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AHC Media

An Approach to Household Toxicological Emergencies in the Pediatric Patient

Patients with toxic ingestions most often will present to the emergency department (ED) in one of two ways: 1) a well-appearing patient with a known ingestion, or 2) an ill-appearing patient with an unknown or suspected ingestion. This article will present the approach to both of these circumstances, discussing treatment and monitoring of specific overdoses as well as the initial approach to an ill child with a suspected overdose. The focus will be on common and accidental ingestions of toxins by pediatric patients. As such, intentional overdoses by adolescents, iatrogenic overdose, and management of ingested foreign bodies will not be discussed.

—Ann M. Dietrich, MD, FAAP, FACEP

Children are naturally curious as part of their normal development. Sometimes, despite the most conscientious parents and the most rigorous prevention methods, children's exploration leads them to ingest potentially toxic household substances.[1] From cleaning supplies to beauty products to pesticides to pharmaceuticals, the home is full of potential toxins. Fortunately, many ingestions are benign and can be observed at home with regular check-in by poison control. Still, the fraction of ingestions that are dangerous can pose a significant morbidity and mortality threat. The wide range of chemicals and availability of new pharmaceuticals require emergency providers (EPs) to be well prepared for the presentation of any ingestion. Local and national poison centers are always available for consultation, but EPs should be familiar with common ingestions and their initial management.

Definition and Epidemiology

Annually, more than 2 million people report toxic exposures to the American Association of Poison Control Centers in the United States. Nearly half of those exposures are in children younger than 6 years of age, making these ingestions a significant public health issue.[2] The vast majority of exposures in this pediatric subgroup occur in the home as an unintentional oral ingestion of a single substance. The peak age for unintentional childhood poisoning is between ages 1 and 3 years, corresponding to a normal developmental increase in mobility and independence.[3-7]

The most common ingestions are readily available to children in many homes. Approximately half of poisonings involve exposure to prescription or over-the-counter (OTC) drug.[2] These are most commonly analgesics, antihistamines, gastrointestinal preparations (such as antacids), and vitamins. The other half involves nondrug substances such as cosmetics, personal care items, cleaning solutions, plants, carbon monoxide, and foreign bodies.

The top four ingestions in each class have remained relatively consistent through the years,[2,8-11] although recent trends have shown a decrease in some of the "under the

EXECUTIVE SUMMARY

- Two percent of childhood fatalities in the United States are due to poisoning found in the home and include carbon monoxide, aspirin, lamp oils, and gasoline.
- Ingestions of sedative-hypnotics, cardiac medications, and opiates carry the highest morbidity and are becoming increasingly more available to children, as older family members are on such prescription.
- Emergency physicians should evaluate unconscious patients for specific toxidromes, paying specific attention to level of consciousness, reflexes, and pupil size and reactivity. Seizures resulting from toxic ingestions often can be difficult to control but can provide clues to the ingestion simultaneously. Seizures refractory to benzodiazepines should be evaluated and treated for hypoglycemia, isoniazid, tricyclic, or organophosphate toxicity.
- Many long-acting opiates, such as methadone, have a much longer half-life than naloxone. A single dose of naloxone may cause an initial improvement but likely will wear off long before the methadone, and respiratory depression will return.
- Flumazenil and naloxone can precipitate severe physiologic withdrawal or seizures in children who have had long-term exposure and should be used with caution in such patients.

sink" ingestions and an increase in pharmaceutical ingestions. Ingestions of bleach, detergents, and other household products remain substantial but have shown some decline.[7] Pharmaceutical exposure is increasingly problematic despite preventive measures taken by caregivers, pharmaceutical companies, and regulatory agencies.[12]

It is estimated that 2% of childhood fatalities in the United States are due to poisoning.[2] Agents involved generally are found in the home and include carbon monoxide, aspirin, lamp oils, and gasoline.[2,13] Not all exposures lead to medical emergencies, but they must be identified rapidly given the risk of morbidity and mortality with ingestion of potentially highly toxic substances in small amounts.[5] (*See Table 1.*) Ingestions of sedative-hypnotics, cardiac medications, and opiates carry the highest morbidity and are becoming increasingly more available to children, as older family members are on such prescriptions.[14] Opiate ingestion, specifically, is becoming more common and deadly with the increasing prevalence of long-acting opiates such as methadone and sustained-release morphine in the home.[15]

The role of prevention in addressing the scope of pediatric poisoning cannot be overstated. Although most children presenting to the ED are past the prevention stage, these visits are often "near-misses" with regard to serious exposures and can serve as great opportunities for patient and parent education. During the peak age of accidental exposure, children are often curious enough to get into toxins but not old enough to understand the potential danger. A developmental history is important to ensure that the history provided is appropriate for the child's developmental stage. For example, the story of a 3-month-old crawling to the laundry room and drinking laundry detergent is inconsistent with the child's expected development, and concern for child abuse should be high. Unfortunately, some poisonings occur in the setting of serious neglect or intentional abuse, and a good social history should be obtained to identify potential sources of exposures and social stressors.

Parents should be educated on typical child development and the need for constant supervision and barrier devices until the child has adequate understanding.[6] Installing child safety locks, storing hazardous materials out of reach, and avoiding the storage of chemicals in mislabeled or re-used containers are common preventive methods that can be discussed with parents during such visits. Parents also should be educated on teaspoons vs. milliliters to avoid an accidental medication overdose. Still, no single preventive method is fully protective against accidental ingestion; therefore, constant supervision must be stressed above all.

Pathophysiology

The toxicology maxim attributed to Paracelsus is often abbreviated as "The dose makes the poison." This is clearly evident in pediatric poisoning, where one could rephrase the quote to say, "The size of the child makes the poison." Pediatric medications are almost uniformly weight-based so, in kind, the same dose of toxin will have varying effects depending on the age or weight of the child. Children also have a higher body surface-to-weight ratio and a relatively higher minute ventilation, making them more susceptible to dermal and pulmonary absorption of a toxin.[19] The pathophysiology of a toxic exposure varies based on the substance to which the child has been exposed, and can cause disturbance in many different organ systems. Many substances of exposure can cause metabolic acidosis, hypoglycemia or hyperglycemia, hypocalcemia, rhabdomyolysis, pneumonitis, respiratory distress, myocardial ischemia, or dysrhythmias. Toxins that affect the aerobic metabolism (i.e., cyanide), glucose metabolism (i.e., sulfonylureas), or oxygen delivery (i.e., carbon monoxide) will have a much more profound effect on small children who have a higher metabolic rate and poor glucose and oxygen reserves. The course of symptoms depends on the particular toxic exposure and can last from minutes (if the half-life of the substance is short) to days (if there are complications from the exposure; e.g., fulminant hepatic failure in acetaminophen overdose).

Children are generally healthy, although their renal and intestinal excretion can be altered by ingestions. Like adults, children with underlying disease are at much higher risk for significant poisoning. Children with multiple medical problems also are more likely to be prescribed multiple medications that may interact or have unintended side effects with ingestions.

Although household items and medications can cause diverse symptoms, many can be condensed into recognizable toxidromes with relatively uniform management plans and antidotes. EPs should be able to identify several recognizable toxidromes easily in the emergency setting to begin empiric treatment. (*See Table 2.*)

The Approach to the Undifferentiated Poisoned Child

Initial Management

The approach to treating any critically ill child should start with an evaluation of the child's appearance, work of breathing, and circulation (ABCs), according to

Table 1. Xenobiotics That May Cause Severe Toxicity to an Infant After a Small Adult Dose, a Single Pill, or a Small Volume[2,16-18]

- β-adrenergic antagonists (sustained-release)
- Benzocaine
- Bupropion
- Calcium-channel blockers (sustained-release)
- Camphor
- Clonidine
- Cyclic antidepressants
- Diphenoxylate and atropine (Lomotil)
- Methanol or ethylene glycol
- Methyl salicylate
- Opioids (buprenorphine, codeine, methadone, oxycodone)
- Pesticides/herbicides/rodenticides
- Phenothiazines (especially chlorpromazine, thioridazine)
- Quinine or chloroquine (antimalarials)
- Sulfonylureas
- Theophylline

the pediatric assessment triangle,[21] paying specific attention to toxicologic features of the patient's presentation.[22] Toxic exposure always should be considered in a child who presents with altered mental status or abnormal work of breathing.[23-26] Many toxic ingestions can by hypothesized by a brief evaluation of presenting vital signs. (*See Table 3.*) Initial management should begin with a rapid assessment of the ABCs with simultaneous intravenous (IV) access, cardiorespiratory monitoring with pulse oximetry, and rapid bedside glucose testing.[3,22]

Airway: Children with stridor, profuse secretions, profuse vomiting, stupor, or respiratory failure should have an advanced airway placed for stabilization. EPs may consider empiric administration of naloxone in respiratory failure if the clinical scenario suggests an opiate overdose. The airway and oropharynx also should be examined for the presence of caustic burns that would benefit from early intubation.

Breathing: Patients with respiratory distress also may have a metabolic cause, such as in salicylate overdose, and should have their breathing managed cautiously to avoid worsening acid-base disturbances. Oxygen can be applied as needed based on the patient's oxygen saturation.

Circulation: Initially, poor perfusion can be managed with a 20 mL/kg bolus of crystalloid but may require inotropes depending on the ingestion. Dysrhythmias, whether fast or slow, generally can be managed according to American Heart Association guidelines; however, some wide complex tachycardias due to tricyclic antidepressants or anticholinergic ingestions may not be responsive to traditional management.[27] Similarly, digitalis toxicity may cause dysrhythmias that will respond only to Fab antibody therapy.

Disability/Dextrose: EPs should evaluate unconscious patients for specific toxidromes, paying specific attention to level of consciousness, reflexes, and pupil size and reactivity. Seizures resulting from toxic ingestions often can be difficult to control but can provide clues to the ingestion simultaneously. Seizures refractory to benzodiazepines should be evaluated and treated for hypoglycemia, isoniazid, tricyclic, or organophosphate toxicity. Increasingly, antidepressants are a cause of toxicologic seizures and require management of serotoninergic effects.[28] Increasing availability of liquid nicotine for e-cigarettes has led to some cases of high-dose nicotine overdose with refractory seizures.[29]

Exposure: The patient's clothes should be removed and the skin decontaminated in the case of a dermal exposure. EPs and staff should use caution to avoid contaminating themselves during this time. (*See Tables 3 and 4.*)

History

If caregivers are available, a brief, directed history should be obtained to ascertain the potential for toxic exposure as well as the patient's pertinent medical history, allergies, and medication use. Parents should be questioned specifically about potential toxins available in the home, such as household cleaning agents, alcohol, and prescription and OTC medications. It is helpful to ask for a list of all medications in the child's environment, even those that the parents think are "out of reach" or "locked up."

Parents and emergency medical personnel should be asked about the estimated time of exposure and where the patient was found. A child found in the bathroom is likely to have ingested medication whereas a child found in the garage is likely to have had exposure to a pesticide or hydrocarbon.[30] If a chemical or pill bottle was found near the child, the substance or product name, specific ingredients, and dosage or concentration (if applicable) can be incredibly helpful in treatment. Whenever possible, ask a caregiver to bring the products, pills, and/or containers to assist with identifying the substance and quantifying the exposure, or provide a picture of the label. Counting pills or measuring the remaining volume of a liquid medication or toxin can be useful in generating estimates for amount ingested. Poison control center specialists also can help to identify possible ingredients and review the potential toxicities of each component based solely on the name of the product, although it must be the exact name. In cases of suspected ingestion, poison center specialists, websites, or smart-phone applications can help identify pills based on markings, shape, and color.

Caregivers also should be asked about drugs of abuse available in the home, although they may be reluctant to divulge this information. Emergency personnel should be asked about the state of the home and if there is concern for drug use.

Physical Exam

Once the primary survey has been performed and the ABCs stabilized, a thorough head-to-toe physical exam will help further identify an unknown toxin. It is important to mention that non-toxicologic reasons for altered mental status are equally common and should be investigated and treated simultaneously. Sepsis and trauma can cause very similar symptoms, so imaging and empiric antibiotic therapy should be employed as clinically appropriate.

Diagnostic Studies

In the absence of a good history or in the presence of a significant overdose, laboratory studies can provide important information to guide diagnosis, monitoring, and treatment of the child with toxic exposure. Although routine testing is not

Table 2. Recognizable Toxidromes[3,20,24]

Toxidrome	Signs						Examples
	Vital Signs	Mental Status	Pupils	Skin	Bowel Sounds	Other	
Sympathomimetic	Hypertension, tachycardia, hyperthermia	Agitation, psychosis, delirium, violence	Dilated	Diaphoretic	Normal to increased		Amphetamines, cocaine, PCP, bath salts (cathinones), ADHD medication
Anticholinergic	Hypertension, tachycardia, hyperthermia	Agitated, delirium, coma, seizures	Dilated	Dry, hot	Diminished	Ileus urinary retention	Antihistamines, tricyclic antidepressants, atropine, jimson weed
Cholinergic	Bradycardia, blood pressure and temperature typically normal	Confusion, coma, fasciculations	Small	Diaphoretic	Hyperactive	Diarrhea, urination, bronchorrhea, bronchospasm, emesis, lacrimation, salivation	Organophosphates (insecticides, nerve agents), carbamates (physostigmine, neostigmine, pyridostigmine), Alzheimer's medications, myasthenia treatments
Opioids	Respiratory depression, bradycardia, hypotension, hypothermia	Depression, coma, euphoria	Pinpoint	Normal	Normal to decreased		Methadone, buprenorphine, morphine, oxycodone, heroin
Sedative–hypnotics	Respiratory depression, heart rate normal to decreased, blood pressure normal to decreased, temperature normal to decreased	Somnolence, coma	Small or normal	Normal	Normal		Barbiturates, benzodiazepines, ethanol

indicated for all toxic ingestions, those in extremis can benefit from a broad laboratory screening.[31-33] (*See Table 5.*) In these patients, electrolytes, blood urea nitrogen and serum creatinine, acid base status, thyroid stimulating hormone and free T4 level, pregnancy test (if appropriate), and urine drug screen should be sent. Rapid quantitative blood tests for several common ingestions may be available depending on a specific hospital's laboratory.

If there is concern for rhabdomyolysis (such as with ingestion of sympathomimetics or any agent that causes extreme agitation, hyperthermia, seizures, or prolonged coma), a urinalysis and creatine kinase should be ordered. Serum osmolality can help calculate osmolar gap for identification of toxic alcohols. A 12-lead electrocardiogram should be obtained in those with occult toxic exposure, with the potential for cardiac conduction disturbances such as minor QT changes, wide QRS complex form, or AV blockade.

The Approach to the Poisoned Child with an Identifiable Toxin

Fortunately, most children do not present in extremis when exposed to a toxin. Instead they often are found near a toxin with pill fragments in their mouth, chemical stains on their clothes, or toxin scattered around them. These children often can be managed at home,[34] although the potential for toxicity may warrant a period of ED observation. With guidance from the local poison center, most children can be observed for change in clinical status and discharged home if they are stable after the time of peak effect has passed. Children with a known ingestion who present ill or have a clinical status change while in the ED can be managed according to Advanced Hazmat Life Support guidelines.[22] Briefly, this involves altering the absorption of the toxin, administering antidotes when available, and enhancing the elimination.

Altering Absorption

Prevention is key with pediatric poisoning, but even after a toxin has been ingested, further absorption sometimes can be prevented through methods like activated charcoal. Given orally, activated charcoal can bind certain compounds in the stomach before these toxins can be absorbed into the bloodstream. Activated charcoal should be used in conjunction

Table 2. Recognizable Toxidromes (cont.)[3,20,24]

Toxidrome	Signs						Examples
	Vital Signs	Mental Status	Pupils	Skin	Bowel Sounds	Other	
Serotonin syndrome (similar findings with neuroleptic malignant syndrome)	Hyperthermia, tachycardia, hypertension or hypotension (autonomic instability)	Agitation, confusion, coma	Dilated	Diaphoretic	Increased	Neuromuscular hyperexcitability: clonus, hyperreflexia (lower extremities > upper extremities)	SSRIs, lithium, MAOIs, linezolid, tramadol, meperidine, dextromethorphan
Salicylates	Tachypnea, hyperpnea, tachycardia, hyperthermia	Agitation, confusion, coma	Normal	Diaphoretic	Normal	Nausea, vomiting, tinnitus, ABG with primary respiratory alkalosis and primary metabolic acidosis; tinnitus or difficulty hearing	Aspirin and aspirin-containing products, methyl salicylate
Withdrawal (sedative–hypnotic)	Tachycardia, tachypnea, hyperthermia	Agitation, tremor, seizure, hallucinosis, delirium tremens	Dilated	Diaphoretic	Increased		Lack of access to ethanol, benzodiazepines, barbiturates, GHB, or excessive use of flumazenil
Withdrawal (opioid)	Tachycardia	Restlessness, anxiety	Dilated	Diaphoretic	Hyperactive	Nausea, vomiting, diarrhea	

Table 3. Potential Toxic Ingestions Based on Abnormal Presenting Vital Signs[24]

Temperature	Heart Rate	Respiratory Rate	Blood Pressure	Mental Status	Potential Ingestions
High	High	High/normal	High	Agitated or obtunded	Anticholinergic Sympathomimetics Nicotine Tricyclics Antihistamines
Low	Low/normal	Low	Low/normal	Sedate or comatose	Ethanol Opioids Clonidine Benzodiazepines Beta-blockers Calcium channel blockers

Source: Author adapted.

with local poison center recommendations, as there can be several drawbacks to its use. The binding effect begins to wane approximately one hour after the ingestion, and binding does not work with all toxins.[35] Several xenobiotics, such as ethylene glycol, iron, and lithium, will not absorb, and children bear all of the risk without any benefit. The most common risks are vomiting, aspiration, and difficulty visualizing the airway if endotracheal intubation is needed. Vomiting risk is not insignificant and is the reason the American Academy of Pediatrics has discontinued the recommendation for syrup of ipecac or induced vomiting.[36] Roughly one in five children given activated charcoal will vomit, so the timing and clinical scenario should be optimized as much as possible to achieve the greatest benefit.[37] Activated charcoal should not be given to children who are poorly responsive, as they are more likely to aspirate and are likely to have absorbed enough toxin to nullify the potential benefits.

Similarly, ingested pills can be pushed through the gastrointestinal tract using whole bowel irrigation. An enteral tube is placed and a cathartic infused to promote bowel motility. This can be useful in the case of gastric pill bezoars, pills with enteric-coating, or toxins not absorbed by activated charcoal.[38] However, much like activated charcoal, whole bowel irrigation

requires enteral administration or infusion of a noxious substance and is generally poorly tolerated by children. For this reason, the use of either method is waning nationally.[2,38]

Antidotes

An antidote is a powerful weapon in an EP's arsenal and, when available, can change a critical patient into one who can be discharged home.[39] These antidotes often are used off-label but, in conjunction with local poison centers, can be lifesaving. A list of common and important antidotes is described in Table 6.

A patient with an opioid overdose often will have a dramatic response to administration of naloxone, as any clinician who has been in this scenario can attest. Sadly, not every toxin has such a powerful antidote, and most other antidotes have varying degrees of clinical effect. Special care should be taken to understand the basic pharmacology of these antidotes, including their duration of action, maximum doses, and potential side effects. Many long-acting opiates, such as methadone, have a much longer half-life than naloxone. A single dose of naloxone may cause an initial improvement but likely will wear off long before the methadone, and respiratory depression will return. The dosage of some antidotes, such as sodium bicarbonate in tricyclic overdose, may depend on the amount ingested and should be titrated to clinical effect. Lastly, flumazenil and naloxone can precipitate severe physiologic withdrawal or seizures in children who have had long-term exposure and should be used with caution in such patients.

Another "antidote" worthy of mention is lipid emulsion infusion. The mechanism of this method is still debated,[41] although it has been shown to be highly effective in local anesthetic toxicity.[42] This has led to its use in other lipophilic drugs, such as antidepressants, antipsychotics, calcium-channel blockers, and beta-blockers, with varying results. Current expert consensus does not recommend for or against use of lipid emulsion in non-anesthetic toxicity.[43]

Enhanced Elimination

Availability of antidotes can be a problem in some hospitals, and although expert consensus is that these should be stocked in all hospitals, supportive care should be used in times of shortage.[44] Once a toxin has been ingested and absorbed, enhancing the elimination of the toxin may be the only supportive care measure left to use. Urinary alkalization for aspirin overdose is the classic example in which sodium bicarbonate infusion enhances the amount of salicylate excreted by the kidneys, thus lowering the effective serum concentration.[45] Hemodialysis and hemoperfusion are more drastic methods of elimination and are used only in critical cases. Hemodialysis is effective for smaller molecules, such as alcohols, iron, lithium, and heavy metal poisoning, although larger drugs do not dialyze off as readily.

Table 4. Physical Findings in Poisoning[3,20,24,25]

Sign	Toxin
Odor	
Bitter almonds	Cyanide
Acetone	Isopropyl alcohol, methanol, paraldehyde, salicylates
Alcohol	Ethanol
Wintergreen	Methyl salicylate
Garlic	Arsenic, thallium, organophosphates, selenium
Ocular Signs	
Miosis	Opioids (except propoxyphene, meperidine, and pentazocine), organophosphates and other cholinergics, clonidine, phenothiazines, sedative–hypnotics, olanzapine
Mydriasis	Anticholinergics (e.g., antihistamines, TCAs, atropine), sympathomimetics (cocaine, amphetamines, PCP), post-anoxic encephalopathy, opiate withdrawal
Nystagmus	Anticonvulsants, sedative–hypnotics, alcohols, PCP, ketamine, dextromethorphan
Lacrimation	Organophosphates, irritant gas or vapors
Retinal hyperemia	Methanol
Cutaneous Signs	
Diaphoresis	Cholinergics (organophosphates), sympathomimetics, withdrawal syndromes
Alopecia	Thallium, arsenic
Erythema	Boric acid, elemental mercury, cyanide, carbon monoxide, disulfiram, scombroid, anticholinergics, vancomycin
Cyanosis (unresponsive to oxygen)	Methemoglobinemia (e.g., benzocaine, dapsone, nitrites, phenazopyridine), amiodarone, silver
Oral Signs	
Salivation	Organophosphates, salicylates, corrosives, ketamine, PCP, strychnine
Oral burns	Corrosives, oxalate-containing plants
Gum lines	Lead, mercury, arsenic, bismuth
Gastrointestinal Signs	
Diarrhea	Antimicrobials, arsenic, iron, boric acid, cholinergics, colchicine, opioid withdrawal
Hematemesis	Arsenic, iron, caustics, NSAIDs, salicylates
Constipation	Lead

TCA: tricyclic antidepressants, PCP: phencyclidine, GHB: gamma hydroxybutyrate, LSD: lysergic acid diethylamide, NSAID: nonsteroidal anti-inflammatory drug

Source: Author adapted.

Common Toxins and Management

Alcohols

Alcohols commonly are found around

Table 4. Physical Findings in Poisoning (cont.)[3,20,24,25]

Sign	Toxin
Cardiac Signs	
Tachycardia	Sympathomimetics, anticholinergics, antidepressants, antipsychotics, methylxanthines (theophylline, caffeine), salicylates, cellular asphyxiants (cyanide, carbon monoxide, hydrogen sulfide), withdrawal (ethanol, sedatives, clonidine, opioids), serotonin syndrome, neuroleptic malignant syndrome
Bradycardia	β-blockers, calcium channel blockers, digoxin, clonidine, organophosphates, opioids, sedative–hypnotics
Hypertension	Sympathomimetics, anticholinergics, monoamine oxidase inhibitors, serotonin syndrome, neuroleptic malignant syndrome, clonidine withdrawal
Hypotension	β-blockers, calcium channel blockers, cyclic antidepressants, iron, antipsychotics, barbiturates, clonidine, opioids, arsenic, amatoxin mushrooms, cellular asphyxiants (cyanide, carbon monoxide, hydrogen sulfide), snake envenomation
Respiratory Signs	
Depressed respirations	Opioids, sedative–hypnotics, alcohol, clonidine, barbiturates
Tachypnea	Salicylates, sympathomimetics, caffeine, metabolic acidosis, carbon monoxide, hydrocarbon aspiration
Central Nervous System Signs	
Ataxia	Alcohols, anticonvulsants, sedative–hypnotics, lithium, dextromethorphan, carbon monoxide, inhalants
Coma	Opioids, sedative–hypnotics, anticonvulsants, antidepressants, antipsychotics, ethanol, anticholinergics, clonidine, GHB, alcohols, salicylates, barbiturates
Seizures	Sympathomimetics, anticholinergics, antidepressants (especially TCAs, bupropion, venlafaxine), cholinergics (organophosphates), isoniazid, camphor, lindane, salicylates, lead, nicotine, tramadol, water hemlock, withdrawal
Delirium/psychosis	Sympathomimetics, anticholinergics, LSD, PCP, hallucinogens, lithium, dextromethorphan, steroids, withdrawal
Peripheral neuropathy	Lead, arsenic, mercury, organophosphates

TCA: tricyclic antidepressants, PCP: phencyclidine, GHB: gamma hydroxybutyrate, LSD: lysergic acid diethylamide

Source: Author adapted.

the house where children are apt to explore. Although ethanol in a liquor cabinet, ethylene glycol in the garage, and methanol in paint supplies are generally known toxins, most cosmetics, cleaning solutions, and hygiene products contain alcohols in varying concentrations.[46] Ethanol is tolerated poorly by small children and also can deplete glucose stores quickly, leading to hypoglycemia.[47] Supportive care is generally best for therapy. Ethylene glycol and methanol will produce toxic active metabolites if not appropriately managed. Prevention of this metabolite is best achieved with fomepizole infusion, which competes with ethylene glycol and methanol for metabolism by alcohol dehydrogenase. This competitive zero-order metabolism also can be accomplished with intravenous ethanol, although fomepizole is preferred.[48]

Acetaminophen

Each year, acetaminophen is in the top five most common pediatric ingestions.[2,8-11] The potential toxicity of acetaminophen led to the withdrawal of the concentrated "infant" formulation of acetaminophen from pharmacies. There are four stages of acetaminophen toxicity. Stage 1 occurs within the first 24 hours, and patients are largely asymptomatic or have mild gastrointestinal upset. If untreated, stage 2 begins with resolution of nausea and vomiting, but the start of right upper quadrant pain as hepatotoxicity develops. During this time, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels may elevate as a quantifiable sign of liver damage. By days 3-5, stage 3 continues and shows hepatic failure indicated by jaundice, coagulopathy, encephalopathy, and extremely elevated AST and ALT. In stage 4 after five days, there is normalization of AST and ALT. In this stage, if so far untreated, patients die from hepatic failure. The lethality of acetaminophen overdose is counteracted with N-acetylcysteine (NAC), and although it should be given early for maximum effect, it may show some benefit up to 72 hours after ingestion. Use of the Rumack-Matthew nomogram can reliably predict the risk of toxicity if a level is drawn within 1-4 hours of ingestion.[49,50] Previously, oral NAC was tolerated poorly by children because of the rancid odor and taste. Although IV NAC has helped eliminate that problem, IV NAC is not without its own issues, with reported cases of seizures or even death with inadvertent overdosing of NAC associated with treatment of acute acetaminophen overdose.[51,52]

Salicylates

Salicylates also are potentially deadly toxins that continue to be high on the list of pediatric ingestions. However, the ingestion of pharmaceutical salicylates is equaled by the ingestion of household salicylates contained in cosmetics, topical balms, and other household products. Methyl salicylates often are found in high concentrations in many sports creams or muscle pain reliever salves and have an odor that is enticing for children. To further complicate matters, children often present with a mixed acid-base disturbance that can delay diagnosis.[53,54] Finally, the Done nomogram, which attempts to predict toxicity, is not nearly as reliable as the Rumack-Matthew nomogram.[55] Management is characterized by alkalinization of the urine to enhance excretion and supportive care of resultant pulmonary edema and electrolyte disturbances.

Antidepressants

Novel uses of tricyclic antidepressants (TCA) and selective serotonin reuptake inhibitor (SSRI) antidepressants have made them even more available to the pediatric population through the adults and teenagers in the home. Left untreated,

TCAs can lead to an anticholinergic syndrome and wide complex tachycardia due to the sodium channel blockade.[56,57] Children in extremis may require increasing doses of sodium bicarbonate to overcome the sodium blockade effect. SSRI ingestion also can cause dysrhythmias, although not to the same effect as TCAs. SSRIs are more likely to cause neurologic symptoms, with seizures and rigidity seen in serotonin syndrome.

Antihypertensives

Patterns of toxicity with antihypertensive ingestions depend on the class of drug ingested. Typically, these classes are ß-blockers, calcium-channel blockers, and α2-inhibitors; others, such as angiotensin-converting enzyme inhibitors, occur but they generally are not as dangerous as these three.[57] Within each class, medications may be managed quite differently. For example, atenolol is a dialyzable drug, whereas most other ß-blockers are not. The common thread between all of these drugs is the end effect of hypotension and cardiovascular collapse.

Patients who ingested ß-blockers often will present with a profound bradycardia and hypotension. Along with this, the ß2 effect of many ß-blockers also can cause hypoglycemia and sedation. Although glucagon once was the standard treatment to reverse systemic ß-blockade, high doses of insulin and glucose (nearly 10-fold the standard dose) have been shown to be effective in treatment of severe toxicity.[58] Calcium-channel blockers are treated similarly with the addition of IV calcium gluconate or calcium chloride to compete at the calcium channels. Calcium-channel blocker overdose also tends not to be sedating, and patients will have normal or high blood sugar levels.[25] This difference can help distinguish the most prominent type of ingestion in the setting of an unknown or mixed ingestion.

Although used therapeutically as a cardiovascular agent, α2-blockers, such as clonidine, guanfacine, and tizanidine, increasingly are being used in school-aged children and teenagers as a mood-altering agent, making them more common in households. Hypotension is a prominent feature in overdose, but equally common is a pronounced respiratory and neurologic effect in overdose that is very similar to opioids. In particular, clonidine is partially responsive to naloxone, which may prevent intubation in some cases.[59]

Table 5. Suggested Diagnostic Evaluation for Undifferentiated Poisoned Child

- Complete blood count
- Electrolytes
- Blood urea nitrogen/creatinine
- Liver function tests
- Ethanol and other toxic alcohol screen
- Rapid urine drug screen
- Electrocardiogram
- Serum osmolarity
- Arterial blood gas
- Salicylates
- Anticonvulsants (phenytoin, valproic acid, carbamazepine, etc.)
- Acetaminophen
- Iron
- Digoxin
- Lithium

Table 6. Selected Toxin/Antidote Pairings[40]

Toxin	Antidote
Acetaminophen	N-acetylcysteine
Anticholinergic poisoning	Physostigmine
Benzodiazepines	Flumazenil
β-blocker	Glucagon Insulin/glucose
Calcium-channel blockers	Calcium chloride Glucagon Insulin/glucose
Cholinergic poisoning	Atropine Pralidoxime
Digoxin	Digoxin-immune Fab
Ethylene glycol	Fomepizole
Iron	Deferoxamine
Isoniazid	Pyridoxine
Methemoglobin	Methylene blue
Opiates	Naloxone
Sulfonylureas	Glucose Octreotide
Tricyclic antidepressants	Sodium bicarbonate

Source: Author adapted.

Laundry Detergent Pods

Household ingestions of cleaning agents make up a fairly large proportion of poison center calls,[2] but as new products begin to emerge on the market, new patterns of poisonings are seen. In the case of laundry detergent pods, a known class of cleaning agent (laundry detergent) has been repackaged and rebranded to cause a new class of toxicity.[60] Bright-colored packaging and a self-contained, easily ruptured pod allow for children to ingest more toxin than they would from traditional powder or liquid formulations. Most ingestions are mild and cause vomiting and cough with some oral irritation and drowsiness.[61] Severe ingestions can result in an altered level of consciousness,

respiratory distress, and metabolic acidosis. Fortunately, current data suggest that this is temporary and is managed best with supportive care of airway and breathing. Ocular exposure also can result in chemical corneal injury, so fluorescein staining should be considered for patients who are exposed.

Nicotine

The knowledge about nicotine toxicity from tobacco products is fairly extensive, given the historical abundance of tobacco in mainstream culture. Calls to poison centers are common for ingestion of whole cigarettes or cigarette butts.[62,63] One cigarette butt contains between 5-7 mg of nicotine, which is far less than the reported lethal dose of 40-60 mg. A whole cigarette contains about 10 mg of nicotine, which is certainly enough to cause toxicity symptoms of nausea, vomiting, tachycardia, and diaphoresis. More profound ingestions can cause altered levels of consciousness, seizures, and dysrhythmias. Fortunately, the bitter taste of cigarettes and nicotine usually precludes children from ingesting the entire object. That remained the case until recently, when new technology developed far more concentrated forms of nicotine delivery, such as nicotine patches (as much as 114 mg) and e-cigarette nicotine liquid (as much as 36 mg/mL). If these products are not kept out of reach, a child could quickly consume a 30 mL bottle with disastrous consequences.[64,65] Often, supportive care for the cholinergic responses of vomiting and salivation is all that is needed, with the addition of mild sedation for the agitation of the nicotinergic effects. More significant overdoses may be responsive to atropine or benzodiazepines.

Marijuana

Pediatric marijuana exposure is becoming more prevalent with the legalization of marijuana in many U.S. states.[66] Secondhand inhalation of marijuana smoke is unlikely to cause intoxication, although ingestion is more potent and more likely to occur given children's explorative nature. Generally, cannabinoids act as a depressant, although synthetic cannabinoids can have a quite potent stimulant effect.[25,67,68] There is no known antidote for cannabis toxicity, and symptoms of lethargy, coma, and seizures should be treated supportively with airway intervention and benzodiazepines as warranted.

Conclusion

The number of available toxins is ever increasing, and children are incredibly prone to morbidity and mortality from ingestion. Initial stabilization of ABCs is standard regardless of the toxin ingested. Identification of the toxin should be of next importance to determine whether interventions to alter the absorption, enhance the elimination, or administer an antidote will be most beneficial. Therefore, a good history is paramount after initial stabilization, paying special attention to any and all medications in the home and where the child was found. In an undifferentiated patient, sepsis and trauma are equally common and should be investigated concurrently. With an identified toxin, coordination of care with local poison control centers will result in the best possible outcome for the child. Similarly, in a well-appearing child with ingestion or suspected ingestion, local poison centers can provide observation and management guidelines and allay the fears of worried parents and providers. Prevention is key in pediatric poisoning and any "close-call" can be a valuable opportunity for parental education to prevent a second visit.

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CME/CE Questions

1. Which of the following can cause severe toxicity in a child with a single dose?
   a. Acetaminophen
   b. Metoprolol
   c. Ciprofloxacin
   d. Aspirin
   e. Diphenhydramine
2. A 6-year-old boy was found playing outside, and suddenly became confused and "twitching." On physical exam he is noted to be diaphoretic, bradycardic, drooling, and vomiting. His symptoms are consistent with which toxidrome?
   a. Sympathomimetic
   b. Anticholinergic
   c. Cholingeric
   d. Opioid
   e. Serotonin syndrome
3. The majority of accidental ingestions in the pediatric population occurs in which of the following age ranges?
   a. Birth to 1 year
   b. 1 to 3 years
   c. 4 to 6 years
   d. 8 to 12 years
   e. 13 to 17 years
4. A 3-year-old girl presents after ingestion of "one of grandma's pills." The child has respiratory depression, hypotension, pinpoint pupils, and is unresponsive. What medication should be given immediately to attempt reversal of the ingested agent?
   a. Deferoxamine
   b. Pyridoxine
   c. Flumazenil
   d. Naloxone
   e. Glucagon
5. For which of the following toxin/drug toxicities will hemodialysis be most effective?
   a. Amlodipine
   b. Heparin
   c. Glyburide
   d. Lithium
   e. Levothyroxine
6. A 7-year-old boy presents to the ED with confusion, agitation, dry mouth, and blurred vision. The mother is prescribed amitryptiline, and she thinks "he may have taken some." What finding is common on ECG?
   a. Wide QRS
   b. Short QTc
   c. T wave inversion
   d. J wave
   e. ST depression

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PEDIATRIC EMERGENCY MEDICINE **REPORTS**

Practical, Evidence-Based Reviews in Pediatric Emergency Care

An Approach to Household Toxicological Emergencies in the Pediatric Patient

Recognizable Toxidromes[3,20,24]

Toxidrome	Signs						Examples
	Vital Signs	Mental Status	Pupils	Skin	Bowel Sounds	Other	
Sympathomimetic	Hypertension, tachycardia, hyperthermia	Agitation, psychosis, delirium, violence	Dilated	Diaphoretic	Normal to increased		Amphetamines, cocaine, PCP, bath salts (cathinones), ADHD medication
Anticholinergic	Hypertension, tachycardia, hyperthermia	Agitated, delirium, coma, seizures	Dilated	Dry, hot	Diminished	Ileus urinary retention	Antihistamines, tricyclic antidepressants, atropine, jimson weed
Cholinergic	Bradycardia, blood pressure and temperature typically normal	Confusion, coma, fasciculations	Small	Diaphoretic	Hyperactive	Diarrhea, urination, bronchorrhea, bronchospasm, emesis, lacrimation, salivation	Organophosphates (insecticides, nerve agents), carbamates (physostigmine, neostigmine, pyridostigmine), Alzheimer's medications, myasthenia treatments
Opioids	Respiratory depression, bradycardia, hypotension, hypothermia	Depression, coma, euphoria	Pinpoint	Normal	Normal to decreased		Methadone, buprenorphine, morphine, oxycodone, heroin
Sedative–hypnotics	Respiratory depression, heart rate normal to decreased, blood pressure normal to decreased, temperature normal to decreased	Somnolence, coma	Small or normal	Normal	Normal		Barbiturates, benzodiazepines, ethanol
Serotonin syndrome (similar findings with neuroleptic malignant syndrome)	Hyperthermia, tachycardia, hypertension or hypotension (autonomic instability)	Agitation, confusion, coma	Dilated	Diaphoretic	Increased	Neuromuscular hyperexcitability: clonus, hyperreflexia (lower extremities > upper extremities)	SSRIs, lithium, MAOIs, linezolid, tramadol, meperidine, dextromethorphan
Salicylates	Tachypnea, hyperpnea, tachycardia, hyperthermia	Agitation, confusion, coma	Normal	Diaphoretic	Normal	Nausea, vomiting, tinnitus, ABG with primary respiratory alkalosis and primary metabolic acidosis; tinnitus or difficulty hearing	Aspirin and aspirin-containing products, methyl salicylate
Withdrawal (sedative–hypnotic)	Tachycardia, tachypnea, hyperthermia	Agitation, tremor, seizure, hallucinosis, delirium tremens	Dilated	Diaphoretic	Increased		Lack of access to ethanol, benzodiazepines, barbiturates, GHB, or excessive use of flumazenil
Withdrawal (opioid)	Tachycardia	Restlessness, anxiety	Dilated	Diaphoretic	Hyperactive	Nausea, vomiting, diarrhea	

Potential Toxic Ingestions Based on Abnormal Presenting Vital Signs[24]

Temperature	Heart Rate	Respiratory Rate	Blood Pressure	Mental Status	Potential Ingestions
High	High	High/normal	High	Agitated or obtunded	Anticholinergic Sympathomimetics Nicotine Tricyclics Antihistamines
Low	Low/normal	Low	Low/normal	Sedate or comatose	Ethanol Opioids Clonidine Benzodiazepines Beta-blockers Calcium channel blockers

Source: Author adapted.

Xenobiotics That May Cause Severe Toxicity to an Infant After a Small Adult Dose, a Single Pill, or a Small Volume[2,16-18]

- β-adrenergic antagonists (sustained-release)
- Benzocaine
- Bupropion
- Calcium-channel blockers (sustained-release)
- Camphor
- Clonidine
- Cyclic antidepressants
- Diphenoxylate and atropine (Lomotil)
- Methanol or ethylene glycol
- Methyl salicylate
- Opioids (buprenorphine, codeine, methadone, oxycodone)
- Pesticides/herbicides/rodenticides
- Phenothiazines (especially chlorpromazine, thioridazine)
- Quinine or chloroquine (antimalarials)
- Sulfonylureas
- Theophylline

Suggested Diagnostic Evaluation for Undifferentiated Poisoned Child

- Complete blood count
- Electrolytes
- Blood urea nitrogen/creatinine
- Liver function tests
- Ethanol and other toxic alcohol screen
- Rapid urine drug screen
- Electrocardiogram
- Serum osmolarity
- Arterial blood gas
- Salicylates
- Anticonvulsants (phenytoin, valproic acid, carbamazepine, etc.)
- Acetaminophen
- Iron
- Digoxin
- Lithium

Selected Toxin/Antidote Pairings[40]

Toxin	Antidote
Acetaminophen	N-acetylcysteine
Anticholinergic poisoning	Physostigmine
Benzodiazepines	Flumazenil
β-blocker	Glucagon Insulin/glucose
Calcium-channel blockers	Calcium chloride Glucagon Insulin/glucose
Cholinergic poisoning	Atropine Pralidoxime
Digoxin	Digoxin-immune Fab
Ethylene glycol	Fomepizole
Iron	Deferoxamine
Isoniazid	Pyridoxine
Methemoglobin	Methylene blue
Opiates	Naloxone
Sulfonylureas	Glucose Octreotide
Tricyclic antidepressants	Sodium bicarbonate

Source: Author adapted.

Supplement to *Pediatric Emergency Medicine Reports*, November 2016: "An Approach to Household Toxicological Emergencies in the Pediatric Patient." Authors: Aaron N. Leetch, MD, Assistant Professor of Emergency Medicine & Pediatrics, Assistant Residency Director, EM and EM/Peds Programs, Banner University Medical Center, Tucson, AZ, and Molly Wormley, MD, Chief Resident, University of Arizona, Tucson.