Poisoning

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INTRODUCTION

Paediatric poisoning is common; most occurrences are accidental and harmless. The accurate assessment of lethality, appropriate supportive care and avoidance of unnecessary and dangerous decontamination techniques prevent harmful intervention.

EPIDEMIOLOGY

There were 1.08 million toxic ingestions by children <6 years old in the USA during 1998 (Litovitz et al, 1999). Poisoning accounts for 2% of all injury deaths in developed countries, and 5% in developing countries (World Health Organization, 1993).

Common substances are easily accessible, e.g. analgesics, cough preparations, cleaning solutions and cosmetics. As most paediatric poisonings are accidental, packaging legislation and education on safe storage is important (Rodgers, 1996). Vigilance is needed for intentional poisoning. In children over 5 years, drug/alcohol experimentation, abuse and attempted suicide becomes more common. Substances with greatest risk of death include iron, anticonvulsants, antidepressants and cocaine.

ASSESSMENT

History

A high index of suspicion in children presenting with an acute, unexplained illness is the key to recognition of poisoning. History is notoriously unreliable. Important questions include what poison was involved, dose, timing, route and co-ingestants. Less commonly parenteral, dermal, ophthalmic and inhalational exposures occur.

Examination

Systematic assessment of the child's airway, breathing and circulation is of prime importance and supersedes any specific poisoning concerns. Early protection of the compromised airway to prevent obstruction or aspiration, especially if decontamination is to be used, is vital. Oxygenation and ventilation should be rapidly assessed and augmented if necessary.

Hypotension, bradycardia and arrhythmias should be treated using paediatric guidelines as they are detected (Mackway-Jones et al, 2001). Hypotension is usually caused by hypovolaemia secondary to fluid and blood loss (iron) or vasodilation (barbiturates and benzodiazepines), and should be treated with fluid boluses. Occasionally, hypotension is caused by bradycardia or negative inotropy (beta-blockers), requiring inotropic support. An early baseline temperature is essential.

Hypothermia and hyperthermia are common complications in poisonings and require early intervention. Serial assessment of pupils and conscious level using the Glasgow Coma Score (GCS)/AVPU* scores will aid management and diagnosis. Seizures should be treated urgently, usually with benzodiazepines, and sedation for agitation should be avoided.

Toxidromes: Toxidromes are a group of signs and symptoms that suggest the pharmacological activity of a poison. The most common are listed in *Table 1*.

Monitoring and investigation

Minimal monitoring is pulse oximetry, continuous electrocardiogram and non-invasive blood pressure. Basic investigations should include:

- Urea and electrolytes, including chloride, to allow calculation of the anion gap (anion gap = $Na^+ - (HCO_3^- + Cl^-)$, usually <12–14mmol)

*AVPU = alert, responsive to voice, responsive to pain, unresponsive

TABLE 1. Toxidromes

Syndrome and causes	Manifestations	
Anticholinergic Atropine Antihistamines Tricyclics	**Peripheral antimuscarinic** Hot, dry skin Thirst and dysphagia Fixed, dilated pupils Tachycardia Hyperthermia Abdominal distension Urinary retention	**Central** Lethargy Confusion, delirium Hallucinations Ataxia Seizures Rapid, shallow breathing
Sympathomimetic Aminophylline Amphetamines Caffeine Cocaine Ritalin	CNS excitation Seizures Hypertension Tachycardia	
Cholinergic Acetylcholine Pilocarpine	Same as for muscarinic effects (see below)	
Acetylcholinerase inhibition Organophosphates	**Muscarinic effects** Sweating Constricted pupils Hypersalivation Bradycardia, tachycardia Urinary incontinence Wheezing Vomiting, diarrhoea	**CNS effects** Anxiety Ataxia Coma Respiratory and cardiac failure
Narcotics Opiates	CNS depression Pinpoint pupils Hypotension Slowed, deep respirations Rhabdomyolysis	

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- Blood gas analysis, preferably arterial, to assess respiratory function and metabolic acidosis
- Serum osmolar gap (osmolar gap = measured osmolarity – ([1.86 x Na in mmol/litre] + urea in mmol/litre + glucose in mmol/litre)/ 0.93, usually <10 mOsm/kgH_2O*)*
- Serum glucose and liver function
- Urine and possibly serum toxicology
- Specific drug levels at the appropriate time, if known to be useful (*Table 2)*
- Consider chest X-ray for aspiration and abdominal X-ray for radio-opaque drugs.

MANAGEMENT

Supportive care

'Treat the patient, not the poison'. Most patients who present with a poisoning will survive if there is prompt and appropriate management of the airway, breathing and circulation. Decontamination, although important, should not be of primary concern and should never be undertaken before the patient's airway, breathing and circulation has been stabilized.

Gut decontamination

The use of methods to decontaminate the gut has reduced in recent years because of their lack of efficacy and complications (*Figure 1*).

Ipecac syrup: Induced vomiting with ipecac syrup was historically a first-line method of decontamination. There have been reports of adverse events such as prolonged vomiting, Mallory–Weiss tears, gastric rupture and fatal aspiration. The American Academy of Clinical Toxicology (AACT) and European Association of Poison Centres and Clinical Toxicologists (EAPCCT) have issued position statements concluding that there is no value in the use of ipecac syrup, and that its administration may significantly delay the use of activated charcoal (AACT and EAPCCT, 1997a); ipecac syrup is therefore not recommended.

Gastric lavage: Gastric lavage has been shown to cause an increased risk of oesophageal injury, hypothermia, hyponatraemia, water intoxication and fatal misplacement of the tube in the trachea, thus is also not recommended (AACT and EAPCCT, 1997b).

Activated charcoal: Adsorptive agents decrease the amount of the toxin available for absorption by the gut. Current guidelines for the use of activated charcoal recommend a single dose of 1g/kg. When administered within 1 hour of ingestion of an adsorbable poison (*Table 3*), activated charcoal has been shown to reduce absorption by up to 75% (AACT and EAPCCT, 1997c). Multiple doses are recommended only in ingestions of life-threatening amounts of carbamazepine, theophylline, quinine, dapsone or phenobarbitone. The initial dose is 1g/kg, followed by 0.5–1 g/kg every 4 hours until visible in stools or clinical effects of poisoning have resolved (Bates et al, 1997).

The main complications of charcoal are vomiting and aspiration, which can result in empyema, pneumothorax or acute respiratory distress syndrome, and can be fatal. There is no evidence for the use of activated charcoal in poisonings presenting after 1 hour of ingestion, and it is therefore not recommended in these cases (AACT and EAPCCT, 1997c).

Catharsis: Cathartic agents are used to increase gastric motility and thereby shorten the absorption time for toxins in the gut. Reviews (AACT and EAPCCT, 1997d), however, suggest that there is no increased benefit in the combined use of cathartics and charcoal. There is an increased risk of hypernatraemic dehydration and cardiovascular collapse with the use of sorbitol in chil-

TABLE 2. Substances with clinically useful serum levels

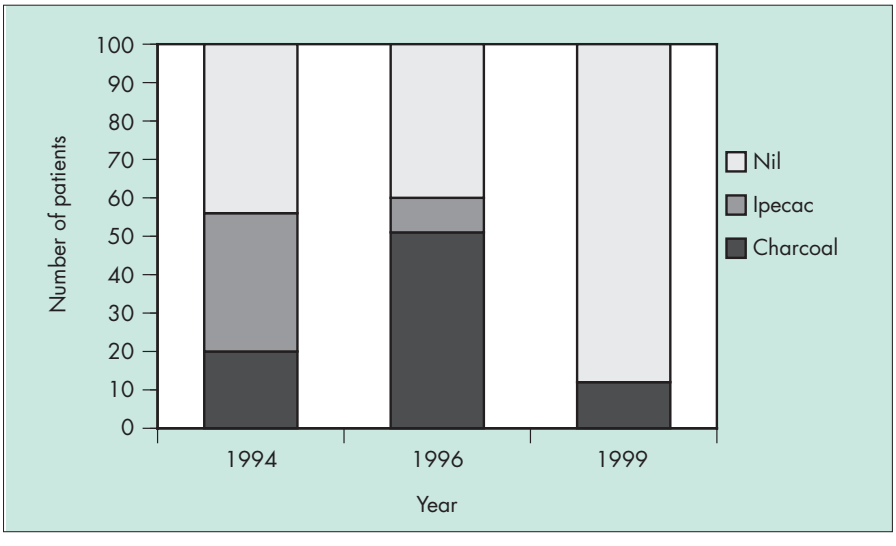
Anticonvulsants (phenytoin, phenobarbitone, carbemazepine)
Carboxyhaemoglobin
Digoxin
Ethanol, methanol, ethylene glycol
Heavy metals
Iron
Lead
Lithium
Paracetamol
Salicylate
Theophylline

TABLE 3. Substances not adsorbed by charcoal

Acids and alkalis
Alcohols (e.g. methanol, ethanol, ethylene glycol)
Cyanide
Metals (e.g. iron, lead, lithium)
Oils and petrolium
Sodium chloride
Sodium hypochlorite (bleach)

From Shannon (2000)

Figure 1. Reduction in use of decontamination methods for paediatric accidental ingestions in New Zealand. From Dillon and Gee (2002).



dren (Farley, 1986). Cathartics are not recommended for use in paediatric poisonings (AACT and EAPCCT, 1997d).

Whole bowel irrigation: There is insufficient evidence to warrant the routine use of whole bowel irrigation (AACT and EAPCCT, 1997e). Theoretically, it may be useful in poisoning with iron, enteric-coated drugs, lead and zinc, but clinical data are limited. Therefore, whole bowel irrigation should only be used following consultation with national poison centres.

Enhanced elimination

Enhanced elimination is rarely indicated and has potentially serious side effects.

Forced diuresis: Forced acid (amphetamines) or alkaline (salicylate) diuresis may increase renal clearance, but increases the risk of fluid and electrolyte imbalance.

Haemodialysis and filtration: Haemodialysis and filtration can be useful for massive overdoses of lithium, salicylates, ethanol and methanol; they are rarely indicated and have significant risks. Haemoperfusion, where blood is passed over a charcoal filter, is rarely used in children.

SPECIFIC ANTIDOTES

Table 4 lists the few poisons that have a specific antidote or treatment.

SPECIFIC POISONINGS

Paracetamol

Paediatric mortality from acute ingestion of paracetamol (*Table 5*) is probably much lower than the 0.5% mortality quoted in adults. Paracetamol is 95% metabolized by glucuronidation and sulphation in children. In overdose (>150 mg/kg) these pathways are overwhelmed, leading to increased metabolism via the P450 pathway, producing a toxic metabolite, n-acetyl-p-benzoquinone-imine. This is normally detoxified by liver glutathione, but if stores become depleted then liver toxicity ensues. N-acetylcystein replenishes glutathione stores. Chronic overdosage is a more serious problem in children.

Tricyclic antidepressants

Tricyclic antidepressant overdoses (*Table 6*) account for 2% of paediatric poisoning admissions, but a disproportionate number of deaths. They have three main toxic effects:

- Anticholinergic toxidrome (*Table 1*)
- Cardiotoxicity
- Neurotoxicity.

The duration of the QRS complex is prognostic; >0.12 s signifies an increased risk of seizures, >0.16 s signifies an increased risk of arrhythmias. Alkalinization with sodium bicarbonate and correction of respiratory acidosis reduces toxicity by reducing the unbound drug and reducing binding to sodium channels.

Recreational drugs

Experimentation is common in adolescents, but accidental overdose is not uncommon in children of drug-abusing parents. Accidental overdose with methadone is more common than with heroin. Toxic effects of narcotics are listed in *Table 1*. Treatment is supportive, and care is needed with naloxone as it has a short half-life (1–2 hours) and re-sedation is likely.

Ecstasy is a common accidental toxin. Cocaine is more commonly taken intentionally; features are listed in *Table 7*.

Iron

Iron is the leading cause of death from poisoning in children <6 years. The toxicity (*Table 8*) depends on the elemental iron dosage. Toxicity starts at 20–60 mg/kg, and doses above 120 mg/kg are lethal. Potentially toxic doses should be treated with whole bowel lavage if presenting within

TABLE 4.
Specific treatments/antidotes used in acute poisoning

Poison	Antidote
Warfarin	Vitamin K/fresh frozen plasma
Benzodiazepines	Flumazenil (caution, may fit)
Beta-blockers	Atropine, glucagon
Carbon monoxide	Oxygen
Cyanide	Dicobalt edentate, hydoxycobalamin, sodium thiosulphate, sodium nitrite
Digoxin	Digoxin-specific antibody fragments
Ethylene glycol/methanol	Ethanol
Iron	Desferrioxamine
Methaemagloninaemia	Methylene blue
Opioids	Naloxone
Organophosphates	Atropine, pralidoxime
Paracetamol	N-acetylcysteine

TABLE 5.
Paracetamol overdose

Specific investigations	Levels at 4 hours post-ingestion and 2-hourly until peaked, plot on Rumack–Mathew nomogram International normalized ratio (INR) Baseline liver function tests, urea and electrolytes, and glucose
Clinical features	Minimal for 24–4 hours, then signs of progressive liver failure. Isolated renal failure can occur. Death from fulminant hepatic failure 4–18 days
Specific treatment	N-acetylcystein if above line on nomogram at any time up to 36 hours post-ingestion, and probably longer. Lower threshold for chronic ingestion or comorbidities. If severe N-acetylcystein reaction, can use oral methionine if not had charcoal
Indications for liver transplant	Very rare in children. Adult criteria: pH<7.3, encephalopathy grade 3/4, prothrombin time day 4 >180
Discharge	If 4-hour level non-toxic and timing certain, or after 48 hours with normal INR, liver function tests and creatinine

4 hours and having undissolved tablets on X-ray.

Serum iron levels are prognostic. Levels <60 mmol/litre are rarely toxic, and levels >90 mmol/litre should always be treated with desferrioxamine (15mg/kg/hour intravenously); intermediate levels should be treated if symptomatic. Supportive care is by replacement of fluid and blood losses, and support for multiple organ dysfunction. Renal replacement therapy is needed to remove the iron–desferrioxamine complex. HM

TABLE 6.
Tricyclic antidepressant overdose

Specific investigations	12-lead plus continuous electrocardiogram Arterial blood gas with electrolytes
Indications for 8.4% sodium bicarbonate (1–2 ml/kg)	Widened QRS >0.1 Arrhythmias, hypotension Seizures
Treatment of arrhythmias	Correct acidosis and electrolytes Avoid anti-arrhythmic drugs (lignocaine best option) Consider DC cardioversion
Treatment of hypotension	Fluids and bicarbonate. Inotropes including glucagon
Convulsions	Airway and breathing, Benzodiazepines
Cardiac arrest	Prolonged cardiopulmonary resuscitation (case reports for 5 hours) Magnesium, extracorporeal membrane oxygenation
Admit to PICU if	GCS<8, QRS>0.1, fits, hypotension, arrhythmias
Discharge	If 6 hours without symptoms, tachycardia or QRS> 0.1

GCS = Glasgow Care Scale, PICU = paediatric intensive care unit

TABLE 7.
Features of cocaine and ecstasy poisoning

	Cocaine	Ecstasy
Features	Agitation and delirium Tachycardia and arrhythmias Hypertension Myocardial infarction Convulsions Hyperthermia Metabolic acidosis	Agitation and delirium Hyperreflexia and nystagmus Hypertension followed by hypotension Acute respiratory distress syndrome Convulsions Hyperthermia Rhabdomyolysis
Management	Benzodiazepines for seizures Vasodilators for hypertension (avoid beta-blockers) Dantrolene for hyperthermia	Benzodiazepines for seizures Electrolyte correction and renal support for rhabdomyolysis

TABLE 8.
Phases of iron toxicity

Phase 1 (0–6 hours)	Vomiting, abdominal pain, diarrhoea, gastrointestinal bleeding and leucocytosis
Phase 2 (6–24 hours)	Apparent improvement
Phase 3 (12–48 hours)	Coma, shock, convulsions, acidosis, hepatic and renal failure
Phase 4 (2–8 weeks)	Gastrointestinal stricture and obstruction

KEY POINTS

- Paediatric toxic ingestions are common and preventable, and most are harmless.
- A high index of suspicion in children presenting with an acute, unexplained illness is the key to recognition of poisoning.
- Unnecessary and ineffective attempts at decontamination do more harm than good.
- Specific antidotes are few.
- Supportive care is essential.
- Some poisons require specific management.
- Always call a national poisons centre for advice.

Figure 1 *is reproduced by kind permission of the New Zealand Medical Journal.*

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