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A Critical Review of Potentially Deadly Pediatric Ingestions

Awareness of common agents that have the potential to be fatal is essential for all acute care providers. The authors give a comprehensive review of the the most critical common pediatric ingestions and focus on initial presentation and management.
— Ann M. Dietrich, MD, FAAP, FACEP, Editor

Background

Accidental poisonings remain a common, but preventable, cause of pediatric harm.[1-3] In 2019, the American Association of Poison Control Centers (AAPCC) reported 2,148,141 toxic exposures, with 1,411 resultant fatalities.[4] Of these total exposures, more than 1.2 million were for unintentional pediatric exposures — nearly 58% of all calls for that year.[4] Most unintentional medication exposures occur in children younger than 6 years of age, with the highest prevalence between ages 1 and 2 years.[5] In 2019, children younger than 3 years of age were involved in 31.3% of all toxic exposures, and children younger than 5 years accounted for 42.8% of human exposures.[4]

Developmental status plays an important role in understanding the circumstances leading to pediatric poisonings.[3,6] As children become more mobile and explore their environment between the ages of 1 and 5 years, the risk of unintentional ingestion increases significantly.[3,5,6] During this time, increased mobility via crawling and cruising, development of the pincer grasp, and oral exploratory behavior put them at greater risk for unintentional exposures.[2,3,6] Conversely, preteens and teenagers are at a higher risk for illicit drugs and intentional overdoses.[3,6]

Campaigns to increase education about safe medication storage and advances in child-resistant packaging and labels have seen promising results, with emergency department (ED) visits for unintentional medication ingestions declining from 2010-2018.[3,7,8] Despite these advances, accidental pediatric poisonings remain an ongoing, but preventable, public health problem.

To this end, it is imperative for the emergency provider to be aware of common agents that have the potential to cause life-threatening toxicity or death should accidental ingestions occur. This article focuses on substances that are potentially catastrophic if ingestions occur and how to manage them accordingly.

General Management Principles for Pediatric Ingestions

Resuscitation of the poisoned pediatric patient must be the first priority, including stabilization of airway, breathing, and circulation.[9,10] Early intubation and mechanical ventilation may be required for patients showing signs of respiratory depression.[9,10] Intravenous (IV) access should be obtained, and continuous cardiac monitoring should be initiated.[9,10] Hypotension may require IV fluid resuscitation with crystalloid solution and/or vasopressor support as detailed in the specific sections later.[9,10]

EXECUTIVE SUMMARY

- Resuscitation of the poisoned pediatric patient must be the first priority, including stabilization of airway, breathing, and circulation.
- Administration of activated charcoal is most effective within one hour but should be undertaken only if the conditions are appropriate. The patient should have a normal mental status and a secure airway. Activated charcoal can be considered in pediatric patients meeting these criteria, but it is not recommended following the ingestion of alcohols or heavy metals or with caustic ingestions.
- The most common presenting symptoms of beta-blocker poisoning are bradycardia and hypotension. Supportive care is the initial treatment. Hypotension initially should be managed with intravenous (IV) fluid administration of small boluses of 5 mL/kg to 10 mL/kg of isotonic crystalloid. Medical management of beta-blocker toxicity begins with administration of IV glucagon. IV calcium has been used to improve hemodynamics while increasing inotropy.
- The use of sodium bicarbonate in tricyclic antidepressants poisonings is the mainstay of reversing cardiotoxic effects. Sodium bicarbonate should be given to hemodynamically unstable patients, patients who are seizing, and patients with QRS prolongation of more than 100 milliseconds. An initial bolus of 1 mEq/kg to 2 mEq/kg should be administered, followed by an intravenous infusion containing sodium bicarbonate.
- Camphor is a common ingredient found in a wide variety of over-the-counter nasal decongestants, ointments, and topical anesthetic rubs for musculoskeletal pain. Toxicity primarily results from oral ingestion, although there have been reports of toxicity from dermal and inhalational exposure among toddlers. Signs and symptoms of camphor ingestion include gastrointestinal distress and central nervous system (CNS) effects. Toxic effects occur as soon as 10 minutes to 20 minutes after ingestion. Treatment is mainly supportive, including airway management and seizure control.
- The principal manifestation of sulfonylurea toxicity is hypoglycemia. Hypoglycemia has been reported in young children after ingestion of just a single tablet, with reports of delayed hypoglycemia up to 16 hours or 18 hours after ingestion. Management of sulfonylurea toxicity should be directed at the prompt recognition and treatment of hypoglycemia.
- Loperamide, a widely used nonprescription antidiarrheal medication, has reported toxicity in the pediatric population after ingestion of one-half tablet, and death in children has been reported after ingesting fewer than five tablets.
- Opioid overdose in children is characterized by delayed onset of toxicity, unexpectedly severe poisoning, and prolonged toxic effects. Toxicity typically manifests as respiratory depression, miosis, and CNS depression, although additional findings may include hyporeflexia, hypothermia, pruritus, bradycardia, hypotension, and decreased bowel sounds. In addition to supportive management after a suspected opioid ingestion, reversal of the offending drug with the opioid antagonist naloxone should be administered to children and adolescents with significant signs or symptoms of opioid intoxication, including coma, depressed respirations, or miosis.

Gastrointestinal (GI) decontamination is the practice of removing an ingested toxin from the GI tract to decrease its absorption or increase its clearance and should be considered with most presentations of overdose and exposure.[11] Activated charcoal is an intragastric binder that helps prevent gastrointestinal absorption and subsequent toxicity.[11]

Administration of activated charcoal is most effective within one hour but should be undertaken only if the conditions are appropriate.[9,11] The patient should have normal mental status and a secure airway.[11] Activated charcoal can be considered in pediatric patients meeting these criteria, but it is not recommended following ingestion of alcohols or heavy metals or with caustic ingestions.[9,11]

Care should be taken with patients who are vomiting or if there is concern for impending intubation, since aspirated charcoal can be caustic to the airway.[12] The recommended dose is 10 g to 25 g or 0.5 to 1 g/kg for children up to 1 year of age and 25 g to 50 g or 0.5 to 1 g/kg for children 1 to 12 years of age.[12]

Whole-bowel irrigation with polyethylene glycol may assist with increasing transit time of toxins in the GI tract, decreasing total absorption time.[11] In appropriate scenarios, recommended dosing is as follows: 500 mL/h in children 9 months to 6 years old and 1,000 mL/h in children 6 to 12 years old.[11]

Sodium bicarbonate and magnesium may be needed to treat QRS prolongation and ventricular arrhythmias in certain overdoses.[9,13,14] The dose of sodium bicarbonate is 1 mEq/kg to 2 mEq/kg given as an IV push that can be repeated if effective. An infusion then can be started with 132 mEq of sodium bicarbonate mixed into 1 L of 5% dextrose in water at twice the maintenance fluid rate in children.[13]

Table 1 (view online at https://bit.ly/3GatCU1) summarizes the clinical features and treatments for common pediatric poisonings, and Table 2 (view online at https://bit.ly/3da84KE) outlines particularly dangerous medications to be mindful of when working with pediatric patients.

Calcium Channel Blockers

Clinical Manifestations

Calcium channel blockers (CCBs) are commonly prescribed medications used for a variety of indications, including hypertension, coronary artery disease, and atrial fibrillation.[12] CCBs work by directly inhibiting voltage-gated L-type calcium channel opening and calcium influx into myocardial and vascular smooth muscle cells.[14,15] By hindering the influx of calcium into vascular smooth muscle cells and cardiac myocytes, CCBs cause vasodilation, reduce myocardial contractility and conduction, and lower blood pressure.[14-16] Patients who overdose on CCBs classically present with bradycardia, conduction abnormalities (e.g., second- or third-degree heart block), hypotension, and hypoglycemia.[15,16] Effects from extreme negative inotropy can present as cardiogenic shock or even cardiac arrest.[17] Patients can present with symptoms in as little as one to five hours after ingesting immediate-release CCBs. However, signs and symptoms may be present for more than 24 hours with sustained-released

preparations.[17] Hypotension may last for more than 24 hours in some cases, and cardiac conduction abnormalities have been reported for as long as seven days postexposure.[17]

Management

Clinical management of CCB toxicity begins with early assessment and management of hemodynamic stability. Shock typically will require management in an intensive care unit (ICU).[14] Ingestion of sustained-released CCBs will require at least 24 hours of monitoring, even if the patient is initially asymptomatic. Cardiac monitoring is an essential first step, and the patient should be managed in an institution where transcutaneous or transvenous pacing is available.[17]

For patients who are hypotensive, smaller boluses of 5 mL/kg to 10 mL/kg of normal saline or lactated Ringer's is an appropriate initial fluid challenge to avoid overloading patients with myocardial depression.[12] Vasopressors should be initiated for refractory hypotension; norepinephrine or epinephrine is a good initial single agent because of efficacy in improvement in contractility and heart rate, respectively (pediatric norepinephrine dosing starts at 0.05 mcg/kg/min to 0.1 mcg/kg/min; epinephrine dosing starts at 0.1 mcg/kg/minute; both can be titrated to effect).[12] Atropine and glucagon rarely are effective.[12,14,17] IV calcium can be administered as a first-line agent for its inotropic effects, and there have been some reports of its benefit in cases of mild toxicity.[12,15,17] There are conflicting data as to its clinical utility with significant toxicity, but it remains a first-line therapy, since calcium is readily available in EDs and adverse effects are rare.[12,15]

High-dose insulin has the strongest evidence as an effective antidote for CCB overdose.[12,15] It is recommended for all patients presenting with signs of myocardial dysfunction, in combination with IV fluids, calcium, and vasopressor therapy.[12] High-dose insulin increases inotropy and overcomes hypoinsulinemia and insulin resistance caused by the CCB overdose.[12] Current recommendations are for a 1 unit/kg bolus of regular insulin followed by an insulin infusion of 1 unit/kg/hour.[12] Maintenance infusion rates range from 0.1 unit/kg/hour to 3 units/kg/hour.[12] The most common adverse effect from this therapy is hypoglycemia. If necessary, euglycemia can be maintained through continuous IV infusion of 5% to 10% dextrose.[18] The proposed initial rate for such an infusion is 0.5 g/kg/hour to 1 g/kg/hour of dextrose.[18]

IV lipid emulsion therapy may be considered in patients refractory to first-line therapies after CCB overdose and may be useful in patients who are hemodynamically unstable.[12,15] Lipid emulsions are fats used in total parenteral nutrition and are widely accepted antidotes for acute local anesthetic toxicity.[12,18] Recently, IV lipid emulsion therapy has been studied as an antidote for overdoses of highly lipophilic medications, such as CCBs.[12,18] Although robust data remain limited, there are a growing number of case reports supporting IV lipid emulsion therapy for this use.[12] As an antidote, it is commonly administered as a bolus of 1 mL/kg to 1.5 mL/kg lipid emulsion 20% over one minute followed by an infusion of 0.25 mL/kg/min for 30 minutes to 60 minutes.[12,18]

In cases of refractory cardiac arrest and multi-organ failure, there have been case reports demonstrating modest success with extracorporeal membrane oxygenation (ECMO).[12] Transcutaneous or transvenous pacing could be a consideration in patients with unstable bradycardia with high nodal heart block.[12]

Beta-Blockers

Clinical Manifestations

Beta adrenergic antagonists (beta-blockers) are used for a wide range of clinical indications including hypertension, ischemic heart disease, heart failure, arrhythmias, and migraine headache.[13] Beta-blockers competitively antagonize myocardial beta-1 adrenoceptors, which decreases cellular levels of cyclic adenosine monophosphate, resulting in a reduction of calcium entry into cardiomyocytes.[13,14,19] This results in depressed myocardial contractility, decreased automaticity in pacemaker cells, and decreased conduction velocity through the atrioventricular node.[13,14,19] Symptoms typically start within two hours of ingestion, and nearly all patients are symptomatic within six hours.[13] The most common presenting symptoms of beta-blocker poisoning are bradycardia and hypotension.[13,14,19] Severe overdoses can result in profound myocardial depression and cardiogenic shock, ventricular dysrhythmias, respiratory depression, mental status changes (including delirium, coma, and seizures), hypoglycemia, bronchospasm, and hypothermia.[13,14,19]

Management

Supportive care is the initial treatment of symptomatic beta-blocker poisoning. Hypotension initially should be managed with IV fluid administration of small boluses of 5 mL/kg to 10 mL/kg of isotonic crystalloids to avoid fluid overloading patients.[13,14] A 12-lead electrocardiogram (ECG) should be obtained to evaluate for conduction abnormality or QRS prolongation.[19]

Medical management of beta-blocker toxicity begins with administration of IV glucagon.[19] High-dose glucagon produces positive inotropic and chronotropic actions on heart muscles.[14,19] The onset of clinical effect, specifically increases in pulse and blood pressure, is expected within a few minutes of a single dose and may persist for 10 minutes to 15 minutes.[14] The initial pediatric dose is 50 mcg/kg administered intravenously over one minute.[13] If there is no observed effect after 10 minutes following a second dose, it is unlikely an infusion will provide benefit.[13]

IV calcium has been used to improve hemodynamics while treating beta-blocker toxicity by increasing inotropy. Calcium chloride (20 mg/kg; maximum dose is 1 g) or calcium gluconate (60 mg/kg/dose; maximum dose is 3 g) may be used.[13]

Refractory hypotension should be treated with vasopressors to maintain a mean arterial pressure > 60 mmHg, with norepinephrine or epinephrine as the first-line choices.[13,14] IV atropine can be administered at a dose of 0.5 mg to 1 mg every three to five minutes up to a total of 0.03 mg/kg to 0.04 mg/kg to try to reverse bradycardia, but it rarely is effective.[13,14]

Patients with evidence of hemodynamic instability or shock secondary to beta-blocker overdose should be treated with high-dose insulin.[13,19,20] High-dose insulin (regular, short-acting) should be initiated at 1 U/kg/hour titrated up to 10 U/kg/hour, and a regional poison control center should be consulted.[19,20] As with CCB overdoses, IV lipid emulsion may be considered as second-line therapy for some cases of refractory beta-blocker toxicity.[13,19] Overdoses of lipophilic beta-blockers, such as propranolol and carvedilol, may be more amenable to this therapy than hydrophilic agents, such as atenolol and metoprolol.[19]

Tricyclic Antidepressants

Clinical Manifestations

Tricyclic antidepressants (TCAs) are widely used for a number of indications,

including treatment of depression, chronic and neuropathic pain, and migraines.[21-23] After oral administration, TCAs are rapidly absorbed by the GI tract and bind strongly to plasma albumin.[23,24] TCAs are highly protein bound, resulting in large volumes of distribution and a long half-life of elimination that often can exceed 24 hours.[23] TCAs exert their toxic effects on the peripheral nervous system (anticholinergic effects), the cardiovascular system, and the central nervous system.[23]

Mortality associated with TCA overdose most commonly is associated with their cardiotoxic effects manifested as ECG abnormalities, arrhythmias, and hypotension.[17,25] TCAs cause cardiac sodium channel blockade, which increases the duration of the cardiac action potential and refractory period, delaying atrioventricular conduction.[24,25] This can lead to heart block and bradycardia.[26]

The classic ECG with TCA toxicity shows sinus tachycardia, right axis deviation of the terminal 40 milliseconds of the QRS complex, and prolongation of the PR, QRS, and QT intervals.[21] Right axis deviation caused by delayed right ventricular activation from interventricular conduction delays produces a large (> 3 mm) positive terminal R wave in lead aVR and a deep, negative S wave in lead I.[21] Widening of the QRS complex from baseline and positive deflection of the terminal QRS complex in lead aVR are not specific to TCA overdose alone but are pathognomonic for a sodium channel blockade.[21]

Less common ECG findings include nonspecific ST segment and T wave changes, right bundle branch block, high-degree atrioventricular blocks, and the Brugada pattern.[21,25] The risk of seizures increases if the QRS complex is > 100 milliseconds and if the QRS duration is > 160 milliseconds, patients are highly likely to experience ventricular dysrhythmias.[21] Hypotension after TCA overdose may be a result of arrhythmia-induced cardiogenic shock and decreased peripheral vascular resistance from blockade of alpha-adrenergic receptors.[17,26]

Anticholinergic features, such as mydriasis, flushing, dry mucous membranes, tachycardia, and hyperthermia, are common in TCA overdose but often do not cause serious clinical problems.[17,23] Central nervous system (CNS) findings, such as delirium, hallucinations, seizures, and coma, also can be present.[22]

Management

Pediatric patients with suspected TCA overdose may present as altered, agitated, seizing, or even comatose.[21] Respiratory depression may be present, and the emergency provider should be prepared to manage the patient's airway.[21] GI decontamination with activated charcoal is not recommended unless immediately post-ingestion because of the risk of seizures.[26] An urgent ECG should be obtained to evaluate for QRS prolongation and evidence of cardiac dysrhythmia.[17,21,25] Patients who are asymptomatic with normal vital signs, normal ECG findings, and no other concerning findings may be observed for a six-hour period.[17]

The use of sodium bicarbonate in TCA poisonings is the mainstay of reversing cardiotoxic effects.[17,21,23] Sodium bicarbonate should be given to hemodynamically unstable patients, patients who are seizing, and patients with QRS prolongation of more than 100 milliseconds.[26] An initial bolus of 1 mEq/kg to 2 mEq/kg should be administered, followed by an intravenous infusion containing sodium bicarbonate.[17,26]

The goal of this therapy is to narrow the QRS and keep the serum pH between 7.5 and 7.55.[17,26] Efforts should be made to avoid acidosis, since this can worsen cardiac and neurologic toxicity.[26] Sodium bicarbonate is the first-line agent for all TCA-induced ventricular dysrhythmias.[17] Seizures should be treated with benzodiazepines as a first-line agent, with prompt administration of anticonvulsants, such as phenobarbital or propofol, for refractory cases.[17,26] Phenytoin is not recommended because it has the potential to worsen ventricular dysrhythmias.[17]

Camphor

Clinical Manifestations

Camphor is a common ingredient found in a wide variety of over-the-counter nasal decongestants, ointments, and topical anesthetic rubs for musculoskeletal pain.[16,17] Common over-the-counter medications with camphor include Vick's VapoRub, Orajel, Tiger Balm, Bengay, Absorbine, and Save the Baby. Camphor originates from the product of the bark of the camphor tree and has a distinct odor and pungent taste.[16,17]

Toxicity primarily results from oral ingestion, although there have been reports of toxicity from dermal and inhalational exposure among toddlers.[16] Signs and symptoms of camphor ingestion include GI distress and CNS effects. Toxic effects occur as soon as 10 minutes to 20 minutes after ingestion.[16,17] GI effects include oropharyngeal burning and irritation, nausea, and vomiting.[16,17] Initial CNS effects may manifest as hyperactivity characterized by agitation, excitement, restlessness, anxiety, delirium, hallucinations, hyperreflexia, myoclonic jerks, and seizures.[16,17] Patients subsequently can progress to a phase of CNS depression, coma, and apnea.[16,17] Death can result from respiratory depression or intractable seizures.[16,17]

Management

There are no specific antidotes for camphor toxicity. Treatment is mainly supportive, including airway management and seizure control.[17] The patient should be decontaminated early to ensure no residual camphor present on the skin is being continuously absorbed.[27] There is no indication for GI decontamination or activated charcoal.[27] Asymptomatic patients with reassuring vital signs and no evidence of seizure-like activity should be observed for six to eight hours.[17] Seizures should be treated with benzodiazepines as first-line agents.[16,17,27] Phenobarbital or propofol should be considered for refractory cases.[17] Aspiration and seizure precautions should be maintained for the duration of observation due to the risk of acute onset of CNS depression and seizures with associated emesis.[17]

Sulfonylureas

Clinical Manifestations

Sulfonylureas are oral hypoglycemic agents used to treat diabetes mellitus. Commonly used sulfonylureas include glipizide, glyburide, glimepiride, and chlorpropamide. Sulfonylurea medications help to regulate serum glucose concentration by directly inhibiting ATP-sensitive potassium channels in pancreatic beta cell membranes.[17,28] The resulting increase in intracellular potassium results in membrane depolarization and insulin release independent of circulating serum glucose concentrations.[17,28] Sulfonylureas also potentiate the action of insulin on target tissues and suppress compensatory gluconeogenesis.[17,28]

The principal manifestation of sulfonylurea toxicity is hypoglycemia. Hypoglycemia has been reported in young children after ingestion of just a single tablet, with reports of delayed hypoglycemia

up to 16 hours or 18 hours after ingestion.[17,28] Clinical signs and symptoms of hypoglycemia include lethargy, dizziness, weakness, confusion, headache, irritability, coma, and seizure.[17,28] Autonomic symptoms, including trembling, palpitations, diaphoresis, and nausea, also may be present.[28] If untreated, the patient may progress to permanent neurological impairment and death.[17,28]

Management

Management of sulfonylurea toxicity should be directed at the prompt recognition and treatment of hypoglycemia.

The initial management of hypoglycemia in infants and children is a weight-based bolus of IV dextrose at 0.5 g/kg to 1 g/kg.[28] Children should be treated with 25% (D25W) or 10% (D10W) dextrose solution, and infants should receive D10W.[28,29] Basic pediatric dosing recommendations are: D10W: 5 mL/kg to 10 mL/kg; D25W: 2 mL/kg to 4 mL/kg; D50W: 1 mL/kg to 2 mL/kg.[29] Intramuscular (IM) glucagon at 5 mg can be administered to raise serum glucose levels temporarily while IV access is being obtained but should not be used as a substitute for dextrose because its short duration of action limits its efficacy.[28,29] Doses of dextrose can be repeated, but excessive IV dextrose can stimulate endogenous insulin production, leading to further hypoglycemia.[28] As such, children symptomatic from a suspected sulfonylurea overdose also should be treated with octreotide.[28]

Octreotide is a somatostatin analogue that inhibits secretion of several hormones, including glucagon and insulin.[17,28] IV dextrose given initially can cause transient hyperglycemia, which may stimulate pancreatic beta-islet cells to release more insulin, causing rebound hypoglycemia. Octreotide can minimize this insulin release. Pediatric dosing of octreotide is 1 mcg/kg to 1.5 mcg/kg (up to 50 mcg) every six to 12 hours.[28,29] Octreotide can be administered both intravenously and subcutaneously and should be administered for a total of 24 hours.[28,29] Octreotide can be given as an IV bolus over several minutes or by continuous IV infusion.[28,29]

Once the patient's initial hypoglycemia is corrected, blood glucose should be monitored twice during an initial 30-minute period and, if stabilized, every four to six hours thereafter.[29] Even if not initially hypoglycemic, current recommendations are that all children suspected or known to have ingested a sulfonylurea be admitted for a minimal 24-hour observation.[17,28,29]

Antimalarials

Clinical Manifestations

Chloroquine and hydroxychloroquine have been used for the treatment of malaria for more than 70 years because of their antiparasitic properties and are now commonly used in the treatment of conditions such as rheumatoid arthritis and systemic lupus erythematous because of their anti-inflammatory properties.[16,30] Toxicity is characterized by cardiotoxic, respiratory, and CNS effects.[16,30,31] Onset of symptoms can start within one to three hours of ingestion, and a single 500-mg tablet of chloroquine can be fatal to children.[31]

Cardiotoxic effects are similar to 1A antiarrhythmics, with cardiac sodium and potassium channels blocked resulting in arrhythmias and intractable hypotension.[16] ECG changes include prolonged PR, QRS, and QT intervals and ventricular dysrhythmias.[31] Profound hypokalemia is another known complication.[30,31] Severe respiratory symptoms, including tachypnea, dyspnea, pulmonary edema, and respiratory failure have been reported.[30] CNS effects range from drowsiness to visual disturbances, agitation, refractory seizures, and coma.[30]

Management

Patients may present with severe hypotension after a chloroquine or hydroxychloroquine overdose requiring fluid resuscitation.[32,33] Vasopressors, specifically epinephrine, may be used to counteract vasodilatation and myocardial depression after an overdose.[32,33] Diazepam is recommended for the management of seizures.[32,33] Profound hypokalemia is a known complication, and potassium replacement may be required, but careful monitoring for rebound hyperkalemia with resulting dysrhythmias is important.[32,33]

IV lipid emulsion in the treatment of hydroxychloroquine overdose has never been studied and should be discussed with a toxicologist before considering its use.[32] ECMO can be considered as the last option for patients with hydroxychloroquine overdose when other options have failed.[32] There are no data supporting serum alkalinization with sodium bicarbonate for drug removal, and it should be avoided because of the potential for further intracellular shift of potassium exacerbating already marked hypokalemia.[32,33]

Salicylates

Clinical Manifestations

Salicylates are found in several over-the-counter products, including aspirin (acetylsalicylic acid), oil of wintergreen (methyl salicylate), and Pepto Bismol (bismuth subsalicylate).[17] The minimal potentially toxic dose in children is 150 mg/kg.[16,17] Oil of wintergreen, found in topical liniments and solutions used in hot vapors, is the most concentrated form of salicylate, with 1 mL of a 98% solution containing 1,400 mg of salicylate and having the potential to be highly toxic.[16,34] One teaspoon of 98% methyl salicylate contains 7,000 mg of salicylate — the equivalent of nearly 90 baby aspirin — which is more than four times the potentially toxic dose for a 10-kg child.[16,17] Less than one teaspoon of oil of wintergreen has previously proved fatal.[17]

Signs and symptoms of salicylate poisoning include metabolic acidosis with respiratory alkalosis, nausea, vomiting, diaphoresis, tinnitus, and mental status changes.[16,17] Toxic levels of salicylates directly stimulate the brainstem respiratory center, causing hyperventilation and hyperpnea.

Severe intoxication may cause noncardiogenic pulmonary edema, cerebral edema, coma, and death.[16,17] More than half of patients with salicylate poisoning have mixed acid-base disturbances, with a combined anion gap metabolic acidosis and respiratory alkalosis.[17,34] Respiratory acidosis suggests presence of pulmonary edema, respiratory muscle fatigue, or a mixed ingestion.[17]

Management

There are no specific antidotes for salicylate poisoning, and the mainstay of management is rapid clinical assessment, initiation of enhanced elimination, and supportive therapy.[34] Early consultation with a toxicologist is recommended.[34] For airway management, clinicians should be cautious when considering intubation. The brief period of apnea associated with endotracheal intubation can lead to a precipitous fall in pH, causing increased amounts of the drug to accumulate in the

CNS through protonation of salicylate.[34] Aggressive volume resuscitation should be provided to hypotensive patients, with the goal of establishing euvolemia and not forcing diuresis, which has been associated with an increased pulmonary edema risk.[34]

Alkalinization of the urine to accelerate kidney clearance of salicylate is essential in the management of acute salicylate intoxication and should be considered at salicylate levels of 30 mg/dL to 40 mg/dL.[17,34] The goal of alkalinization is a urine pH of 7.5 or greater, through administration of IV sodium bicarbonate.[17,34] An initial bolus of 1 mEq/kg to 2 mEq/kg should be followed by a continuous infusion of 5% dextrose in water containing sodium bicarbonate (three ampules, each containing 44 mmol of sodium bicarbonate added to 1 L of solution).[17,34] Care should be taken to avoid serum pH greater than 7.55.[17,34] Careful monitoring of serum potassium concentration is essential, since hypokalemia can be worsened with alkaline diuresis.[17,34]

In the setting of acidemia and hypokalemia, the distal tubule preferentially excretes protons in exchange for sodium, resulting in an inability to alkalinize the urine.[17] If urine output is adequate and there is no evidence of acute kidney injury, 40 mmol of potassium can be added to each liter of solution to help correct the hypokalemia.[34] Hemodialysis is the most efficient way to remove salicylate from the body, can additionally correct electrolyte and acid-base abnormalities rapidly, and is indicated for critically ill patients.[17,34] A plasma salicylate level > 90 mg/dL in acute overdoses (6.5 mmol/L) is an indication for dialysis, while chronic ingestions should prompt consideration for hemodialysis at lower levels.[34]

Antidiarrheals

Clinical Manifestations

Two of the most commonly used antidiarrheal agents are loperamide diphenoxylate/atropine (commonly sold under the brand name Lomotil).[16,17,35] Both diphenoxylate and atropine are rapidly absorbed by the GI tract and inhibit smooth muscle motility, giving the drug its antidiarrheal properties.[17] Diphenoxylate's metabolite difenoxin is five times more active and has an elimination half-life of 12 hours to 14 hours.[17] Children may develop delayed toxicity after accidental ingestion.[35]

Diphenoxylate/atropine toxicity classically is described as biphasic, with initial anticholinergic symptoms manifesting two to three hours after ingestion. followed by delayed narcotic effects.[16,17] Anticholinergic effects reported in children with diphenoxylate/atropine toxicity include lethargy or agitation, mydriasis, tachycardia, dry mucous membranes and skin, facial flushing, urinary retention, ileus, and hyperthermia.[16,35] Opioid effects include miosis and CNS and respiratory depression.[16] Toxicity in the pediatric population has been reported after ingestion of one-half tablet, and death in children has been reported after ingesting fewer than five tablets.[17,35]

Loperamide is a widely used nonprescription antidiarrheal medication that is a synthetic piperidine derivative structurally similar to diphenoxylate and haloperidol. It slows intestinal transit time by stimulating mu opioid receptors in the myenteric plexus and possesses antisecretory properties.[35,36]

The drug has extremely low bioavailability, but, when taken at large doses, loperamide blocks cardiac sodium and potassium channels, which may cause cardiac toxicity manifesting as conduction abnormalities and dysrhythmias, such as prolonged QRS and QT intervals and polymorphic ventricular tachycardia.[35,36] A single acute ingestion of less than 0.4 mg/kg is unlikely to cause serious toxicity, but fatalities, abdominal distension, and paralytic ileus have been reported in children younger than 1 year of age after ingesting 0.6 mg/day to 3 mg/day.[35]

Management

In addition to providing good supportive care, children with diphenoxylate/atropine ingestions should be monitored for opioid effects, including CNS and respiratory depression.[17] Patients with lethargy, apnea, or coma should be administered naloxone, 1 mg to 2 mg IV.[17,35] Repeated doses of naloxone may be required, since symptoms have been reported to recur 24 hours after initial resolution of the narcotic effects.[17,35] There is no evidence for utility of physostigmine, and anticholinergic effects from diphenoxylate/atropine should be managed with supportive care.[35]

In loperamide cardiotoxicity, standard advanced cardiac life support therapy should be followed in patients with cardiac arrest, including cardioversion or defibrillation for shockable rhythms and IV magnesium for polymorphic ventricular tachycardia.[36] For QRS-interval widening caused by the loperamide sodium channel blockade, a trial of IV sodium bicarbonate is reasonable, although supporting evidence is limited.[36]

Because of concerns for abrupt respiratory arrest, current recommendations are that pediatric patients with diphenoxylate/atropine exposures be admitted to an ICU for at least 24 hours of observation.[17,35] Similar precautions should be taken with children after a large ingestion of loperamide.[35,36]

Opioids and Opiates

Clinical Manifestations

Opioid abuse has gained significant attention in the past decade because of the rising number of deaths from unintentional drug overdoses. Although annual deaths are highest among adults 45 to 54 years of age, adolescents and young adults are seen more frequently in the ED for opioid abuse, with a growing number of children requiring hospitalization for prescription opioid poisoning.[37] In children younger than 6 years of age, opioid intoxication typically occurs in the home as an exploratory ingestion of prescription opioids that were intended for adults.[37-39]

Opioid overdose in children is characterized by delayed onset of toxicity, unexpectedly severe poisoning, and prolonged toxic effects.[38] Toxicity typically manifests as respiratory depression, miosis, and CNS depression, although additional findings may include hyporeflexia, hypothermia, pruritus, bradycardia, hypotension, and decreased bowel sounds.[16,17,37] Infants and children may be more susceptible to the toxic effects of opioids compared with adults because of the differing rates of drug absorption, distribution into the CNS, and metabolism.[17,38] Most deaths are secondary to respiratory depression and subsequent hypoxia.[16,17] Aspiration pneumonitis, pulmonary edema, acute respiratory acidosis, and anaphylaxis are additional causes of direct morbidity and mortality.[17,37]

Management

In addition to supportive management after a suspected opioid ingestion, reversal of the offending drug with the opioid antagonist naloxone should be administered to children and adolescents with significant signs or symptoms of opioid

intoxication, including coma, depressed respirations, or miosis.[17,37,38] Patients may require supplemental oxygen, endotracheal intubation, and positive end expiratory pressure if there is an inadequate response to naloxone or if pulmonary edema is present.[37]

Naloxone dosing depends on patient weight and the clinical scenario. Recommended dosing included:

- children < 20 kg: naloxone 0.1 mg/kg IV (maximum 2 mg/dose);
- children ≥ 20 kg: naloxone 2 mg IV; and
- adolescents may receive lower incremental doses of naloxone (0.04 mg or 0.4 mg per dose) with repeat doses every three to five minutes titrated to patient response.[37]

Naloxone may be repeated every three minutes until improvement in respiratory depression is seen.[37] Children often ingest a higher dose of opioids per kilogram of body weight than adults and typically require larger doses of naloxone to reverse the effects.[38] Children with methadone exposure should have an ECG performed to evaluate for QT prolongation.[39] Admission for a 24-hour observation period is indicated for all children 3 years of age or younger who have been exposed to any opioid analgesic other than immediate-release opioid formulations (e.g., methadone, fentanyl patches, and extended-release formulations) and all toddlers exposed to buprenorphine formulations (including buprenorphine-naloxone).[38,39]

Clonidine and Imidazolines

Clinical Manifestations

Clonidine is a centrally acting alpha-2 adrenergic agonist that is a biochemical derivative of imidazoline, originally developed as a topical nasal decongestant and later marketed as an antihypertensive.[17,40-42] Clonidine is now more commonly used in the pediatric populations for behavioral disorders, attention deficit hyperactivity disorder, tic disorders, sleep disturbances, and post-traumatic stress disorder.[17,40-42] Other imidazolines, such as naphazoline, oxymetazoline (Afrin), tetrahydrozoline (Visine), and xylometazoline, still are used as topical nasal decongestants.[17] Guanfacine and the antispasticity agent tizanidine are other commonly prescribed oral central alpha-2 adrenergic agonist medications.[40]

Clonidine poisoning may occur from exploratory ingestion by young children, transdermal exposure from a clonidine patch, suicidal ingestion with older children, and therapeutic error.[40] Clonidine has a narrow therapeutic index (therapeutic range 2 mcg/kg to 4 mcg/kg), and toxicity may occur with doses of more than 5 mcg/kg.[41] Overdose may manifest as an opioid toxidrome, with decreased level of consciousness, miosis, bradycardia, hypotension, respiratory depression, hypotonia, lethargy, drowsiness, or coma.[17,40-42] Clonidine exposure frequently is symptomatic in children and may lead to endotracheal intubation.[40,42] Signs of toxicity usually are seen within 30 minutes to 90 minutes after ingestion, but new findings rarely appear more than four hours after exposure.[17,40] Symptoms may persist for one to three days.[17]

Management

No true antidote for clonidine or related imidazoline intoxication exists, and management largely is supportive. All patients with suspected exposure should undergo continuous cardiac monitoring and have a 12-lead ECG performed because of the risk of bradycardia and atrioventricular nodal blockade.[17,40,42] Hypotension should be addressed with IV isotonic crystalloid fluid resuscitation, and, in refractory cases, vasopressors are recommended.[17,40,42] Norepinephrine and dopamine have been listed as the vasopressor agents of choice.[17,40,42]

Bradycardia typically is mild and transient, but persistent bradycardia may respond to atropine.[17,40,42] Symptomatic patients may respond to naloxone for respiratory depression, but evidence is limited. Patients with severe CNS depression and apnea should receive a trial of IV naloxone.[17,40,42] Clonidine is not amenable to hemodialysis.[40] Patients without any clinical signs of toxicity within several hours of ingestion may be discharged home following a four- to eight-hour observation period.[42] Pediatric ICU admission is recommended for patients with any cardiovascular or CNS symptoms.[42]

Toxic Alcohols

Clinical Manifestations

Methanol, ethylene glycol, and isopropyl alcohol are called the "toxic alcohols," since all can cause cellular dysfunction and even small amounts may cause death.[17,43,44] The toxic alcohols are found in many common household items.

Methanol is found in automotive windshield washer fluid, de-icing solutions, carburetor cleaners, other industrial cleaning products, and illegal spirits.[17,43-45]

Ethylene glycol is a component of antifreeze and also may be found in radiator coolant, de-icing solutions, brake oil, fire extinguishers, inks, adhesives, and illegal spirits.[17,43-45] The glycol component causes a sweet taste, making ethylene glycol attractive to children and pets.[17]

Isopropyl alcohol can be found in rubbing alcohol, hand sanitizer, and various industrial products.[17,43-45] Table 3 (view online at https://bit.ly/3phimhN) summarizes the clinical features and treatments for toxic alcohols.

While the parent alcohols are responsible for early signs of inebriation, it is their metabolites that cause toxicity and end-organ damage. Alcohol dehydrogenase catalyzes the first oxidation reaction of the toxic alcohols, which eventually results in their terminal and toxic metabolites: formic acid from methanol and oxalic and glycolic acid from ethylene glycol.[17,43-45]

Methanol and ethylene glycol cause a profound anion gap metabolic acidosis and result in end-organ damage.[43-45] Toxic metabolites from methanol cause damage to the retina and the brain, leading to blindness and lesions in the basal ganglia.[43-45] Ethylene glycol poisoning may cause renal failure and also can cause hypocalcemia, leading to cardiac dysrhythmias.[43-45] Isopropyl alcohol can be distinguished from other alcohol ingestions because it is associated with an osmolar gap, but not an anion gap.[45] Ketosis without a metabolic acidosis is pathognomonic for isopropyl alcohol.[44] Clinical findings may present early after ingestion but can be delayed as long as 96 hours if ethanol is co-ingested.[17,43]

Symptoms of toxic alcohol ingestion include simple inebriation and nonspecific symptoms of headache, nausea, vomiting, and abdominal pain to altered mental status and coma. Specifically, methanol ingestion may result in pulmonary dysfunction, visual disturbances (blurred, double, hazy vision) and result in complete blindness, and Parkinson's disease-like symptoms.[17,43-45]

Ethylene glycol's effects can manifest as isolated cranial nerve toxicity, presenting

as ophthalmoplegia, facial weakness, hearing loss, dysphagia, or dysarthria.[17,43-45] This presentation may be delayed by days. Isopropyl alcohol can result in hemorrhagic gastritis, pancreatitis, cardiovascular and respiratory collapse, and hypotension/hypothermia from peripheral vasodilation.[17,43-45]

Management

Direct testing for toxic alcohol concentrations can be diagnostic, but it is frequently unavailable early in the clinical course. Therefore, empiric antidotal therapy should be initiated pending confirmatory levels in cases with high suspicion for ingestion or signs of toxicity.[17,43-45] GI decontamination plays no role, since absorption of methanol or ethylene glycol is so rapid.[43]

Alcohol dehydrogenase blockade with fomepizole or ethanol is indicated in patients with clinical concern for ethylene glycol or methanol ingestion with worsening metabolic acidosis of unknown etiology or concurrent acidosis and abnormal osmol gap. An elevated osmol gap has been used as evidence for the presence of an uncounted, osmotically active toxic alcohol.

Fomepizole is effective at low concentrations, has minimal side effects, and does not require monitoring in an ICU.[17,43,46] Fomepizole is loaded at 15 mg/kg IV followed by 10 mg/kg every 12 hours.[43,44,46] Ethanol can be used as an alcohol dehydrogenase blockade when fomepizole is not available.[17,43-46]

However, fomepizole is the superior antidote for methanol or ethylene glycol poisoning because of its fewer adverse effects. It should be used when available.[43,44,46] All patients with methanol poisoning should receive folic acid 50 mg IV every four to six hours, and patients with ethylene glycol poisoning should receive pyridoxine (50 mg IV) and thiamine (100 mg IV) supplementation to promote metabolism of the parent alcohol to its less toxic compounds.[43,46]

Hemodialysis is the best method to rapidly remove both toxic acid metabolites and parent alcohols, and should be used in patients with renal failure or if acidemia is present.[17,43-46]

Treatment of isopropyl alcohol intoxication is mainly supportive. Metabolism of isopropyl alcohol forms acetone, which is eliminated from the body without causing end-organ damage.[43,44,47] As a result, there is no indication to block the metabolism of isopropyl alcohol with fomepizole or ethanol.[43,44,47] Hypotension should be treated with IV crystalloid and vasopressors, if necessary.[47] Patients with hemodynamic instability despite aggressive fluid resuscitation can be considered for hemodialysis, although this almost never is required.[47]

Laundry Detergent Pods

Clinical Manifestations

Individual laundry detergent pods were introduced to the United States market in early 2012. These "pods" are small, single-use packets composed of highly concentrated detergent in a water-soluble membrane.[45,48,49] They often are brightly colored and may resemble candy, making them particularly attractive to young children.[45,48,50]

There has been a rising number of exposures in children younger than 6 years of age, with the most common routes of exposure through ingestion followed by ocular exposure when the pod bursts.[45,48] Several children have required endotracheal intubation and admission to the ICU after ingestion of one of these products.[45,48] The likely mechanism of toxicity is the high concentration of nonionic surfactants present in the capsules, although anionic surfactants, ethanol, and propylene glycol may also contribute.[50]

The most common clinical manifestations of oral ingestion include nausea and vomiting, coughing and choking, drowsiness, or lethargy.[45,48,50] More significant symptoms include coma, seizure, respiratory distress, aspiration pneumonitis, hematemesis, bradycardia, pulmonary edema, and caustic injury to the GI tract.[45,48,50] Obtundation with loss of airway is the most likely cause of respiratory failure, leading to intubation with mechanical ventilation.[49] Ocular exposures can result in conjunctival erythema, corneal abrasions, and, occasionally, ocular burns.[45,48] Hyperlactatemia and mild metabolic acidosis are the most common laboratory derangements.[50]

Management

The majority of patients do not require any specific intervention after ingestion.[50] In young children without signs or symptoms of toxicity, difficulty swallowing, or changes in mental status, a short period of four to six hours of observation may be all that is required.[50] Supplemental oxygen should be administered to those who develop hypoxemia, and bronchodilators can be given for laryngospasm or bronchospasm.[50]

Any clothing saturated in detergent liquid should be removed, and the affected area of skin should be thoroughly irrigated with soap and water.[50] Correction of acid-base disturbances and hypotension usually can be achieved by the administration of crystalloid fluids alone.[50] Copious flushing of eyes with isotonic saline should be initiated for ocular exposure.[45,49,50]

Ophthalmology and gastroenterology specialists should be consulted if there is concern for significant ocular or GI injury.[45] GI decontamination with activated charcoal is not recommended, since this may impair visibility if endoscopy is required and may lead to emesis, which could re-expose the esophagus to the corrosive substance.[45,50] Endoscopy should be performed in patients with swallowing difficulties, drooling, or oropharyngeal burns.[50]

Organophosphates and Carbamates

Clinical Manifestations

Organophosphates and carbamates are powerful cholinesterase inhibitors with the ability to cause severe cholinergic toxicity following inhalation, ingestion, or cutaneous exposure.[51-55] Both compounds are found in insecticides that have been widely used for insect control in the home and in agriculture for more than 50 years.[51,53-55] Organophosphorus nerve agents have been used in terrorist attacks, such as the 1995 sarin gas attack on the Tokyo subway station, as well as more recent assassination attempts. Carbamates are found in medications such as physostigmine and neostigmine, which are commonly used to treat diseases such as glaucoma and myasthenia gravis.[53,54] Insecticide poisoning is the most common cause of organophosphate and carbamate poisoning.[51,53,54]

Although structurally different, clinical signs and symptoms of poisoning with both agents are virtually identical and require similar management following overdose.[51-55] While severe poisonings are potentially lethal, early diagnosis and treatment often lead to a good prognosis.[53] Organophosphates and carbamates inhibit the acetylcholinesterase enzyme, causing an accumulation of acetylcholine in the synapses, neuromuscular junction,

and end organs, resulting in muscarinic and nicotinic symptoms and signs.[51-54] Onset and duration of symptoms may vary depending on the agent's rate of acetylcholinesterase inhibition, route of absorption, enzymatic conversion to active metabolites, and the lipophilicity.[54]

Symptoms typically manifest within three hours after oral or respiratory exposures but may be delayed up to 12 hours after dermal absorption.[54] Early findings may mimic a flu-like illness and include hypersecretion.[51] The dominant clinical features of acute cholinergic toxicity include bradycardia, miosis, lacrimation, salivation, bronchorrhea, bronchospasm, urination, emesis, and diarrhea.[51-55] Muscle fasciculations and weakness also typically are present.[52-54] Progressive symptoms may lead to muscle and respiratory problems.

In severe poisonings, the CNS also may be affected, particularly in children.[51] Between 20% and 30% of children will have seizures and between 50% and 100% of children will have lethargy, stupor, or coma.[51-54] Cardiac arrhythmias, such as heart block and QTc prolongation, are occasionally seen with organophosphate poisonings.[54] The most common cause of mortality in organophosphate and carbamate poisoning is respiratory failure because of a combination of depression of the CNS respiratory center, neuromuscular weakness, excessive respiratory secretions, and bronchoconstriction.[51-54]

Management

Patients with organophosphate or carbamate poisonings may rapidly develop respiratory failure due to a CNS respiratory center depression, nicotinic receptor-mediated diaphragmatic weakness, bronchospasm, copious secretions, and emesis, thus requiring early endotracheal intubation.[51-54] Even patients with normal mental status or normal vital signs may decompensate rapidly.[54] Emergency providers should avoid succinylcholine for rapid sequence intubation because it is metabolized by acetylcholinesterase, which is inhibited in organophosphate or carbamate poisonings, leading to prolonged neuromuscular blockade.[53,54] Nondepolarizing neuromuscular blocking agents, such as rocuronium, can be used but may be less effective because of competitive inhibition at the neuromuscular junction. Therefore, increased doses may be required.[54]

Decontamination is an important part of initial care. Clothing should be removed, and the patient's body should be washed with soap and water.[51-54] Medical personnel should take measures to protect themselves from contaminated skin and clothing to avoid accidental exposure, including wearing proper personal protective equipment and providing treatment in a well-ventilated area.[51,53,54]

Atropine should be given as a nonspecific antidote in both organophosphate and carbamate poisonings.[54] It competes with acetylcholine at muscarinic receptors, which prevents cholinergic activation.[51-54]

For moderate to severe cholinergic toxicity, atropine should be administered at a beginning dose of 0.05 mg/kg IV for children.[51-54] The dose can be doubled every three to five minutes if no initial effect is noted, titrating to the cessation of bronchoconstriction and clearing of respiratory secretions.[51-54] Hundreds of milligrams of atropine by bolus and continuous infusion may be required.[54] Atropine is ineffective in treating neuromuscular dysfunction, since it does not bind to nicotinic receptors.[54] Pralidoxime may be given after atropine to reverse the acetylcholinesterase inhibitor complex and relieve neuromuscular symptoms.[51-54] In children, an initial IV bolus of pralidoxime should be given over 15 minutes to 30 minutes at a dose of 25 mg/kg to 50 mg/kg.[51-54] An infusion can be used after the bolus (10 mg/kg/h to 20 mg/kg/h in children).[51-54]

Seizures are an uncommon complication of organophosphate or carbamate poisonings, and, when they occur, they usually represent severe toxicity.[51-54] Benzodiazepines are first-line therapy for seizures. Benzodiazepine-refractory seizures may be treated with phenobarbital.[53]

Lead Toxicity

Clinical Manifestations

Elevated blood lead levels (BLLs) have been a concern for decades and can lead to serious long-term harm to the physical and mental health of children.[56-59] BLLs of U.S. children rose from the 1900s to the 1970s until prevention strategies, such as banning lead in paint, gasoline, and plumbing, helped reverse the trend.[56,58,59] However, there remain many children at risk of lead poisoning today.[56,59] Lead has no biologic role in the body, and any detectable level is abnormal.[56,58,59] The American Academy of Pediatrics (AAP) and the Centers for Disease Control and Prevention state that there is no safe or nontoxic BLL.[56,59] Children younger than 5 years of age especially are at risk of lead poisoning.[56,59]

The majority of children with elevated blood lead concentrations will be asymptomatic. Even at higher BLLs (45 mcg/dL [2.17 mcmol/L] or greater), symptoms may be subtle and nonspecific.[56,59] Clinical manifestations may include headache, abdominal pain, anorexia, constipation, hyperactivity, and inattention.[56,58,59] Lead can have deleterious effect on renal function, and may cause anemia, bone changes, and hypertension.[56] The most serious symptoms in children are found in the CNS. Acute encephalopathy occurs at BLLs greater than 100 mcg/dL to 150 mcg/dL (4.8 mcmol/L to 7.2 mcmol/L) and may manifest as persistent vomiting, altered or fluctuating state of consciousness, ataxia, seizures, agitation, increased somnolence, hearing loss, peripheral neuropathy, convulsions, or coma.[56,59]

Symptomatic lead poisoning is a medical emergency and should be addressed immediately if there is clinical suspicion.[56,59] Even low BLLs (< 5 mcg/dL [0.24 mcmol/L]) have been associated with impaired neurocognitive functioning, behavioral development, and permanent CNS injury in young children.[56,59]

Management

Symptomatic lead poisoning is an emergency, and immediate hospitalization is indicated.[56,57,59] Management of asymptomatic children and primary prevention will not be discussed here, but emergency providers should question parents about potential exposures in patients incidentally found to have elevated BLLs. The exception is that even asymptomatic patients found to have a BLL ≥ 70 mcg/dL should have a repeat BLL within 24 hours and, if confirmed, they should be hospitalized and chelation therapy should be initiated.[56,57,59]

Children with lead encephalopathy require admission to a pediatric ICU.[57,59] Patients with lead encephalopathy may present with altered mental status or refractory seizures from increased intracranial pressure.[56,57] Benzodiazepines are the first-line therapy for seizures. If additional medications are needed, phenobarbital is the preferred second-line agent.[57,59] Adequate urine output must be maintained to permit chelation and excretion of blood and tissue lead.[57] Patients should be given fluids containing dextrose and maintenance amounts of sodium at a rate to obtain a daily urine output between

300 mL/m^2 and 350 mL/m^2.[57] Children with lead flecks or foreign bodies noted on abdominal radiographs should receive GI decontamination with whole bowel irrigation.[57]

Chelation therapy in symptomatic children can be life-saving. Chelating agents remove lead from the blood and soft tissues, including the brain.[57,59] Chelating agents include dimercaprol (also called British anti-Lewisite), calcium disodium edetate ($CaNa_2EDTA$), and succimer (also known as dimercaptosuccinic acid or DMSA).[57,60] Dimercaprol crosses the blood-brain barrier and is indicated when neurotoxicity is present or with high BLLs.[60] Dimercaprol is administered as an intramuscular injection at a dose of 75 mg/m^2 (or 3 mg/kg to 5 mg/kg) every four hours and should be used in conjunction with $CaNa_2EDTA$ to prevent lead from being transported to the brain.[57] Notably, IM dimercaprol can be painful, and pretreatment with diphenhydramine is recommended to avoid the adverse effects of dimercaprol-related histamine release.[57] $CaNa_2EDTA$ can be given as a single agent for symptomatic patients with elevated BLL ≥ 70 mcg/dL without CNS findings or given with dimercaprol in children with lead encephalopathy.[57,60] IV dosing is 1,000-1,500 mg/m^2/day (or 35 mg/kg/day to 50 mg/kg/day) infused over eight to 12 hours.[61] Care should be taken to ensure $CaNa_2EDTA$ is given for chelation therapy, since mistaken use of disodium edetate (Na_2EDTA), which is used to treat hypercalcemia, has resulted in severe hypocalcemia and death.[57,60]

Succimer is an oral analogue of dimercaprol that effectively chelates lead.[60] Succimer is preferred by many toxicologists for all but the most severe case of lead toxicity and is first-line for chelation of asymptomatic children with BLL 45 mcg/dL to 69 mcg/dL.[60] It also may be used in combination with $CaNa_2EDTA$ for chelation of children with symptomatic lead poisoning (BLL ≥ 70 mcg/dL), although dimercaprol still is preferred for children with lead encephalopathy.[60] Dosing of succimer is 10 mg/kg or 350 mg/m^2 three times per day for five days, followed by the same dose two times per day for 14 days.[60]

Recommendations and Conclusion

Accidental poisonings remain a preventable cause of pediatric morbidity and mortality.[1-3] Reduction of unintentional pediatric ingestions depends on prevention messages and campaigns. The AAP and United States Preventive Services Task Force both have recommended that healthcare providers provide periodic anticipatory guidance for poisoning prevention to families with young children.[62,63] The emergency provider has an opportunity at each pediatric patient encounter to provide brief counseling, especially to families with young children. Parents should be encouraged to evaluate their home periodically for all potential injury hazards, not just those related to poisoning.[62]

The AAP has provided specific information for parents on poison prevention through their Healthy Children webpage (https://www.healthychildren.org/English/safety-prevention/all-around/Pages/Poison-Prevention.aspx). The emergency provider can refer parents to this website or discuss these recommendations during a patient encounter. These recommendations include:[62,64]

- Keep harmful products locked up and out of the child's sight and reach.
- Use safety latches or locks on drawers and cabinets where you keep dangerous items.
- Purchase and keep all medicines in containers with safety caps.
- Discard unused medication.
- Check the label each time you give a child medicine to ensure proper dosage.
- Never leave containers of alcohol or electronic cigarettes/nicotine refill cartridges within a child's reach.
- Never place poisonous products in food or drink containers.
- Read labels with care before using any product.
- Take extra care during stressful times (when you may be distracted).
- Teach children not to drink or eat anything unless it is given to them by an adult.
- Never refer to medicine as "candy" or another appealing name.
- Check your home often for old medications and get rid of them.
- Be especially vigilant when there is a change in routine. Holidays, visits to and from grandparents' homes, and other special events may bring greater risk of poisoning.
- Get rid of substances used for old-fashioned treatments, such as oil of wintergreen, boric acid, ammoniated mercury, oil of turpentine, and camphorated oil.

Parents should be encouraged to post the universal poison control telephone number for the United States near their home phone or enter this information as a contact in their cellular phone.[61] Emergency providers also are encouraged to call this number at every suspected poisoning for guidance and recommendations. The national toll-free number for poison control centers is 1-800-222-1222.[62] More information about poison control centers can be obtained by visiting the AAPCC website at www.aapcc.org.[62]

References

To view the references, visit https://bit.ly/3EduRkJ.

CME/CE Questions

1. What are the classic electrocardiogram findings in tricyclic antidepressant overdose?
   a. Large S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III
   b. QRS prolongation, shortened PR interval (< 120 ms), slurring slow rise of initial portion of the QRS complex
   c. Widening of the QRS complex, right axis deviation, large (> 3 mm) positive terminal R wave in lead aVR
   d. T wave inversions in right precordial leads V1-V3, epsilon wave after QRS in V1, QRS widening, prolonged S wave upstroke in V1-V3
2. A 3-year-old male presents to the emergency department approximately 30 minutes following the ingestion of an unknown amount of his grandmother's propranolol. Triage vital signs are as follows: blood pressure 61/32 mmHg, heart rate 51 beats per minute, temperature 98.1°F, respiratory rate 12 breaths per minute, oxygen 99% on room air. He remains hypotensive and bradycardic despite treatment with intravenous (IV) crystalloids, glucagon, IV calcium, atropine, and vasopressors. What is the next line therapy for

his hemodynamic instability and refractory shock?
   a. Naloxone 0.1 mg/kg IV
   b. Octreotide 1 mcg/kg to 1.5 mcg/kg every six to 12 hours
   c. Sodium bicarbonate started at 1 mEq/kg to 2 mEq/kg
   d. High-dose insulin with glucose initiated at 1 U/kg/hour

3. A 2-year-old female is brought in the emergency department by her mother 20 minutes after ingestion of an unknown substance. On exam, the patient appears restless, agitated, confused, and is not appropriately responding to her mother's questions. Initial blood glucose is 122 mg/dL. The patient appears to have muscle fasciculations in her extremities and subsequently has two episodes of nonbloody, nonbilious emesis in the room. While being placed on cardiac monitor, the patient begins to have a seizure. What is the most likely ingestion that caused this presentation?
   a. Camphor
   b. Sulfonylurea
   c. Calcium channel blocker
   d. Opioid

4. A 5-year-old male presents after an ingestion of oil of wintergreen. What are the most likely laboratory derangements seen in this patient?
   a. Metabolic acidosis with respiratory alkalosis
   b. Hypochloremic, hypokalemic metabolic alkalosis
   c. Profound hyperkalemia with hyperglycemia
   d. Hyponatremia with hyperkalemia

5. A 4-year-old girl is brought into the emergency department after swallowing two of her grandfather's glipizide pills. She is trembling, diaphoretic, nauseated, and appears lethargic. Point-of-care glucose is 23 mg/dL. She remains hypoglycemic despite multiple repeated doses of dextrose. What is the next best step in management?
   a. Intramuscular glucagon 5 mg
   b. Initiate octreotide 1 mcg/kg to 1.5 mcg/kg
   c. Continuous lactated Ringer's infusion with IV dextrose pushes
   d. IV glucagon 50 mcg/kg

6. A 17-month-old girl presents to the emergency department after ingesting an unknown substance. On arrival, she is lethargic, hypotensive, bradycardic, bradypneic, and hypothermic, with pinpoint pupils. Escalating doses of naloxone are used with transient improvement in symptoms. What is the most likely ingestion that caused this presentation?
   a. Sulfonylurea
   b. Beta-blocker
   c. Clonidine
   d. Hydroxychloroquine

7. What is the recommended chelation therapy for patients presenting with lead encephalopathy?
   a. Oral succimer
   b. Succimer and dimercaprol
   c. Succimer and $CaNa_2EDTA$
   d. Dimercaprol and $CaNa_2EDTA$

8. A 13-year-old boy presents to the emergency department with vomiting, diarrhea, diaphoresis, copious tearing, miotic pupils, and muscle fasciculations. He was playing on his parent's farm prior to the onset of symptoms. Which of the following is an antidote for this patient's exposures?
   a. Flumazenil
   b. Fomepizole
   c. Atropine
   d. Physostigmine

9. Which cofactor should be supplemented in methanol poisoning?
   a. Folic acid
   b. Pyridoxine and thiamine
   c. Vitamin B12
   d. Magnesium

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Upon completion of this educational activity, participants should be able to:

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- formulate a differential diagnosis and perform necessary diagnostic tests;
- apply up-to-date therapeutic techniques to address conditions discussed in the publication;
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A Critical Review of Potentially Deadly Pediatric Ingestions

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PEDIATRIC EMERGENCY MEDICINE **REPORTS**

Practical, Evidence-Based Reviews in Pediatric Emergency Care

A Critical Review of Potentially Deadly Pediatric Ingestions

"One Pill Can Kill" List	
Calcium channel blockers	• Amlodipine • Diltiazem
Beta-blockers	• Propranolol • Carvedilol • Atenolol • Metoprolol
Tricyclic antidepressants	• Amitriptyline • Nortriptyline • Imipramine
Camphor	• Vick's VapoRub • Orajel • Tiger Balm • Bengay • Absorbine • Save the Baby
Sulfonylureas	• Glipizide • Glyburide • Glimepiride • Chlorpropamide
Antimalarials	• Chloroquine • Hydroxychloroquine
Salicylates	• Aspirin • Oil of wintergreen • Pepto Bismol
Antidiarrheals	• Diphenoxylate/ atropine • Loperamide
Opioids and opiates	• Fentanyl • Oxycodone • Methadone
Alpha-adrenergic agonists	• Clonidine
Imidazolines	• Naphazoline • Oxymetazoline (Afrin) • Tetrahydrozoline (Visine) • Xylometazoline
Toxic alcohols	• Methanol • Ethylene glycol • Isopropyl alcohol

Methanol, Ethylene Glycol, and Isopropyl Alcohol Toxicity and Treatment

Alcohol	Common Sources	Clinical Findings	Lab Findings	Treatment
Methanol	• Windshield washer fluid • De-icing solutions • Industrial cleaning products • Illegal spirits ("moonshine")	• Visual disturbances (blurred, double, or hazy vision) • Nausea, vomiting, abdominal pain • Gastrointestinal bleeding • Parkinson-like symptoms	• Profound anion gap • Metabolic acidosis	• Fomepizole • Ethanol • Folic acid • Hemodialysis
Ethylene glycol	• Antifreeze • Radiator coolant, • De-icing solutions • Brake oil • Fire extinguishers • Adhesives • Illegal spirits	• Cranial nerve toxicity: ophthalmoplegia, facial weakness, hearing loss, dysphagia, dysarthria • Renal failure • Hypocalcemia • Multisystem organ failure	• Profound anion gap • Metabolic acidosis	• Fomepizole • Ethanol • Pyridoxine and thiamine • Hemodialysis
Isopropyl alcohol	• Rubbing alcohol • Hand sanitizer	• Pancreatitis • Gastrointestinal bleeding • Hypotension • Central nervous system depression • Coma	• Elevated osmolar gap • Ketosis without metabolic acidosis	• Supportive care

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