Suspected poisoning in children results in about 40,000 annual Emergency Department attendances in England and Wales, with approximately half of these admitted for observation or treatment. The majority of poisonings are accidental, especially in the under-5 age group, although intentional overdoses and substance abuse are seen in older children. Rarely, children present with symptoms as a result of deliberate administration of compounds by adults.

Deaths in children from poisoning are becoming increasingly rare with only two deaths reported in 2006 and a decline in mortality rates of ~85% since 1976. Factors responsible for this decline include the introduction of child-resistant containers, reducing the pack sizes of aspirin and acetaminophen, and more effective management and the support provided by the National Poisons Information Service (NPIS). The NPIS has provided information and advice to health-care professionals since 1963 by telephone (+44 870 600 6266) and now has an internet-based database (www.toxbase.org). Appropriate advice often prevents unnecessary hospital admissions and also reduces morbidity and mortality.

In 2006–7, the NPIS received a total of 18,873 telephone enquiries involving children under the age of 10, and over 250,000 TOXBASE accesses. The types of product involved are shown in Table 1; the most common agents are acetaminophen and ibuprofen.

**Developmental considerations**

Paediatric patients may be particularly vulnerable to certain toxins at specific stages of childhood. Breast fed infants may be exposed to drugs or toxins excreted in breast milk; neonates have immature metabolic capabilities, and toddlers, as they develop exploratory hand-to-mouth activity, may be exposed to a wide range of potential hazards.

**Physical considerations**

As physiology evolves from infancy to adulthood, there are many age-related changes in vital signs (Table 2). Infants have a limited ability to increase stroke volume and therefore their cardiac output is primarily dependent on heart rate. They also have a limited ability to recruit alveoli and rely heavily on ventilatory frequency to vary their minute ventilation.

**Initial assessment and management**

The initial priority in treating poisoned children is the standard ABC (airway, breathing, and circulation) resuscitation approach.

**A**: Assess airway patency by looking, listening, and feeling for air movement. If there is no air movement, try to open the airway with simple manoeuvres such as the jaw thrust or the use of airway adjuncts. Certain ingested agents may predispose to airway oedema and obstruction, including caustic agents, angiotensin-converting enzyme inhibitors, and plants containing calcium oxalate crystals (e.g. *Dieffenbachia* and *Philodendron* houseplants).

**B**: Assess the adequacy of breathing by observing ventilatory frequency, use of accessory muscles, breath sounds, and oxygen saturations. Reduced respiratory effort may require bag-valve-mask ventilation until a definitive airway can be secured. It is important to remember that succinylcholine may cause prolonged block in children who have a reduced cholinesterase concentration due to exposure to

**How children differ from adults**

Although there has been widespread study of adult-based toxicology, it is not always possible to translate knowledge derived from the study of adults to the care of children. Whenever possible, in this article, we shall be advocating clinical decisions based on documented experience in paediatric patients.
Poisoning in children

Table 1 Types of agents involved in telephone enquiries to the NPIS for children aged 0–9 years in 2006/7

<table>
<thead>
<tr>
<th>Agent</th>
<th>Number of enquiries</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharmaceuticals</td>
<td>7055</td>
</tr>
<tr>
<td>Plants</td>
<td>1084</td>
</tr>
<tr>
<td>Cleaning product</td>
<td>727</td>
</tr>
<tr>
<td>Plastic</td>
<td>456</td>
</tr>
<tr>
<td>Surfactant/detergent</td>
<td>448</td>
</tr>
<tr>
<td>Essential oil</td>
<td>415</td>
</tr>
<tr>
<td>Inert compound</td>
<td>271</td>
</tr>
<tr>
<td>DIY/building product</td>
<td>256</td>
</tr>
<tr>
<td>Baby care product</td>
<td>209</td>
</tr>
<tr>
<td>Insecticide</td>
<td>31</td>
</tr>
<tr>
<td>Food</td>
<td>26</td>
</tr>
<tr>
<td>Alcohols</td>
<td>25</td>
</tr>
<tr>
<td>Battery</td>
<td>22</td>
</tr>
<tr>
<td>Other</td>
<td>7848</td>
</tr>
<tr>
<td>Total</td>
<td>18 873</td>
</tr>
</tbody>
</table>

Table 2 Normal vital signs in children by age

<table>
<thead>
<tr>
<th>Ventilatory frequency (bpm)</th>
<th>&lt;1 yr</th>
<th>1–2 yr</th>
<th>2–5 yr</th>
<th>5–12 yr</th>
<th>&gt;12 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–2 yr</td>
<td></td>
<td>110–160</td>
<td>100–150</td>
<td>95–140</td>
<td>80–120</td>
</tr>
<tr>
<td>2–5 yr</td>
<td></td>
<td>110–160</td>
<td>100–150</td>
<td>95–140</td>
<td>80–120</td>
</tr>
<tr>
<td>5–12 yr</td>
<td></td>
<td>80–120</td>
<td>80–120</td>
<td>60–100</td>
<td></td>
</tr>
<tr>
<td>&gt;12 yr</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>&lt;1 yr</td>
<td>1–2 yr</td>
<td>2–5 yr</td>
<td>5–12 yr</td>
<td>&gt;12 yr</td>
</tr>
<tr>
<td></td>
<td>70–90</td>
<td>80–95</td>
<td>80–100</td>
<td>90–110</td>
<td>100–120</td>
</tr>
</tbody>
</table>

C: Assess the circulation in terms of cardiovascular status (heart rate, arterial pressure, and capillary refill) and the effect of circulatory inadequacy on other organs (mental state, urine output, skin temperature, and colour). Hypotension should initially be treated with a 20 ml kg\(^{-1}\) crystalloid bolus, remembering that if it is caused by specific toxins such as \(\beta\)-blockers, the specific antidote should also be given, for example, glucagon. If the arterial pressure remains resistant to therapy, adequate filling must be ensured in conjunction with the judicious use of inotropic or vasopressor support. However, the use of inotropes may worsen cardiovascular toxicity and should first be discussed with a clinical toxicologist.

Arrhythmias associated with poisoning are best treated by correcting precipitating factors (e.g. hyperkalaemia and acidosis) and by administering the appropriate antidote; antiarhythmics may be pro-arrhythmic and negatively inotropic and make the situation worse.

D: Assess neurological function in terms of:

- level of consciousness using the Alert-Voice-Pain-Unconscious score or the Glasgow coma scale;
- pupillary size and reaction;
- posture and the presence of any seizure activity;
- bedside blood glucose concentration;

Sustained seizures should be treated using benzodiazepines such as lorazepam 100 µg kg\(^{-1}\) or diazepam 0.25 mg kg\(^{-1}\) i.v. Resistant seizures will require sedation, intubation, and ventilation with subsequent EEG monitoring.

E: Record the child’s core temperature. A fever suggests poisoning with cocaine, sympathomimetics, salicylates, anticholinergics, and dissociative drugs such as ketamine. The appropriate antidote must be administered expediently. Children with hyperthermia must be treated aggressively to avoid serious complications; they should be cooled to a core temperature of \(<39^\circ\text{C}\) with continuous temperature monitoring. There is no role for antipyretic medication, but external and internal cooling measures should be considered. Excessive heat production due to agitation or muscle rigidity can be controlled with benzodiazepines or dantrolene, or paralysis and mechanical ventilation.

Toxin-induced hypothermia is usually mild unless environmental exposure has also occurred. It is associated with poisonings by hypoglycaemic agents, opioids, ethanol, and phenoxyzines.

Diagnostic testing

All children with features of toxicity should have measurement of serum electrolytes, renal and hepatic function, blood glucose, and an assessment of acid–base balance from venous or arterial blood gas analysis.

Plasma drug concentrations are not routinely helpful with the exception of acetaminophen, salicylate, iron, lithium, digoxin, theophylline, ethylene glycol, methanol, carboxyhaemoglobin (COHb), methaemoglobin, and the anticonvulsants. These should always be measured when exposure to these toxins is suspected, although often the delay in processing the results prevents an impact on management in acute poisoning. Similarly, bedside urine tests are of limited use: they are non-specific and do not give reliable results regarding the timing of exposure to the toxin.

Neonates require special consideration. There are several important differences in the normal ranges for certain laboratory measurements that may falsely indicate poisoning. COHb concentrations typically range from 2% to 5% because carbon monoxide is a by-product of protoporphyrin metabolism. Neonates also have a lower baseline cholinesterase activity (studies have found a reduction in fetal red blood cell cholinesterase of 50–70%) which

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coke or organophosphate compounds: prolonged apnoeas of up to 7 h have been described.4

B: Consider the possibility of poisoning by a non-obvious agent.

D: Assess the circulation in terms of cardiovascular status (heart rate, arterial pressure, and capillary refill) and the effect of circulatory inadequacy on other organs (mental state, urine output, skin temperature, and colour). Hypotension should initially be treated with a 20 ml kg\(^{-1}\) crystalloid bolus, remembering that if it is caused by specific toxins such as \(\beta\)-blockers, the specific antidote should also be given, for example, glucagon. If the arterial pressure remains resistant to therapy, adequate filling must be ensured in conjunction with the judicious use of inotropic or vasopressor support. However, the use of inotropes may worsen cardiovascular toxicity and should first be discussed with a clinical toxicologist.

Arrhythmias associated with poisoning are best treated by correcting precipitating factors (e.g. hyperkalaemia and acidosis) and by administering the appropriate antidote; antiarhythmics may be pro-arrhythmic and negatively inotropic and make the situation worse.

Children in cardiac arrest should be treated according to standard guidelines (e.g. The Advanced Cardiac Life Support protocol), although it is important to address the need for a specific antidote, for example, sodium bicarbonate for tricyclic antidepressant (TCA) poisoning.

D: Assess neurological function in terms of:

- level of consciousness using the Alert-Voice-Pain-Unconscious score or the Glasgow coma scale;
- pupillary size and reaction;
- posture and the presence of any seizure activity;
- bedside blood glucose concentration;

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may make them more susceptible to the effects of cholinesterase inhibition, for example, organophosphate poisoning.

An ECG should be performed which may detect conduction abnormalities of diagnostic and prognostic importance; a widening of the QRS complex may be the first sign of TCA toxicity.

### Pharmacological manipulation

#### Gastrointestinal decontamination

The role of gut decontamination is one of the more controversial topics in toxicology. Guidance has been provided by consensus statements from both the American Academy of Clinical Toxicologists and the European Association of Poison Centres and Clinical Toxicologists. In asymptomatic children with non-toxic ingestions, no decontamination is necessary. However, if the ingestion is recent, the child is symptomatic, or the toxin may cause delayed toxicity, then gastrointestinal (GI) decontamination is recommended. Several modes are available and have been studied to determine their efficacy. However, few children have been included in the trials.

Activated charcoal (AC) is the safest mode and is probably effective at reducing the amount of drug absorbed into the bloodstream. It should be given as a 1 g kg\(^{-1}\) dose, if the child has taken a potentially toxic overdose within the previous hour. There is little evidence for its use after 1 h, although it may be considered for drugs when delayed gut absorption may occur (e.g. TCAs and sustained-release preparations). It is an odourless, tasteless, black powder that is effective at adsorbing many toxins with the exception of metals, alcohols, and petroleum distillates. Children may be averse to its gritty texture and colour; if they cannot be cajoled with flavouring, an opaque cup, and straw, then it can be administered via a fine-bore nasogastric tube. Multiple dosing may be needed (every 2–3 h) for drugs such as theophylline and carbamazepine which undergo entero-enteric circulation.

Gastric lavage is usually reserved for children who present within 1 h of ingesting a potentially life-threatening poison. It is often difficult to remove the toxic agent from the GI tract because of the small size of lavage tube needed in paediatric patients, and the child will often need to be intubated to facilitate this technique. It is contraindicated in poisonings by most hydrocarbons, acids, and alkalis. The administration of ipecac syrup to induce vomiting has no role in the GI decontamination of acutely poisoned patients.

Whole-bowel irrigation is a newer technique used to flush the toxin through the bowel, thereby preventing further absorption. Polyethylene glycol 500 ml h\(^{-1}\) is given orally and continued until the rectal effluent is clear (usually in 4–6 h). Although much of the evidence is anecdotal, it has been used in the paediatric population with minimal side-effects. It is particularly useful for ingestions that are not absorbed by AC such as lead paint, iron tablets, and batteries. In these cases, serial abdominal radiographs may also be used to demonstrate its effectiveness.

### Antidotes

An antidote is an agent used to neutralize or counteract the effects of a poison. There are a limited number of antidotes used in clinical practice and these are shown in Table 3. They should not be used indiscriminately in patients with unknown poisonings because this complicates the clinical situation, and there may be adverse reactions to their administration.

#### Enhanced elimination

Several methods exist to increase the elimination of toxins and their metabolites. Urinary alkalinization by the administration of sodium bicarbonate effectively increases the elimination of drugs with a low pKa such as salicylates and chlorpropamide. Alkalization increases the ionized fraction of the drug in the tubular lumen, preventing its reabsorption.

Less than 0.05% of all toxic exposures require extracorporeal therapy. These techniques carry substantial risks (e.g. hypotension, bleeding, and venous air embolism) and should always be discussed with a clinical toxicologist before their institution. They are of particular use for severe toxicity associated with theophylline, lithium, salicylates, methanol, and ethylene glycol and can be used

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**Table 3** Antidotes used in the management of paediatric poisonings

<table>
<thead>
<tr>
<th>Toxic</th>
<th>Antidote</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>β-blocker</td>
<td>Glucagon</td>
<td>Bolus 0.1 mg kg(^{-1}); infusion 0.07 mg kg(^{-1}) h(^{-1})</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Digoxin-specific antibody (Digibind)</td>
<td>1 vial (38 mg) binds digoxin 0.5 mg</td>
</tr>
<tr>
<td>Ethylene glycol/methanol</td>
<td>Ethanol</td>
<td>10 ml kg(^{-1}) loading; maintenance: 1–2 ml kg(^{-1}) h(^{-1}) (Aim 100 mg dl(^{-1}))</td>
</tr>
<tr>
<td>Cyanide</td>
<td>Hydroxocobalamin [Cyanokit (R)]</td>
<td>70 mg kg(^{-1})</td>
</tr>
<tr>
<td>Organophosphate</td>
<td>Atropine</td>
<td>Test 0.05 mg kg(^{-1}); double dose every 5 min</td>
</tr>
<tr>
<td>Iron</td>
<td>Desferrioxamine</td>
<td>10–15 mg kg(^{-1}) h(^{-1}) until acidosis resolved</td>
</tr>
<tr>
<td>Heavy metals</td>
<td>EDTA</td>
<td>20–30 mg kg(^{-1}) day(^{-1})</td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>N-acetylcysteine</td>
<td>150 mg kg(^{-1}) over 15 h</td>
</tr>
<tr>
<td>Opioids</td>
<td>Naloxone</td>
<td>50 mg kg(^{-1}) over 4 h</td>
</tr>
<tr>
<td>Sulphonylureas</td>
<td>Octreotide</td>
<td>100 mg kg(^{-1}) over 16 h</td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td>Sodium bicarbonate</td>
<td>0.1 mg kg(^{-1}) max 2 mg</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Vitamin K</td>
<td>1 µg kg(^{-1}) i.v./s.c. 6 h(^{-1})</td>
</tr>
</tbody>
</table>

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to correct metabolic anomalies associated with the toxin and also removing the toxin itself. Haemodialysis is the most common technique, but charcoal haemoperfusion, plasmapheresis, exchange transfusion, and continuous ultrafiltration techniques may also be used.

**Specific poisons**

**Acetaminophen**

Owing to its widespread availability, acetaminophen is a leading cause of paediatric poisoning. Its treatment is based on the adult guidelines, although there is some evidence to suggest that children have a lower incidence of hepatotoxicity after acetaminophen overdose with a toxic dose of $200 \text{ mg kg}^{-1}$ ($150 \text{ mg kg}^{-1}$ in adults). The guidelines, disseminated by the Royal College of Paediatrics and Child Health, are based upon the use of N-acetylcysteine (NAC) which acts as a glutathione donor and conjugates the toxic metabolite N-acetyl-P-benzoquinone imine in the liver. In late presentations (>24 h after ingestion), the evidence for the use of NAC is more controversial and should be discussed with a clinical toxicologist.

**Salicylate**

Intentional aspirin use in children has significantly decreased since its association with Reye syndrome became apparent. However, there is still a significant incidence of accidental poisoning with aspirin in children. Ingestions of $<150 \text{ mg kg}^{-1}$ are considered non-toxic; patients who take $150–300 \text{ mg kg}^{-1}$ exhibit signs of mild to moderate toxicity and can be treated by rehydration. Ingestions of $>500 \text{ mg kg}^{-1}$ are considered potentially life-threatening. Plasma salicylate concentrations should be measured at least 4 h after ingestion.

Children do not usually display the respiratory alkalosis associated with adult salicylate overdose. However, symptoms of acute toxicity develop more rapidly and are more severe in children than adults. They often present with nausea and vomiting, followed by tinnitus or deafness and fever. Fits, coma, hypotension, and pulmonary oedema may be seen as acidemia progresses with severe fluid and electrolyte imbalances.

The management of salicylate poisoning includes preventing further absorption (gastric lavage and multiple doses of AC may be warranted), enhancing elimination by urinary alkalinization, and correcting fluid and electrolyte abnormalities. Aim for a urinary pH of 8 and a urine output of $2 \text{ ml kg}^{-1} \text{ h}^{-1}$. The pKa of aspirin is 3.0; therefore, a large fraction will be ionized and held in the tubular luminal fluid at this pH. Children are also at high risk of hypoglycaemia and this must be guarded against. Haemodialysis effectively removes salicylate and corrects the fluid and electrolyte disturbance; it should be considered in cases of coma, seizures, pulmonary oedema, renal failure, and severe metabolic disturbance.

**Tricyclic antidepressants**

There is a high risk of toxicity to children from taking even one tablet of a TCA because they have a small therapeutic index and 10–20 mg kg$^{-1}$ can be fatal. The clinical picture can deteriorate suddenly and most patients will show maximal symptoms only 4 h after ingestion. The toxic effects are caused by four main pharmacological properties: (i) norepinephrine uptake inhibition; (ii) direct α-adrenergic block; (iii) myocardial membrane-stabilizing effect; and (iv) anticholinergic action.

Cardiovascular toxicity is manifested as conduction delays, dysrhythmias, and hypotension. The child may also have CNS involvement with a reduced level of consciousness, agitation, and fits and there may be other anticholinergic signs (e.g. dry mouth, dilated pupils, and hyperthermia).

The keystones of management are GI decontamination (gastric lavage for early presentation and up to two doses of AC), supportive care, and close monitoring. Sodium loading with sodium bicarbonate to achieve a pH of 7.50–7.55 has been shown to have beneficial effects, although the mechanism is unclear. Certainly, TCA protein binding is reduced in an alkaline environment and this appears to be associated with an improvement in myocardial contractility, arterial pressure, QRS intervals, and dysrhythmias. Seizures are usually short and self-limiting, but, where necessary, benzodiazepines are the treatment of choice. Tricyclic-specific antibody fragments have been developed, but their use is limited by cost, possible renal toxicity, and the extremely large amounts required.

**Household poisons and pesticides**

Household chemicals are an extremely diverse group of compounds that make up the third most common group of poison referrals after pharmaceuticals and plants. They include cleaning agents, glues, cosmetics, hydrocarbons, and pesticides. Most are packaged in non-toxic concentrations and serious clinical effects are uncommon. However, ingestions of caustic agents and pesticides can cause fatalities.

Caustic exposures occur more frequently in children than in adults as a result of exploratory behaviour; household products such as bleaches are often brightly packaged and readily attract attention. Injuries may be to the airway or GI tract, or as splash injuries to skin and mucous membranes, and may have profound long-term effects, such as oesophageal strictures and the need for permanent tracheostomy. Decontamination techniques are contraindicated, although immediate dilution with water may be of benefit. Management is supportive; patients may require early intubation and endoscopy. The use of steroids is under constant debate.

Organophosphate and carbamate pesticides produce a distinct initial clinical syndrome identified by the excessive release of acetylcholine. They may present with central nicotinic effects such as agitation, seizures, and coma, or with the muscarinic effects of miosis, lachrymation, bronchospasm, diarrhoea, and vomiting, or a
combination. Often they are tachycardic because the nicotinic effect on the sympathetic ganglion over-rides the classically described parasympathetically mediated bradycardia. Respiratory effects are the most common cause of death because these children are difficult to ventilate due to excessive secretions and severe bronchoconstriction. Two other phases are also described: (i) the intermediate syndrome in the first 2 weeks which is characterized by profound respiratory muscle weakness requiring ventilatory support; and (ii) a delayed polyneuropathy at 7–14 days which is responsible for disability rather than death. Management is supportive with gastric lavage in life-threatening ingestions and the use of atropine to antagonize the muscarinic receptors. High doses are required (0.02 mg kg\(^{-1}\) i.v. every 5–10 min). Pralidoxime 25–50 mg kg\(^{-1}\) over 30 min may be effective for the nicotinic effects. Remember that agents that rely on plasma cholinesterase for their hydrolysis such as succinylcholine and mivacurium may have a prolonged action and should be avoided.\(^4\)

**Prevention**

Nearly one-third of children under the age of 6 yr who present with an accidental poisoning will subsequently have a second episode.\(^10\) Although the mortality rates from poisoning have been dramatically reduced in the last 40 yr, the numbers of toxic exposures with their toll on the health-care system remain high. To reduce this impact, the clinician must acknowledge the role of prevention and management of paediatric poisonings. This area must also be addressed by the primary care trusts, health boards, community pharmacies, and the pharmaceutical industry. We must all be urged to support ‘used drugs dump’ and ‘safe storage at home’ campaigns, particularly as ~85% of paediatric poisonings occur in the home. Other education strategies may focus on adequate supervision and safe placement of medications and toxic chemicals with child-safe cabinets and containers. Home-based social support and General Practitioner advice that have been shown in health promotion research programmes to impact upon preventing unintentional injuries may also have a valid role in preventing accidental poisonings.\(^11\)

**References**


Please see multiple choice questions 5–9