 222–3
Methanol	Ethanol, fomepizole†	See ➲ Methanol poisoning, p. 211
Opioids	Naloxone	See ➲ Opioid poisoning, p. 196
Organophosphates	Atropine, pralidoxime†	See ➲ Organophosphate poisoning, p. 214
Paracetamol	Acetylcysteine	See ➲ Paracetamol poisoning, pp. 198–201
Serotonin syndrome (eg SSRI overdose)	Cyproheptadine	See ➲ Serotonin syndrome, p. 224
Sulfonylureas	Glucose, octreotide	See ➲ Sulfonylurea poisoning, p. 205
Tricyclic antidepressants	Sodium bicarbonate, Intralipid®	See ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195; ➲ Tricyclic antidepressant poisoning, pp. 202–3
Warfarin	Vitamin K, prothrombin complex concentrate, FFP	See ➲ Patients on anticoagulants, pp. 178–9
Adder bites	Snake venom antiserum	See ➲ Snake bites, p. 423
Foreign snakes	Antivenoms†	Expert advice (see ➲ National Poisons Information Service, pp. 188–9)

† Very rarely needed—get expert advice. See main text.
‡ Flumazenil is not licensed for use as a standard antidote, except in specific circumstances. See main text.

Antidotes are also available for arsenic, lead, mercury, thallium, and certain other metals. Some antidotes (marked†) are very rarely needed—get expert advice (see ➲ National Poisons Information Service, pp. 188–9) about when and how to use these antidotes (and where to obtain them). Flumazenil (marked‡) is not licensed for use as a standard antidote, except in specific circumstances (see ➲ Benzodiazepine poisoning, p. 204).

Increasing elimination of poisons

The vast majority of poisoned patients recover with supportive care plus appropriate antidotes, if necessary. Active removal of absorbed poison is only needed in special circumstances. Alkalinization of the urine may help in salicylate poisoning (see ➲ Salicylate poisoning, p. 197). Haemodialysis is occasionally used for severe poisoning with salicylates, ethylene glycol, methanol, lithium, phenobarbital, and chlorates. Haemoperfusion is rarely needed but might be helpful (on specialist advice) in severe poisoning with barbiturates, chloral hydrate, or theophylline.

Lipid emulsion (Intralipid®) therapy for drug toxicity

IV lipid emulsion is rarely needed but can be life-saving in overdoses of local anaesthetics such as lidocaine or bupivacaine (see ➲ Local anaesthetic toxicity, p. 294). Haemodialysis is occasionally used for severe poisoning with salicylates. It may be useful in cardiac arrest caused by some other drugs—the indications are unclear, but case reports record dramatic recovery from cardiac arrests due to a variety of drugs. Consider lipid emulsion in drug-induced cardiac arrest unresponsive to standard treatment (see ➲ Cardiac arrest management, pp. 52–3). EDs, theatres, and ICUs should stock it.

Lipid emulsion acts as a 'lipid sink', binding lipophilic drugs and reducing the amount of active free drug. It may also affect myocardial metabolism. Lipid emulsion is not licensed for use in drug overdose, and the safety of rapid infusion is unknown. Lipid interferes with analysis of blood samples, so if possible, take these before starting lipid emulsion, including blood for later measurement of drug concentrations.

Give 20% Intralipid® 1.5mL/kg IV as a bolus (for a 70kg patient, give 100mL), followed by 0.25mL/kg/min for 20–30min to an initial maximum of 500mL. Consider repeating the bolus once or twice for persistent cardiovascular collapse or asystole. Titrate the infusion rate against the clinical response.

Report cases in which lipid emulsion is used to the Poisons Information Service (see ➲ National Poisons Information Service, pp. 188–9).

Insulin therapy in poisoning

Poisoning with cardiac drugs, such as calcium channel blockers (see ➲ Calcium antagonist poisoning, p. 206) and β-blockers (see ➲ Beta-blocker poisoning, p. 206), may cause severe hypotension. If standard treatments are ineffective, get expert advice (see ➲ National Poisons Information Service, pp. 188–9) and consider using insulin therapy, which may improve myocardial carbohydrate metabolism and ↑ BP and cardiac output.

Low toxicity substances

Many ingestions by children, particularly within the household, cause extreme anxiety for parents but are, in reality, of low toxicity. Sometimes mild abdominal discomfort may occur, but severe features are unlikely. Mild symptoms can be treated with small amounts of oral fluids. These substances are numerous and include soil, fresh dog faeces, crayons, felt tip pen ink, and aftersun lotion. A more comprehensive list and poster are available from NPIS (🖱 http://www.npis.org/lowtoxposter2017.pdf).

Opioid poisoning

The opioids include morphine, diamorphine (heroin), pethidine, codeine, buprenorphine, and methadone. These are used as analgesics (sometimes combined with paracetamol, as in co-codamol and co-dydramol), cough suppressants, and anti-diarrhoeal agents. Acute opioid poisoning often occurs in recreational users. Consider opioid poisoning in patients presenting with coma of unknown aetiology (remembering to check for transdermal patches).

Clinical features

Opioid poisoning causes the triad of coma, ↓ RR, and pinpoint pupils. Cyanosis, apnoea, convulsions, and hypotension may occur. Effects of opioids are potentiated by alcohol. Non-cardiogenic pulmonary oedema may result from injecting heroin or other opioids.

Respiratory depression may cause death within 1hr of an opioid overdose. However, delayed respiratory depression can occur in poisoning with co-phenotrope (diphenoxylate and atropine), in which the opioid effects usually predominate over atropine toxicity. Delayed toxicity may occur with slow-release formulations of drugs, and also with methadone which has a very long duration of action (half-life 15–60hr).

Treatment

Clear and maintain the airway. If breathing is inadequate, ventilate with O_2 using a bag and mask or an ET tube. *Naloxone* is a specific antagonist for opioids and reverses coma and respiratory depression if given in sufficient dosage. Give naloxone as a therapeutic trial in suspected opioid poisoning—record coma level, pupil size, and RR, and check for any response. The usual initial dose of naloxone for adults is 0.4mg IV, followed by a further dose of 0.8mg after 60s if no response. The aim is to reverse respiratory depression, not to restore full consciousness. In suspected toxicity due to therapeutic excess or reduced elimination in chronic opioid users or palliative care patients, consider lower doses.

For children, give 100mcg/kg (IV, IM or IN) up to 2mg, repeated as necessary. Intranasal naloxone, given by dripping or spraying the IV solution into the nose over 60s, enables rapid absorption. For children at risk of opioid withdrawal, give 1–10mcg/kg every 60s, titrated according to the response.

Naloxone has a much shorter duration of action than most opioids and so coma and respiratory depression often recur when naloxone wears off. More naloxone is often needed, given IV, by IVI, or IM, the dose adjusted depending on the response. Observe for at least 6hr after the last dose of naloxone and up to 24hr with methadone overdose. If repeat doses are required, consider starting a naloxone infusion.

Persuade patients at risk of respiratory depression to stay in hospital—consider using the Mental Capacity Act (see ➲ Mental Capacity Act, p. 645) if a patient is determined to leave.

Salicylate poisoning

If <125mg/kg body weight of aspirin is ingested and the patient is asymptomatic, harm is unlikely. If the patient denies taking salicylate and has no clinical evidence of poisoning, blood tests to check salicylate levels are not necessary.

Clinical features

- *Commonly*: vomiting, tinnitus, deafness, sweating, vasodilatation, hyperventilation, and dehydration. Hypokalaemia may occur.
- *Severe poisoning* may produce confusion, coma, and convulsions.
- *Children* are prone to developing hyperpyrexia and hypoglycaemia.
- *Rare features* include non-cardiogenic pulmonary oedema, cerebral oedema, and renal failure.

Metabolic and acid–base disturbances

May be complex—adults usually have mixed metabolic acidosis and respiratory alkalosis, but respiratory effects predominate. In small children (± a few adults), acidosis predominates, often with confusion or coma.

Management

Consider giving activated charcoal if a patient has ingested >125mg/kg of salicylate in the previous 1hr (or any amount of methyl salicylate).

Gastric lavage may be of benefit if a patient has ingested >500mg/kg body weight in the previous 1hr. Measure plasma salicylate concentration after at least 2hr in symptomatic, and 4hr in asymptomatic, patients. Take a repeat sample after a further 2hr in those who are symptomatic or have an initial level of ≥200mg/L. Check U&E, glucose, clotting, and ABG/VBG. Consider a second dose of charcoal if the plasma salicylate ↑, suggesting delayed gastric emptying or if enteric-coated tablets have been taken.

Mild poisoning Asymptomatic patients with plasma salicylate <300mg/L (2.2mmol/L) and a normal VBG are medically fit for discharge at 6hr.

Moderate poisoning Patients with salicylate levels of 300–700mg/L (2.2–5.1mmol/L) require treatment. Replace K^+ if low (max 20mmol/hr IV). Give sodium bicarbonate 50–100mmol over 30min. If plasma level is >500mg/L (or 350mg/L in children), consider urinary alkalinization to enhance elimination. Aim for a urinary pH of 7.5–8.5. In adults, this can be achieved by giving 3–5mmol/kg of sodium bicarbonate (eg ~1.5L of 1.26% over 1hr). Beware using 4.2% or 8.4% sodium bicarbonate solution as it is a venous irritant and can cause tissue necrosis if extravasation occurs.

Severe poisoning CNS features, acidosis, or salicylate >700mg/L (5.1mmol/L) are associated with significant mortality. Consider urgent haemodialysis/haemodiafiltration. In life-threatening poisoning (coma and extreme hyperventilation), consider paralysis and IPPV, whilst haemodialysis removes salicylate and corrects the electrolyte disturbances.

Paracetamol poisoning

Paracetamol may cause severe liver damage if >150mg paracetamol/kg body weight are taken. Severe toxicity is unlikely if <75mg/kg has been ingested. In obese patients (>110kg), calculate the toxic dose in mg/kg, and the dose of acetylcysteine using a weight of 110kg, rather than the patient's actual weight.

A metabolite of paracetamol (*N*-acetyl-*p*-benzoquinone imine, NAPQI) binds glutathione in the liver and causes hepatic necrosis when stores of glutathione are exhausted. Renal failure from acute tubular necrosis occurs occasionally, but renal failure without liver failure is rare.

Risk factors for paracetamol toxicity

Previously, some patients were deemed to be at ↑ risk of liver damage (eg alcoholics and patients on enzyme-inducing drugs) and were treated differently. However, this is of historical interest only, as all patients are now treated as if 'high risk', with a low threshold for acetylcysteine use.

Clinical features

Nausea, vomiting, and abdominal discomfort are common within a few hours. In untreated patients developing liver damage, vomiting continues beyond 12hr and there is pain and tenderness over the liver (from 24hr), jaundice (at 2–4 days), and sometimes coma from hypoglycaemia (at 1–3 days) and hepatic encephalopathy (onset at 3–5 days). Loin pain, haematuria, and proteinuria suggest incipient renal failure. Hepatic failure causes bleeding from coagulation abnormalities and hyperventilation from metabolic acidosis. In fatal cases, cerebral oedema, septicaemia, and DIC are common. However, many patients survive severe liver damage and recover completely.

LFTs are normal until >18hr after the overdose. The most sensitive lab evidence of liver damage is often a prolonged INR (from 24hr after overdose). Liver enzymes (ALT and AST) may reach >10,000U/L at 3–4 days. Bilirubin rises more slowly (max at about 5 days).

Paracetamol antidotes

Acetylcysteine is given by IVI in 5% glucose. Initial dose is 150mg/kg body weight in 200mL of glucose over 1hr, 50mg/kg in 500mL over 4hr, then 100mg/kg in 1L over 16hr. Acetylcysteine can cause side effects (which are more likely if the plasma paracetamol level is low): erythema and urticaria around the infusion site or more generalized rashes, itching, nausea, angio-oedema, bronchospasm, and rarely hypotension or hypertension. Side effects are dose-related and usually start in the first hour of treatment. If they occur, stop the infusion and give an antihistamine (eg chlorphenamine 10mg IV over 1min). When symptoms have settled, resume acetylcysteine at a slower rate—consider giving the first bag over 2hr and the rest at the normal rate.

Evidence suggests a modified 12hr regimen may be as efficacious with fewer side effects, but it is not yet in widespread use. *Note:* in rare circumstances (eg lack of venous access), oral acetylcysteine has been given, but it remains an unlicensed indication.

Children

Serious paracetamol poisoning is rare in children. Young children rarely take large amounts of paracetamol, and they metabolize it differently from adults and may have lower risk of hepatotoxicity. However, there are no data for assessing the risk in children, so use the same treatment guidelines as for adults.

If it is certain that <75mg/kg has been taken, then no investigation or treatment is needed—discharge with advice to return if symptoms develop.

Treatment with acetylcysteine is rarely needed in children. Doses are as for adults (see ➲ Paracetamol antidotes, p. 198), but with smaller volumes of fluid for IVI.

Pregnancy

Assess the risk of toxicity and treat as for non-pregnant patients. Acetylcysteine does not seem to carry any risk to the fetus and may protect the fetal liver from damage. Calculate the dose ingested based on the patient's pre-pregnancy weight, and the treatment dose of acetylcysteine based on the current weight.

Paracetamol overdose does not appear to cause teratogenic effects.

Staggered overdoses

If paracetamol has been taken in excess (≥75mg/kg) over >1hr, consider this to be a 'staggered overdose'. Do not use the graph to guide treatment for patients with staggered overdoses. If the patient has symptoms of toxicity or the amount taken was >75mg/kg, take blood for INR, LFTs, U&E, and paracetamol level (which may confirm that some was taken), and treat with acetylcysteine. If in doubt, start treatment and get expert advice.

Outcome of treatment

Treatment with acetylcysteine within 8hr of an overdose is very effective in preventing liver and renal damage. Later treatment is less effective, but still worthwhile.

Late presentation after paracetamol poisoning

Patients who present late are more likely to be severely poisoned than those who present soon after ingestion. Late presenters often have continuing vomiting and abdominal pain, which are symptoms of liver damage. The treatment graph (see Fig. 4.1) may be unreliable at >15hr, because of insufficient data on untreated patients.

Liver transplantation

Liver transplantation is occasionally needed for hepatic failure due to paracetamol overdose in patients who presented or were treated late. Aim to identify and refer patients to a liver transplant unit as soon as possible. Transplant criteria include arterial pH <7.30 (H^+ >50nmol/L) after resuscitation, or PT >100s (INR >6.7), and creatinine >300 micromoles/L in patients with grade 3 or 4 hepatic encephalopathy.

Management of paracetamol poisoning

The time since ingestion is crucial in interpreting paracetamol concentrations and assessing the need for specific treatment. Record the time of ingestion as accurately as possible. When taking blood for paracetamol levels, record the precise time in the notes and on blood forms. Start treatment immediately if the time of ingestion is unknown.

Management within 4hr of ingestion

Consider activated charcoal (see ➲ Activated charcoal, p. 192) if 150mg/kg paracetamol has been taken in the previous 1hr. Take blood at 4hr from ingestion and use the treatment graph (see Fig. 4.1) to assess the risk of liver damage; if the result is above the treatment line, give IV acetylcysteine (see ➲ Paracetamol antidotes, p. 198).

Management at 4–8hr from ingestion

Measure paracetamol level, and use the graph to assess the risk of liver damage. If above the treatment line, or only just below it, give IV acetylcysteine (for doses, see ➲ Paracetamol antidotes, p. 198). Treatment is most effective if started before 8hr—start it at once if the paracetamol level is not available by this time and >150mg/kg has been taken. Patients treated with acetylcysteine within 8hr of an overdose should be medically fit for discharge at the end of the treatment course.

Management at 8–24hr from ingestion

Urgent action is needed—start treatment with IV acetylcysteine immediately if >150mg/kg paracetamol has been taken. Measure plasma paracetamol level (plus creatinine, LFTs, and INR), and use the treatment graph to assess the risk of liver damage. If the paracetamol level is well below the line and the patient is asymptomatic, stop acetylcysteine treatment. Continue acetylcysteine if the level is above the treatment line, if there is doubt about the time of ingestion, or if the patient has nausea or vomiting. After 12–15hr, the graph is less reliable and some laboratories may have a higher limit of detection than the treatment line. If there is any doubt, treat with acetylcysteine, especially if ALT is ↑, even if the level is below the treatment line.

Management at >24hr from ingestion

Measure paracetamol level, LFTs, U&E, creatinine, INR, and ABG. Start treatment with IV acetylcysteine if the patient is clinically jaundiced or has hepatic tenderness. Otherwise, wait for investigation results—treat with acetylcysteine if abnormal and seek advice from NPIS or a liver unit. If the patient is asymptomatic, with non-detectable paracetamol level and normal bloods, treatment is not required.

Management of staggered overdose

Commence acetylcysteine and take bloods at least 4hr after the last ingestion. If the patient is asymptomatic, has normal ALT and U&E, INR <1.3, and a paracetamol level <10mg/L, consider discontinuing acetylcysteine.

Treat similarly for patients with therapeutic excess—see TOXBASE.

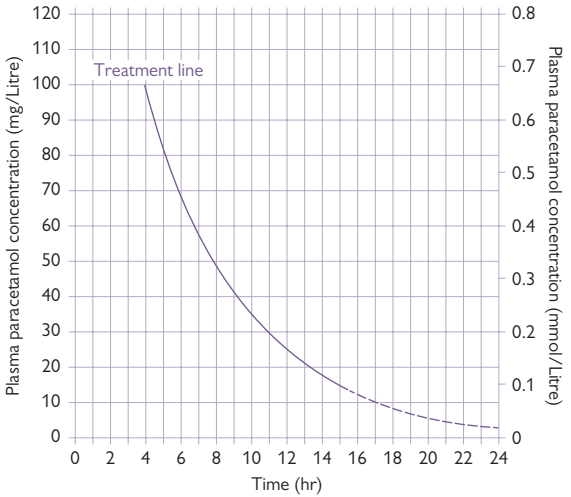

Fig. 4.1 Paracetamol treatment graph.

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Note: check whether the laboratory reports paracetamol in mg/L or mmol/L. Start treatment at once if in doubt about the time of the overdose or if the plasma paracetamol level is only just below the treatment line.

Tricyclic antidepressant poisoning

Tricyclic antidepressants are highly toxic in overdosage. They have anticholinergic effects. They also cause α-receptor and Na^+ channel blockade.

Clinical features

Commonly: tachycardia, dry skin, dry mouth, dilated pupils, urinary retention, ataxia, jerky limb movements, and drowsiness leading to coma. Unconscious patients often have a divergent squint, ↑ muscle tone and reflexes, myoclonus, and extensor plantar responses. The pupils may be dilated and unreactive. In deep coma, there may be muscle flaccidity with undetectable reflexes and respiratory depression requiring IPPV. Fits occur in ~10% of unconscious patients and may precipitate cardiac arrest. Patients recovering from coma often suffer delirium with hallucinations and have jerky limb movements and severe dysarthria.

ECG changes

Sinus tachycardia is usual, but as poisoning worsens, the PR interval and QRS duration ↑. These help to confirm clinical diagnoses of tricyclic poisoning in unconscious patients. The P wave may be superimposed on the preceding T wave, so the rhythm can look like VT when it is sinus tachycardia with prolonged conduction. In severe poisoning, ventricular arrhythmias and bradycardia may occur, especially in hypoxic patients (see Figs. 4.2, 4.3, and 4.4).

Management

- Clear airway, intubate, and ventilate if necessary, and give nursing care.
- Observe continuously, in view of the potential for rapid deterioration.
- Monitor ECG and check ABG in unconscious or post-ictal patients.
- Consider activated charcoal by mouth or gastric tube if a toxic dose has been taken within 1hr—see TOXBASE.
- Do not give anticonvulsants for single brief fits, but give lorazepam or diazepam IV if fits are frequent or prolonged.
- Most arrhythmias occur in unconscious patients within a few hours of overdose. Treat arrhythmias by correcting hypoxia and acidosis. *Sodium bicarbonate* (8.4%, adult: 50–100mL IV; child: 1mL/kg) may dramatically improve the cardiac rhythm and output (by altering protein binding and ↓ active free tricyclic drug). Consider further bicarbonate, depending on the clinical response (especially hypotension), ECG (especially QRS >120ms), and arterial pH. Aim for pH 7.5–7.55, avoiding excessive alkalosis (pH >7.65), which may be fatal.
- Avoid anti-arrhythmic drugs. If arrhythmias do not respond to bicarbonate, discuss with a poisons specialist (see ➲ National Poisons Information Service, pp. 188–9).
- Correct hypotension by giving IV fluids. Glucagon may help in severe hypotension (see ➲ Beta-blocker poisoning, p. 206). Vasopressors or inotropes may be required, on specialist advice.
- Consider Intralipid® (see ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195) for severe arrhythmias or cardiac arrest.
- Do not use physostigmine or flumazenil (risk of precipitating fits).
- Unconscious patients usually improve in ~12hr and regain consciousness in 36hr. In extreme cases, veno-arterial extracorporeal membrane oxygenation or cardiac bypass may be required.

ECG changes in tricyclic antidepressant poisoning

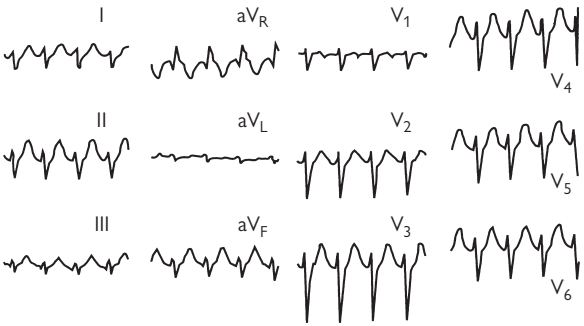

Fig. 4.2 ECG in tricyclic antidepressant poisoning, showing sinus tachycardia with prolonged conduction, which may be mistaken for VT.

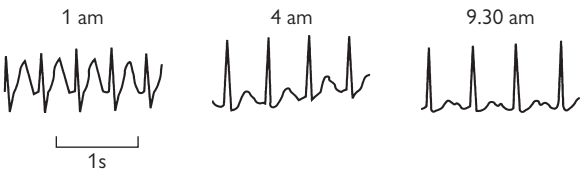

Fig. 4.3 Serial ECG rhythm strips in amitriptyline poisoning, showing spontaneous recovery with supportive care.

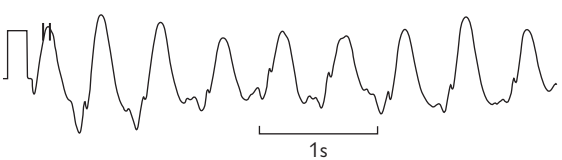

Fig. 4.4 ECG trace in very severe tricyclic antidepressant poisoning. The patient was unconscious, with GCS of 3, and was intubated and ventilated, with BP of 70/50mmHg.

Benzodiazepine poisoning

Benzodiazepines (eg diazepam, nitrazepam, temazepam) rarely cause serious poisoning when taken alone in overdose. However, they potentiate other CNS depressants (eg alcohol, tricyclics, and barbiturates).

Clinical features

Drowsiness, dizziness, ataxia, and dysarthria. Rarely, coma, respiratory depression, and mild hypotension. Fatal poisoning is unusual but may occur from respiratory depression in elderly patients and those with chronic COPD.

Management

Clear the airway and maintain ventilation if necessary. Provide supportive care. Consider giving activated charcoal if the patient has taken a potentially toxic dose within the past 1hr.

Many benzodiazepines have long-acting metabolites, which may affect driving and other motor skills for several days or even weeks after an overdose. Give appropriate warnings about this.

Flumazenil is a specific benzodiazepine antagonist. It reverses the effects of benzodiazepines within 1min but has a short duration of action (<1hr)—as a result, toxic effects may recur. Flumazenil can cause convulsions and cardiac arrhythmias and may precipitate a withdrawal syndrome in patients who are dependent on benzodiazepines. It is particularly dangerous in patients with combined benzodiazepine and tricyclic antidepressant poisoning, in whom it may cause convulsions and cardiac arrest. Flumazenil can be considered to improve ventilation in patients who would otherwise require mechanical ventilation.

Clomethiazole poisoning

Clomethiazole overdosage may cause coma, respiratory depression, ↓ muscle tone, hypotension, and hypothermia. Excessive salivation and a characteristic smell of clomethiazole on the breath are often noticeable. Treat supportively. In severe cases, consider flumazenil and IPPV.

Phenothiazine poisoning

Phenothiazines (eg chlorpromazine), butyrophenones (eg haloperidol), and related drugs are used as antipsychotics and antiemetics. In overdosage, they may cause drowsiness, coma, hypotension, and hypothermia. Deep coma and respiratory depression are uncommon. Some conscious patients suffer *dystonic reactions* with oculogyric crises and muscle spasms causing torticollis or opisthotonus. Convulsions may occur. ECG changes of prolonged PR, QRS, and ST intervals and arrhythmias are seen particularly with thioridazine poisoning.

Treat supportively. Activated charcoal may help. If cardiac arrhythmias occur, correct hypoxia, acidosis, and electrolyte abnormalities before giving any anti-arrhythmic drug. Consider sodium bicarbonate if QRS is >120ms. Treat dystonic reactions with procyclidine (5–10mg IV or IM), or alternatively diazepam.

Barbiturate poisoning

Barbiturate poisoning may cause coma, respiratory depression, hypotension, and hypothermia, with effects potentiated by alcohol. There are no specific neurological signs. Skin blisters and rhabdomyolysis may result from prolonged immobility. Treat supportively, with IPPV if necessary. Repeated doses of activated charcoal may help remove barbiturates. Very rarely, charcoal haemoperfusion or haemodialysis helps in some patients with deep/prolonged coma and respiratory complications.

Lithium poisoning

Clinical features Often due to chronic accumulation from therapeutic overdosage or drug interactions (eg with diuretics or NSAIDs), rather than self-harm. Acute-on-chronic overdose is particularly likely to cause serious toxicity. Symptoms may start up to 24hr after an overdose, especially with slow-release tablets. Nausea, vomiting, and diarrhoea are followed by tremor, ataxia, confusion, ↑ muscle tone, and clonus. In severe cases, there may be convulsions, coma, and renal failure. Lithium-induced nephrogenic diabetes insipidus may complicate treatment.

Investigations Measure lithium levels immediately in those on chronic therapy, and at 6hr if lithium-naive (plain tube, not lithium heparin!). Check U&E, creatinine, LFTs, Ca^{2+}, and TFTs. Therapeutic lithium levels are 0.4–1.0mmol/L. In poisoning, interpret levels with caution, with treatment dictated by the type of overdose. Toxic effects are often seen at >1.5mmol/L. Soon after a large overdose, higher levels may occur with few clinical effects, before lithium is distributed to tissues.

Management Activated charcoal does not absorb lithium. Whole-bowel irrigation (see ➲ Whole-bowel irrigation, p. 193) may be considered for slow-release tablets—discuss this with a poisons specialist (see ➲ National Poisons Information Service, pp. 188–9). Use standard supportive measures and control convulsions with diazepam. Observe all patients for >24hr. Give oral fluids in conscious patients. Forced diuresis is contraindicated. Haemodialysis is the best treatment in severe poisoning but often has to be repeated because of rebound release of lithium from tissue stores.

Sulfonylurea poisoning

Overdosage causes hypoglycaemia, which may recur over several days after long-acting drugs such as glibenclamide. Check U&E, glucose, and LFTs. Correct hypoglycaemia with PO or IV glucose (see ➲ Hypoglycaemia, pp. 158–9). Observe and check BMG hourly. To prevent recurrent hypoglycaemia, give 10% glucose IVI; in severe cases, 20% glucose may be needed, via a central line because of venous irritation. Hypokalaemia may occur. In severe poisoning, get expert advice (see ➲ National Poisons Information Service, pp. 188–9) and consider octreotide (unlicensed indication) which blocks pancreatic insulin release—initial dose for adults 50mcg SC or IV. Consider discharge after 6hr (12hr in sustained-release preparations) if asymptomatic and normal BMG not requiring glucose.

Beta-blocker poisoning

Clinical features

Overdose usually causes sinus bradycardia, but the heart rate can be normal. Hypotension, coma, convulsions, and cardiac arrest may occur. ECG changes include marked QRS prolongation and ST and T wave abnormalities. Propranolol may cause bronchospasm in asthmatics and hypoglycaemia in children. Sotalol can cause prolonged QTc and VT, with torsades de pointes.

Management

Monitor ECG, heart rate, and BP. Obtain venous access. Check U&E, Ca^{2+}, FBC, and blood glucose. Consider activated charcoal (see ➲ Activated charcoal, p. 192). Bradycardia and hypotension may respond to atropine (0.5–1.2mg for adult; 0.02mg/kg for child), but this is often ineffective.

Glucagon is the best treatment for severe cardiotoxicity and seems to work by activating myocardial adenylyl cyclase in a way not blocked by β-blockade. Glucagon 5–10mg IV (50–150mcg/kg for a child) usually produces a dramatic improvement in pulse and BP, with return of cardiac output and consciousness. Glucagon often causes vomiting—anticipate this and position the patient appropriately. In severe poisoning, glucagon has a transient effect on cardiac output—commence an infusion after the initial bolus (adults: 50–150mcg/kg/hr; children: 50mcg/kg/hr).

If glucagon is unavailable or ineffective, consider vasopressors (eg metaraminol) or inotropes (eg adrenaline) under expert guidance—see TOXBASE.

Cardiac pacing is an option for bradycardia but is often ineffective. Occasionally, circulatory support is needed by prolonged chest compressions or extracorporeal cardiac bypass whilst more glucagon is obtained or the β-blocker metabolized. Consider insulin therapy (see ➲ Insulin therapy in poisoning, p. 195) for severe hypotension, and Intralipid® (see ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195) in cardiac arrest.

Calcium antagonist poisoning

Poisoning with verapamil, nifedipine, diltiazem, or other Ca^{2+} channel blockers is rare but may be fatal. Nausea, vomiting, dizziness, and confusion may occur. Bradycardia and AV block may lead to AV dissociation, with profound peripheral vasodilatation, hypotension, and cardiac arrest (especially in patients taking β-blockers). Metabolic acidosis, hyperkalaemia, and hyperglycaemia may occur.

Treat supportively—monitor ECG and BP. Consider charcoal. Check U&E, glucose, and Ca^{2+}. Give atropine (0.5–1.2mg; child 0.02mg/kg) for symptomatic bradycardia. Pacing may be needed. Calcium chloride (0.2mL/kg of 10% IV up to 10mL over 5min, observing ECG) may ↓ prolonged intracardiac conduction—consider repeating every 10–20min up to four doses. Glucagon may help, as in β-blocker poisoning. Inotropic support with dobutamine, isoprenaline, adrenaline, or high-dose insulin therapy (see ➲ Insulin therapy in poisoning, p. 195) may be needed to maintain cardiac output. In severe poisoning or cardiac arrest, consider Intralipid® (see ➲ Lipid emulsion (Intralipid®) therapy for drug toxicity, p. 195).

Digoxin poisoning

Toxicity from the therapeutic use of digoxin is relatively common. Acute poisoning is rare but may be fatal. Similar effects occur with digitoxin and, very rarely, with plants containing cardiac glycosides (foxglove, oleander, and yew).

Clinical features

Nausea, vomiting, malaise, delirium, and xanthopsia (yellow flashes or discoloration of vision). Acute poisoning usually causes bradycardia with PR and QRS prolongation. There may be AV block, AV dissociation, and escape rhythms, sometimes with ventricular ectopics or VT. Hyperkalaemia occurs and, in severe cases, metabolic acidosis due to hypotension and ↓ tissue perfusion.

Management

Provide supportive treatment. Monitor ECG and BP. Obtain venous access. Give activated charcoal to ↓ absorption and prevent entero-hepatic recycling of digoxin (see Activated charcoal, p. 192). Measure U&E, Mg^{2+}, plasma digoxin (after 6hr), and ABG/VBG in severe poisoning. Get expert help for severely poisoned patients. Digoxin levels do not correlate well with toxic features, but treat hyperkalaemia actively. Use digoxin-specific antibodies for severe bradycardia, VT, and severe hyperkalaemia (K^+ >6.5mmol/L)—consider repeat doses. Correct severe metabolic acidosis with sodium bicarbonate. Bradycardia and AV block often respond to atropine IV (total 1.2mg; child 0.02mg/kg). Cardiac pacing may be helpful. Treat severe poisoning with digoxin antibodies (DigiFab®), which, together with insulin/glucose, may rapidly reduce serum K^+ level. Anaphylactoid reactions occur in up to a quarter of patients.

ACE inhibitor poisoning

Overdosage with ACE inhibitors (eg captopril, enalapril, lisinopril) may cause drowsiness, hypotension, hyperkalaemia, and, rarely, renal failure. Monitor BP and ECG. Give IV 0.9% saline if BP is low. Check U&E, FBC, and LFTs. Consider activated charcoal (see Activated charcoal, p. 192).

Theophylline poisoning

Theophylline and aminophylline can cause fatal poisoning. Many preparations are slow-release and may not produce serious toxicity until 12–24hr after ingestion, so ensure careful observation.

Features

Nausea, vomiting (often severe and not helped by antiemetics), abdominal pain, haematemesis, restlessness, ↑ muscle tone, ↑ reflexes, headache, and convulsions. Coma, hyperventilation, hyperpyrexia, and rhabdomyolysis may occur. Sinus tachycardia may be followed by supraventricular and ventricular arrhythmias and VF. BP may initially ↑ but later ↓ in severe poisoning. Complex metabolic disturbances include respiratory alkalosis, followed by metabolic acidosis, hyperglycaemia, severe hypokalaemia, and hypomagnesaemia.

Management

- Treat supportively and monitor ECG, heart rate, and BP.
- Measure U&E, Ca^{2+}, Mg^{2+}, PO_4^-, glucose, ABG, and plasma theophylline (repeated after a few hours). If symptomatic, repeat K^+ hourly, as correcting hypokalaemia may prevent dangerous arrhythmias. Correct hypokalaemia with K^+ (no faster than 20mmol/hr).
- Consider gastric lavage if <1hr since ingestion. Give repeated activated charcoal (see ➲ Activated charcoal, p. 192), by NG tube if necessary.
- Consider ondansetron (8mg slowly IV in adult) for intractable vomiting.
- GI bleeding may require transfusion and ranitidine.
- Observe closely if there is tachycardia with an adequate cardiac output. Non-selective β-blockers (eg propranolol) may help severe tachyarrhythmias and hypokalaemia, but cause bronchospasm in asthmatics. Lidocaine and mexiletine may precipitate fits, so disopyramide is preferable for ventricular arrhythmias.
- Control convulsions with diazepam or lorazepam.
- Treat ventricular arrhythmias with direct current (DC) cardioversion.
- Consider charcoal haemoperfusion or haemodialysis in severe poisoning, especially if PO or NG activated charcoal is impracticable due to vomiting. Serious hyperkalaemia may occur during recovery from theophylline poisoning if large amounts of K^+ were given earlier.

Salbutamol poisoning

Poisoning with β_2-agonists may cause vomiting, agitation, tremor, tachycardia, palpitations, hypokalaemia, and hypertension. Rarely, hallucinations, hyperglycaemia, delayed hypoglycaemia, ventricular tachyarrhythmias, myocardial ischaemia, and convulsions occur.

Treat supportively:

- Correct hypokalaemia by infusion of K^+ (max 20mmol/hr).
- Monitor ECG and BP.
- Activated charcoal may ↓ drug absorption.
- Do not treat tachycardia if there is an adequate cardiac output. Metoprolol or esmolol may help severe tachyarrhythmias and hypokalaemia, but can precipitate bronchospasm in asthmatics.

Iron poisoning

Iron tablets may resemble sweets and so can be ingested by inquisitive children. Serious poisoning is uncommon, but fatalities can occur. Note that iron is present in some weed/seed preparations.

Different preparations contain the equivalent of 35–105mg of elemental iron per tablet, sometimes in slow-release form.

Features

In the first few hours after ingestion, nausea, vomiting, diarrhoea, and abdominal pain are common. Vomit and stools are often grey or black and may contain blood. Hyperglycaemia and ↑ WCC may occur. Most patients do not develop further features.

In severe poisoning, early effects include haematemesis, drowsiness, convulsions, coma, metabolic acidosis, and shock.

Early symptoms settle after 6–12hr, but a few patients then deteriorate 24–48hr after ingestion, with shock, hypoglycaemia, jaundice, metabolic acidosis, hepatic encephalopathy, renal failure, and occasionally bowel infarction. Survivors may develop gastric strictures or pyloric obstruction 2–5 weeks after the overdose.

Management

- Employ supportive measures, as required.
- Check serum iron, FBC, glucose, LFTs, INR, and also ABG/VBG in severe poisoning. Severe poisoning causes metabolic acidosis and may cause RBCs to haemolyse.
- Do not give charcoal, as it does not absorb iron. Iron tablets are radio-opaque and can be counted on a plain abdominal X-ray film. Whole-bowel irrigation (see ➲ Whole-bowel irrigation, p. 193) may be useful if many tablets remain in the gut, especially with slow-release formulations.
- Obtain expert advice in serious poisoning. Coma and shock indicate severe poisoning needing immediate treatment with *desferrioxamine* by IVI (15mg/kg/hr, until a max of 80mg/kg has been given). If there are features of severe poisoning, commence desferrioxamine prior to obtaining the serum iron concentration. Measurement of total iron-binding capacity may give misleading results after iron poisoning.
- Desferrioxamine causes hypotension if infused too rapidly and can produce rashes and, rarely, anaphylaxis, pulmonary oedema, or ARDS. The iron–desferrioxamine complex makes the urine orange or red, which confirms that free iron has been bound and that desferrioxamine was required.
- Patients who still have no symptoms 6hr after an iron overdose have probably not ingested toxic amounts and may be discharged, with advice to return if symptoms develop.
- Pregnancy does not alter the treatment needed for iron poisoning—use desferrioxamine if indicated.

Ethanol poisoning

Features

Alcohol initially causes disinhibition and later ataxia, dizziness, dysarthria, and drowsiness. It potentiates the CNS-depressant effects of many drugs. In addition to the characteristic odour of alcoholic beverages, there may be nystagmus to horizontal gaze.

Severe poisoning

Patients who present with severe alcohol intoxication may be comatose, with respiratory depression, hypotension, hypothermia, and metabolic acidosis.

Hypoglycaemia is a particular problem in children who have consumed alcohol and may occur after some hours.

Death may result from respiratory failure or aspiration of vomit. For an adult, the *fatal dose of ethanol* alone is ~300–500mL of absolute alcohol—whisky and gin usually contain 40–50% ethanol. *Note*: the UK legal limit for driving is 80mg/dL; patients who have a blood alcohol level of >350mg/dL are at significant risk of death from acute alcohol intoxication. The rate of clearance of alcohol from the blood varies enormously between individuals, with typical quoted values of 10–20mg/dL/hr in most adults, although this can be much higher in some chronic alcoholics.

Never assume that ↓ GCS is due to alcohol until other causes have been excluded (especially hypoglycaemia, head injury, post-ictal state, meningitis/encephalitis, hepatic encephalopathy, and intoxication with other drugs).

Management

- Maintain a clear airway and adequate ventilation. Nurse in the recovery position to protect the airway.
- Measure blood alcohol—if low, it may challenge the diagnosis and indicate an alternative aetiology.
- Check blood glucose every 1–2hr in severe poisoning. Correct hypoglycaemia with glucose, not glucagon (unless IV access is difficult).
- Look for signs of injury, especially head injury, and adopt a low threshold for a CT brain scan to search for intracranial haemorrhage.
- Treat co-ingested poisons appropriately.
- If obtunded, check CK for evidence of rhabdomyolysis (especially if there is a history of the patient having been lying for several hours).
- Gastric lavage and activated charcoal are ineffective in ethanol intoxication.
- Involve ICU and consider dialysis in extreme cases.

Methanol poisoning

Ingestion of 10mL of pure methanol may cause blindness, and 30mL can be fatal, the toxic effects being due to the metabolites formaldehyde and formic acid. *Methylated spirits* contain toxicologically insignificant amounts of methanol—toxicity is almost entirely due to ethanol.

Clinical features

Methanol initially causes only mild transient drowsiness. Serious toxicity develops after a latent period of 12–24hr with vomiting, abdominal pain, headache, dizziness, blurring of vision, and drowsiness leading to coma. There is severe metabolic acidosis, hyperglycaemia, and ↑ serum amylase. Survivors may be blind from optic nerve damage and develop Parkinsonian problems.

Management

- Consider gastric lavage if <1hr since ingestion. Do not give charcoal.
- Measure ABG, U&E, Cl^-, HCO_3^-, glucose, FBC, LFTs, ethanol, osmolality, and plasma methanol if possible—if not available locally, discuss with Clinical Chemistry. Calculate the osmolar gap and anion gap.
- Read TOXBASE advice (see ➲ National Poisons Information Service, pp. 188–9). Discuss with NPIS.
- Observe for at least 6hr after ingestion, even if asymptomatic.
- Early use of fomepizole or ethanol (as for ethylene glycol—see ➲ Ethylene glycol poisoning, p. 212) minimizes methanol toxicity and should be started if poisoning is likely, especially if there is a high anion gap metabolic acidosis.
- Consider sodium bicarbonate to correct metabolic acidosis (aim for pH 7.44). Hypernatraemia is a risk if a large amount is needed.
- Give folinic acid (30mg IV every 6hr for 48hr).
- In severe poisoning, refer to ICU for haemodialysis and possibly IPPV.

Ethylene glycol poisoning

Ethylene glycol is used mainly as antifreeze. The fatal dose for an adult is about 100g (90mL of pure ethylene glycol). Toxic effects are due to the metabolites glycolaldehyde, glycolic acid, and oxalic acid. Fomepizole or ethanol block ethylene glycol metabolism, preventing toxicity.

Clinical features

In the first 12hr after ingestion, the patient appears drunk but does not smell of alcohol. Ataxia, dysarthria, nausea, vomiting, and sometimes haematemesis occur, followed by convulsions, coma, and severe metabolic acidosis.

From 12–24hr after ingestion, hyperventilation, pulmonary oedema, tachycardia, cardiac arrhythmias, and cardiac failure may develop. Hypocalcaemia may be severe. Acute tubular necrosis and renal failure occur at 24–72hr. Cranial nerve palsies may develop.

Urine microscopy shows calcium oxalate monohydrate crystals which are diagnostic of ethylene glycol poisoning. Some makes of antifreeze contain fluorescein, which makes urine fluoresce in ultraviolet light (eg a Wood's lamp from a dermatology department). This helps to confirm ethylene glycol poisoning, but the absence of fluorescence does not exclude poisoning.

Management

- Consider gastric lavage if <1hr since ingestion. Do not give charcoal.
- Measure ABG, U&E, Cl^-, HCO_3^-, glucose, FBC, LFTs, osmolality, and plasma ethylene glycol if possible (check with the local Clinical Chemistry department). Calculate the osmolar gap and anion gap.
- Read TOXBASE advice and discuss with NPIS (see ➲ National Poisons Information Service, pp. 188–9).
- Observe for at least 6hr after ingestion, even if asymptomatic.
- Monitor ECG, pulse, BP, RR, and urine output.
- High anion gap metabolic acidosis (see ➲ The anion gap, pp. 102–3) occurs in ethylene glycol poisoning (and also methanol poisoning, DKA, alcoholic ketoacidosis, and renal failure), but acidosis only develops after some ethylene glycol has been metabolized.
- Early use of fomepizole or ethanol minimizes toxicity, so commence this if poisoning is likely. Consider fomepizole—discuss with NPIS (see ➲ National Poisons Information Service, pp. 188–9) about indications, dosage, and where to obtain it.
- If fomepizole is not available, give a loading dose of ethanol PO as whisky, gin, or vodka (40% ethanol, 2.5mL/kg PO or 10mL/kg of 10% ethanol IV over 30min). Follow by IVI of ethanol—the dose depends on the usual alcohol consumption and whether dialysis is being used.
- Use sodium bicarbonate to correct metabolic acidosis which has not improved with adequate ventilation and fluid resuscitation (aim for pH 7.44). Large amounts may be needed and hypernatraemia may occur.
- Correct hypocalcaemia with calcium gluconate (10–20mL of 10% slowly IV) only if there are seizures or QTc >500ms. Note that correction risks calcium oxalate stone formation.
- Correct hypomagnesaemia.
- Consider haemodialysis in severe poisoning, with frequent measurements of blood ethylene glycol concentrations (and ethanol if this is used) ± ICU and IPPV.

Paraquat poisoning

Paraquat is a weedkiller which is very toxic if ingested—death is likely after 10mL of liquid paraquat ingestion. Paraquat poisoning is now rare in the UK where paraquat is no longer approved for sale or use.

Clinical features of paraquat ingestion

Paraquat is corrosive and causes immediate burning pain in the mouth and throat, nausea, and vomiting, followed by abdominal pain and diarrhoea. Large amounts result in rapid deterioration, with shock, pulmonary oedema, metabolic acidosis, coma, convulsions, and death within 24hr.

Paraquat lung usually develops by 5–7 days, with pulmonary oedema and fibrosis causing breathlessness and cyanosis. Lung shadowing is seen on CXR. Death from hypoxia occurs 7–14 days after poisoning.

Management

- Avoid supplemental O_2, where possible, as it may ↑ pulmonary toxicity.
- Avoid gastric lavage as oesophageal perforation may occur.
- Consider activated charcoal PO.
- Measure lactate—this may be of prognostic value.
- Send urine to test for paraquat (TOXBASE can advise which hospital labs provide this). A negative test after 4hr of suspected ingestion excludes significant poisoning.

Petrol and paraffin poisoning

Petrol, paraffin (kerosene), and other petroleum distillates contain mixtures of hydrocarbons, often with small quantities of other chemicals. Unintentional poisoning occurs after liquids have been stored in inappropriate containers or siphoning fuel from a vehicle. The major problem is pneumonitis caused by aspiration of hydrocarbons into the lungs.

Clinical features

In many cases, no symptoms occur. There may be nausea, vomiting, and occasionally diarrhoea. Aspiration into the lungs causes choking, coughing, wheeze, breathlessness, cyanosis, and fever. X-ray changes of pneumonitis (shadowing in the mid or lower zones) may occur without respiratory symptoms or signs. Occasionally, pleural effusions or pneumatoceles develop. In severe cases, there may be pulmonary oedema, drowsiness, convulsions, or coma.

Management

Many patients remain well and need no treatment. Avoid gastric lavage. Aim to obtain a CXR at 6–8hr, but request this earlier if required.

Discharge (with advice to return if symptoms develop) those patients who are free of symptoms or signs 8hr after ingestion.

If symptoms occur, treat supportively with O_2 and bronchodilators, together with steroids. Consider CPAP/IPPV if the patient deteriorates.

Organophosphate poisoning

Organophosphates are widely used as insecticides. Poisoning is rare in the UK, but common in many developing countries. Organophosphates are absorbed through the skin, bronchial mucosa, and gut and inhibit cholinesterases, causing accumulation of acetylcholine at nerve endings and neuromuscular junctions. The speed of onset, severity, and duration of toxicity vary between different compounds. Irreversible binding of cholinesterase ('ageing') develops after some minutes or hours. Pralidoxime reactivates cholinesterase if given promptly, before ageing occurs.

Organophosphate nerve gas agents, such as sarin, may be released deliberately by terrorists. Information is available from TOXBASE (see ➲ National Poisons Information Service, pp. 188–9).

Carbamate insecticides act similarly to organophosphates, but poisoning with carbamates is generally less severe and pralidoxime is not needed.

Clinical features

Minor exposure to organophosphates may cause subclinical poisoning with ↓ cholinesterase levels, but no symptoms or signs. Symptoms may be delayed by 12–24hr after skin exposure.

Early features of toxicity include anxiety, restlessness, insomnia, tiredness, headache, nausea, vomiting, abdominal colic, diarrhoea, sweating, hypersalivation, and miosis. Muscle weakness and fasciculation may develop.

In severe poisoning, there is widespread paralysis with respiratory failure, pulmonary oedema, profuse bronchial secretions, bronchospasm, convulsions, and coma. Hyperglycaemia and cardiac arrhythmias may occur. Occasionally, delayed effects of poisoning develop 1–4 days after acute poisoning, with cranial nerve palsies, muscle weakness, and respiratory failure which resolve after 2–3 weeks. Peripheral neuropathy may develop after 2 weeks, usually involving the legs.

Management

- Ensure all staff in contact with the patient wear protective clothing to avoid getting contaminated.
- Provide supportive treatment as needed.
- Clear the airway and remove secretions. Give O_2 and IPPV if needed.
- Insert IV cannulae. Take blood for cholinesterase (EDTA tube on ice).
- Give diazepam to treat agitation and control convulsions.
- If there are profuse bronchial secretions or bronchospasm, give atropine IV (adult 2mg; child 0.02mg/kg), repeated every 5min, with the dose doubled each time until the chest sounds clear, systolic BP >80mmHg, and pulse >80. Some patients need >100mg of atropine. If atropine is required, give pralidoxime in addition.
- In moderate or severe poisoning, give pralidoxime. The dose of pralidoxime is 30mg/kg IV over 30min, followed by an IVI at 8mg/kg/hr. Improvement is usually apparent within 30min.
- NPIS can advise on pralidoxime supply and use. In a terrorist incident, the UK has stocks of antidotes in reserve for mass casualty poisonings.

Cyanide poisoning

Cyanide compounds are widely used in industry and may be ingested or inhaled inadvertently or deliberately. Cyanides produced by burning polyurethane foam ↑ mortality from smoke inhalation—if there is severe acidosis, consider cyanide toxicity. Cyanide poisoning may be caused by the drug sodium nitroprusside or ingestion of amygdalin (laetrile) from the kernels of apricots, cherries, and other fruits. Solutions for removing artificial fingernails may contain acetonitrile (methyl cyanide).

Cyanides inhibit cytochrome oxidase, blocking the tricarboxylic acid cycle and stopping cellular respiration. This process is reversible. Inhalation of hydrogen cyanide often causes death within minutes. Ingestion of cyanides can produce rapid poisoning, but food in the stomach may delay absorption and the onset of symptoms. Delayed poisoning may follow absorption of cyanides through the skin. Ingested cyanide compounds react with gastric acid to form hydrogen cyanide, which could poison first-aiders giving mouth-to-mouth resuscitation.

Clinical features

Acute poisoning causes dizziness, anxiety, headache, palpitations, breathlessness, and drowsiness. In severe cases, there may be coma, convulsions, paralysis, pulmonary oedema, cardiac arrhythmias, and cardiorespiratory failure, with metabolic acidosis. Most of the clinical features result from severe hypoxia, but cyanosis is uncommon. Classically, there is a smell of bitter almonds on the breath, but many people cannot detect this.

Management

- Avoid staff getting contaminated.
- Provide supportive measures—give 100% O_2 and monitor ECG.
- Remove contaminated clothing and wash exposed skin.
- Consider activated charcoal or gastric lavage within 1hr of ingestion.
- In mild poisoning, reassurance, O_2, and observation may be all that is required. Exposure to cyanide causes great anxiety—it may be hard to distinguish fear of poisoning and early symptoms of toxicity.
- *Specific antidotes* should be available but are not always needed.

Some specific antidotes to cyanide are dangerous in the absence of cyanide—only give if poisoning is moderate or severe (eg coma). In severe cyanide poisoning, give *dicobalt edetate* (Kelocyanor®) 300mg IV over 1min, repeated if there is no improvement after 1min. In the absence of cyanide, dicobalt edetate may cause cobalt poisoning with facial, laryngeal, and pulmonary oedema, vomiting, tachycardia, and hypotension. In mild poisoning, treat with *sodium thiosulfate* (adult dose 25mL of 50% solution IV over 10min; child 400mg/kg) or with *sodium nitrite* (adult dose 10mL of 3% solution IV over 5–20min; child dose 0.13–0.33mL of 3% solution/kg, ie 4–10mg/kg). Sodium thiosulfate often causes vomiting. Sodium nitrite may cause hypotension. High doses of *hydroxocobalamin* (5g IV over 15min, Cyanokit®) are useful and relatively safe in cyanide poisoning, especially in victims of smoke inhalation.

Carbon monoxide poisoning

Carbon monoxide (CO) is a tasteless and odourless gas produced by incomplete combustion. Poisoning may occur from fires, (old) car exhausts, and faulty gas heaters. CO is also produced by metabolism of methylene chloride (used in paint strippers and as an industrial solvent). CO ↓ the O_2-carrying capacity of blood by binding haemoglobin (Hb) to form carboxyhaemoglobin (COHb). This impairs O_2 delivery from blood to the tissues and also inhibits cytochrome oxidase, blocking O_2 utilization. These effects combine to cause severe tissue hypoxia.

The elimination half-life of CO is 320min on breathing air, 80min on 100% O_2, and 23min on O_2 at 3 atmospheres pressure.

Clinical features

Early features are headache, malaise, nausea, and vomiting (sometimes misdiagnosed as a viral illness or gastroenteritis, especially if several members of a family are affected).

In severe poisoning, there is coma with hyperventilation, hypotension, ↑ muscle tone, ↑ reflexes, extensor plantars, and convulsions. Cherry-red colouring of the skin may be seen in fatal CO poisoning but is rare in live patients. Skin blisters and rhabdomyolysis may occur after prolonged immobility. Pulmonary oedema, MI, and cerebral oedema can occur. Neurological and psychiatric problems sometimes develop later.

Management

- Clear the airway and maintain ventilation with as high a concentration of O_2 as possible. For a conscious patient, use a tight-fitting mask with an O_2 reservoir, but if unconscious, intubate and provide IPPV on 100% O_2.
- Record ECG and monitor cardiac rhythm—look for arrhythmias and signs of acute MI.
- Check VBG or ABG—SpO_2 measurements are misleading in CO poisoning, as are p_aO_2 values, but acidosis indicates tissue hypoxia.
- Check COHb levels (in blood or with a special pulse oximeter)—although these correlate poorly with clinical features, COHb >20% after arrival at hospital suggests serious poisoning. COHb may be up to 8% in smokers without CO poisoning. A nomogram (see ➲ Nomogram of decay of COHb with time, p. 403) can help to estimate COHb at the time of exposure.
- Correct metabolic acidosis by ventilation and O_2—try to avoid bicarbonate, which may worsen tissue hypoxia.
- Consider mannitol if cerebral oedema is suspected.
- *Hyperbaric O_2* therapy is logical, but of no proven benefit for CO poisoning. Transfer to a hyperbaric chamber and pressurization may take hours, and so hyperbaric treatment may be no more effective than ventilation on 100% normobaric O_2. Caring for a critically ill patient in a small-pressure chamber may be impracticable. NPIS no longer recommends hyperbaric O_2 treatment. Previously used criteria were: if a patient has been unconscious at any time, has COHb >20%, is pregnant, or has cardiac complications or neurological or psychiatric features. Details of some hyperbaric chambers are shown in Table 6.2.

Chlorine poisoning

Chlorine gas causes lacrimation, conjunctivitis, coughing, wheezing, breathlessness, and chest pain. Laryngeal and pulmonary oedema may develop within a few hours.

- Give O_2, with bronchodilators if necessary. If there is laryngeal or pulmonary oedema, get senior help and give prednisolone in high dosage (adult 60–80mg/day initially). In severe cases, consider the need for tracheal intubation, IPPV, and admission to ICU.
- If the eyes are painful, irrigate with water or saline, and examine with fluorescein for corneal damage.
- Allow home casualties with minor exposure to chlorine but no symptoms, with advice to rest and return if symptoms develop.
- Patients with symptoms when seen in hospital usually need admission for at least 12hr for observation.

CS gas (tear gas)

CS (orthochlorobenzylidene malononitrile) is used for riot control and police self-protection. It is an aerosol or smoke, rather than a gas. Exposure to CS causes immediate blepharospasm and lacrimation, uncontrollable sneezing and coughing, a burning sensation in the skin and throat, and tightness of the chest. Vomiting may occur. These symptoms usually improve within 10min in fresh air, but conjunctivitis may persist for 30min. Exposure in a confined space may cause symptoms for some hours and is particularly dangerous in people with pre-existing lung disease. Redness or blistering of the skin may develop, due to the solvent in the spray.

Treat patients exposed to CS gas in a well-ventilated area. Ensure that staff wear gloves and close-fitting goggles. Remove contaminated clothes and wash affected skin thoroughly. Remove contact lenses, and give O_2 and bronchodilators if necessary. Reassure the patient that the symptoms will resolve.

If the eyes remain painful, instil local anaesthetic drops and irrigate them with water or saline. When symptoms have settled, record visual acuity and examine the corneas using fluorescein. Refer to an ophthalmologist if symptoms persist.

CN gas (chloroacetophenone) is used in some countries for riot control and in personal defence devices. CN has similar effects to CS but is more toxic.

Chemical incidents

Chemical incidents involving single or multiple casualties may result from accidents (eg release of chlorine gas) or deliberate release of chemicals (by terrorists or others). CBRN (chemical, biological, radiological, and nuclear) incidents have many features in common.

If you know or suspect that a patient has been involved in a chemical incident:

- Inform senior ED staff.
- Avoid contaminating other staff or patients.
- Ensure that you are wearing suitable PPE, unless the patient has already been decontaminated.
- Decontaminate the patient according to departmental guidelines if this has not been done already (see ➲ Decontamination of patients, p. 218).
- Resuscitate as necessary—airway, breathing, and circulation.
- Assess the clinical features and toxic agent.
- Give antidotes if appropriate, and reassess the patient.
- Enquire whether other patients are expected.
- Inform the local health protection team.
- Get expert advice from TOXBASE (see ➲ National Poisons Information Service, pp. 188–9) or the Department for Environment, Food, and Rural Affairs (Defra) CBRN emergency contact: tel 0300 1000 316.
- If deliberate release is suspected, inform the police and involve other agencies and the press officer.

Chemicals which might cause a chemical incident include: chlorine—see ➲ Chlorine poisoning, p. 217; CS gas (tear gas)—see ➲ CS gas (tear gas), p. 217; cyanide—see ➲ Cyanide poisoning, p. 215; and organophosphates—see ➲ Organophosphate poisoning, p. 214.

Information about chemical incidents

- Liaise with NPIS.
- TOXBASE (see ➲ National Poisons Information Service, pp. 188–9) gives details of toxicity and antidotes, with medical, public health, and public briefing documents about 60 chemicals that might be deliberately released.

Infection control and prevention See ➲ Infection control and prevention, pp. 36–7.

Major incidents See ➲ Major incidents, pp. 40–1.

Radiation incidents See ➲ Radiation incidents, pp. 278–9.

Decontamination of patients

Decontamination after exposure to a chemical, biological, or radiation hazard is intended to reduce the risks to the patient and to other people.

Casualties should be decontaminated at the scene after a CBRN incident, but some contaminated patients may arrive at the ED without warning. Many people will be worried about contamination but not actually be at risk. Even with advance planning, it will be challenging to organize the ED, keep 'clean' areas clean, maintain order, and communicate between the 'decon' team (in PPE) and other ED staff.

Plants, berries, and mushrooms

Plants and berries

Many children eat plant leaves or brightly coloured berries, but serious poisoning from plants is very rare. Identify the plant if possible, using reference books. Advice on toxicity and any necessary treatment is available from Poisons Information Services. Many garden and house plants are non-toxic and no treatment is needed after ingestion.

Serious poisoning from *laburnum* is very rare, with only one death recorded in the UK in 50y. No treatment needs to be provided for children who eat laburnum seeds, except for the very few with symptoms (nausea, salivation, vomiting, headache, and rarely convulsions).

Mushroom poisoning

Serious poisoning from mushrooms or fungi is rare. Most deaths are due to *Amanita phalloides* (death cap mushroom). Reference books are useful, but identification of mushrooms from the description or fragments available is often uncertain. Advice on toxicity and treatment is available from Poisons Information Services (see ➲ National Poisons Information Service, pp. 188–9).

Mushrooms found in gardens are unlikely to produce severe poisoning but may cause vomiting and occasionally hallucinations, usually within 2hr of ingestion. Mushrooms which cause symptoms within 6hr are unlikely to be seriously toxic. Delayed toxicity occurs with *Amanita phalloides* and some other species, which grow throughout the UK.

Amanita phalloides poisoning causes vomiting and profuse watery diarrhoea after a latent period of 6–12hr, followed by hepatic and renal failure. The interval between ingestion and the onset of symptoms is crucial in distinguishing between non-serious and potentially fatal poisoning.

Try to ascertain if:

- More than one variety of mushroom was eaten (since poisonous and edible mushrooms often grow together).
- Whether the mushrooms were cooked (since some toxins are inactivated by heat).
- Whether alcohol was taken (since disulfiram-like effects may occur with *Coprinus* species, ink cap mushrooms).

For most toxic mushrooms, only symptomatic treatment is required. Activated charcoal may ↓ absorption if given within 1hr. Get expert advice immediately if *Amanita* poisoning is suspected (see ➲ National Poisons Information Service, pp. 188–9).

Button batteries

Small children often swallow button or disc batteries intended for toys, watches, hearing aids, and other electrical equipment. Older patients sometimes mistake them for tablets.

Larger batteries may become stuck in the oesophagus, causing perforation or, later, stenosis. They can be mistaken for coins on X-ray.

Most batteries that reach the stomach pass through the gut without any problem. Corrosive damage could occur from electrical discharge, but toxicity from battery contents is rare. Mercury poisoning is very unlikely since mercuric oxide batteries are no longer sold. The NPIS may identify the type of battery involved from the reference number, if this is available on the packet or on a similar battery to that ingested.

Management

See advice on TOXBASE.

X-ray the chest and abdomen or use a metal detector to find the battery. A battery stuck in the oesophagus should be removed immediately by endoscopy, which allows inspection for oesophageal damage.

An asymptomatic child with a battery in the stomach can be sent home, with advice to return if any symptoms develop. If the battery has not been passed after 2 days, use a metal detector or repeat X-ray to look for the battery. If it is still in the stomach (which is rare), refer to consider removal by endoscopy to avoid any risk of perforation or absorption of battery contents. Note that ingestion of honey prior to removal may limit GI injury.

Batteries in the small or large bowel almost always pass spontaneously. Encourage stool inspection, but bear in mind that it may take up to 2 weeks to pass. If abdominal pain, vomiting, diarrhoea, or rectal bleeding occur, an abdominal X-ray is needed to localize the battery, which may require removal by endoscopy or surgery.

Batteries in the nose or ear

Button batteries lodged in the nose may cause corrosive burns and bleeding, sometimes with septal perforation after a few weeks. A battery in the ear may perforate the tympanic membrane and cause facial nerve injury. Liaise with a ear, nose, and throat (ENT) specialist to remove batteries as soon as possible.

Ingestion of magnets

Occasionally, patients (usually children) ingest magnets. When a single magnet is ingested on its own, it usually passes without incident. However, when more than one magnet has been ingested (or one magnet together with another ferrometallic object), there is a risk of bowel necrosis and perforation—refer for removal.

Novel psychoactive substances

Previously known as 'legal highs', new or novel pyschoactive substances (NPS) contain one or more chemical substances which produce similar effects to illegal drugs such as cocaine, cannabis, and ecstasy. In the UK, it is currently illegal to sell or distribute NPS, but possession is not a criminal offence.

NPS are often taken with other recreational drugs ± alcohol, which may potentiate their effects. Sometimes, NPS may be adulterated with other chemicals such as caffeine or lidocaine.

NPS comprise an unknown combination of chemicals, and therefore, treatment relies upon identification of the toxidrome and managing the symptoms.

The four groups of NPS are summarized below (most commonly encountered are stimulants and synthetic cannabinoids).

Stimulant NPS (eg mephedrone)

Synthetic cathinone compounds are sometimes known as 'plant food' or 'bath salts' which are snorted or swallowed. Toxic effects are similar to those of amphetamines: agitation, sweating, tachycardia, palpitations, and hypertension. Some have nausea, hallucinations, fits, muscle spasms, nausea, peripheral vasoconstriction, and myocardial ischaemia. Nasal irritation and epistaxis may occur after snorting these. Treat as for MDMA/amphetamines.

Cannabinoid NPS

Synthetic cannabinoid receptor agonists (SCRAs), such as 'spice' and 'noids', are often smoked or inhaled (eg using vaporizers). Suspect if there is a history of smoking a herbal product and the patient has features similar to those of cannabis intoxication: relaxation, altered consciousness, and disinhibition.

Some patients may become agitated and confused, with evidence of adrenergic stimulation (see TOXBASE section on SCRAs).

Depressant (sedative) NPS

Features include reduced conscious level, hypoventilation, and bradycardia. Treat as for benzodiazepine or opioid poisoning (see ➲ Benzodiazepine poisoning, p. 204; ➲ Opioid poisoning, p. 196).

Hallucinogenic NPS

These may have psychedelic effects (similar to LSD—see ➲ Recreational drugs, pp. 222–3), in which case treat as for tryptamine toxicity (see TOXBASE).

Some patients present with dissociative symptoms (similar to ketamine—see ➲ Ketamine, p. 287), in which case treat as for dissociative drugs, as per TOXBASE.

A general rule for any patient who has taken an unknown recreational drug is that if there are no symptoms present at 4hr and no treatment has been required, then they are safe to be discharged with advice regarding returning for review if symptoms develop.

Recreational drugs

Toxicity is often seen from heroin (see ➲ Opioid poisoning, p. 196), cocaine, ecstasy, and related drugs. Street names for drugs vary and may be confusing. TOXBASE (see ➲ National Poisons Information Service, pp. 188–9) has lists of slang names about drugs.

Illicit drugs vary in strength and are often mixed with other drugs or chemicals, which may cause unexpected effects. Drugs may be smoked, sniffed ('snorted'), swallowed, or injected. Injecting drug users are at ↑ risk of hepatitis (see ➲ Hepatitis, p. 249), HIV (see ➲ Human immunodeficiency virus, pp. 250–1), necrotizing fasciitis (see ➲ Necrotizing fasciitis, p. 244), botulism (see ➲ Botulism, p. 247), anthrax (see ➲ Anthrax, p. 243), and endocarditis (see ➲ Infective endocarditis, p. 244).

Ecstasy (MDMA)

'Ecstasy' (3,4-methylenedioxymetamphetamine, MDMA) is an amphetamine derivative used as an illegal stimulant drug. The name 'ecstasy' is also used for benzylpiperazine (BZP), another illegal drug. 'Liquid ecstasy' is GHB (see ➲ Gammahydroxybutyrate (GHB, GBH, 'liquid ecstasy'), p. 223). MDMA is taken PO as tablets or powder, often at raves or parties. Some people who have previously tolerated the drug have idiosyncratic reactions, with severe toxicity from a single MDMA tablet.

MDMA causes release of serotonin, catecholamines, and other hormones. Inappropriate ADH secretion, abnormal thirst, and excessive water intake may result in hyponatraemia and cerebral oedema, especially in women.

Clinical features

Euphoria, agitation, sweating, dilated pupils, ataxia, teeth grinding, headache, tachycardia, and hypertension. Severe poisoning can cause hyperpyrexia, muscle rigidity, rhabdomyolysis, convulsions, coma, cardiac arrhythmias, renal failure, hepatic failure, cerebral haemorrhage, and DIC. Metabolic acidosis is common. Features of serotonin syndrome may occur, as may hypoglycaemia, severe hyponatraemia, and hyperkalaemia.

Treatment

Consider activated charcoal (see ➲ Activated charcoal, p. 192) if <1hr since ingestion. Observe asymptomatic patients for at least 4hr. Monitor ECG, pulse, BP, and T°. Record ECG, and check U&E, creatinine, glucose, LFTs, and CK. Test urine for blood. In severe cases, check ABG and coagulation.

Support ABC. Get expert advice (see ➲ National Poisons Information Service, pp. 188–9) and ICU help in severe poisoning. RSI may be needed because of trismus and fits—avoid suxamethonium which may cause hyperkalaemia. Control agitation with PO or IV diazepam or lorazepam—large doses may be needed. For severe hypertension, give IV diazepam and GTN. Do not treat single short fits, but give diazepam or lorazepam for repeated or prolonged fits.

Correct metabolic acidosis (possibly with sodium bicarbonate), checking ABG and U&E. Treat hyperkalaemia (see ➲ Hyperkalaemia, pp. 170–1). Treat mild hyponatraemia by fluid restriction. IV saline may be needed for severe hyponatraemia—rapid correction of chronic hyponatraemia can cause brain injury (central pontine myelinolysis), but this is less likely with acute hyponatraemia caused by MDMA. Cool as for heat stroke (see ➲ Heat illness, pp. 274–5) if hyperpyrexial. If rectal T° is >40°C, consider dantrolene 1–2.5mg/kg IV (up to 10mg/kg in 24hr). (See also ➲ Serotonin syndrome, p. 224.)

Amphetamine (amfetamine)

Can be swallowed, snorted, smoked, or injected. Body packers may suffer severe poisoning. Toxic features are euphoria, agitation (excited or agitated delirium), psychosis, sweating, dilated pupils, tachycardia, hypertension, vomiting, abdominal pain, fits, hyperpyrexia, and metabolic acidosis. Severe poisoning may cause stroke, MI, rhabdomyolysis, renal failure, and DIC. Cardiac arrest can occur in violent agitated patients who need physical restraint. Treat amphetamine poisoning as for MDMA (see ➲ Ecstasy (MDMA), p. 222).

Cocaine

Cocaine base ('crack') is usually smoked. Cocaine salt ('coke') is snorted, eaten, or injected. Toxic effects (due to catecholamines, serotonin, and amino acid stimulation and Na^+ channel blockade) are euphoria, agitation, delirium, ataxia, dilated pupils, sweating, vomiting, fits, tachycardia, arrhythmias, and hypertension. Chest pain may be due to myocardial ischaemia or MI (from ↑ catecholamines, ↑ O_2 demand, coronary vasospasm, and thrombosis), aortic dissection, or pneumothorax. Cerebral haemorrhage, hyperpyrexia, rhabdomyolysis, renal failure, gut ischaemia, and serotonin syndrome may occur. Cocaine is an LA, so hot air from smoking crack can cause airway burns.

Treatment

Treat as for MDMA (see ➲ Ecstasy (MDMA), p. 222). Give diazepam for agitation (5–10mg IV, repeated every 5min if needed, up to 100mg). Treat chest pain with diazepam, GTN, O_2, and aspirin. GTN and phentolamine may ↓ BP and ↑ coronary blood flow. Avoid β-blockers (may cause paradoxical hypertension and ↑ coronary vasoconstriction), except labetalol which may be an option (antagonizes both α- and β-adrenoceptors). If ECG suggests acute MI, consider angioplasty or thrombolysis.

Gammahydroxybutyrate (GHB, GBH, 'liquid ecstasy')

GHB is used illegally as a body-building agent and psychedelic drug. It is ingested or injected. Intoxication may cause vomiting, diarrhoea, drowsiness, confusion, ataxia, and agitation. Severe poisoning results in coma, respiratory depression, fits, bradycardia, and hypotension. Consider activated charcoal (see ➲ Activated charcoal, p. 192) if <1hr since ingestion. Observe for at least 4hr and monitor pulse rate, BP, and breathing. Provide supportive treatment as needed. Control agitation and convulsions with diazepam.

LSD (lysergic acid diethylamide)

Causes visual hallucinations, agitation, excitement, tachycardia, and dilated pupils. Hypertension and pyrexia may occur. Paranoid delusions may require sedation. Massive overdose of LSD is rare but may cause coma, respiratory arrest, and coagulation disturbances. Treat supportively.

Amyl nitrite

This volatile liquid is often inhaled recreationally. As a vasodilator, it can cause headache and hypotension. Oxidation of iron in Hb to the ferric form causes methaemoglobinaemia, causing ↓ O_2-carrying capacity. SpO_2 may be normal. Give high-flow O_2 and measure methaemoglobin level. Treat as per TOXBASE—consider methylene blue (methylthioninium chloride) 1–2mg/kg over 5min, which can be repeated after 30–60min.

Serotonin syndrome

Background

The clinical picture of serotonin syndrome is increasingly recognized amongst those taking SSRIs. The syndrome can occur in patients who have taken therapeutic doses of SSRIs, and this is especially likely if they have recently started on the medication or if it is taken in combination with other drugs which ↑ production, availability, or release of serotonin (eg cocaine, MDMA, amphetamines) or reduce metabolism (eg MAOIs). Serotonin syndrome can also occur after an acute overdose. Numerous drugs have been implicated—in addition to those mentioned previously, they include: tricyclic antidepressants, venlafaxine, tramadol, pethidine, buprenorphine, St John's wort, olanzapine, and lithium.

Clinical features

Altered mental status

Confusion, hallucinations, and agitation may occur, with drowsiness and reduced conscious level in severe cases.

Neuromuscular features

Rigidity, shivering/tremor, teeth grinding, ataxia, and hyper-reflexia (especially affecting the lower limbs) may occur.

Autonomic effects

These include tachycardia, hypertension (or hypotension), flushing, diarrhoea, vomiting, and hyperthermia.

Severe cases can result in fits, rhabdomyolysis, renal failure, and coagulopathy.

Differential diagnosis

This includes neuroleptic malignant syndrome, malignant hyperthermia, severe infection (eg encephalitis), and other direct effects of drug overdose or withdrawal.

Investigations

Check U&E, glucose, LFTs, CK, FBC, urinalysis, ABG/VBG, and ECG.

Treatment

Provide supportive measures and obtain expert advice (TOXBASE or call NPIS—see ➲ National Poisons Information Service, pp. 188–9). Agitation, hyperthermia, myoclonic jerking, and fits may benefit from diazepam therapy. Treat rhabdomyolysis with IV fluids and urine alkalinization.

Cyproheptadine (12mg PO, then 4–8mg 6-hourly) is a serotonin receptor antagonist. Although data are lacking, there is a good theoretical basis for its use in serotonin syndrome. Consider chlorpromazine (12.5–25mg IV) in severe cases.

Body packers

Body packers try to smuggle drugs such as cocaine or heroin by ingesting multiple packages of drugs wrapped in condoms, latex, foil, or even fibreglass. Packages may also be hidden in the rectum or vagina (individuals who do this are sometimes referred to as '*body pushers*'). Serious or even fatal poisoning may occur if any packages leak and the drugs are absorbed.

Patients are often in police custody—take care to ensure that correct procedures are undertaken (see https://www.rcem.ac.uk).

In England and Wales, imaging and intimate examination to search for class A drugs requires the patient's written consent and an authorization from a police inspector. In Scotland, the police will seek a Sheriff's warrant.

Rectal and/or vaginal examination should ideally be performed by a forensic physician (police surgeon). Limit the number of individuals handling any retrieved packages to the police or forensic physician. If the patient lacks capacity, consider whether radiological investigation can be undertaken in their best interests (get senior advice).

Management

Advice is available from NPIS (see ➲ National Poisons Information Service, p. 190). Ensure that suspected body packers receive careful assessment and observation. Features of toxicity depend upon the concealed drug and any accompanying adulterants (eg strychnine). Consider the need for intimate examination (as outlined previously).

Try to establish the drug involved, the number of packages, and the type of packaging used. Check basic signs. Urine toxicology screening is not widely available but may detect heroin or cocaine and provide an indication of those occasions when leakage has occurred.

Abdominal X-ray is sometimes used as a screening tool (97% specificity) but is less sensitive than CT. Gastric packages can sometimes be seen on an erect CXR. USS or MRI may play a role in pregnant patients.

Isotonic laxatives and whole-bowel irrigation may aid expulsion of packages. Consider a naloxone infusion (see ➲ Opioid poisoning, p. 196) for symptomatic heroin body packers. In symptomatic patients, refer early as surgery may be required—this is particularly urgent if cocaine or amphetamines are implicated. Endoscopy may help to remove small gastric packages, but there is a risk of damaging packaging and ↑ drug leakage.

Body stuffers

This term is sometimes applied to individuals who swallow drugs (or hide them rectally or vaginally as body pushers) immediately prior to being apprehended by the police. The quantity of drugs ingested in this way may be less than that by body packers. However, any packaging is likely to be much less robust than that used by body packers, thereby ↑ the risk of the packages leaking. Consider activated charcoal and admission for 6–8hr (up to 24hr if suspicion is high).

Parachuting

Recreational drugs are ingested in delicate packaging, with the aim of delaying absorption. Treat as body stuffers (see ➲ Body stuffers, p. 225).

Chapter 5

Infectious diseases

Incubation periods

Incubation period usually <1 week

Staphylococcal enteritis	1–6hr
Salmonella enteritis	6–48hr (usually 12–24hr)
Bacillary dysentery (*Shigella*)	1–7 days (usually 1–3 days)
Botulism	12–96hr (usually 18–36hr)
Cholera	12hr to 6 days (usually 1–3 days)
COVID-19	7–14 days (median 5 days)
Dengue	4–7 days
Diphtheria	2–5 days
Gas gangrene	6hr to 4 days
Legionnaires' disease	2–10 days (usually 7 days)
Meningococcaemia	1–7 days (usually 3 days)
Scarlet fever	1–4 days
Yellow fever	3–6 days

Incubation period usually 1–3 weeks

Brucellosis	7–21 days (occasionally some months)
Chickenpox	10–20 days (usually about 14 days)
Lassa fever	6–21 days
Leptospirosis	2–26 days (usually 7–12 days)
Malaria (falciparum)	7–14 days (occasionally longer)
Malaria (vivax, malariae, ovale)	12–40 days (occasionally >1y)
Measles	10–18 days (rash usually 14–18 days)
Mumps	14–18 days
Pertussis (whooping cough)	5–14 days (usually 7–10 days)
Rubella	14–21 days
Tetanus	1 day to 3 months (usually 4–14 days)
Typhoid	3–60 days (usually 7–14 days)
Typhus	7–14 days

Incubation period usually >3 weeks

Amoebiasis	2 weeks to many months
Hepatitis A	2–6 weeks (usually 4 weeks)
Hepatitis B and C	6 weeks to 6 months
HIV	2 weeks to 3 months (anti-HIV antibody appears)
Infectious mononucleosis	4–7 weeks
Rabies	4 days to 2y (usually 3–12 weeks)
Syphilis	10 days to 10 weeks (usually 3 weeks)

Duration of infectivity of infectious diseases

Chickenpox	3 days before rash until last vesicle crusts
Hepatitis A	2 weeks before until 1 week after jaundice starts
Measles	4 days before rash until 5 days after rash appears
Mumps	3 days before to 1 week after salivary swelling
Pertussis	3 days before to 3 weeks after start of symptoms (5 days if on appropriate antibiotic)
Rubella	1 week before to 1 week after onset of rash
Scarlet fever	10–21 days from onset of rash (1 day if on penicillin)

Notifiable infectious diseases

In the UK, certain infectious diseases are 'notifiable'. A doctor who knows or suspects that a patient has one of these diseases is obliged to notify the local health protection department. Use the special notification form, if available. Telephone the consultant in communicable disease control if investigation or control of an outbreak may be needed (see https://www.gov.uk).

Notifiable infectious diseases list

The list of notifiable diseases from Public Health England is as follows:

- Acute encephalitis, acute infectious hepatitis, acute meningitis, acute poliomyelitis, anthrax, botulism, brucellosis, cholera, COVID-19, diphtheria, enteric fever (typhoid or paratyphoid fever), food poisoning, haemolytic uraemic syndrome, infectious bloody diarrhoea, invasive group A streptococcal disease, legionnaires' disease, leprosy, malaria, measles, meningococcal septicaemia, mumps, plague, rabies, rubella, SARS, scarlet fever, smallpox, tetanus, TB, typhus, viral haemorrhagic fever (VHF), whooping cough, yellow fever.

Note that there is a separate list of notifiable organisms (causative agents) which laboratories report—there is some inevitable overlap between the two lists.

Childhood infectious diseases

Children at risk

Unimmunized children are at risk of infections which would be prevented by the standard immunization schedule (see ➲ Standard immunization schedule, pp. 652–3). Always ask about vaccination status in any febrile, unwell child. The common infectious diseases of childhood can be very serious in children with *immune deficiency* or those on *immunosuppressant drugs*. Refer such children for specialist advice if they develop an infectious disease or have been in contact with one. Children with cystic fibrosis can become very ill with measles, whooping cough, or chickenpox—refer these also. Neonates rarely develop the common exanthems of childhood, but refer if these present to the ED. Chickenpox can be particularly serious in this age group.

Measles[ND]

A viral infection spread by airborne droplets. It is very contagious.

Incubation period Is 10–18 days. Infectious from just before the onset of symptoms until 5 days after the rash appears.

Initial features (lasting ~3 days) Fever, malaise, coryza, conjunctivitis, and cough. Koplik's spots (small white spots like grains of salt) appear inside the cheeks. One to 2 days later, a red maculopapular rash starts behind the ears and spreads to the face and down the body and onto the limbs.

Treatment Is symptomatic unless there are complications (eg otitis media or bacterial pneumonia). Febrile convulsions may occur. Encephalitis is relatively rare (~0.1%) but can be fatal. Hospital admission is rarely needed, unless the child is very ill or has pre-existing disease. In the tropics, many malnourished children die from measles, but in the UK, mortality is very low.

Mumps[ND]

Mumps is a virus infection spread by saliva and respiratory droplets. Infectivity is greatest at the onset of symptoms, but many subclinical cases also spread infection. There has been a large increase in cases in the UK amongst young adults (especially those at college/university), including many who did receive all childhood immunizations.

Incubation period 14–18 days.

Typical features Fever with pain and swelling in one or both parotid glands. The submandibular glands may sometimes be affected. Aseptic meningitis may occur. Orchitis affects 10–15% of post-pubertal ♂ but rarely causes sterility. The pain of orchitis may be relieved by analgesia and a short course of steroids. Orchitis is uncommon before puberty, so consider torsion of the testis if a child presents with testicular pain and swelling (see ➲ Inguinal and scrotal swellings, pp. 722–3).

Rubella (German measles)[ND]

Rubella is usually a mild disease, but infection during pregnancy may cause severe congenital disorders, particularly eye defects, heart defects, and deafness. Guidance on the management of, and exposure to, rubella during pregnancy is available from the Health Protection England/Wales/Scotland websites (https://www.gov.uk). The virus is spread mainly by the airborne route, with an incubation period of 2–3 weeks and infectivity from 1 week before symptoms until 1 week after the rash appears. A macular rash occurs on the face and trunk, with mild fever, occipital lymphadenopathy, and sometimes transient arthralgia. Rare complications are encephalitis and thrombocytopenia.

Treatment Generally symptomatic. The clinical diagnosis of rubella is unreliable—similar rashes may occur with enterovirus and parvovirus infections. If there is concern about rubella infection in pregnancy, take blood for viral antibody levels and arrange urgent follow-up by the GP or obstetrician.

Whooping cough[ND]

See Whooping cough[ND], p. 700.

Infectious mononucleosis (glandular fever)

Infection with *Epstein–Barr virus* is common in children and young adults and is spread by saliva or droplets. Infection often occurs without clinical disease. In glandular fever, there is malaise, fever, a sore throat, and cervical lymphadenopathy. The throat may be very red, and in 25% of cases, there is also infection with β-haemolytic *Streptococcus*. In severe cases, there is marked oedema of the throat, with tonsillar swelling and a membranous exudate ('anginose' infectious mononucleosis), with difficulty in swallowing and breathing. A rash is uncommon, unless ampicillin or amoxicillin are given, causing a widespread erythematous maculopapular rash (which does not signify allergy to penicillins in general).

Complications Include respiratory obstruction, ruptured spleen (spontaneously or after minor trauma, so advise avoid contact sports for 1 month), thrombocytopenia, jaundice, meningitis, encephalitis, facial palsy, and acute polyneuritis (occasionally causing respiratory failure).

Investigations FBC and blood film (for atypical lymphocytes), Monospot test or Paul–Bunnell test (which may be –ve initially).

Differential diagnosis Includes CMV and toxoplasmosis.

Treatment Unnecessary in most patients. Severe or complicated cases need specialist assessment and follow-up. In anginose infectious mononucleosis, a short course of high-dose oral steroids gives rapid relief of symptoms (prednisolone 80mg on day 1; 15mg tds on days 2–3; 10mg tds on days 4–5; 5mg tds on days 6–7). Steroids are also helpful in patients with neurological complications. Concurrent β-haemolytic streptococcal infection requires erythromycin (500mg qds), which would also treat the rare unrecognized case of diphtheria.

Meningitis

Causative organisms

Meningitis may be *bacterial, viral*, or occasionally *fungal*. Bacterial causes of meningitis include meningococci, pneumococci, *Haemophilus influenzae, Listeria*, and TB. Other bacteria may also cause meningitis in neonates, the elderly, and immunosuppressed patients.

Clinical features of bacterial meningitis

Some patients have classic features of headache, neck stiffness, photophobia, fever, and drowsiness. However, the clinical diagnosis of meningitis may be very difficult in early cases. Neonates may present with anorexia, apnoea, or fits. Meningitis may start as a 'flu-like' illness, especially in the immunosuppressed or elderly. Consider meningitis in any febrile patient with headache, neurological signs, neck stiffness, or ↓ conscious level.

Meningococcal meningitis[ND] Caused by *Neisseria meningitidis*. It can result in septicaemia, coma, and death within a few hours of the first symptoms. Skin rashes occur in 50% of patients, often starting as a maculopapular rash before the characteristic petechial rash develops. There may be DIC and adrenal haemorrhage (Waterhouse–Friderichsen syndrome). Meningococcal septicaemia (see ➲ Meningococcal disease, pp. 682–3) may occur without meningitis.

Management

Resuscitate, giving O_2 as required, and obtain venous access.

Start antibiotics *immediately* (without waiting for investigations) if the patient is shocked or deteriorating or there is any suspicion of meningococcal infection (especially a petechial or purpuric rash)—give IV ceftriaxone (adult 2g; child 80mg/kg) or cefotaxime. Chloramphenicol is an alternative if there is a history of anaphylaxis to cephalosporins (see *BNF*). In adults >55y, add ampicillin 2g qds to cover *Listeria.* Give vancomycin ± rifampicin if penicillin-resistant pneumococcal infection is suspected. Give IV dexamethasone (0.15mg/kg, max 10mg, qds for 4 days), starting with or just before the first dose of antibiotics, especially if pneumococcal meningitis is suspected.

Initial investigations FBC, U&E, glucose, clotting screen, VBG, CRP, blood cultures, EDTA sample for polymerase chain reaction (PCR), and clotted blood for serology. LP is needed if meningitis is suspected, unless there is a coagulopathy or ↑ ICP—do a CT scan if there is suspicion of ↑ ICP (confusion/coma, hypertension, bradycardia, or papilloedema) or focal neurological signs.

Provide supportive treatment, including

- IV fluids.
- Pressure area care.
- Monitor conscious level, T°, BP, ECG, SpO_2, and fluid balance.

Get expert help promptly and organize ICU care.

For latest advice, see https://www.meningitis.org and https://www.nice.org.uk.

For meningitis and LP in children, see ➲ Meningococcal disease, pp. 682–3.

Prophylaxis of meningococcal infection

Whilst intubating a patient with suspected meningococcal infection, wear a suitable mask (eg FFP3) and a face shield to reduce the risk of infection.

Meningococcal infection is spread by droplets from the nose of an infected carrier, who may be well. Notify the consultant in communicable disease control (see ➲ Notifiable infectious diseases, p. 229) immediately about any suspected meningococcal infection and obtain advice about antibiotic prophylaxis. Prophylactic antibiotics (rifampicin, ciprofloxacin, or ceftriaxone) are needed for the patient's family and close contacts. Hospital and ambulance staff do not need prophylaxis unless they have given mouth-to-mouth ventilation or intubated the patient without using protective equipment.

Rifampicin is given 12-hourly for 2 days (5mg/kg for a child aged <1y; 10mg/kg at 1–12y; 600mg at age >12y). It makes the urine orange or brown, discolours soft contact lenses, and ↓ effectiveness of OCP for ~4 weeks (see *BNF*)—give appropriate warnings and record this in the notes.

Ciprofloxacin is given as a single PO dose of 500mg (adults), 250mg (child 5–12y), or 125mg (child 2–5y), although it is not licensed for chemoprophylaxis of meningitis.

Ceftriaxone is given as a single IM dose of 250mg (adults and children >12y) or 125mg (children <12y).

Warn contacts of meningococcal patients to report to a doctor at once if they develop symptoms.

TB meningitis

Often gradual onset, with malaise, anorexia, vomiting, headache, and eventually signs of meningitis. Cranial nerve palsies, spastic paraplegia, and coma can occur. Meningitis may be part of miliary TB (see ➲ Tuberculosis[ND], p. 242), which may be apparent on CXR. Ophthalmoscopy may show choroidal tubercles and papilloedema, which is found more commonly than in other forms of meningitis. Refer for specialist investigation and treatment.

Viral meningitis

Viral causes of meningitis include Coxsackie virus, mumps, and echoviruses. Viral meningitis produces similar clinical features to those of bacterial infection, but the illness is often less severe. Initial management is the same as for suspected bacterial meningitis. Refer for admission and investigation.

See also ➲ Acute encephalitis, p. 234.

Fungal meningitis

Fungal meningitis is usually part of disseminated infection in immunosuppressed patients (eg those with AIDS—see ➲ Human immunodeficiency virus, pp. 250–1), or lymphoma, or on steroid therapy). *Cryptococcus neoformans* is the most common organism. Symptoms usually develop slowly, as with TB meningitis. There may be papilloedema and focal neurological signs. Admit for specialist investigation and treatment.

Acute encephalitis[ND]

Causative organisms

Acute encephalitis is most commonly attributed to herpes simplex virus in the UK (hence, early treatment with aciclovir is a high priority). Other viral causes include CMV, EBV, herpes varicella-zoster virus, measles, mumps, HIV, and rabies. Non-viral causes include TB and malaria, in addition to the wide range of causes of bacterial meningitis.

Presentation

The diagnosis of acute encephalitis is not always an easy one to make. There may not always be a history of headache and fever. Patients may present with odd or bizarre behaviour ± confusion. Sometimes the presentation is acute with collapse, seizures, or ↓ conscious level. Keep the diagnosis in mind when assessing patients with unexplained neurological symptoms and/or focal neurological signs.

Investigation

- Take blood for FBC, ESR, CRP, U&E, clotting, blood glucose, viral PCR, and cultures.
- If there is a relevant history of foreign travel, send thin and thick films for malaria (see Malaria[ND], p. 255).
- Contrast-enhanced CT may reveal changes suggestive of herpes simplex encephalitis. MRI is an alternative, if available.
- LP may yield CSF which has ↑ lymphocytes. Send a sample for viral PCR.

Management

- Involve ICU and resuscitate with O_2 and IV fluids as required.
- Start IVI aciclovir (10mg/kg IVI over 1hr).
- Consider treating with IV antibiotics (eg IV ceftriaxone 2g) for bacterial meningitis (see Meningitis, pp. 232–3).

Herpes simplex virus

Primary herpes simplex infection causes painful vesicles and ulceration of the mouth or genitalia (see Sexually transmitted diseases, p. 234). The virus may be inoculated into skin by trauma (herpes gladiatorum, scrumpox) or by contamination of fingers causing herpetic paronychia (whitlow). Infection of the cornea may cause dendritic ulcers (see Ulcerative keratitis, p. 559).

Herpes simplex meningitis and *encephalitis*[ND] are uncommon but may be fatal, especially in immunodeficient patients.

Herpes simplex virus persists in sensory ganglia and may be reactivated by stimuli such as sun, cold, trauma, or viral infections. Recurrence of cold sores of the lips is often preceded by tingling—aciclovir cream or tablets may prevent the development of vesicles. Secondary bacterial infection may require antibiotics. Do not incise a suspected whitlow. Cover it with a dressing and advise care to avoid spreading infection to the lips or eyes.

Herpes varicella-zoster virus

Chickenpox results from primary infection with varicella-zoster virus, which then remains dormant in the dorsal root ganglia. Reactivation of the virus causes *shingles*. Chickenpox is usually a mild disease of childhood. An itchy vesicular rash appears, most densely on the trunk and face, but ↓ peripherally. The lesions appear in crops and crust over in 3–4 days. Fever, malaise, and muscle aches may occur in adults. Infectivity starts 3 days before the rash appears and continues until the last lesion has crusted.

Treat symptomatically, eg calamine lotion for itching and paracetamol for fever. Avoid NSAID use in chickenpox—this is associated with ↑ risk of skin and soft tissue infections. Occasionally, antibiotics are needed for secondary bacterial skin infection (usually *Staphylococcus* or *Streptococcus*). Pneumonia is rare and in children is usually staphylococcal, but in adults it may be caused by chickenpox virus. Chickenpox may be severe in neonates and in those with cystic fibrosis or immune deficiency—refer for specialist assessment and treatment with aciclovir and/or varicella-zoster immune globulin. Consider aciclovir also for adults and older adolescents (see *BNF*).

Shingles often occurs in the elderly and may affect any dermatome, most often thoracic. The pain of shingles may cause diagnostic difficulty until the rash appears, usually after 1–4 days. Erythema is followed by vesicles and then crusting of lesions in a unilateral distribution over one dermatome or two adjacent dermatomes. Ophthalmic shingles may affect the eye via the long ciliary nerves—skin lesions on the side of the tip of the nose imply a high risk of eye involvement (Hutchinson's sign). Oral lesions occur in maxillary and mandibular shingles. Infection of the geniculate ganglion causes a facial palsy, with lesions in the pinna of the ear and on the side of the tongue and hard palate (*Ramsay–Hunt syndrome*). In severe shingles, there may be weakness of muscles supplied by nerves of the same spinal root.

Antiviral treatment (aciclovir, famciclovir, or valaciclovir) ↓ the risk of postherpetic pain if given early (within 72hr of start of rash). *Dose*: aciclovir 800mg five times daily for 7 days. In renal failure, antiviral drugs may cause severe toxicity, so use much smaller or less frequent doses. Patients with immune deficiency or ophthalmic zoster need immediate specialist referral and antiviral treatment. Give analgesia. Antibiotics may be required for secondary infection.

Zika virus

Zika virus infection is mainly transmitted to humans by mosquitos in the Caribbean and South America, although human-to-human transmission can occur. It causes a usually mild illness, which may be subclinical or cause fever, rash, headache, myalgia, and conjunctivitis lasting up to a week. The incubation period is 3–14 days. The principal concern regarding zika virus infection is that infection in pregnancy produces fetal abnormalities (especially microcephaly) in a significant number of pregnant women.

Gastroenteritis/food poisoning[ND]

Diarrhoea

This is the usual presenting symptom of gastroenteritis, but it is also a feature of many other conditions as diverse as otitis media, appendicitis, and ulcerative colitis. Antibiotics often cause diarrhoea. Constipation may present as diarrhoea if there is overflow around an obstructing stool. A rectal tumour may present similarly.

Diarrhoea and vomiting May be caused by many types of bacteria and viruses (eg norovirus), and also by some toxins and poisons. Many episodes of gastroenteritis result from contaminated food, usually meat, milk, or egg products, which have been cooked inadequately or left in warm conditions. The specific cause is often not identified. Some infections are spread by faecal contamination of water (eg cryptosporidiosis from sheep faeces). Rotavirus infection (common in children) may be transmitted by the respiratory route. Severe illness with bloody diarrhoea, haemolysis, and renal failure may result from infection with verocytotoxin producing *Escherichia coli* (VTEC O157).

Food poisoning Is a notifiable disease (see ➲ Notifiable infectious diseases, p. 229). Immediate notification by telephone is mandatory if an outbreak is suspected. The food eaten, symptoms, and incubation period may suggest the organism or toxin involved (see Table 5.1). CO poisoning (see ➲ Carbon monoxide poisoning, p. 216) may cause malaise and vomiting in several members of a family and be misdiagnosed as food poisoning.

Ensure that patients who present with diarrhoea are flagged up as posing a potential risk of infection, so that they can be isolated appropriately to protect other patients from cross-infection.

History

Record the duration of symptoms and the frequency and description of stools and vomit, including the presence of blood. Document other symptoms (eg abdominal pain, fever), food and fluid ingested, and any drugs taken. Enquire about affected contacts, foreign travel, and occupation (especially relevant if a food handler).

Examination

Check vital signs, including pulse rate, RR, BP, SpO_2, and T°. Examine for abdominal tenderness and other signs of infection. Record the patient's weight and compare this with any previous records. Assess the *degree of dehydration*—this is especially important in children and is traditionally classified as mild (<5%), moderate (5–10%), or severe (>10%) (see also ➲ Gastroenteritis in children, pp. 718–19).

Evidence of severe dehydration includes: weakness, confusion, shock, and ↓ urine output.

Table 5.1 Food poisoning characteristics

Cause	Incubation	Food	Symptoms*
Staphylococcus aureus	1–6hr	Meat, milk	D, V, P, shock
Bacillus cereus	1–16hr	Rice	D, V, P
Salmonella	6–48hr	Meat, eggs	D, V, P
Escherichia coli	1–2 days	Any food	D, V, P
E.coli VTEC O157	1–2 days	Meat, milk	D, V, P
Campylobacter	1–3 days	Meat, milk	Fever, P, D
Shigella	1–3 days	Any food	Bloody D, V, fever
Vibrio parahaem	2–3 days	Seafood	Watery D
Cholera	12hr to 6 days	Water, seafood	D (watery), shock
Rotavirus	1–7 days		D, V, fever, cough
Botulism	12–96hr	Preserved food	V, paralysis
Histamine fish poisoning (scombrotoxin)	<1hr	Fish	Flushing, headache, D, V, P (see ➲ Fish poisoning, p. 239)
Ciguatera fish poisoning	1–6hr (rarely 30hr)	Fish from tropical coral reef	D, V, P, paraesthesiae, muscle weakness (see ➲ Fish poisoning, p. 239)
Paralytic shellfish poisoning	30min to 10hr	Shellfish	Dizziness, paraesthesiae, weakness, respiratory failure (see ➲ Fish poisoning, p. 239)
Chemicals	<2hr	Food, water	Various
Mushrooms	<24hr	Mushrooms	D, V, P, hallucinations (see ➲ Plants, berries, and mushrooms, p. 219)

* D, diarrhoea; V, vomiting; P, abdominal pain.

Investigations

Consider sending blood tests (including U&E) if the patient is shocked or unwell.

Stool culture is unnecessary in most cases of gastroenteritis, but obtain if the patient is systemically unwell, has blood or pus in the stool, is immunocompromised, has recently been hospitalized and/or has had antibiotics or PPI, has been abroad or has prolonged symptoms, is a resident in a care home, or works as a food handler, or the diagnosis is uncertain.

Managing gastroenteritis/food poisoning[ND]

Treatment

(See also ➲ Gastroenteritis in children, pp. 718–19.)

- Isolate and take precautions to prevent spread of infection to staff or other patients (see ➲ Infection control and prevention, pp. 36–7).
- Rehydrate aggressively with IV fluids those patients who are shocked and/or severely dehydrated, then reassess and refer for admission. Admit patients who have persistent vomiting and cannot keep down oral fluids. Most illnesses are self-limiting and do not require hospital admission, but remember that some patients may need special attention and/or cannot manage to cope at home (eg significant comorbidity, frail elderly living alone). Other features which may influence the decision to admit include: fever, bloody diarrhoea, abdominal pain, recent foreign travel, diarrhoea for >10 days, drugs that may exacerbate renal impairment, and dehydration.
- Aim to discharge less severe cases with oral fluids (± soup and fruit juice) and advice. Consider supplementing diet with oral rehydration therapy (eg Dioralyte®) in patients >60y or with comorbidities. Warn that home-made salt and sugar mixtures may be dangerously inaccurate versions of the professional oral rehydration products on offer. Advise patients to recommence normal diet after symptoms settle. Most patients can return to work 48hr after the first normal stool (advise food handlers to contact their employer and/or public health authorities).

Antiemetic drugs are sometimes helpful in gastroenteritis. In adults, an antiemetic (eg metoclopramide 10mg IM or prochlorperazine 12.5mg IM or 3mg buccal) may help, but do not use in children as it often causes troublesome side effects.

Anti-diarrhoeal drugs (eg loperamide) are contraindicated in children and rarely needed in adults—they may aggravate nausea and vomiting and occasionally cause ileus. However, they may provide symptomatic control in mild to moderate diarrhoea, but avoid if there is bloody diarrhoea and/or possible *Shigella* infection.

Antibiotics are only needed in special circumstances. Most episodes of gastroenteritis are brief, and many are caused by viruses and not helped by antibiotics. Patients with amoebiasis, giardiasis, and *Campylobacter* or *Shigella* infections may need antibiotics—refer to a specialist and/or an infectious diseases unit for treatment and follow-up.

Antibiotics are occasionally useful in traveller's diarrhoea before a long journey or an important meeting (ciprofloxacin 500mg bd PO for 3 days—see the *BNF* or data sheet about side effects and warnings).

Fish poisoning

Histamine fish poisoning

Also known as scombroid fish poisoning or scombrotoxin poisoning, this is caused by ingesting toxins in fish such as tuna, mackerel, and other dark-meat fish, which have been stored improperly. If the fish is not cooled rapidly after it is caught, an enzyme in bacteria converts histidine into histamine and other toxins, which are heat-stable and so are unaffected by cooking. The patient may notice that the fish tastes metallic, bitter, or peppery and the flesh looks honeycombed. Symptoms start within a few minutes to 2hr, with flushing of the face and upper body, headache, nausea, vomiting, abdominal pain, diarrhoea, dizziness, and palpitations. Urticaria and bronchospasm are less common. Symptoms usually settle within 6hr without treatment but resolve more quickly with antihistamines (eg chlorphenamine 10mg IV in adults, 250mcg/kg in children). In severe cases, cimetidine and, rarely, adrenaline might be needed, with O_2, IV fluids, and bronchodilators.

Tell the patient that histamine fish poisoning is caused by improper fish handling and storage. It is not an allergic reaction, and so the patient would not have to avoid eating fish in future.

Ciguatera fish poisoning

This is caused by a neurotoxin called ciguatoxin which is produced by a dinoflagellate (a unicellular plankton) associated with coral reefs. Fish imported from the tropics may cause ciguatera poisoning in the UK and elsewhere. Symptoms usually start 1–6hr after ingestion with nausea, vomiting, watery diarrhoea, and abdominal pain, followed by neurological symptoms, including paraesthesiae of the lips, tongue, and feet, ataxia, and muscle weakness. A classic feature is paradoxical temperature reversal (cold objects feel hot and hot objects feel cold). Alcohol makes these symptoms worse. Bradycardia and hypotension may occur. Treatment is symptomatic and supportive. GI symptoms usually settle within a day, but paraesthesiae may persist for weeks or months.

Paralytic shellfish poisoning

This can be caused by eating molluscs such as mussels, clams, cockles, and scallops which concentrate a neurotoxin called saxitoxin produced by dinoflagellate plankton. This plankton proliferates when sea temperatures rise in summer and may make the sea look red ('red tide'). Symptoms start 30min to 10hr after ingestion with dizziness, ataxia, paraesthesiae, and muscle weakness, which may progress to respiratory failure. Treatment is supportive, with assisted ventilation if necessary. Complete recovery is usual within 24hr.

Infestations

Worms

The most common helminthic infection seen in the UK is the threadworm *Enterobius vermicularis*. This causes anal itching, especially at night. Sometimes intact worms (length 5–13mm, diameter 0.1–0.5mm) are seen in the faeces. Unwashed fingers transmit ova from the perianal skin to the mouth. Personal hygiene is important in treatment and preventing reinfection (handwashing and nail-scrubbing before each meal and after every visit to the toilet). A bath immediately after getting up removes ova laid overnight. Treat all members of the family with mebendazole, unless contraindicated (see *BNF*), and arrange GP follow-up.

Other helminthic infections include roundworms, hookworms, and tapeworms. Obtain advice from departments of infectious diseases or tropical medicine (see ➲ Imported infectious diseases, p. 254).

Lice

Humans may be infected by the body louse (*Pediculosis humanis corporis*), head louse (*Pediculosis humanis capitis*), or 'crab'/pubic louse (*Phthirus pubis*).

Head lice Common in schoolchildren. Infection is not related to lack of hygiene or the length of hair. Adult lice are 3–4mm long, vary in colour from white to grey-black, and attach themselves firmly to the scalp at the base of hairs. The egg cases ('nits') are white and 1–2mm in diameter, glued firmly to the base of hairs and moving outwards as the hair grows. Head lice cause intense itching, which may suggest the diagnosis. Secondary infection may result in impetigo. Treatment options include physical insecticide, chemical insecticide (malathion), or wet combing (see https://www.nice.org.uk), and advise GP follow-up.

Infection by body lice Related to poor hygiene and infrequent washing of clothes. Body lice are found in the seams of clothing and sometimes in body hair. Treat with malathion. Clothes can be disinfected by boiling or machine laundering and ironing. Body lice may transmit rickettsial diseases (louse-borne typhus) and other infections.

Crab lice Usually transmitted sexually. They cause itching in pubic hair areas. Occasionally, children become infested on eyelashes or eyebrows. Treat with permethrin cream or malathion 0.5% aqueous solution (see *BNF*). Sexual partners or other family members may also need treatment. There may be other coexisting sexually transmitted infections.

Fleas

There are many different types of flea. They cause itchy bites with linear erythematous papules. Treat with calamine lotion and an oral antihistamine (eg chlorphenamine) if itching is severe. Consider a long-acting insecticide in the house, especially in cracks in the floor and under furniture. Advise all household cats and dogs be treated for fleas. Fleas can transmit many infections, including plague, typhus, and Q fever.

Scabies

Scabies is caused by infestation with the mite *Sarcoptes scabiei*, which is about 0.2–0.4mm long and burrows into the skin. It is most often found in the finger webs and on the flexor aspect of the wrists. After 4–6 weeks, intense itching occurs, especially at night or after a hot shower. Burrows (3–15mm long) may be apparent, especially on palpation of affected skin. Genital lesions are reddish and nodular. Secondary bacterial infection may occur. Scabies can be confirmed by microscopy of scrapings from suspected lesions. Treat with topical permethrin 5% cream or malathion aqueous 0.5% (see *BNF*). Treat all members of the household at once. Calamine lotion and an oral antihistamine may help to relieve itching.

Ticks

Ticks may be acquired from domestic animals or whilst walking through undergrowth or exploring caves. Aim to remove the tick completely. Remove ticks by grasping them with tweezers or curved forceps close to the skin and pulling them out straight and perpendicular to the skin. Ticks can carry several diseases, including Lyme disease (see below), tick-borne encephalitis, typhus, and Rocky Mountain spotted fever. Tick paralysis occurs in North America and Australia, with progressive paralysis which is often misdiagnosed as poliomyelitis.

The risk of infection from tick bites is low in most areas, and so routine prophylaxis with antibiotics is not recommended. However, do warn patients to seek medical attention if a rash develops at the site of the bite or they develop a fever.

Lyme disease (Lyme borreliosis) Is caused by the tick-borne spirochaete *Borrelia burgdorferi* and occurs in the UK, most of Europe, the USA, and parts of Asia and Australia. Most cases occur in summer and early autumn and are transmitted by ticks from deer or sheep. The initial tick bite may go unnoticed. Clinical illness develops after about 7–14 days (range 2–30 days), with an expanding red area around the site of the bite (erythema migrans). The second clinical stage of the disease occurs some weeks or months later, with fever, muscle and joint pains, and sometimes facial palsy or other cranial nerve or peripheral nerve palsies. Meningitis, encephalitis, and arthritis may develop. Myocarditis and heart block occur occasionally. Refer to an infectious diseases specialist for confirmation (usually initially including an enzyme-linked immunosorbent assay for Lyme disease) and treatment if Lyme disease is suspected. Treatment for adults and older children is usually with oral doxycycline, unless there is CNS involvement or carditis with haemodynamic instability, when IV ceftriaxone is preferred (see https://www.nice.org.uk).

Tuberculosis[ND]

The *Mycobacterium* genus is characterized by acid-fast staining (ie it is not decolourized by acid after staining with hot carbol fuchsin).

Infection with *Mycobacterium tuberculosis* is common throughout the world. There is growing concern about the re-emergence of TB in the UK and other countries. Many cases of TB occur in the lower socio-economic groups, ethnic minorities, and the immunocompromised. The incidence of TB ↑ with age. Transmission is by the inhalation route.

Presentation

TB can involve almost any organ of the body.

Primary infection

This is usually pulmonary and often asymptomatic.

Post-primary infection

This may present with malaise, weight loss, and night sweats, with localized symptoms, depending on the organs involved.

Pulmonary TB

A relatively common way for TB to present, pulmonary TB may cause cough (initially dry, then productive), haemoptysis, pneumonia, and pleural effusion (see ➲ Pleural effusion, p. 107). CXR typically shows fibronodular/linear opacities in the upper lobes, but they can be seen in the middle and lower lobes ± calcification, cavitation, lymphadenopathy, and pleural effusion.

Miliary TB

This involves blood-borne infection of many organs and develops over 1–2 weeks with fever, weight loss, malaise, and breathlessness. CXR may show multiple small opacities throughout the lung fields, and choroidal tubercles may be visible in the optic fundi.

TB meningitis

This causes headaches and vomiting, sometimes with neck stiffness, cranial nerve palsies, and papilloedema (see ➲ Meningitis, pp. 232–3).

Tuberculous osteomyelitis

This usually affects the spine and progresses slowly over weeks/months, with collapse of adjacent vertebrae and the development of paravertebral abscesses.

Tuberculous lymphadenitis

Patients may present with swollen lymph nodes from tuberculous lymphadenitis or with sinuses or cold abscesses from bone or soft tissue infection. Microscopy of the discharge will show acid-fast bacilli.

Treatment

Refer patients with suspected TB to an appropriate specialist for assessment and treatment. Initial treatment of confirmed TB involves the combination of rifampicin, isoniazid, ethambutol, and pyrazinamide. Isolate patients with untreated pulmonary TB. Notify the local health protection department (see ➲ Notifiable infectious diseases, p. 229).

Anthrax[ND]

Anthrax is caused by the bacterium *Bacillus anthracis* which affects cows and other herbivorous animals, especially in warm climates. The bacterium forms spores, which may remain infective for years. Most human cases of anthrax are *cutaneous anthrax* caused by direct skin contact with infected tissues and occur in people working with animal products such as imported hides. Less common, but more serious, are *inhalation anthrax* caused by inhalation of anthrax spores, and *intestinal anthrax* which is a rare form of food poisoning caused by undercooked infected meat. Anthrax spores released deliberately in terrorist attacks could cause cutaneous anthrax or inhalation anthrax, which is often fatal.

Cutaneous anthrax starts 2–7 days after infection, with a red papule which develops into an ulcer with a black leathery eschar, surrounded by non-pitting oedema. The lesion is painless but may itch. Small satellite lesions may surround the original lesion. Malaise and fever may occur, with septicaemia in 10–20% of cases. Penicillin ↓ the risk of complications from cutaneous anthrax. Clinical diagnosis is confirmed by microscopy and culture of the pustule.

Inhalation anthrax starts within 48hr of exposure (rarely up to 6 weeks) with a flu-like illness, followed by breathlessness, cyanosis, stridor, and sweating, often with subcutaneous oedema of the chest and neck. CXR and CT show mediastinal widening from lymphadenopathy and pleural effusions. Shock, septicaemia, and meningitis are common and usually fatal, despite antibiotics and intensive treatment.

Airborne transmission of anthrax from one person to another does not occur, but cutaneous anthrax could result from direct contact with anthrax lesions. Obtain expert advice immediately if anthrax is suspected. It is a notifiable disease (see ➲ Notifiable infectious diseases, p. 229). Post-exposure antibiotics (eg ciprofloxacin) can prevent anthrax if started early enough. Anticipate press enquiries after any case of anthrax, especially if anthrax has been released deliberately.

Anthrax in drug users

After a serious anthrax outbreak in heroin users in Scotland in 2010, Health Protection Scotland advised doctors to suspect anthrax in a drug user presenting with any of the following:

- Severe soft tissue infection and/or signs of severe sepsis/meningitis.
- Clinical features of inhalational anthrax.
- Respiratory symptoms + features of meningitis or intracranial bleeding.
- GI symptoms (eg pain, bleeding, nausea, vomiting, diarrhoea, ascites).

Approach Get expert help early to advise on management (microbiology, hospital infection control team, Public Health, ICU, surgeons). Start IV antibiotics according to advice (eg combination of ciprofloxacin, clindamycin + penicillin) or if there is soft tissue infection (ciprofloxacin, clindamycin, penicillin, flucloxacillin + metronidazole). Experts will advise on the use of anthrax immune globulin.

Streptococcal infections

Streptococcus pyogenes and other streptococci may reside in the pharynx without symptoms but can cause sore throats (see ➲ Sore throat, pp. 570–1), soft tissue infections (see ➲ Infected wounds and cellulitis, p. 419; ➲ Cellulitis and erysipelas, p. 545), scarlet fever, endocarditis, and septicaemia. Later, non-suppurative sequelae of streptococcal infections include erythema nodosum, rheumatic fever (see ➲ Acute arthritis: 2, p. 513), and glomerulonephritis. Streptococci and staphylococci may cause necrotizing fasciitis, impetigo, and toxic shock.

Scarlet fever[ND]

Some streptococcal infections are associated with scarlet fever. A diffuse blanching scarlet rash often involves the neck, chest, axillae, and groin. Occlusion of sweat glands makes the skin feel rough, like sandpaper. During the first 1–2 days of illness, there is a 'white strawberry tongue', with red papillae protruding through white furry material. After a few days, the white fur separates, leaving a shiny 'raspberry tongue'. Ten to 14 days after onset of the rash, the skin may peel from the palms and soles. Treat with penicillin V (or azithromycin if penicillin-allergic) for 14 days. Complete recovery is usual.

Infective endocarditis

Endocarditis may develop on previously normal heart valves, as well as on diseased or prosthetic valves. The most common organism is *Streptococcus viridans*. Many acute cases present with heart failure and involve *Staphylococcus aureus*. Injecting drug users are liable to staphylococcal infection of the tricuspid valve, with fever and pneumonia from septic PE.

Clinical features Fever and changing murmurs suggest endocarditis. Emboli may cause strokes. Ask about weight loss, malaise, and night sweats. Look for clubbing, splinter haemorrhages, splenomegaly, anaemia, and microscopic haematuria.

Treatment On suspicion of endocarditis, admit immediately for investigation (blood cultures, echocardiography) and treatment.

Necrotizing fasciitis

Rare and severe bacterial infection of soft tissues. It can occur with or without obvious trauma and may follow illicit IM heroin injection ('muscle popping'). *Streptococcus* pyogenes is often involved, sometimes with *S. aureus* or other bacteria. Often there are both aerobic and anaerobic organisms. Infection involves the fascia and subcutaneous tissues, with gas formation and the development of gangrene. Infection may spread to adjacent muscles, causing myonecrosis or pyogenic myositis. Similar infections may involve the abdomen and groin (Fournier's gangrene).

Initial symptoms and signs May be vague, with severe pain, but little on examination—the affected area may be tender, sometimes with slight erythema and swelling. Pyrexia is usual. Infection can spread rapidly and cause marked soft tissue swelling with discoloration, bruising, haemorrhagic blisters, or overlying skin necrosis. Toxic shock may develop—mortality is high. X-rays may show gas in the soft tissues.

Treatment Resuscitate with IV fluids and antibiotics (eg meropenem 2g and clindamycin 900mg), urgent surgery to debride the affected area and excise necrotic tissues, and ICU.

Staphylococcal infections

S. aureus is involved in many infections of wounds, soft tissues (see ➔ Infected wounds and cellulitis, p. 419), joints, and bones (see ➔ Acute arthritis: 2, pp. 512–13; ➔ Osteomyelitis, p. 727; ➔ Septic arthritis, p. 511). Staphylococci also cause impetigo, scalded skin syndrome, food poisoning, toxic shock syndrome, endocarditis, pneumonia, septicaemia, and meningitis.

Impetigo

This highly infectious superficial skin infection is caused by staphylococci or streptococci. It may involve normal skin or complicate a pre-existing condition such as eczema or scabies. Lesions often start around the mouth and nose, spreading rapidly on the face and to other parts of the body. Irregular golden-yellow crusted lesions occur, particularly in streptococcal infections. Staphylococci may cause bullous impetigo, with bullae containing pus which rupture and dry to form crusts. Treat with topical fusidic acid (usually qds for 7 days) and give PO flucloxacillin for 7 days (or azithromycin if allergic) if lesions are widespread or there is cellulitis or pyrexia.

Scalded skin syndrome

S. aureus may produce an exotoxin causing separation of the outer layers of the epidermis, large sections of which slide off with minimal pressure, leaving large raw areas resembling a severe scald. Drug allergies can cause similar lesions. Most cases of scalded skin syndrome (toxic epidermal necrolysis, Lyell's syndrome) occur in children. Admit for nursing and medical care.

Toxic shock syndrome

Caused by exotoxins from *S. aureus* or (less commonly) *S. pyogenes*. Some cases during menstruation are related to tampons, whilst other cases occur after surgical operations, burns, other trauma, or local infections. There is high fever, a generalized erythematous rash, confusion, diarrhoea, muscle pains, hypotension, and renal failure. Subsequently, scales of skin separate from the hands and feet. Death may occur from multi-organ failure. Treat for septic shock with IV fluids and anti-staphylococcal antibiotics. Remove tampons and send for culture. Refer to ICU. Involve a surgeon if an associated abscess requires drainage.

Staphylococcal septicaemia

Occurs particularly in debilitated or immunocompromised patients and in injecting drug users. There may be endocarditis, with metastatic infection of lungs, bone, or soft tissues and gangrene due to emboli or arterial thrombosis. Signs of meningitis and DIC may suggest meningococcal septicaemia (see ➔ Meningitis, pp. 232–3) and the rash may be similar.

Meticillin-resistant *Staphylococcus aureus* (MRSA)

MRSA causes particular concern because of antibiotic resistance and is carried by many asymptomatic people (patients and staff). Transmission is minimized by handwashing (see ➔ Infection control and prevention, pp. 36–7) and other infection control measures. Information about MRSA for patients is available online at https://www.nhs.uk

Tetanus[ND]

An acute and often fatal disease, common in much of Asia, Africa, and South America, especially in neonates. Now rare in developed countries—30–40 cases/y in the UK, many involving the elderly. Injecting drug users (eg those 'skin popping') are also at particular risk. Spores of the Gram +ve organism *Clostridium tetani* (common in soil and animal faeces) contaminate a wound, which may be trivial. The spores germinate in anaerobic conditions, producing tetanospasmin, an exotoxin which blocks inhibitory neurones in the CNS and causes muscle spasm and rigidity.

Incubation period is usually 4–14 days but may be 1 day to 3 months. In 20% of cases, there is no known wound. Tetanus occasionally occurs after surgery or IM injections.

Clinical features

Stiffness of the masseter muscles causes difficulty in opening the mouth (trismus, lockjaw). Muscle stiffness may spread to all facial and skeletal muscles and muscles of swallowing. Characteristically, the eyes are partly closed and the lips pursed and stretched (risus sardonicus). Spasm of chest muscles may restrict breathing. There may be abdominal rigidity, stiffness of limbs, and forced extension of the back (opisthotonus). In severe cases, prolonged muscle spasms affect breathing and swallowing. Pyrexia is common. Autonomic disturbances cause profuse sweating and tachycardia and hypertension, alternating with bradycardia and hypotension. Cardiac arrhythmias and arrest may occur.

Differential diagnoses

Dystonic reaction to metoclopramide or phenothiazines, strychnine poisoning, quinsy, dental abscess, meningitis, and rabies. Procyclidine relieves muscle spasms from drug-induced dystonia but will not affect tetanus; diazepam may relieve dystonia or tetanic spasms.

Management

Obtain senior medical and anaesthetic help. Monitor breathing, ECG, and BP. Refer to ICU. Control spasms with diazepam. Paralyse and ventilate if breathing becomes inadequate. Clean and debride wounds. Give penicillin, metronidazole, and human tetanus immunoglobulin.

Prognosis

Depends on severity of disease and quality of care. Short incubation (<4 days) and rapid progression suggest severe disease, with a high mortality. With expert intensive care, mortality in adults is <10%, but neonatal tetanus is often fatal.

Immunization

Tetanus is eminently preventable by immunization and proper care of wounds (see ➲ The approach to wounds, p. 410; ➲ Tetanus prophylaxis, p. 424).

Gas gangrene

This is a rapidly spreading infection of muscle caused by toxin-producing clostridial bacteria (anaerobic Gram +ve bacilli), usually *Clostridium perfringens*. It is fatal if untreated. It may involve wounds of the buttocks, amputations for vascular disease, or severe muscle injuries (eg gunshot wounds). Occasionally gas gangrene of the perineum occurs without trauma.

Incubation period

Is usually <4 days (sometimes a few hours). Sudden severe pain occurs at the wound site. Generalized toxicity develops, with tachycardia, sweating, and fever. Swelling and skin discoloration occur around the wound, with a serous ooze, marked tenderness, and sometimes haemorrhagic vesicles and crepitus. Shock and AKI develop, with death often within 2 days of the first symptoms.

Diagnosis

Depends on clinical features. Severe pain necessitates wound inspection (remove or window any POP). Obtain immediate senior surgical advice if gas gangrene is suspected. Wound discharge may contain Gram +ve bacilli. X-rays may show soft tissue gas, but its absence does not exclude gas gangrene.

Treatment

IV antibiotics (eg penicillin and clindamycin), immediate surgical removal of all infected tissue, and ICU. Hyperbaric O_2 and gas gangrene antitoxin are rarely available and of no proven benefit.

Botulism[ND]

Clostridium botulinum exotoxin paralyses autonomic and motor nerves by blocking acetylcholine release at neuromuscular junctions and nerve synapses. Infection follows eating tinned or preserved food contaminated with *C. botulinum* spores. Rarely, *C. botulinum* infects wounds or colonizes the gut. Injecting drug users may develop botulism after IM or SC injections of contaminated drugs.

Incubation period 12–72hr. Initial symptoms may be GI (nausea, vomiting, abdominal discomfort, dryness of the mouth) or neurological (dizziness, blurred vision, diplopia). Later problems include dysarthria, dysphagia, muscle weakness or paralysis, constipation and urinary retention, respiratory failure, and sudden death. Susceptibility varies—some people who eat contaminated food develop no symptoms or suffer only mild fatigue.

Clinical signs Result from involvement of autonomic and motor nerves: dry mouth, cranial nerve palsies (ptosis, squint, fixed pupils, weakness of tongue), and limb weakness with flaccid muscles. Consciousness and sensation are preserved. Hypotension and ileus may occur. Fever is unusual.

Differential diagnoses Guillain–Barré syndrome, myasthenia, brainstem stroke, diphtheria, and rabies. It may be misdiagnosed as staphylococcal food poisoning, paralytic shellfish poisoning, and CO or mushroom poisoning.

Management Get senior help. Assess breathing; ventilate if necessary, and admit to ICU. Botulinum antitoxin ↓ mortality and morbidity—see *BNF* and TOXBASE (see ➲ National Poisons Information Service, pp. 188–9). Inform Public Health—others who have eaten contaminated food may need urgent treatment. Anticipate media enquiries.

Sexually transmitted infections

The most common sexually transmitted infection (STI) is *Chlamydia*. Other common diseases include gonorrhoea, genital herpes, trichomoniasis, genital warts, *Mycoplasma genitalium*, *Pediculosis pubis*, HIV, and syphilis. Many patients have more than one disease. Suspicion of STI necessitates prompt referral to a GU medicine clinic for proper diagnosis, treatment, and follow-up of the patient and contacts. Some GU departments allow self-referral. Others provide an on-call service. Only prescribe antibiotics for suspected STIs on the advice of a GU specialist.

Genital ulcers and sores

Most genital ulcers/erosions are either multiple and painful or single and painless. In the UK, multiple genital ulcers are most often due to herpes simplex; other causes are Behçet's disease, chancroid, Stevens–Johnson syndrome, and scabies. Multiple painful sores may occur with gonorrhoea, *Candida*, or other conditions. Syphilis can cause painless or painful ulceration. Primary chancre is a single ulcer, and secondary syphilis often multiple—both are highly infectious—and the incidence has ↑ recently. Other causes of painless ulcers include carcinoma and trauma.

Urethritis

In men, dysuria and urethral discharge are the most common presenting symptoms of an STI. However, 5–10% of men with gonococcal or non-gonococcal urethritis have no symptoms. Urethritis may result from physical trauma, FBs, or attempts at self-treatment with intra-urethral chemicals.

Gonorrhoea Usually has a shorter incubation period (3–5 days) than non-gonococcal urethritis (eg *Chlamydia* 7–14 days) but do not rely on a clinical diagnosis—refer to a GU clinic for diagnosis, management, and follow-up. If no GU advice is available and treatment cannot wait for attendance at a GU clinic, give ceftriaxone 500mg IM + azithromycin 1g PO. If possible, make a glass slide of the discharge, dried in air, for the patient to take to the clinic. Advise the patient not to pass urine for 2hr before the appointment, in order to allow serial urine samples to be taken.

Reactive arthritis (previously called Reiter's syndrome) Is a rare complication of non-gonococcal urethritis. There is arthritis (mainly of the knees, ankles, and feet) and sometimes conjunctivitis, rashes, and cardiac and neurological problems.

Gonorrhoea

Gonorrhoea may infect the urethra, cervix, rectum, pharynx, or conjunctiva. Men usually have dysuria and urethral discharge, with rectal discharge and tenesmus in rectal infection. Women are often asymptomatic but may have dysuria and vaginal discharge.

Complications Include prostatitis, epididymitis, salpingitis, and Bartholin's abscess; rarely, septicaemia with arthritis, fever, rash (maculopapular initially, then pustular), and endocarditis.

Hepatitis[ND]

Hepatitis A (infectious hepatitis)[ND]

Hepatitis A occurs throughout the world but is particularly common in the tropics and subtropics. It is transmitted by contamination of food or water with infected faeces or urine. Many infections are asymptomatic. The incubation period is 2–6 weeks (usually ~4 weeks). Fever, malaise, anorexia, and nausea may last for 2–7 days before jaundice develops. Jaundice is more common in adults than in children and is associated with dark urine, pale stools, and tender hepatomegaly.

Treatment Is symptomatic, but advise avoidance of alcohol. Infectivity is greatest before jaundice develops, so isolation is of little value. Arrange follow-up by a specialist or GP. Complete recovery is usual. Consider hepatitis A vaccine for close contacts (see *BNF*).

Hepatitis B[ND]

Hepatitis B is transmitted by infected blood (eg shared needles in drug users, tattooing, needlestick injury) and sexual intercourse. Incubation period is 6 weeks to 6 months. Symptoms are similar to hepatitis A, often with arthralgia and skin rashes. Most patients with hepatitis B recover completely. A few develop liver failure or chronic hepatitis, with a risk of liver cancer. Asymptomatic carriers of hepatitis B virus are common (~0.1% of UK population, but ~20% in parts of Africa and Asia). All health care workers should be immunized against hepatitis B and use 'standard precautions' (see ➲ Infection control and prevention, pp. 36–7) when handling all blood samples and 'sharps'. The management of needlestick injury is described in ➲ Needlestick injury, p. 425.

Hepatitis C, D, and E[ND]

Hepatitis C and D Are spread in the same way as hepatitis B and may cause hepatic failure or chronic liver disease. New treatments for hepatitis C (eg ledipasvir + sofosbuvir) have transformed the prognosis.

Hepatitis E Is similar to hepatitis A but has high mortality in pregnancy. Refer to a specialist for follow-up.

Leptospirosis (Weil's disease)[ND]

Leptospirosis, caused by the spirochaete *Leptospira interrogans* and other *Leptospira* species, is spread by contact with infected rat's urine, often in rivers, canals, or sewers. Leptospires enter the body through small breaks in the skin or via mucous membranes of the eyes or nose. About 10 days after exposure (range 2–26 days), the illness starts with fever, severe muscle pains, headache, sore throat, nausea, and vomiting. Conjunctival reddening is common. A haemorrhagic rash, jaundice, renal failure, and pulmonary haemorrhage may occur (Weil's disease).

Refer to an infectious diseases unit. Treatment is with penicillin or doxycycline, with supportive care and haemodialysis if necessary. Prophylactic doxycycline is reasonable for people who fall into waterways likely to be contaminated with leptospires.

Human immunodeficiency virus

First reports of AIDS involved the homosexual community in the USA in 1981. HIV was identified as the causative agent in Paris in 1983.

Structure and pathogenesis

HIV is an RNA retrovirus. Retroviruses are characterized by having the enzyme reverse transcriptase. This allows viral RNA to be transcribed (copied) into DNA and incorporated into host cells, which then make a new virus. This mechanism has proved difficult to overcome—despite much effort, no 'vaccine' is yet available and a cure is elusive.

Glycoproteins on the surface of HIV bind to specific receptors on target cells. The cellular receptor for HIV is the CD4 molecule. CD4 receptors are found on a variety of cells, particularly helper/inducer T lymphocytes ('CD4 cells'), but also monocytes and macrophages. CD4 cells normally play a crucial role in co-ordinating the immune response—as HIV infection progresses and CD4 cell counts ↓, the patient develops profound cellular immunodeficiency. Although other complex mechanisms are also involved, CD4 cell counts provide a useful index of disease stage and progress.

Transmission

HIV has been found in many body fluids but is mostly transmitted via blood, semen, cervical secretions, and perhaps breast milk. It may be acquired by:

- Sexual intercourse (vaginal or anal), with an ↑ risk of transmission where individuals already have a genital mucosal breach (eg coexisting STI).
- Risk of transmission from HIV +ve pregnant mother to baby is ~15%.
- Transfusion of unscreened blood/blood products (screening started in 1985 in the UK).
- Contaminated needles shared amongst IV drug users. Needlestick injuries from an HIV +ve source carry a risk of ~0.3%.

Prevention

Post-exposure prophylaxis can significantly ↓ the risk of acquiring HIV after a needlestick injury or unprotected sexual contact (see ➲ Needlestick injury, p. 425).

An ↑ number of those at high risk of acquiring HIV are taking antiretroviral therapy as pre-exposure prophylaxis ('PrEP').

Diagnosis and HIV testing

Antibodies to HIV provide evidence of infection and form the basis of many blood tests, but these antibodies may not appear until 3 months after exposure. Over-the-counter test kits are now available.

Many HIV +ve patients attending the ED are aware of their HIV status. Some patients, however, present with HIV-related illness, without knowing (or admitting) that they are HIV +ve. Test for HIV (with consent) in ED patients where it may influence immediate management.

Routine screening for HIV in ED patients in the UK has been proposed where there is a relatively high prevalence (>2/1000 population).

Natural history of HIV infection

Acute infection is often subclinical, but 2–6 weeks after exposure, there may be non-specific febrile illness with lethargy, myalgia, sore throat, lymphadenopathy, and often a maculopapular rash on the face and trunk. This illness usually resolves after 1–2 weeks but sometimes persists for longer. A long asymptomatic period (~10y) follows the initial illness.

Some patients develop persistent generalized lymphadenopathy (PGL), with lymphadenopathy (>1cm) at two non-inguinal sites for 3 months.

Following a latent phase of chronic infection, patients (especially without treatment) are at risk of developing symptoms as immunity ↓, developing unusual infections and tumours.

Antiretroviral therapy delays the progression of HIV-related illness and ↑ length of survival.

Initial presentation of HIV to the ED

Presentation of any of the diseases listed below should arouse particular suspicion.

Centers for Disease Control HIV infection classification

A patient's CD4 cell count helps to provide information on immune function and disease progression—the combination of this with clinical status underpins the Centers for Disease Control (CDC) classification:

- Stage 1—no AIDS-defining condition and either a CD4 cell count of ≥500 cells/mm^3 or CD4 percentage of ≥29%.
- Stage 2—no AIDS-defining condition and either a CD4 cell count of 200–400 cells/mm^3 or CD4 percentage of 14–28%.
- Stage 3 (AIDS)—a documented AIDS-defining condition or a CD4 cell count of <200 cells/mm^3 or a CD4 percentage <14%.

Some AIDS-defining diseases in HIV +ve patients

- *Pneumocystis jiroveci* pneumonia (previously called *P. carinii*).
- Kaposi's sarcoma.
- Tracheobronchial or oesophageal candidiasis.
- Cerebral toxoplasmosis.
- Pulmonary TB.
- Recurrent pneumonia.
- CMV retinitis.
- Cerebral lymphoma.
- Recurrent *Salmonella* septicaemia.
- Disseminated histoplasmosis.
- Invasive cervical carcinoma.
- Disseminated or extrapulmonary coccidioidomycosis.
- Extrapulmonary cryptococcosis.
- Chronic intestinal cryptosporidiosis for >1 month.
- Progressive multifocal leukoencephalopathy.
- Oesophageal or bronchial herpes simplex for >1 month.
- Histoplasmosis.
- Wasting syndrome attributed to HIV.

Presentation of HIV +ve patients

Some patients with symptomatic HIV infection bypass the ED and liaise directly with the specialist unit caring for them. Assessment of HIV +ve patients is difficult in the ED where advanced infections may present with relatively few signs and little past history is available. Similarly, interpretation of investigations is difficult without knowledge of previous results. On this basis, adopt a low threshold for specialist referral. HIV +ve patients may present with a variety of complications.

Respiratory problems

As CD4 counts ↓, pneumonia due to *Pneumocystis jiroveci* (previously *Pneumocystis carinii*) becomes more likely and it is a common indicator diagnosis of AIDS. A non-productive cough occurs with dyspnoea and fever. CXR may show bilateral interstitial mid-zone shadowing but may be normal. Obtain blood and sputum cultures; rehydrate with IV fluids as necessary, and refer urgently for IV co-trimoxazole or pentamidine ± steroids. Occasionally, *Pneumocystis* infection may present with fulminant respiratory failure, needing emergency tracheal intubation and IPPV. Other common infections include *Aspergillus, Cryptococcus*, and TB. Injecting drug users are at an ↑ risk of bacterial infection, especially *Haemophilus influenzae* and *Streptococcus pneumoniae*.

Neurological problems

Cryptococcus neoformans meningitis may present with headache, fever, and sometimes ↓ conscious level. Neck stiffness and photophobia are rare. Obtain a CT scan to exclude space-occupying lesions before LP and CSF examination. *Cerebral toxoplasmosis* may present similarly, often with focal signs or fits. Neurological problems may also be caused by cerebral lymphoma, progressive leucoencephalopathy (focal deficits secondary to papovaviruses), CMV encephalitis (retinopathy is usually present—see below), and HIV-associated delirium or dementia.

Eye problems

The most significant eye problem is *CMV retinitis*, occurring in 15% of patients. This presents with blurred vision, blind spots, 'floaters' or flashing lights, and ↓ VA. Characteristic retinal changes are irregular yellow-white lesions and perivascular haemorrhages that have been called 'pizza pie'. Retinal detachment may occur. Refer urgently for ophthalmological assessment and treatment with ganciclovir.

Mucocutaneous problems

Oral candidiasis, seborrhoeic dermatitis, and oral hairy leukoplakia (white ridges on the lateral border of the tongue) are often seen before AIDS develops. As immunity ↓, patients may develop herpes simplex, herpes zoster, and molluscum contagiosum. Gum bleeding and dental problems are common—the former may be due to thrombocytopenia. Kaposi's sarcoma is seen in the skin and mucous membranes. It is rarely life-threatening but requires specialist evaluation and treatment.

Gastrointestinal problems

Nausea, vomiting, diarrhoea, and weight loss are common complaints and can be due to drug therapy. Dysphagia may result from oesophageal candidiasis, herpes simplex, CMV, or Kaposi's sarcoma, all of which require specialist investigation and treatment.

CMV colitis can cause a serious illness, characterized by abdominal pain, diarrhoea, and fever. Obtain plain X-rays if the recognized complication of toxic dilatation is suspected. Other frequently implicated infective causes of diarrhoea include *Cryptosporidium, Giardia, Microsporidium*, and *Salmonella*. Send stool specimens (including for *Clostridium difficile*) and treat severe diarrhoea by IV rehydration and correction of electrolyte imbalance before referral.

Hepatitis viruses are likely to complicate the picture in injecting drug users, many of whom are infected with hepatitis B and C.

Drug reactions and side effects

Some patients present with symptoms due to drug therapy. This may not be initially apparent—the safest approach is to exclude tumours and opportunistic infection first.

HIV and ED staff

ED staff are often concerned about the possibility of acquiring HIV from patients. The need to perform invasive emergency procedures on 'high-risk' patients makes these concerns understandable. Additionally, apparently 'low-risk' patients with untreated HIV may also pose a greater threat than patients known to have HIV who are taking antiretroviral therapy. Therefore, treat every patient as 'high risk'. The risk to ED staff is largely in the form of needlestick injury [although the risk of acquiring HIV following a needlestick injury from a HIV +ve source can be ↓ by post-exposure prophylaxis (see ➲ Needlestick injury, p. 425]. Safe practice is reflected in the recommended standard precautions (see ➲ Infection control and prevention, pp. 36–7)—follow these in all patients. Pregnant staff should not treat patients with AIDS (because of concern about CMV and herpes simplex virus).

Handling HIV +ve patients

Despite vigorous attempts to educate the general public, HIV remains a taboo subject amongst many in society. It is imperative to treat all patients, including those who are HIV +ve, with sensitivity and compassion. In view of prevailing attitudes, patient confidentiality is of the utmost importance. Remember that family and friends accompanying the patient may be unaware of the patient's HIV status.

HIV +ve staff

The risk to patients from ED staff infected with HIV is minimal but remains a theoretical possibility. Staff who believe that they may be HIV +ve must obtain and follow occupational health advice.

Needlestick injury

See ➲ Needlestick injury, p. 425.

Imported infectious diseases

Patients may present to the ED with infectious diseases acquired abroad. It is essential to ask where a patient has been, especially in the 6 weeks before the onset of symptoms. The most common imported diseases are bowel infections causing diarrhoea (see ➔ Gastroenteritis/food poisoning[ND], pp. 236–7). Less common, but very important, diseases include malaria (see ➔ Malaria[ND], p. 255), typhoid (see ➔ Typhoid[ND] and parathyroid[ND] (enteric fever), p. 256), legionnaires' disease (see ➔ Pneumonia, pp. 114–15), and hepatitis (see ➔ Hepatitis[ND], p. 249). Rabies (see ➔ Rabies[ND], p. 257) and viral haemorrhagic fevers, such as Lassa fever (see ➔ Viral haemorrhagic fevers[ND], p. 258), are very rare in the UK.

Occasionally, tropical diseases are acquired in Britain from bites by infected insects carried by plane (eg 'airport malaria').

Advice about tropical diseases is available from departments of infectious diseases or tropical medicine, including:

- The Hospital for Tropical Diseases (based in London) (🔗 http://www.thehtd.org; telephone 0203 456 7890.
- A public access website provided by the NHS which gives information for people travelling abroad from the UK (🔗 https://www.fitfortravel.scot.nhs.uk).
- An alternative is 🔗 https://travelhealthpro.org.uk

Pyrexia of unknown origin in travellers

Think of, and check for, malaria (see ➔ Malaria[ND], p. 255) in any febrile patient who has been in a malarious area. Consider Lassa fever (see ➔ Viral haemorrhagic fevers[ND], p. 258) in someone who has been in West Africa in the previous 3 weeks. Typhoid (see ➔ Typhoid[ND] and parathyroid[ND] (enteric fever), p. 256) often presents as a septicaemic illness with constipation, rather than diarrhoea. TB (see ➔ Tuberculosis[ND], p. 242) and brucellosis may cause fever and sweating at night.

Investigations

Warn the lab of possible risk of infection.

- FBC, thick and thin blood films for malaria.
- U&E, blood glucose, blood culture.
- Urine stick testing, microscopy, and culture.
- CXR.

Further investigations may include LFTs and viral titres.

Management

Barrier nurse in a cubicle (ideally use a negative-pressure room if available). Wear gown, gloves, goggles, and mask. Record vaccination and prophylaxis history, with countries and areas visited and dates of travel and onset of symptoms. Look particularly for confusion, dehydration, jaundice, rashes, chest signs, liver and spleen enlargement and tenderness, lymphadenopathy, neck stiffness, and photophobia. Seek expert advice at once if the patient is very ill or there is concern about typhoid or Lassa fever or other VHFs. Refer to an infectious diseases specialist.

Malaria[ND]

Malaria is very common in the tropics and subtropical regions, and is a parasitic infection transmitted by mosquitos. The five species which cause malaria in humans are *Plasmodium falciparum, P. vivax, P. malariae, P. ovale*, and *P. knowlesi*. Falciparum ('malignant tertian') malaria is the most important, since it may be rapidly fatal and drug-resistant strains are common. Serious complications are unusual in the other types of malaria, but they may cause febrile convulsions in children.

In the UK, malaria occurs in travellers from malarious areas, especially *P. vivax* from the Indian subcontinent and *P. falciparum* from Africa, South East Asia, and Central and South America. Malaria often develops despite antimalarial tablets, because of drug resistance or incorrect dosage. Check for malaria in any febrile illness within 2 months of visiting a malarious area. Common misdiagnoses are influenza and viral hepatitis.

Specific country information about the risk of malaria is available on https://travelhealthpro.org.uk

Clinical features

The incubation period is usually 7–14 days for *P. falciparum* and 12–40 days for other types of malaria, but occasionally it is much longer (>1y), especially in *P. malariae* and *P. vivax* infections. Malaise, fatigue, fever, and headache are followed by paroxysms (lasting 8–12hr) of rigors, vomiting, and then severe sweating. Fever may be periodic (classically 48hr in *P. ovale* or *P. vivax*, and 72hr in *P. malariae*). Haemolytic anaemia, jaundice, and splenomegaly may occur, but lymphadenopathy is not a feature. *P. falciparum* may cause cerebral malaria with coma, fits, and focal neurological signs. Diarrhoea, cardiac failure, pulmonary oedema, and shock may occur. Deterioration can be rapid.

Investigations

Consider Lassa fever (see Viral haemorrhagic fevers[ND], p. 258) in recent visitors to West Africa. Send blood for thin and thick film examination for malaria in any ill person who has visited a malarious area. Repeated blood films may be needed. Also arrange FBC (malaria may cause anaemia, thrombocytopenia, neutropenia), blood glucose (hypoglycaemia may occur), and U&E (AKI possible), and test the urine for blood ('black water fever').

Treatment of malaria

Involve ICU and get urgent expert advice from a tropical disease specialist (see Imported infectious diseases, p. 254). Artemisinin derivatives (such as artemether or artesunate) are more effective than quinine for the management of falciparum malaria—treat with an artemisinin combination, such as artemether–lumefantrine (Riamet®) according to the UK malaria treatment guidelines (2016) (see https://www.journalofinfection.com/article/S0163-4453(16)00047-5/pdf). These guidelines also outline the management of less severe non-falciparum malaria.

Typhoid[ND] and paratyphoid[ND] (enteric fever)

These fevers, caused by *Salmonella typhi* and *S. paratyphi A, B,* or *C*, occur throughout the world, especially where hygiene is inadequate. They are spread by contamination of food or water by urine or faeces from a patient or an asymptomatic carrier. Typhoid may occur despite immunization. Typhoid and malaria are the first diseases to consider if fever develops soon after a visit to the tropics. The *incubation period* is usually 7–14 days but may range from 3 to 60 days.

Initial symptoms

Headache, fever, and a dry cough, with abdominal discomfort and anorexia. Constipation is common, but diarrhoea may occur, especially in children. Confusion and hallucinations may develop.

Physical examination

This may be normal, except for fever. There may be relative bradycardia (ie less than the usual 10 beats/min ↑ in pulse rate per °C of fever). Splenomegaly and abdominal tenderness occur, but there is no lymphadenopathy. 'Rose spots' are pink macular spots on the lower chest or upper abdomen which blanch on pressure. There may be signs of pneumonia or dehydration. Intestinal perforation or haemorrhage occur occasionally.

Investigations

FBC (mild anaemia is common, WCC usually normal), blood films for malaria, U&E, LFTs, blood cultures, and CXR (for signs of TB or pneumonia).

Treatment

Isolate and barrier nurse. Admit suspected cases to an infectious diseases unit and notify the local consultant in communicable disease control. The usual drug treatment is with ciprofloxacin or cefotaxime, but other antibiotics may be needed for drug-resistant infections.

Dengue

Dengue is a mosquito-borne viral infection which is common in Southern Asia, the Western Pacific, Central Africa, and Central and South America. Most infections are asymptomatic. Symptoms start after an incubation period of 4–7 days with fever, malaise, nausea and vomiting, headache, and severe muscle and bone pains ('break bone fever'). Some patients have a transient macular rash, petechiae, lymphadenopathy, hepatomegaly, ↓ WCC, ↓ platelets, and ↑ liver enzymes.

Most patients recover after 3–7 days with symptomatic treatment. A few develop dengue shock syndrome (DSS) with hypotension, pleural effusions, ascites, ↓ plasma protein, and bleeding problems. Abdominal pain may be severe. Treatment is supportive, with careful fluid balance management and IV fluids in DSS. With expert care, most patients with severe dengue eventually make a full recovery.

Poliomyelitis[ND]

Paralytic poliomyelitis is rare in developed countries where vaccination is routine. Fever is followed by signs of meningitis, pain, and spasm in limb muscles. Respiratory failure may be fatal.

Resuscitate and ventilate if necessary and refer to ICU. The differential diagnosis includes Guillain–Barré syndrome (see Acute generalized weakness, pp. 148–9) and organophosphate poisoning (see Organophosphate poisoning, p. 214).

Rabies[ND]

Rabies is a viral infection of mammals that occurs in most parts of the world, including much of the Arctic, as well as tropical and temperate regions. At present, it is not endemic in the UK, Norway, Sweden, Iceland, Australasia, or Japan. Human and animal rabies is most common in the Indian subcontinent, China, Thailand, the Philippines, and parts of South America. Most human infections result from dog bites, but rabies can be transmitted by many other domesticated or wild animals such as cats and foxes. Rabies virus in an animal's saliva may cause infection by contamination of a bite or scratch, or by absorption through mucous membranes of the eye, mouth, or nose. Rarely, infection occurs from inhalation of the virus in bat-infested caves.

Prevention of rabies after a bite is described in Bite wounds, pp. 420–1. Detailed information about rabies is available at http://www.gov.uk/government/publications/rabies-the-green-book-chapter-27. This document includes detailed advice on both pre-exposure prophylaxis and post-exposure treatment.

Clinical features

The *incubation period* of rabies is usually 3–12 weeks but can vary from a few days to >2y.

The first symptoms are itching, tingling, or pain at the site of the bite wound. Headache, fever, and malaise occur, with spreading paralysis and episodes of confusion, hallucination, and agitation. Hydrophobia is characteristic—attempts at drinking cause spasm of muscles involved in breathing and swallowing and also profound terror. In ~20% of cases, there is 'dumb rabies', with ↑ paralysis, but no episodes of spasm or hyperactivity. Rabies is almost always fatal, even with ICU treatment.

Management

If rabies is suspected, barrier nurse the patient in a quiet room with a minimum of staff who must wear gowns, gloves, eye protection, and masks. Obtain advice immediately from a specialist in infectious diseases. Anticipate press enquiries. Record the names of all staff involved, so that they can be offered rabies immunization.

Viral haemorrhagic fevers[ND]

Lassa fever

Lassa fever occurs in many rural parts of West Africa. It is a viral infection acquired from infected blood or secretions, transmitted by inadvertent innoculation (eg needlestick injuries) or contamination of mucous membranes or broken skin. In Africa, it is transmitted by multimammate rats. The incubation period is up to 3 weeks. There is high mortality.

Early symptoms Are non-specific with fever, malaise, headache, sore throat, retrosternal chest pain, and backache. Periorbital oedema, swelling of the neck, and conjunctival injection are common. Suspect Lassa fever in any pyrexial patient who has been in rural West Africa (south of the Sahara) in the previous 3 weeks. However, malaria and typhoid are much more common and need urgent diagnosis and treatment.

Management If Lassa fever is possible, barrier nurse the patient in a cubicle by staff wearing gloves, gowns, goggles, and masks. Take special care to avoid needlestick injuries, which may cause fatal infection. Before taking any blood samples, discuss the case with a tropical diseases specialist and the local consultant in communicable disease control. Start treatment immediately for falciparum malaria (see ➲ Malaria[ND], p. 255). Warn the laboratory about Lassa fever and send blood for examination for malaria. The patient will be admitted to an isolation bed, possibly in a high-security infectious diseases unit.

Ebola fever and Marburg fever

These are VHFs which occur in West and Central Africa (Democratic Republic of the Congo, Uganda, Kenya, and Sudan) and have similar clinical features and high mortality. Transmission is usually by infected blood, but the viruses may be acquired from monkeys or apes. The incubation period is usually 4–10 days. Illness starts suddenly with severe headache, high fever, and generalized pains, especially in the back, followed by severe diarrhoea, abdominal pain, dry throat, a maculopapular rash, conjunctivitis, and GI bleeding. Isolate and treat as for suspected Lassa fever.

Other viral haemorrhagic fevers

Diseases with similar features (plus, in some cases, jaundice) include dengue (see ➲ Dengue, p. 256), Crimean–Congo fever (Central Africa, parts of Eastern Europe, and Asia), and yellow fever (Africa and South America). The initial management is the same as for Lassa fever.

Middle East respiratory syndrome

Due to a coronavirus, this is also known as 'MERS' or 'MERS-CoV' which was first identified in 2012. It is a severe respiratory illness (mortality is >30%) which presents with fever, cough, and breathlessness. Consider it in patients with respiratory symptoms who have returned from Saudi Arabia and neighbouring areas. Obtain specialist and ICU help. Treatment is supportive, and at the present time, the risk of human-to-human transmission appears to be relatively low.

Severe acute respiratory syndrome

Background

Severe acute respiratory syndrome (SARS) is a viral respiratory illness caused by a coronavirus. SARS was first recognized in March 2003 but probably originated in November 2002 in the Guangdong province of China where the virus has been found in wild animals. SARS spread to several countries, causing deaths in South East Asia and Canada in March to May 2003. Few cases have occurred since then. No cases are known at the time of writing, but there is concern that SARS may re-emerge from China.

Spread

SARS is spread by respiratory droplets produced when an infected person coughs, sneezes, or uses a nebulizer. The virus can also spread when someone touches an object contaminated by infectious droplets and then touches his/her mouth, nose, or eyes.

Features

The incubation period of SARS is usually 2–7 days but may be up to 10 days. The illness starts with fever (>38°C), usually associated with rigors, headache, muscle pains, and malaise. Diarrhoea may occur. Some patients have mild respiratory symptoms initially. A dry cough develops after 2–7 days, with ↑ breathlessness from hypoxia caused by pneumonia. CXR may be normal or may show patchy infiltrates, and later areas of consolidation. WCC is usually normal or ↓ initially (lymphopenia).

Management

If SARS is suspected, get expert help (ED consultant, infectious diseases specialist, and infection control staff) and isolate the patient (if possible, in a negative-pressure room). Ensure that a minimum number of staff have contact with the patient. Staff who do have contact must wear masks or respirators (of FFP3 standard), goggles, gowns, and gloves, with strict handwashing and careful disposal of all items. Provide the patient with an N95 mask or a surgical mask. Record SpO_2 and give O_2 if necessary, but avoid flow rates of >6L/min to minimize virus aerosolization. If bronchodilators are needed, use a spacer inhaler, rather than a nebulizer. Maintain a list of all contacts. Expect press enquiries.

An expert will help to assess to decide about admission. Those admitted should ideally be placed in a negative-pressure isolation room with full infection control measures. Treat as for community-acquired pneumonia (see ➲ Pneumonia, pp. 114–15).

Further information about SARS is available at 🖱 https://www.hse.gov.uk

COVID-19

This section has been written whilst current knowledge of this illness is at an early stage. Refer to latest guidance (https://www.gov.uk).

Background, origin, and spread

In 2019, illness from a coronavirus (SARS-CoV-2) emerged from Wuhan in China and spread throughout the world. It was quickly apparent that it was a relatively mild illness for most, but a small proportion developed ARDS requiring IPPV, with an estimated 2% mortality rate. The initial response to this coronavirus disease 2019 (COVID-19) varied considerably between countries, in terms of testing, contact tracing, availability of ventilators, and PPE for health care workers. Within 6 weeks of a senior politician telling the public that the 'US has contained the virus', that country had the highest number of deaths in the world and the illness had been declared a pandemic.

Transmission and incubation period

COVID-19 is spread principally by close contact between individuals, especially by respiratory droplets (coughing, sneezing, breathing), some of which may contaminate surfaces. The incubation period is 2–14 days, typically 5 days, with individuals believed to be infectious before displaying symptoms.

Clinical features

In March 2020, an ED consultant in Cornwall, UK reported: 'An initial feeling of headache, intense tiredness and cold, progressive over several hours, gave way to fever, shivering, and myalgia.' He also described 'some tight feelings in my chest and loss of sense of smell'. He recovered completely within 10 days.

The main symptoms are fever, persistent cough, and breathlessness. Some patients also experience headache, myalgia, chest tightness, GI disturbance, and loss of sense of smell. There are no distinctive features on examination, even in those with CXR abnormalities. Patients who become seriously ill typically deteriorate 7–10 days after onset of symptoms. They may have hypoxia on minimal exertion, a high NEWS2, and features of ARDS.

High risk factors

The mortality rate is much higher for older individuals, especially men and those with pre-existing health problems (including immunosuppression, diabetes, hypertension, cardiovascular and respiratory disease). Healthy children and young adults may still require hospital treatment, including IPPV, but are at relatively low risk of death. Of relevance to health care workers, there is evidence that being exposed initially to a large amount of virus may predispose to more severe illness.

Management

- On initial suspicion, isolate the patient and put a mask on him/her; protect staff and other patients: don appropriate PPE ('full PPE' including FFP3 respirator, visor, long-sleeved gown and gloves for high risk procedures such as airway management/intubation, suctioning, NIV).
- Consider early discharge with advice for patients not seriously ill who have NEWS2 of <3 and no hypoxia on minimal exertion. High risk patients seen early (in the first 7 days) may benefit from community follow up at 7–10 days to check for deterioration.
- Take blood for FBC, U&E, CRP, D-dimer, LFTs, coagulation, ferritin, procalcitonin, troponin, cultures.
- Take upper respiratory swabs for COVID-19.
- Consider imaging, depending upon local resources. CXR may show peripheral and basal ground glass opacities in patients predisposed to ARDS. USS correlates well with CXR (and CT) findings.
- Provide O_2 as required: target SpO_2 94–98% (88–92% in COPD).
- Give paracetamol and oral fluids. Avoid IV fluids in ARDS. If needed, start with 250mL IV 0.9% saline bolus.
- Consider alternative diagnoses (eg bacterial pneumonia) and treat accordingly.
- Refer seriously unwell patients early to ICU to consider intubation and IPPV.
- Decide early about the ceiling of care/treatment escalation plan.
- Do not start CPR (or other aerosol-generating procedures) unless all staff are wearing PPE.

Health care workers

Tragically, many health care workers have already died as a result of treating patients with COVID-19 (see dedication). Strict adherence to correct use (including donning and doffing) of PPE helps reduce risks.

Community strategy

Measures to prevent spread of COVID-19 in the community have included social distancing, isolation, testing, contact tracing, shutdown of schools, restaurants, and businesses, with dramatic social and economic consequences.

Future treatments

There is hope that in the short term, antiviral and antibody treatments will reduce the mortality rate. Longer term hopes are focussed on a vaccine.

Influenza pandemics, avian flu, and swine flu

Background

Influenza is common in the UK and many other countries, particularly during winter. Most people are ill for only a few days with fever, muscle aches, coughing, and nausea, but there are some deaths, especially in elderly people.

Pandemic influenza occurs when a new subtype of influenza A emerges, which can spread easily from person to person and which is different from previous strains (so there is no pre-existing immunity). Influenza pandemics occurred in 1918–1919 (with 40–50 million deaths worldwide, including many children and young adults) and also in 1957 and 1968. Another pandemic could develop at any time. There was concern about influenza A subtype H5N1, which infected poultry in Hong Kong in 1997 and 2003 and spread to birds across South East Asia, with carriage by migrating birds across Asia and to Europe and Africa. This avian flu infected many millions of birds and some people in South East Asia and Turkey who had been in close contact with infected chickens. Mortality in these cases was high. In 2009, influenza A subtype H1N1 caused a pandemic of swine flu which started in Mexico and spread to many other countries. Most patients with swine flu had only mild illness, but a minority developed severe infection, and some died.

Human-to-human spread of H1N1 or H5N1 flu is rare at the time of writing, but another pandemic could develop if the virus mutates again.

Spread

Like SARS (see ➲ Severe acute respiratory syndrome, p. 259), flu is spread by droplets coughed or sneezed into the air, or by direct contact with hands contaminated with the virus.

Features

Consider the possibility of avian flu or swine flu in a patient with fever of ≥38°C and cough or breathlessness, who, in the last 7 days, has been in an area affected by H1N1 or H5N1 influenza. Laboratory staff and health care workers in contact with cases of severe unexplained respiratory illness could also be at risk.

Management

Isolate the patient and treat with precautions against transmission of the virus, as for SARS (see ➲ Severe acute respiratory syndrome, p. 259). Antiviral treatment with oseltamivir or zanamivir may be considered, depending on current guidelines.

Clinical guidelines about the assessment of suspected cases and the management of influenza patients are updated as the situation changes and if another pandemic develops.

Chapter 6

Environmental emergencies

Hypothermia: presentation

Definitions

Hypothermia is defined as a core T° of <35°C.

Hypothermia may be classified as in Table 6.1.

Table 6.1 Classification of hypothermia

Mild hypothermia	32–35°C
Moderate hypothermia	30–32°C
Severe hypothermia	<30°C

Background

Hypothermia in young adults often reflects environmental exposure (eg hill-walking or cold water immersion) or immobility and incapacity from alcohol and/or drugs. The elderly more typically become hypothermic indoors; common factors include unsatisfactory housing, poverty, immobility, lack of cold awareness (autonomic neuropathy, dementia), drugs (sedatives, antidepressants), alcohol, acute confusion, hypothyroidism, and infection.

Clinical features

Severe hypothermia can mimic death. As core T° ↓, cerebral and cardiovascular function deteriorate. At 32–35°C, apathy, amnesia, ataxia, and dysarthria are common. At <32°C, consciousness falls progressively, leading to coma, ↓ BP, arrhythmias (check pulse for at least 1min before diagnosing cardiac arrest), respiratory depression, and muscular rigidity. Shivering is an unreliable sign. VF may occur spontaneously when T° falls <28°C and may be provoked by limb movement or invasive procedures (especially in the presence of hypoxia).

Diagnosis

Check tympanic T° (or rectal T° with an electronic probe or low-reading thermometer). Tympanic and rectal T° may lag behind core (cardiac) T° during rewarming. Oesophageal T° reflects core levels more accurately but requires special equipment.

Investigations

- U&E, FBC, toxicology (including alcohol level), and clotting screens. *Note*: hypothermia can cause or aggravate coagulation disturbances.
- Blood glucose (BMG reading may be falsely ↓).
- Amylase or lipase (↑ levels are common but do not necessarily imply pancreatitis).
- Blood cultures and ABG.
- ECG: look for prolongation of elements in the PQRST complex, J waves (also called Osborn waves), and arrhythmias (AF and bradycardias are the most common) (see Fig. 6.1).
- CXR: look for pneumonia, aspiration, and LVF. Consider other X-rays after rewarming (eg suspected fractured hip).
- Consider CT scan if underlying head injury or stroke is suspected.

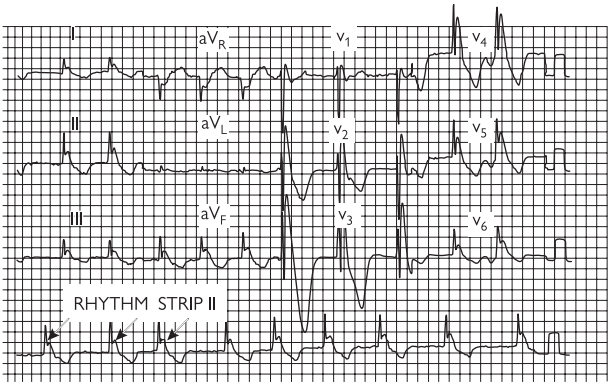

Fig. 6.1 ECG in hypothermia.

Notes on the ECG shown in Fig. 6.1

- Rhythm disturbance: AF with slow ventricular response.
- Prolongation of QRS.
- Delayed repolarization 'J waves' (arrowed).
- ST–T wave abnormalities.

Hypothermia: management

Principles

- Treat in a warm room (>21°C), remove wet clothes, and dry the skin.
- Handle the patient gently and place on a cardiac monitor.
- Give warmed, humidified O_2 by mask.
- Intubation, if needed, should be preceded by oxygenation.
- Secure IV access. IV fluid is rarely required unless there is loss of volume from another cause. If BP ↓ during rewarming, give 300–500mL of warmed 0.9% saline or colloid. In unstable patients, consider CVP and urinary catheter. Warm IV fluid administration is an inefficient rewarming method and runs the risk of fluid overload and precipitating arrhythmias.
- Correct hypoglycaemia with IV glucose.
- In cardiac arrest, give CPR at standard rates. Note that the heart may be unresponsive to defibrillation, pacing, and drug therapy. Drug metabolism is ↓ and unpredictable—avoid drugs until core T° >30°C.
- Defibrillation is appropriate at normal energy levels if VF/VT occurs. If three shocks are unsuccessful, defer further shocks until core T° >30°C.

Rewarming methods

Choice depends on the severity and duration of the condition, available facilities, and the individual patient.

Passive rewarming Easy, non-invasive, and suitable for mild cases (T° >32°C). ↓ evaporative and conductive losses by wrapping in warm blankets (remember to cover the back and sides of the head) ± polythene sheets. Avoid space blankets which are noisy and have no advantages over polythene sheets. Endogenous metabolism and shivering usually generate enough heat to allow spontaneous rewarming. Aim for a rate of 0.5–2°C/hr, but do not rewarm the elderly with prolonged hypothermia too rapidly (>0.6°C/hr), as hypotension or cerebral/pulmonary oedema may develop.

Active rewarming A water bath at ~41°C is rapid and useful for immersion hypothermia but cannot be used in injured patients or if CPR is required. Airway care, ventilation, and monitoring are difficult, and hazards include core T° afterdrop and BP ↓ due to peripheral vasodilatation. Hot water bottles and heat pads are less efficient and can cause burns. A hot air blanket is more convenient than a water bath, provides some heat, and reduces heat loss.

Core rewarming

- Airway warming with heated (40–45°C) humidified O_2 provides some additional heat and reduces heat loss. It can be combined with other rewarming methods. It may reduce the risk of cardiac arrhythmias.
- Consider peritoneal lavage—run in saline at 45°C via a catheter; leave for 10–20min, then replace with a fresh warm supply. The fluid directly heats the liver and blood in the IVC. Other options are warm irrigation of the pleural cavity, stomach, or bladder.

Extracorporeal rewarming with cardiopulmonary bypass maintains brain and organ perfusion and, if available, is the method of choice in patients with severe hypothermia or cardiac arrest. Cardiopulmonary bypass can result in rapid rewarming, with core T° ↑ at 1−2°C/5min.

Frostbite and non-freezing cold injury

Frostbite

Frostbite[1] occurs when tissues freeze at sub-zero temperatures. Predisposing factors include inadequate clothing/footwear, hypothermia, exhaustion, alcohol (which impairs judgement), drugs (eg β-blockers), peripheral vascular disease, smoking, and previous cold injury. Frostbite usually involves extremities, especially the fingers, toes, nose, and ears.

Frostnip May precede frostbite. The skin of the nose, face, or fingers goes white and numb but recovers rapidly on protection from the cold, with transient paraesthesiae, but no tissue loss and no permanent damage.

Superficial frostbite Involves the skin and subcutaneous tissues. The frozen area is numb and looks white and waxy. Tissues feel firm or hard but are still pliable. Rewarming is painful. Oedematous hyperaemic skin becomes mottled or purple, with serum-filled blisters. A hard black eschar forms, and after ~3 weeks, this separates, revealing sensitive red, shiny skin.

Deep frostbite Involves muscles, nerves, and sometimes bone, as well as the skin and superficial tissues. The damaged area is hard and remains grey or white after rewarming. Blood-filled blisters develop. The dead tissue mummifies and then separates after several weeks or months.

Treatment of frostbite Varies with the situation and facilities. Only frostnip should be treated in the field. Frostbitten tissues need rewarming as soon as possible, but further damage from refreezing needs to be avoided. Treat hypothermia before frostbite. Rewarm frostbitten limbs in water at 37–39°C until skin circulation returns (usually ~30min). Give analgesia and ibuprofen (which inhibits prostaglandins). After rewarming, let the area dry in warm air (do not towel dry). Elevate the limb. Expose the area, with a bed cradle to avoid pressure of bedclothes. Clean the area daily in a whirlpool bath, and encourage movement. If necessary, split eschar to relieve stiffness, but avoid surgical debridement and amputations and allow the eschar to separate spontaneously; premature surgery causes avoidable tissue loss. Expert advice is helpful in severe frostbite—the British Mountaineering Council (https://www.thebmc.co.uk) has a frostbite advice service. Bone scans or MRI/magnetic angiography (MRA) may help to define deep tissue injury. In severe frostbite, early thrombolysis with tPA may reduce the risk of eventual amputations.

Non-freezing cold injury[1]

Trench foot (immersion foot) Caused by prolonged immersion in cold water or wet boots at temperatures just above freezing. Vasoconstriction causes tissue ischaemia and nerve damage. The feet are initially cold, numb, and pale or mottled. On rewarming, they become red, swollen, and very painful. Blisters may develop.

Treatment Keep the feet clean, warm, dry, and elevated to reduce oedema.

Outcome Most patients recover fully, but some have continued pain, paraesthesiae, and sensitivity to cold.

1 *State of Alaska Cold Injury Guidelines, 2014*. Available at: http://dhss.alaska.gov/dph/Emergency/Documents/ems

Drowning and near drowning

Definitions

Drowning Death by suffocation from submersion in any liquid. Drowning is a common cause of death in young people; 40% of drownings occur in children aged <4y.

Near drowning Survival (at least temporarily). In adults, the most common predisposing factor is alcohol, sometimes with other drugs. A significant proportion reflect attempted suicide. In the UK, marine near drowning is usually associated with hypothermia (see ➲ Hypothermia: presentation, pp. 264–5).

Pathophysiology

Wet drowning Involves significant aspiration of fluid into the lungs. This causes pulmonary vasoconstriction and hypertension with V/Q mismatch, aggravated by surfactant destruction and washout, ↓ lung compliance, and atelectasis. Acute respiratory failure is common. ABG shows hypoxia, hypercarbia, and mixed respiratory/metabolic acidosis. The onset of symptoms can occur rapidly, but in lesser insults, symptoms may be delayed.

Contamination Water contaminated with chemical waste, detergents, etc. may induce further lung injury.

Electrolytes Irrespective of whether aspirated water is salt water, fresh water, or swimming pool water, changes in serum electrolytes and blood volume are similar and rarely immediately life-threatening.

Gastric fluid Swallowing of fluid into the stomach, with gastric dilatation, vomiting, and aspiration, is common.

Dry drowning In ~10–20% of deaths from drowning, a small amount of water entering the larynx causes persistent laryngospasm, which results in asphyxia and an immediate outpouring of thick mucus, froth, and foam, but without significant aspiration—this is 'dry drowning'.

Secondary drowning Deterioration in a previously apparently well patient, following successful resuscitation after submersion. It may occur in 5–10% of initial survivors.

The mammalian diving reflex

This is probably seen only in young children but may explain why successful resuscitation without neurological deficit can occur after prolonged immersion. Cold water stimulates facial nerve afferents, whilst hypoxia stimulates the carotid body chemoreceptors. These effects reflexively ↓ the heart rate and vasoconstrict skin, GI tract, and skeletal muscle vessels, redistributing blood to the brain and heart. Associated hypothermia results in ↓ metabolic demands, delaying cerebral hypoxia.

Management

- Consider associated injury (eg to the cervical spine from diving into a shallow pool or surfing), and treat appropriately.
- Maintain the airway. Remove regurgitated fluid/debris by suction of the upper airway. Ensure adequate ventilation and correction of hypoxia. If the patient does not have a gag reflex or is apnoeic, ventilate with a bag and mask and proceed to early tracheal intubation and IPPV. In spontaneously breathing patients, give the highest FiO_2 possible. IPPV will be required if hypoxia and/or hypercapnia are present despite O_2 therapy or if there are signs of pulmonary oedema. Ventilation with positive end-expiratory pressure (PEEP) may significantly improve oxygenation by ↑ functional residual capacity, improving V/Q mismatch and enhancing fluid resorption from the pulmonary bed. However, PEEP may ↓ venous return to the heart.
- If the patient is in cardiac arrest, commence CPR (see ➲ Cardiac arrest, p. 48). Defibrillation may not be successful until core T° is >30°C (see ➲ Hypothermia: management, pp. 264–5). Appropriate rapid core rewarming techniques are required.
- Remove all wet/cold clothing.
- Monitor core T° and start rewarming (see ➲ Hypothermia: management, pp. 264–5).
- NG tube to relieve gastric dilatation.
- Check U&E, blood glucose, ABG/VBG, FBC, and ECG.
- Obtain CXR if symptomatic.
- Consider the possibility of alcohol, illegal drugs, or drug overdose. Keep urine and blood samples and test if appropriate, eg paracetamol.
- Do not use 'prophylactic' steroids or barbiturates.
- Antibiotics may be warranted if contaminated water (eg sewage) is involved (see ➲ Leptospirosis (Weil's disease), p. 249).
- Inhalation of mud/sand, etc. may require bronchoscopy for clearance.

Outcome

Resuscitation without cerebral deficit is possible after prolonged submersion (even after >60min), particularly if associated with hypothermia. 50% of children recovered apparently lifeless will survive, and even adults with a GCS of 3–4 out of 15 and fixed dilated pupils can survive unimpaired.

Respiratory effort is a sensitive prognostic sign, but in hypothermic patients, its absence does not necessarily imply poor outcome. Note the time to the first spontaneous inspiratory gasp.

Poor prognostic factors Include extremes of age, severe acidosis, immersion for >5min, and coma on admission.

Good prognostic factors Include patients who are alert on admission, hypothermia, older children/adults, brief submersion time, and those who receive rapid on-scene BLS and respond to initial resuscitation measures.

Asymptomatic patients Those who have no abnormality on repeated clinical examination, ABG, and CXR require observation for at least 4–6hr prior to considering discharge. Admit all others to ICU or a general ward as appropriate.

Diving emergencies: 1

Consider any symptom developing within 48hr of a dive as related to the dive until proven otherwise. On suspicion of a diving-related episode, seek specialist advice urgently (see Table 6.2).

Diving-related emergencies fall into four main categories: drowning (see ➲ Drowning and near drowning, pp. 268–9), barotrauma, decompression illness, and marine bites or stings (see ➲ Specific bites and stings, pp. 422–3).

Barotrauma

May occur in any gas-containing body cavity during descent or ascent.

Descent barotrauma ('squeeze') results from compression of gas in enclosed spaces as the ambient pressure ↑. Commonly, the ears, sinuses, and skin are affected. Middle ear squeeze may be precipitated by Eustachian tube congestion and leads to erythema, haemorrhage, or tympanic membrane perforation with conductive hearing loss. Round or oval window rupture (inner ear squeeze) occurs with sudden pressure changes between the middle and inner ear and results in acute tinnitus, vertigo and deafness, and a perilymphatic fistula. ENT opinion is urgently required if a perilymphatic fistula is suspected and for cases of severe or continuing symptoms. If tympanic membrane rupture has not occurred, middle ear squeeze can usually be managed with decongestants/simple analgesics. If it has ruptured, give antibiotics (see ➲ Traumatic tympanic membrane rupture, p. 567). Instruct the patient not to dive until the symptoms have resolved and the drum has completely healed.

Sinus barotrauma has a similar aetiology to middle ear injury and is often associated with upper respiratory tract infection (URTI), mucosal polyps, and sinusitis. Treat similarly to ear barotrauma.

Divers who fail to exhale periodically via the nose into their face mask during descent may develop 'face mask squeeze' (skin barotrauma). Erythema, bruising, and petechial and conjunctival haemorrhages develop in the enclosed area. Skin tightly enclosed by parts of the diving suit can have similar appearances. Usually no treatment is required.

Ascent barotrauma is the reverse of squeeze and particularly affects the lungs. It may be caused by breath-holding during rapid uncontrolled ascent or by air trapping in patients with asthma or congenital lung bullae. Mediastinal emphysema is the most common event and presents with ↑ hoarseness, neck swelling, and retrosternal chest discomfort. Symptoms usually resolve spontaneously with high concentrations of O_2. Pneumothorax is a potentially life-threatening complication if it develops during the dive, as intrapleural gas cannot be vented and ↑ ascent will precipitate tension. Conventional treatment by needle decompression, aspiration, or chest drain insertion (see ➲ Chest drain insertion, p. 346) is required.

Dental pain may occur on ascent or descent barotrauma in carious teeth or those which have had recent fillings. The affected tooth is tender on tapping. Treat symptomatically with analgesics and arrange dental referral.

Table 6.2 Sources of advice on diving emergencies and hyperbaric chambers

England, Wales, Northern Ireland	
Diving Incident Telephone Advice Line, Institute of Naval Medicine, Gosport, Hampshire	Telephone 07831 151523 (24hr) Ask for the Duty Diving Medical Officer
Diving Diseases Research Centre, Plymouth http://www.ddrc.org	Telephone 01752 209999 (24hr) Ask for the Duty Diving Doctor
Scotland	
Hyperbaric Medicine Unit, Aberdeen Royal Infirmary	Telephone 0345 408 6008 State 'diving emergency'. Give your name and telephone number Ask for the Duty Hyperbaric Doctor
In the event of any difficulties in contacting these agencies in the UK, telephone 999 and ask for COASTGUARD	
Other countries	
Divers Alert Network (DAN) https://www.diversalertnetwork.org	DAN Diving Emergency Hotline (USA) +1 919 684 9111
This has links to diving emergency contact numbers throughout the world	

Further information

British Hyperbaric Association. Available at: http://www.hyperbaric.org.uk
Divers Alert Network. Available at: https://www.diversalertnetwork.org
Diving Diseases Research Centre (Hyperbaric Medical Centre, Plymouth, England). Available at: https://www.ddrc.org
Scottish Diving Medicine. Available at: https://www.sdm.scot.nhs.uk

Diving emergencies: 2

Decompression illness

There are two forms of decompression illness. The first occurs when dissolved nitrogen in blood and tissues is not expelled at a sufficient rate to prevent bubble formation. The second occurs when air bubbles are released into the circulation because of pulmonary barotrauma. This follows if air bubbles enter the pulmonary capillaries from ruptured alveoli. The bubbles travel via the left side of the heart to the systemic circulation. Cerebral air embolism usually causes symptoms as the diver surfaces, with loss of consciousness, fits, cardiovascular collapse, and chest pain. Clinically, differentiation between the two forms is difficult and initial management is the same. In general, the sooner the onset of symptoms, the greater the likely severity. Symptoms may be attributed by the patient (and the unwary doctor) to musculoskeletal sprains/strains or other minor injury.

Decompression illness is more likely in divers who have not followed safe ascent recommendations, the obese, in cold water, and when excessive exercise has occurred during the dive. It may be precipitated by air travel if insufficient time is left between diving and flying for residual nitrogen to leave the body in a controlled fashion. Bubbles have direct mechanical and local inflammatory effects, commonly involving the joints, skin, CNS, lungs, and ears.

Joint pain, 'the bends', most often affects the shoulders and elbows. A dull aching sensation, ↑ by movement, but without localized tenderness, is common. Pruritic rashes, local swelling, and a peau d'orange effect may occur. Back pain, limb weakness, sensory abnormalities, or urinary retention imply spinal cord involvement. Central effects include focal deficits, cerebellar disturbance, and mood changes.

Treatment for decompression illness Is recompression. If delayed, the risks of permanent damage to the brain and spinal cord greatly ↑. The diagnosis of decompression sickness may only follow the response to recompression. Pending this, give the highest possible concentration of O_2. Analgesics and sedatives can mask recompression responses and should only be used on specialist advice. Entonox® is absolutely contraindicated.

If intubation is required, inflate the ET tube cuff with sterile water, since during recompression, an air-filled cuff will deflate. IV fluids (0.9% saline or a plasma expander) assist oxygenation of ischaemic tissues and facilitate discharge of excess tissue nitrogen load into the venous system by ensuring adequate circulating volume. Some centres may recommend aspirin and/or dextran solutions to ↓ capillary sludging which accompanies severe decompression sickness.

Despite dry or wet suits, hypothermia is common. Treat with appropriate passive or active rewarming (see ➲ Hypothermia: management, p. 266).

Air evacuation If, after consultation with the diving medical centre, air evacuation is necessary, unpressurized aircraft should not fly above 300m. The diver should breathe 100% O_2. On reaching the diving centre, recompression to a simulated depth of 18m with 100% O_2 occurs, interspersed with periods of air breathing to ↓ O_2 toxicity risk. Slow decompression then follows standard treatment protocols.

Divers usually dive in pairs. If a diver has symptoms of decompression sickness or pulmonary barotrauma, his 'buddy' will be at risk also. Although recompression may not be required in the buddy, transfer him/her, along with the affected diver and their diving equipment, to the recompression facility.

Obtain the following information before referral, if possible

- The patient's current condition, progression since onset, and response to treatment.
- Time of onset of symptoms related to the dive.
- Dive profile and history (depth, duration, activity during the dive, speed of ascent including details of any stoppages, environmental conditions (water temperature, currents, etc.), pre-dive exercise, alcohol, drugs and food, type and condition of diving equipment used, clothing worn, other recent dives). Many divers store much of this information in a dive computer.
- Previous medical history, previous diving-related episodes, and drug history.

Heat illness

Body T° is normally kept at 36–38°C by the hypothalamus. Hyperthermia occurs when homeostatic mechanisms are overwhelmed by factors acting individually or (commonly) together. This can occur even in temperate climates. At-risk groups include the young and the elderly in conditions of ↑ T° and humidity, patients with unaccustomed or prolonged muscular activity (eg at 'raves', associated with ecstasy or other drugs), grand mal fitting, athletes, marathon runners, and armed forces recruits.

Predisposing medical factors

- Alcohol use or withdrawal (including delirium tremens).
- Cardiac disease.
- Any condition which may cause or aggravate Na^+/water (H_2O) loss (eg gastroenteritis, cystic fibrosis).
- Drugs, including: alcohol, diuretics, salicylates, anticholinergics (antihistamines, tricyclic antidepressants), sympathomimetics (amphetamines, ecstasy, LSD, cocaine, phencyclidine, appetite suppressants), phenothiazines, antipsychotics, MAOIs, and SSRIs (see Serotonin syndrome, p. 224).

Heat illness has a spectrum of severity

Heat cramps ⇔ Heat exhaustion ⇔ Heat stroke.

In *heat cramps/exhaustion*, homeostatic mechanisms still function but are overwhelmed.

In *heat stroke*, all thermoregulatory control is lost and body T° ↑ rapidly to very high levels (>41°C), causing widespread severe tissue and organ damage. Mortality ~10%.

Heat cramps

Core T° of 37–39°C. Mental function is normal. Sweating during exercise and replacement with hypotonic fluid lead to Na^+ deficiency. Brief cramps occur in muscles used in heavy work, usually after exertion.

Heat exhaustion

Core T° <40°C. Mental function is normal. Characterized by mixed Na^+/H_2O depletion. Sweating and tachycardia are usually present. Symptoms of weakness, fatigue, headache, vertigo, nausea and vomiting, postural dizziness, and syncope. Patients will recover with rest and fluids.

In mild cases, remove from heat and use simple cooling techniques. Rehydrate with oral electrolyte solutions.

More severe cases require IV 0.9% saline or 0.45% saline/5% glucose. Use clinical assessment, U&E, and Hct to guide infusion rate. Up to 4L of fluid may be required over 6–12hr. Avoid over-rapid infusion which may cause pulmonary and/or cerebral oedema.

Measurement of core temperature

Tympanic or rectal T° measurement is appropriate in the ED but may underestimate core T° and respond slowly as this changes. Oesophageal and intravascular probes give the most accurate readings of core T° but require special equipment.

Heat stroke

Suspect in collapse during or after exercise and in high-risk groups. Core T° is >41°C (but significant cooling can occur before arrival in the ED). Outcome depends upon the height and duration of ↑ T°.

- *CNS*: oedema + petechial haemorrhages cause focal/generalized damage.
- *Muscle injury*: releases enzymes, myoglobin, urate, K^+, PO_4^-.
- *Liver*: jaundice commonly develops after 24hr.
- *Kidneys*: acute renal failure (ARF) from hypovolaemia, muscle breakdown products, acidosis, and DIC.
- *Blood*: DIC, thrombocytopenia, leucocytosis.
- *Metabolic*: ↑ or ↓ K^+, metabolic acidosis, respiratory alkalosis, hypoglycaemia.

Features

Sweating may be present, but the skin surface may feel deceptively cool due to peripheral vasoconstriction.

- *CNS*: confusion, delirium, fitting, coma, oculogyric crisis, dilated pupils, tremor, muscle rigidity, decerebrate posturing, cerebellar dysfunction.
- *CVS*: tachycardia, hypotension, arrhythmias.
- *Coagulopathy*: purpura, conjunctival haemorrhages, melaena, haematuria.

Investigations

- ABG, U&E, BMG, CK, clotting screen, LFTs, urate, Ca^{2+}, PO_4^-, ECG, CXR.

Treatment

- Treat immediately and involve ICU staff. Remove all clothing.
- Secure the airway (intubation/IPPV if needed). Give O_2 as needed.
- Cooling techniques depend upon facilities available and the clinical state of the patient. Do not give 'antipyretics' such as aspirin/paracetamol. Evaporative cooling is the most efficient and applicable treatment. Spray the naked patient with tepid tap water and blow air with fans. Ice-packs can be applied to the axillae, groins, neck, and scalp (but avoid prolonged contact). Consider cold gastric or peritoneal lavage, or cardiopulmonary bypass if these techniques fail. Aim to cool ≥0.1°C/min. Stop active cooling when core T° is <39°C.
- *IV fluids*: give 50mL of 10% glucose IV if BMG is <3mmol/L. Severe hypovolaemia is uncommon, but if hypotension persists despite ↓ T°, give IV 0.9% saline (1–1.5L over 1–2hr). Avoid overloading the circulation with a risk of pulmonary/cerebral oedema. CVP monitoring may be needed. CVP may be initially ↑ due to peripheral vasoconstriction.
- Insert a urinary catheter. If myoglobinuria is present, aim for ↑ urine output and consider giving IV bicarbonate and/or mannitol.
- If fits occur, give IV lorazepam—but beware of respiratory depression.

Neuroleptic malignant syndrome An idiosyncratic reaction in patients on anti-psychotics (especially haloperidol, thioridazine, chlorpromazine). Features are muscle rigidity, extrapyramidal signs, autonomic dysfunction, and severe dyskinesia. Stop the antipsychotic, cool the patient, and give dantrolene.

Malignant hyperpyrexia A rare autosomal dominant condition related to use of suxamethonium and volatile anaesthetics. Dantrolene prevents Ca^{2+} release from skeletal muscle and is very effective—the initial dose is 2–3mg/kg IV, then give 1mg/kg as needed (max total dose 10mg/kg) (see https://www.aagbi.org).

Electrical injuries

Electric shocks can cause cardiac and respiratory arrest. The heart often restarts spontaneously, but respiratory arrest may be prolonged, causing fatal hypoxia. The electric current may produce burns and muscle damage. Spasms from a shock may result in dislocations or fractures or precipitate a fall causing major trauma. Fatal electrocution can occur from domestic electricity (in the UK, 230V, alternating current at 50 cycles/s), but severe injury is more common with high-voltage shocks (>1000V).

Lightning causes a DC shock at a very high voltage (up to 100,000,000V), but with a short duration (0.1–1ms).

Electrical flash and arc burns

An electrical short-circuit near to a person may cause sudden vaporization of metal and deposit a thin layer of hot metal on the skin, without any electricity passing through the casualty. Electrical flash burns may look dramatic because of skin discoloration but are often superficial and heal well. In contrast, electrical arcing produces high temperatures and may cause deep dermal or full-thickness burns, especially if clothing is set alight.

Contact burns

If electricity has passed through the patient, there are usually two or more entry or exit wounds, comprising full-thickness burns with white or charred edges. Tissue damage is more extensive than the visible burns, especially with high-voltage injuries. Deeper layers of skeletal muscle may be involved and muscle damage can cause myoglobinuria and renal failure. Myonecrosis and oedema of muscles may produce compartment syndrome (see ➲ Crush syndrome, pp. 406–7).

If current passes through the torso (especially from arm to arm), cardiac arrhythmias are more likely than if only a single limb is involved. Myocardial damage may occur, often in association with vascular injuries.

Neurological effects of electric shocks include coma, fits, headaches, transient paralysis, peripheral neuropathy, and mood disturbances.

Ophthalmic injuries are common after electrical burns of the head. Cataracts and glaucoma may develop later.

Electrocution in pregnancy carries risks to the fetus (spontaneous abortion may occur). Obtain obstetric advice.

Lightning

Sudden vaporization of sweat and rainwater caused by lightning may explode clothes and shoes off the victim and rupture ear drums. Lightning burns are superficial, often with a characteristic feathered or fern-like appearance (Lichtenberg figures). The limbs are often cold and mottled due to arterial spasm, which usually resolves over a few hours. Deep muscle damage and myoglobinuria are rare. Coma may result from direct brain injury, head injury due to a fall, or cardiac arrest. CPR, if indicated, may be successful, even if required for prolonged periods. Survivors may be confused and amnesic for several days and may have fits and temporary paralysis. Cataracts are common.

Management

- At the scene, make sure that the current is turned off before anyone approaches or touches the casualty. Remember that high-voltage electricity can arc through the air or pass through the ground.
- Check the airway, breathing, and circulation. Electrical burns of the mouth and throat may cause oedema and airway obstruction.
- Perform CPR as necessary, remembering that a good outcome may follow prolonged resuscitation. Minimize movement of the spine in case there has been spinal trauma.
- Examine thoroughly for head, chest, abdominal, and skeletal injuries.
- Examine all over for skin entry/exit burns, and check pulses and sensation.
- Check the ECG—there may be arrhythmias (eg AF), conduction defects, ST elevation, and T wave changes—if present, place on a cardiac monitor.
- Test the urine for blood (except in an asymptomatic young healthy person who has suffered a domestic shock, when it is unnecessary). If the stick test is +ve for blood, but there are no RBCs on microscopy, treat for myoglobinuria to prevent AKI—obtain specialist advice; maintain a high urine output, and consider using mannitol ± isotonic sodium bicarbonate.
- Except in minor low-voltage (domestic) electrical injury with no associated worrying features (such as burns, ECG abnormalities, hypotension, reduced conscious level), check FBC, U&E, and CK.
- If there is a reduced conscious level or there are focal neurological abnormalities, request a CT brain scan.
- Significant electrical injuries may cause fluid loss into muscle, resulting in hypovolaemia—if there are significant burns or soft tissue damage, treat with IV fluid (start with 1000mL of 0.9% saline).
- High-voltage injuries with associated burns and tissue damage may require widespread fasciotomies—involve surgical experts to help with excision or amputation of non-viable tissues and inspection and further debridement after 48hr.

Admission or discharge

Allow home asymptomatic patients with domestic and minor low-voltage burns who have a normal ECG, no history suggestive of arrhythmia (eg palpitations), no pre-existing history of cardiac problems, no significant skin burns, and no myoglobinuria, but advise review if any problem develops. Note that late-onset arrhythmias are very unlikely.

Admit children who bite electric flexes for observation, because of the risk of delayed bleeding from labial blood vessels.

Admit for observation (± treatment) all patients with high-voltage conduction injuries (including lightning) and those with cardiac arrhythmias, chest pain or ECG abnormalities, vascular injury, significant skin burns, or myoglobinuria.

Radiation incidents

In the UK, 24hr advice and assistance is available via NAIR (National Arrangements for Incidents involving Radioactivity) by telephone (0800 834153) or via the police. Try to distinguish between external irradiation of a person and contamination with radioactive material. Someone exposed to X-rays or to gamma rays in a radiation sterilizing unit receives no further radiation after removal from the source, and there is no risk of contaminating anyone else. However, a person contaminated with radioactive material is still exposed to radiation and needs urgent, careful decontamination to minimize the risks to himself and other people. Some hospitals are officially designated for the care of casualties contaminated with radioactive substances, but in an emergency, a patient may be taken to any ED where a plan for such events should exist.

Anticipation of a radiation incident

- Inform the ED consultant immediately if a patient from a radiation incident arrives or is expected.
- Get advice and help from a radiation physicist (from the medical physics or radiotherapy department).
- Implement the appropriate Radiation Incident Plan to deal with the patient.
- Expect media enquiries.

Treatment of contaminated casualties

Where possible, treatment should take place in a designated decontamination room. This room should have a separate entrance, ventilation arrangements, decontamination facilities with a shower, and contaminated water collection facilities. Cover the floor of this room and entrance/exit corridors with disposable sheeting. All staff must themselves be decontaminated and checked before leaving this area.

- Turn off air conditioning.
- Pregnant and potentially pregnant staff should not be involved.
- Provide any necessary life-saving treatment, but avoid spreading contamination.
- 'Barrier nurse', as for an infectious disease.
- Assume patients are contaminated until they have been checked by the radiation physicist.
- Instruct patients and staff not to eat, drink, or smoke.
- Involve a minimum number of staff who should wear face masks, theatre clothing with impermeable gowns or plastic aprons, two pairs of gloves, and overshoes or rubber boots.
- Restrict and record the movements of people in and out of the room.
- Ensure that the ambulance crew waits for monitoring of themselves and their vehicle.
- Keep everything that may be contaminated for radiation testing.
- Collect the patient's clothes, dressings, swabs, and any equipment used in plastic bags, and keep them in the decontamination room.
- All blood/urine samples must be specially labelled and the laboratories informed of the radiation risk.
- Life-threatening injuries may take precedence over all of the above, such that patients may need to be managed in the resuscitation room.

Decontamination of the patient

The radiation physicist should determine the sites of contamination and monitor the effectiveness of treatment. The object is to remove any contaminating substance and minimize absorption into the body, especially via the mouth, nose, and wounds.

- Cover any wounds prior to decontamination.
- Avoid splashing.
- Radioactive material can usually be removed from intact skin by washing with soap and water. Gentle scrubbing may be needed, but it is important to avoid damaging the skin. Carefully clean wounds and irrigate with water or saline.
- Clean the mouth using a mouthwash and a soft toothbrush, with care to avoid swallowing any fluid.
- Instruct the patient to blow their nose into paper handkerchiefs. If the nose is still contaminated, irrigate it with small amounts of water.
- Irrigate each eye from the medial side outwards to avoid draining contaminated water into the nasolacrimal duct.
- Clean the hair by washing with shampoo and by clipping if contamination persists, but do not shave the scalp.
- If monitoring shows that all contamination has been removed, treat the patient as for an irradiated, but uncontaminated, patient. However, if contamination persists or if radioactive material has been ingested or inhaled, further treatment will be needed after discussion with a radiation specialist.
- Check all staff involved in treating the patient for radioactive contamination before they leave the treatment area.

The irradiated patient

A patient who has been irradiated or contaminated with radiation may be at risk of radiation sickness or other ill effects. Admit to a designated unit for assessment and follow-up by a radiotherapist or other specialist.

Initial symptoms of radiation sickness are malaise, nausea, vomiting, and diarrhoea, starting a few hours after exposure. There is then a latent period before the main effects of radiation sickness appear. Record any symptoms and the time of onset. The effects of anxiety and stress may be similar to the early features of radiation sickness.

Take blood for FBC, U&E, and blood group, recording the time on the blood tubes and in the notes. Measurement of the lymphocyte count and analysis of chromosomes at known times after exposure are helpful in assessing the amount of radiation received and determining the prognosis. A low ($<1.0 \times 10^9$/L) or falling lymphocyte count indicates serious radiation exposure.

Further information

National Operational Guidance. https://www.ukfrs.com

Chapter 7

Analgesia and anaesthesia