

lorazepam or intravenous diazepam (preferably in emulsion form) may be required to treat convulsions. Activated charcoal given within 1 hour of the overdose reduces absorption of the drug. Although arrhythmias are worrying, some will respond to correction of hypoxia and acidosis. The use of anti-arrhythmic drugs is best avoided, but intravenous infusion of sodium bicarbonate can arrest arrhythmias or prevent them in those with an extended QRS duration. Diazepam p. 236 given by mouth is usually adequate to sedate delirious patients but large doses may be required.

Selective serotonin re-uptake inhibitors (SSRIs)

Symptoms of poisoning by selective serotonin re-uptake inhibitors include nausea, vomiting, agitation, tremor, nystagmus, drowsiness, and sinus tachycardia; convulsions may occur. Rarely, severe poisoning results in the serotonin syndrome, with marked neuropsychiatric effects, neuromuscular hyperactivity, and autonomic instability; hyperthermia, rhabdomyolysis, renal failure, and coagulopathies may develop.

Management of SSRI poisoning is supportive. Activated charcoal given within 1 hour of the overdose reduces absorption of the drug. Convulsions can be treated with lorazepam p. 238, diazepam, or buccal midazolam p. 239 (see *Convulsions*). Contact the National Poisons Information Service for the management of hyperthermia or the serotonin syndrome.

Antimalarial poisoning

Overdose with quinine, chloroquine, or hydroxychloroquine is extremely hazardous and difficult to treat. Urgent advice from the National Poisons Information Service is essential. Life-threatening features include arrhythmias (which can have a very rapid onset) and convulsions (which can be intractable).

Antipsychotic poisoning

Phenothiazines and related drugs

Phenothiazines cause less depression of consciousness and respiration than other sedatives. Hypotension, hypothermia, sinus tachycardia, and arrhythmias may complicate poisoning. Dystonic reactions can occur with therapeutic doses (particularly with prochlorperazine and trifluoperazine), and convulsions may occur in severe cases. Arrhythmias may respond to correction of hypoxia, acidosis, and other biochemical abnormalities, but specialist advice should be sought if arrhythmias result from a prolonged QT interval; the use of some anti-arrhythmic drugs can worsen such arrhythmias. Dystonic reactions are rapidly abolished by injection of drugs such as procyclidine hydrochloride p. 273 or diazepam (emulsion preferred).

Second-generation antipsychotic drugs

Features of poisoning by second-generation antipsychotic drugs include drowsiness, convulsions, extrapyramidal symptoms, hypotension, and ECG abnormalities (including prolongation of the QT interval). Management is supportive. Charcoal, activated p. 898 can be given within 1 hour of ingesting a significant quantity of a second-generation antipsychotic drug.

Benzodiazepine poisoning

Benzodiazepines taken alone cause drowsiness, ataxia, dysarthria, nystagmus, and occasionally respiratory depression, and coma. Charcoal, activated can be given within 1 hour of ingesting a significant quantity of benzodiazepine, provided the patient is awake and the airway is protected. Benzodiazepines potentiate the effects of other central nervous system depressants taken concomitantly. Use of the benzodiazepine antagonist flumazenil p. 899 [unlicensed indication] can be hazardous, particularly in mixed overdoses involving tricyclic antidepressants or in benzodiazepine-dependent patients. Flumazenil may prevent the need for ventilation,

particularly in patients with severe respiratory disorders; it should be used on expert advice only and not as a diagnostic test in children with a reduced level of consciousness.

Beta blockers poisoning

Overdoses with beta-blockers may cause cardiac effects such as bradycardia, hypotension, syncope, conduction abnormalities, and heart failure. Bradycardia is the most common arrhythmia, but some beta-blockers may induce ventricular tachyarrhythmias secondary to prolongation of QT interval (e.g. sotalol) or QRS duration (e.g. propranolol). Although the predominant effects of beta-blocker overdose are on the heart, other features may also occur, such as central nervous system effects (including drowsiness, confusion, convulsions, hallucinations, and in severe cases coma), respiratory depression, and bronchospasm. The effects of overdose can vary from one beta-blocker to another; propranolol overdose in particular may cause coma and convulsions.

The following guidance provides only an overview of the management of beta-blocker overdose from the *National Poisons Information Service TOXBASE database*.

All children who have been exposed to beta-blockers as a result of self-harm should be referred for assessment. Medical assessment is recommended for all children who are symptomatic or have ingested more than a toxic dose; and in those who are treatment naive or have taken more than their therapeutic dose, and have a significant cardiac history or asthma. All children who have exceeded their prescribed daily dose of 2 or more cardiotoxic agents should also be referred for medical assessment irrespective of the dose ingested. Hospital clinicians are encouraged to discuss all serious cases with the UK National Poisons Information Service.

For children presenting with overdose, maintain a clear airway and adequate ventilation. Although the benefit of gastric decontamination is uncertain, charcoal, activated can be considered if the child presents within 1 hour of ingestion of more than a potentially toxic dose.

For the management of hypotension, ensure adequate fluid resuscitation; in an emergency, vasopressors and inotropes can be initiated under the advice of an experienced physician. Intravenous glucagon [unlicensed] p. 502 is a treatment option for severe hypotension, heart failure, or cardiogenic shock. In severe cases, an insulin and glucose infusion can improve myocardial contractility and systemic perfusion, especially in the presence of acidosis. Consider intravenous sodium bicarbonate p. 634 for correction of metabolic acidosis that persists despite correction of hypoxia and adequate fluid resuscitation—rapid correction is particularly important if QRS duration is prolonged. For symptomatic bradycardia give intravenous atropine sulfate p. 869; dobutamine [unlicensed] p. 129 or isoprenaline [unlicensed] (available from 'special-order' manufacturers or specialist importing companies) may be considered if bradycardia is associated with hypotension. A temporary cardiac pacemaker can be used to increase the heart rate.

Treat bronchospasm with nebulised bronchodilators and corticosteroids.

If convulsions occur give oxygen, and correct acid-base and metabolic disturbances as required. Prolonged or frequent convulsions should be controlled with intravenous diazepam, lorazepam, or midazolam. If convulsions are unresponsive to treatment, the child should be referred urgently to critical care.

Calcium-channel blockers poisoning

Features of calcium-channel blocker poisoning include nausea, vomiting, dizziness, agitation, confusion, and coma in severe poisoning. Metabolic acidosis and hyperglycaemia may occur. Verapamil and diltiazem have a profound cardiac depressant effect causing hypotension and arrhythmias,