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## Management of toxic exposure in children

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The belly becomes extremely bound, neither answering to the most drastic purges or sharpest clysters, the latter coming off without wind or stool, the former being soon vomited.

—John Huxam, 1759, on lead poisoning

The recognition of poisons and poisoning dates back thousands of years. The potential magnitude of impact on children's health was not apparent until 1952, when the American Academy of Pediatrics (AAP) reported more than 50% of childhood accidents in the United States involved toxic ingestions. The AAP's study led to the development of the first US poison control center in Chicago the next year. Nearly 50 years later, 61 poison control centers are operating, and the field of medical toxicology has become a formal subspecialty with representatives from the disciplines of preventive medicine, occupational medicine, emergency medicine, and pediatrics. Concentrated expertise has enhanced the ongoing need for accurate direction in toxicology.

This article provides the practitioner with information regarding assessment and management of children who present to the emergency department (ED) with a suspected toxic exposure. The text concentrates on ingestions and emphasizes the potential differences following exposure in the pediatric population as compared with adults.

### Addressing an ongoing problem

More than 4 million poisoning cases are reported annually to poison centers throughout the United States. Most of these cases occur in the pediatric population. Pediatric encounters with nonpharmaceutical agents

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and medications remain a major cause of injury-related morbidity and mortality in the United States [1,2]. Some contrasting features of acute pediatric ingestion are relevant for those who provide medical intervention.

The age of the patient, behavioral events, and physical factors within an environment contribute to the spectrum of toxic encounters. Preteens and teenagers, through their employment or drug experimentation, may be exposed to toxins [3]. Exploratory behavior rather than purposeful harm generally brings this age group to medical attention. It is appropriate, however, to consider suicidal gesture or frank suicide attempt in those who present for ED care older than 9 years. Intentional ingestion becomes more likely after age 5 [4]. For children younger than age 5, especially those aged 2 years and younger, unintentional behavior on the part of the child and parent are typically blamed for intoxication. Improper storage of substances in the home, increasing childhood mobility, and a transient distraction of caregivers is the classic triad leading to toxic ingestion. Children younger than 2 years may be unable to discriminate safe from unsafe liquids, particularly if the agent is stored in a recognized container. They fail to recognize the suitability of the liquid and may consume ingredients before taste aversion leads to discontinuing the drink. A young child may also mistake a medication for an edible ingredient, such as candy. Children younger than 2 years may observe the ritual of self-medication in other family members and imitate that behavior. Intoxications in children younger than 1 year may reflect accidental administration of an incorrect drug or dose by the caretaker [5], administration of a pharmaceutical by an older sibling, or parental abuse [6]. Very young children are also subject to toxicity from passive exposure to agents such as smoke from marijuana and “crack” cocaine [7].

### *Preventative strategies*

Most pediatric intoxications occur in the home. Approximately 15% of exposures occur outside the home, such as a day care center or a sitter’s residence, including relatives [8,9]. The substance ingested reflects the availability of the product [10]. Most of these ingestions are of a single household product, plant, food supplement, or medication. Most of these unintentional encounters result in mild or no symptoms, and significant morbidity or mortality is uncommon. Polypharmaceutic ingestion, with increased morbidity, is facilitated at homes of grandparents, who may inadequately shield their anticholinergic, antihypertensive, dysrhythmic, or psychoactive medications. In addition, access to illicit products manufactured at home or in clandestine laboratories may result in a more severe expression of toxicity [11].

Preventive strategies enacted by federal and state governments, organized physician groups, and individual clinicians have been enacted. Communal preventive strategies have decreased the incidence of poisoning. The 1960 passage of the Federal Hazardous Substance Labeling Act, which required

proper labeling of products, was the first of these strategies. Child-resistant caps, mandated by the Poison Prevention Packaging Act of 1970, have significantly reduced morbidity and mortality from ingestions. Poison control center sticker trials, community programs, clinic- and office-based counseling also have had a measured impact. As a result, there has been a significant decline in the number of pediatric poisoning deaths: 216 in 1972 versus 25 in 1997 [2].

Individual clinicians have succeeded in the role of preventive health care. Emergency physicians (EPs) are not excused from preventive health practices, however. Secondary preventive efforts on the part of EPs regarding ingestions have the potential to improve the future health of children. After sensing the urgency, determining the need for monitoring, and early aggressive intervention when indicated, the EP should explore the social climate for any child brought for the evaluation of an ingestion. If the EP uncovers concerns and provides resources for socially isolated, medically, or psychologically ill parents, he or she may prevent a repeat ingestion [12–15].

### **Initial history and physical examination**

An occult ingestion may occasionally result in an alarming deterioration in health status. A child may then be brought to the ED by a caretaker unaware of the toxic exposure. Equally as uncommon, a parent may bring a child for evaluation after the parent has witnessed the ingestion. Most often, the suspicion of ingestion by a caretaker is a precipitating event for the ED visit.

#### *Patient history*

When not distracted by a patient's tenuous state, the EP benefits from a focused patient history. Information should be sought from emergency medical services, the accompanying caregiver, and, if potentially helpful, by telephone inquiry to others at the site of the incident. Obtaining a reliable history may be crucial to subsequent management.

Management of intoxication may be facilitated by identification of the substance. Knowing the agent ingested and understanding the pharmacology enables appropriate initial decision making. There are a limited number of pharmaceuticals, household products, and plants that are toxic for children (Boxes 1 and 2). Knowing that an exposure includes a substance that is less toxic or nontoxic obviates immediate therapeutic interventions.

A second key element in decision making is the quantity of substance ingested. For many intoxicants, there may be a direct relationship with the volume consumed and the likelihood of adverse clinical manifestations. For various intoxicants, a minimum quantity (mg/kg) that leads to symptoms

### **Box 1. Pharmaceuticals toxic for children**

#### *Analgesics*

Acetaminophen  
Nonsteroidal anti-inflammatory drugs  
Salicylates

#### *Anesthetics*

Benzocaine  
Lidocaine

#### *Anticholinergics*

Cyproheptadine  
Diphenhydramine  
Dimenhydrinate  
Hydroxyzine  
Hyoscyamine  
Orphenadrine  
Scopolamine

#### *Anticonvulsants*

Barbiturates  
Carbamazepine  
Phenytoin

#### *Antidepressants/psychotics*

Chlorpromazine  
Clozapine  
Cyclics  
Lithium  
Monoamine oxidase (MAO) inhibitors  
Sertraline  
Thioridazine

#### *Antihypertensives/dysrhythmics*

Captopril  
Clonidine  
Digoxin  
Nifedipine  
Verapamil

#### *Antimalarials*

Chloroquine

Quinines

#### *Antituberculosis*

Isoniazid

#### *Bronchodilators*

Albuterol  
Caffeine  
Ephedrine  
Theophylline

#### *Fluoride*

Ammonium fluoride,  
bifluoride

#### *Hypoglycemics*

Sulfonylureas

#### *Iron*

Prenatal hematinics

#### *Methylxanthines*

Caffeine  
Theophylline

#### *Opioids*

Codeine  
Diphenoxylate  
Hydrocodone  
Methadone  
Oxycodone  
Pentazocine  
Propoxyphene

#### *Sedatives*

Triazolam

#### *Sympathomimetics*

Nasal/ocular imidazoline  
Amphetamines  
Cocaine  
Phencyclidine  
Phenylpropanolamine  
Pseudoephedrine

*Adapted from Emery D, Singer J. Highly toxic ingestions for toddlers: when a pill can kill. Pediatr Emerg Med Rep 1998;3:111-22.*

and the amount that leads to lethality in 50% of animals or children, or LD<sub>50</sub>, may be known. Predicting the amount consumed may be problematic [46]. For prescribed pills, projecting the amount missing may be possible by subtracting from the expected, remnant number. For liquids, a missing volume may be estimated. Unfortunately, pills or liquid may be unaccounted for when spilled at the scene. Also, there is no guarantee that the labeled content of the container was consistent with the pill or liquid that disappeared. An assessment of quantity consumed is facilitated if the ingestion was witnessed. The volume of a swallow is a function of body mass. The volume of a swallow is 0.27 mL/kg, or roughly 5 cc for a 2-year-old and 20 cc for an adolescent [16]. When two children have potentially coingested a substance, the mg/kg consumption should be calculated based on the principle that each child has taken all the substance and the other none.

The elapsed time since ingestion should provide insight as to the likelihood for deterioration for most pharmaceuticals and nonpharmaceuticals. Intoxication is unlikely if no clinical effects have occurred for those substances typically associated with prompt clinical change. Predicting clinical effects for substances with delayed manifestations, however, is not possible. In any case, the timing should be pursued. The elapsed time since ingestion may determine the use of charcoal, gastric emptying techniques, or specific antidotal therapy.

The route of exposure is also important for the patient and medical caretakers. Besides the most common route of oral toxic exposure, inhalation, ocular, or dermal contamination is possible. Patients who have been exposed to concentrated lipid-soluble compounds require skin decontamination. Special caution is necessary for organophosphate insecticides, which may be present in high enough quantities on skin, in sweat, and in bodily secretions of patients that serious sequelae can occur if proper protection of the staff is not accomplished.

A cause for the ingestion also must be pursued. Dismissing an ingestion as an accident in an “accident-prone” preschooler may lead to further injury or repeat ingestion. Multiple environmental factors can distract even a well-adjusted, supervisory figure; however, the EP should seek stressors, such as the following: ill health, including psychiatric disturbances; marital discontent and separation; spousal abuse; and unemployment or other financial burdens. If support is not offered to a dysfunctional family, a recurrence of ingestion or injury becomes more likely. Further inquiries should be driven by the age of the ingestor. The physician must determine if any discrepancy exists between the proffered history of ingestion with the developmental stage of the patient. Immobility and rudimentary hand-to-mouth coordination generally prevent self-poisoning by very young children and infants. Poisoning by an older sibling or parent should be considered for an ingestor who is younger than 1 year. For the elementary school child, the physician needs to inquire about academic underachievement

**Box 2. Household products and plants toxic for children***Acid/alkali*

Boric acid  
Bowl cleansers  
Clinitest tablet  
Disc battery

*Alcohols*

Ethanol  
Ethylene glycol  
Isopropyl alcohol  
Methanol

*Antiseptics*

Camphor  
Hydrogen peroxide  
Phenol  
Pine oil

*Cyanide**Hydrocarbons*

Aliphatics  
Aromatics

*Industrial chemicals*

Burtyrolactone (solvent for  
acrylate polymers)  
Methylene chloride (paint  
thinner)  
Selenious acid (gun blueing)  
Zinc chloride (soldering flux)

*Mothballs*

Naphthalene

*Nail products*

Acetone (polish remover)  
Acetonitrile (sculptured nail  
remover)  
Methacrylic acid (artificial nail  
primer)  
Nitromethane (artificial nail  
remover)

*Organophosphates*

Carbamate

*Plants*

Aconite  
Cantharidin  
Castor bean  
Clove oil  
Comfrey  
Fox glove  
Na Hwang  
Mushrooms, specific  
Nutmeg  
Oleander  
Pennyroyal oil

*Rodenticides*

Arsenic  
Hydroxycoumarin  
Indanediones  
Strychnine

*Weed/bug killers*

Lindane  
Nicotine  
Paraquat

*Adapted from Emery D, Singer J. Highly toxic ingestions for toddlers: when a pill can kill. Pediatr Emerg Med Rep 1998;3:111–22.*

and school avoidance, including truancy, as these events may reflect underlying pediatric psychopathology. Substance abuse, alcohol abuse, or feared pregnancy may precipitate an ingestion from the age of 9 through the teenage years. Failure to inquire about the possibility of suicide gesture or

attempt may lead to inappropriate disposition and inadequate long-term support.

The pursuit of past medical history should include prior ingestions and injuries requiring medical attention, such as fractures or lacerations. Positive responses to these screening questions suggest that the current ingestion may reflect more than a chance occurrence [14].

### *Physical examination*

Physical signs of pediatric intoxication on admission to an emergency facility are highly variable. They depend on route of exposure, time and therapeutic intervention since exposure, extent of the exposure, type of product, and patient age.

As a rule, clinical consequences of a specific intoxication in the adult and pediatric populations are similar. The undesirable side effects and physical findings seen in adults from a class, product, or category of drug are also seen in children. Some of the impact of an intoxicant on a child may be subtle or, conversely, magnified as compared with adults because of peculiarities in metabolic degradation, pediatric anatomy, or physiology.

In pediatric patients, airway resistance is greater, cardiac output is very dependent on heart rate, and cardiovascular instability may be hidden by “normotensive” blood pressure readings. Young infants are very susceptible following ingestion to thermoregulatory problems, such as hypothermia and hyperthermia and including neuroleptic malignant syndrome [10]. In the first few years of life, mechanisms that typically distort a mental status (eg, hypoglycemia, electrolyte abnormality) or create focal findings may be masked by a reduced pediatric neurologic repertoire.

### **Presenting symptoms and signs**

Most pharmaceuticals and household agents deemed highly toxic for children create disabling alterations of mental status and vital signs [17].

### *Neurologic symptoms*

The central and autonomic nervous system findings may be insidious (opioids, benzocaine, acetaminophen), abrupt (lidocaine, monocyclic and tricyclic antidepressants, theophylline, phenothiazines), transient (hydrocarbons), or fluctuate with alternating excitation and depression (imidazolines, phencyclidine, clonidine). The manifestations can arise directly from neurotoxicity (sympathomimetics, nicotine) or indirectly from hypoxia (oxidizing agents), hypoglycemia (alcohols, salicylates, hypoglycemics,  $\beta$ -blockers), electrolyte disturbance (theophylline, salicylates), hypoperfusion (iron, calcium channel blockers), or acidemia (salicylates, alcohols).

In adults, toxic substances tend to provoke a clearly defined nervous system alteration that is either excitatory or depressed. Toxic stimulation in an adult may be readily recognized. The poisoned adult may become restless, seem anxious or disoriented. On questioning the adult, his or her confusion may be readily apparent. Adults may be able to articulate abnormal sensations, such as paresthesias, difficulty in hearing, visual disturbances such as hallucinations, headache, dizziness, and other abnormal sensations. cursory observation by the clinician may uncover generalized increased motor activity, motor excitation, or obvious dystonia.

Most intoxicants that are excitatory in an adult create a similar physiology in the child, but the clinical expression may be more difficult to discern. Many cynical, adult-oriented EPs believe that “normal behavior” in a toddler includes restlessness, ineffectual motor activity, rapid task changing, and incomprehensible articulation. Wise physicians, however, who possess knowledge of age-dependent variations of norm may recognize an excitatory toxic ingestion from subtle findings that include increased motor tone, tremor, and self-injurious behavior, including repetitive picking of the skin or uncontrolled tongue thrusting. The inability to sit upright, grasp or transfer objects, outright refusal to stand and ambulate, or a broad-based gait may be more prominent in young patients and be more obvious than an ataxic dysarthric-producing ingestion in an adult. Repetitive twitching or jerking movements suggest muscular irritability or a dystonic equivalent in a child.

Intoxicants that depress the sensorium in adults produce the same effects in children. The clear progression from subjective sleepiness, apparent drowsiness, outright lethargy, to frank coma in adults is mirrored in school-aged and older children. In the preschool-aged population, the change in sensorium and motor tone are typically accelerated. Toddlers are much more prone to rapid obtundation.

The intoxicants classically leading to seizure in adults may also cause seizures in children [18] (Box 3). The seizure activity is more likely in a child than in an adult exposed to several products including nicotine, salicylates, camphor, and cocaine [19,20]. In addition, children are uniquely predisposed to seizure as a neurotoxic reaction associated with oral ingestion of diethyltoluamide [21], camphor, ammonium fluoride, and lidocaine [22].

### *Vital signs*

In adults, specific toxic ingestion may be suspected from changes in vital signs. Various intoxicants produce characteristic alterations in temperature, heart rate, respiratory rate, and blood pressure. In the pediatric population, vital sign alterations with various intoxicants may also assist in diagnosis.

Opiates, sedative hypnotics, hypoglycemics, and alcohols classically cause hypothermia. Hypothermia with the pediatric ingestion from these

**Box 3. Intoxicants causing pediatric seizures**

Ammonium fluoride	Ibuprofen
Amphetamines	Imidazolines
Anticholinergics	Isoniazid
Antihistamines	Lead
$\beta$ -blockers	Lidocaine
Caffeine	Lindane
Camphor	Lithium
Carbamates	Lysergic acid
Carbon monoxide	Nicotine
Chlorinated insecticides	Opioids
Cocaine	Phencyclidine
Cyclic antidepressants	Phenothiazines
Diethyltoluamide	Phenylpropanolamine
Dilantin	Physostigmine
Ergotrate	Propoxyphene
Ethanol	Salicylates
$\gamma$ -hydroxy butyrate	Strychnine
Hydrocarbons	Theophylline
Hypoglycemics	

substances may result from exposure or altered metabolic activity. Adult and pediatric hyperpyrexia is common following intoxication with amphetamines, anticholinergics,  $\beta$ -blockers, isoniazid, and sympathomimetics. Salicylate intoxication in the pediatric population consistently leads to hyperthermia. In children, the impact of cyclic antidepressants on temperature regulation is variable.

Anticholinergics, antihistamines, amphetamines, sympathomimetics, cyclic antidepressants, cyanide, propoxyphene, and theophylline create tachycardia. Lower doses on a mg/kg basis of iron create tachycardia in pediatric patients. Bradycardia with pediatric ingestion is similar to adults for sedative hypnotics, calcium channel blockers, clonidine,  $\beta$ -blockers, opiates, digitalis, nicotine, and alcohols.

An increased respiratory rate following ingestion may result from a toxin-induced metabolic acidosis, noncardiogenic pulmonary edema, or direct pulmonary insult. Pediatric patients appear to be more susceptible to pulmonary insult compared with adults following exposure to hydrocarbons, organophosphates, and salicylates. Bradypnea is more common in young patients following ingestion of acetone, barbiturates, ethanol, ibuprofen, nicotine, and sedative hypnotics. Following clonidine ingestion, respiratory depression, including apnea, are more pronounced in children [23].

Sedative hypnotics, heroin, and methadone disproportionately reduce blood pressure in children. ACE inhibitors, calcium channel blockers,

digoxin, nitrites,  $\beta$ -blockers, imidazolines, cyclic antidepressants, theophylline, propoxyphene, and quinidine may produce profound hypotension. Sympathomimetics, amphetamines, phencyclidine, anticholinergics, nicotine, and cocaine raise blood pressure in children. Delayed hypertension may result from pediatric ingestion of thyroid supplements.

### **Early versus delayed manifestations**

As a rule, the products of moderate or severe toxicity create signs and symptoms in pediatric patients within a 2- to 4-hour period of exposure. There are cases, however, that result in clinical sequelae beyond this time frame. Delayed manifestations result from the formation of a toxic metabolite or from delayed onset, peak, or duration of a toxin.

Intoxicants that typically result in delayed onset of symptoms include those that are enteric coated. For example, enteric-coated salicylates permit continued absorption and delayed toxicity [24]. Sustained-release preparations taken in excess also have delayed impact. Products that have shown delayed toxicity include acetaminophen, calcium channel blockers, lithium, and theophylline. Substances that produce physiologic slowing of the gastrointestinal tract or pylorospasm cause delayed pediatric toxicity. Among these are medications with anticholinergic properties (eg, benzotropine mesylate, carbamazepine, dimenhydrinate, diphenhydramine, cyproheptadine, hyoscyamine, hydroxyzine, oxybutynin, and scopolamine), opioids, and sedatives. Delayed toxicity can result from delayed absorption caused by drug concretion. Examples include sustained-release theophylline and sustained-release verapamil [25]. Several pharmaceuticals, chemicals, and plants have delayed manifestations because they are biotransformed to an active product and redistributed to a vulnerable organ, contributing to delayed toxicity (Table 1). In certain circumstances, delayed manifestations may occur and the mechanism is unknown. Examples include delayed central nervous system depression with diphenoxylate/atropine, hypoglycemia with sulfonylureas, and latent dysrhythmia with cyclic antidepressants.

### **Pediatric fatalities**

Most household products, plants, and medications are either nontoxic or have limited toxicity for children. The products of moderate toxicity cause physiologic changes only after high-dose ingestion. Severely toxic products cause devastating effects on a single organ system or affect multiple organ systems. The highly toxic substances also have pronounced pharmacocactivity on individuals with a smaller body mass.

The most frequently fatal pharmaceutical ingestions in children have been prenatal iron supplements, antidepressants, cardiotoxic agents, and

Table 1  
Biotransformed intoxicants with latent manifestations

Substance	Manifestations	Mechanism
Acetaminophen	Hepatotoxicity	Depletion glutathione stores
Acetonitrile	CNS, CV	Cyanide production
Amatoxin	Liver, renal failure	Enzyme depletion
Brodifacoum	Anticoagulation	Compromised vitamin K
Ethylene glycol	Metabolic acidosis	Glycoaldehyde
Fluoride	Dysrhythmia	Hyperkalemia
Isopropanol	CNS, GI	Acetone production
Methanol	Metabolic acidosis	Accumulation formic acid
MAO inhibitors	Hyperadrenergic	Built-up transmitters
Nitromethane	CNS	Methemoglobinemia
Organophosphates	SLUDGE	Excess acetylcholine
Pennyroyal oil	Hepatotoxicity	Accumulation methofuran

*Abbreviations:* CNS, central nervous system; CV, cardiovascular; GI, gastrointestinal; MAO, monoamine oxidase.

salicylates [1]. Hydrocarbons, alcohols, cleaning substances, pesticides, and gun-blueing agents have been the most commonly reported fatal nonpharmaceutical ingestions in children [26].

#### *When a pill or a swallow can kill*

A small group of pharmaceutical and household products can create life-threatening effects when ingested in very small quantities [27]. Approximately 24 agents have the potential to be fatal to individuals with small body mass, such as a child younger than 2 years [22]. The pharmaceutical and household products capable of killing a toddler after consumption of 1 to 2 tablets or 1 to 2 tsp of a marketed-dose unit are listed in Box 4.

### **Selected management issues**

Most pediatric patients who present to the ED are successfully managed with the maintenance of airway, ventilation, oxygenation, cardiovascular, and fluid status. This supportive care is more critical than specific toxicologic management techniques, such as gastric decontamination, enhanced elimination, and antidotes. The latter techniques have been shown to be only selectively of benefit in adults and children. The recommendations for pediatric patients continue to evolve.

#### *Gastrointestinal decontamination*

Gastrointestinal decontamination, a well-established toxicologic technique for asymptomatic and symptomatic patients, has fallen in disfavor. In asymptomatic adults, decontamination has been shown to be of no benefit [28]. In asymptomatic children, evidence of improved outcome following

**Box 4. Products with lethal potential for very young patients**

Acetonitrile  
Ammonium fluoride  
Benzocaine  
Brodifacoum (others)  
Butyrolactone  
Camphor  
Chloroquine  
Chlorpromazine (others)  
Clozapine  
Desipramine (others)  
Diphenoxylate  
Hydrocarbons  
Hyoscyamine sulfate  
Imidazoline products  
Lindane  
Methadone  
Methanol  
Methyl salicylate  
Pennyroyal oil  
Quinine  
Salt  
Selenious acid  
Theophylline

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*Adapted from Emery D, Singer J. Highly toxic ingestions for toddlers: when a pill can kill. Pediatr Emerg Med Rep 1998;3:111–22.*

ingestion also has not been shown. Decontamination has been suggested, however, for the asymptomatic child who is at theoretic risk for significant toxicity [29]. If the pediatric patient has ingested a potentially life-threatening amount of a poison and decontamination can be undertaken promptly, orogastric lavage, activated charcoal, or whole bowel irrigation can be considered [30–32].

Orogastric lavage may be useful for a substance known to delay gastric emptying or to form a concretion. It is an acceptable method for urgent removal of a solid or liquid substance for a child with an adequate gag reflex, where the airway need not necessarily be protected. The child should be placed in the Trendelenburg left-lateral decubitus position and monitored during tube passage. The largest bore possible should be used. A 16- to 28-French (F) tube should be used in a child, and at least a 36-F tube should be used in an adolescent. After tube placement is confirmed, normal saline is introduced in 50 to 100 cc aliquots in young children and up to 200 cc in

adolescents [33]. Fluid is withdrawn by either aspiration or by gravity drainage. Lavage performed in this fashion has been shown to decrease the mean serum bioavailability of specific toxins [34]. Two points are important: recovery of all accounted-for intact tablets does not ensure that some quantity of drug has already been absorbed [35]. Second, lavage continued until the effluent is clear does not ensure that all of the gastric intoxicant has been retrieved. Toxic overdoses that may benefit from lavage include tricyclic antidepressants, calcium channel agonists, or toxins that are not bound by charcoal, such as iron, lithium, and alcohols. If activated charcoal also is to be administered, it should be infused after the last effluent is acquired [36].

Activated charcoal may be given after gastric evacuation. The greatest benefit occurs when it is administered within an hour of ingestion. There are insufficient data to support or exclude its use 1 hour after ingestion [30]. The recommended dose of charcoal is 1 g/kg in children up to the age of 1 year, 25 to 50 g in children aged 1 to 12 years, and 25 to 100 g in adolescents [29]. Activated charcoal may also be administered without gastric emptying procedures. Charcoal may be given by nasogastric tube or offered as a flavored slurry. The addition of chocolate milk, cola, or cherry-flavored syrup improves both the taste and ease of swallowing for children [37].

Whole bowel irrigation has greatest utility for potentially toxic ingestions of sustained-release or modified-release pharmaceuticals (eg, calcium channel antagonist) that have delayed absorption [38]. Whole bowel irrigation has been shown to be effective when initiated within 4 hours after the ingestion of enteric-coated aspirin [39]. Whole bowel irrigation may enhance passage of whole pill fragments that have passed beyond the pylorus (eg, radiographically documented iron tablets). Whole bowel irrigation may also be the preferred route of decontamination for medications that are not absorbed by charcoal. A polyethylene glycol electrolyte solution is offered by mouth or, when declined, by nasogastric tube. For children up to age 6, the recommended rate is 0.5 L/hour; for children aged 6 to 12 years, 1 L/hour; and for children older than 12 years, the recommended rate is 1.5 to 2 L/hour [32].

### *Enhanced elimination*

Supportive care that sufficiently sustains vital functions in pediatric patients can be provided while the clinician awaits the normal process of drug elimination. In certain circumstances, efforts to adsorb and increase the enteric or renal excretion of a drug may improve patient outcome. Two methods that can improve recovery, and which may be begun in an emergency setting, are multiple dosing of activated charcoal and urinary alkalization.

There have been several reports on the use of multiple-dose activated charcoal in the pediatric population. An accelerated rate of drug clearance

has been shown for many drugs with enterohepatic recirculation [40]. The current recommendations include its use for life-threatening ingestions of carbamazepine, dapsone, phenobarbital, quinine, digoxin, and sustained-release preparations [10,41]. A dose of 5 to 10 g every 3 to 4 hours is recommended [40]. Multiple doses should be administered only if the patient has an intact gag reflex, a protected airway, and no evidence of adynamic ileus or mechanical bowel obstruction.

Forced diuresis in association with alkalinization of the urine can enhance elimination of salicylates and phenobarbital. The method can be employed when the patient has a potentially life-threatening amount of either drug, is hemodynamically stable, has normal renal function, and has no evidence of cerebral or pulmonary edema. The goal is to keep the urinary pH at greater than 7.5. This can be accomplished by intermittently providing 1 to 2 mEq/kg of sodium bicarbonate intravenously over a 1 to 2 hour time frame [33]. An alternative method is to provide a renewable, continuous intravenous infusion of sodium bicarbonate every 4 hours based on clinical and laboratory reassessment. The following formula is used:

$$0.6 \text{ (the bicarbonate space)} \times \text{patient weight (kg)} \times 5 \text{ mEq} \\ = \text{the mEq of sodium bicarbonate to be provided over the next} \\ 4 \text{ hours. Add the total mEq to a 4-hour intravenous drip of} \\ \text{glucose with added potassium chloride.}$$

### *Antidotes*

Antidote administration may supplement supportive care and, in certain circumstances, prevent or reverse toxicity. The benefit of an antidote is derived from its mechanism of action. Some antidotes displace an intoxicant from a site of action. Some examples are oxygen for carbon monoxide, naloxone for opiates, and vitamin K preparations for oral anticoagulants. An antidote also may act in competition at a receptor site, such as atropine with acetylcholine. Other antidotes, such as *N*-acetylcysteine, restore function by repair or by bypassing the production of toxic metabolites. Several antidotes hasten the excretion of a detoxification complex. Examples include deferoxamine-ferric iron and cyanmethemoglobin.

The acute intoxications for which antidotes are a proven benefit are few. Much of the information has been derived from studies on adults; however, there is reasonable strength of evidence for pediatric recommendations [42]. Table 2 selectively lists antidotes for pediatric intoxications.

### **Length of observation**

Several considerations come into play when analyzing the optimal length of observation for the potentially intoxicated child. The variables can be separated into categories, which include the quality of home care, the type of

Table 2  
Selected pediatric antidotes for acute intoxication

Toxin	Trigger for use	Antidote	Administration
Acetaminophen Liquid	> 225–250 mg/L at 2 h [46]	N-acetylcysteine	Oral, IV [48] (pending FDA approval), 140 mg/kg; 70 mg/kg q 4 h, at least 36 h [51]
Pills	> 150–200 mg/L at 4 h	Physostigmine	IV, 0.02 mg/kg over 5–10 min [11]
Anticholinergics	Confirm cause of altered mental status	Flumazenil	IV, 0.005–0.01 mg/kg at 0.2 mg/min rate; maximum 1 mg [50]
Benzodiazepines	Confirm cause of altered mental status	Glucagon	IV, 0.15 mg/kg bolus; 0.1 mg/kg/h maintenance
β-blockers	Cardiovascular toxicity	10% Calcium chloride solution	1–2 cc/kg over 5 min; repeat every 10–20 min
Calcium channel blockers	Cardiovascular toxicity		
Cyanide	Any manifestations	3% Sodium nitrite solution and 25% sodium thiosulfate solution	IV, 0.2–0.3 cc/kg over 5 min; maximum 10 cc
Ethylene glycol, methanol	Acidemia; > 20 mg/dL ethylene glycol	10% Ethanol or fomepizole	IV, 1–2 cc/kg over 1–2 min IV, 10 cc/kg; continuous 1.5 cc/kg/h IV, 15 mg/kg bolus; 10 mg/kg q 12 h × 4, 15 mg/kg q 12 h thereafter [38]
Iron	Serum iron > TIBC, or 350–500 mcg/dL	Deferoxamine	IM, 90 mg/kg; maximum 1 g, or IV, 10–15 mg/kg/h
Isoniazid	Neurotoxicity; > 40 mg/kg ingestion	Pyridoxine	IV, 75 mg/kg bolus; maximum 5 g [49]
Opioids	CNS depression	Naloxone	0.1–0.4 mg/kg bolus; 0.16 mg/kg/h continuous
Organophosphate	Nicotinic, muscarinic or CNS manifestations	Atropine and pralidoxime	IV, 0.05–0.1 mg/kg; repeat as necessary 25–50 mg/kg over 15–30 min; continuous 10–20 mg/kg/h up to 500 mg/h [47]
Oxidants	Methemoglobinemia, > 20%–30%	1% Methylene blue solution	1–2 mg/kg over 5 min; repeat 1 mg/kg [52]
Sulfonyleureas	Hypoglycemia	Octreotide	Subcut 1 mcg/kg every 12 h; IV, 15 ng/kg/min [28]

Abbreviations: CNS, central nervous system; IM, intramuscular; IV, intravenous.

Adapted from American College of Emergency Physicians. Clinical policy for the initial approach to patients presenting with acute toxic ingestion or dermal or inhalation exposure. Ann Emerg Med 1999;33:735–61.

ED facility, the medication or medications involved, and the patient's degree of functional derangement.

If a suboptimal home environment was a factor in the poisoning, the physician should not rely on a family to appropriately observe a child or return for repeat assessment. In this circumstance, the physician should perform a more lengthy observation. If, however, a reliable caregiver understands the potential for delayed consequences of a poisoning, has means of transportation, and is receptive to providing further observation, the length of ED observation may be shortened.

If the ED facility has a designated observation unit or physical environment that permits a dedicated physician and nursing staff to retain a patient, an extended observation is facilitated. If resources (eg, staffing, equipment, space, finances) preclude extended observation in an individual ED, the clinician may need to quickly decide whether the patient be discharged earlier, transferred to another health facility that can provide a higher order of observation, or admitted to the hospital.

The risk for toxicity logically drives the length of observation [43]. Patients determined to have a nontoxic unintended exposure may be discharged after the family situation is assessed and secondary preventative strategies have been applied. When historical information indicates exposure to a moderate intoxicant that would be expected to provide immediate clinical impact, and the patient is symptom free, extended observation is not warranted. When the theoretic time of peak toxicity has passed and there are no possibilities of delayed manifestations (eg, no concretion, extended release, or toxic metabolites formed) observation need not be extended. Asymptomatic patients with selected acute intentional ingestion can be released from medical observation before the accepted "routine" 6-hour observation [44,45].

Patients in whom a finite period of toxicity can be predicted and who manifest clinical change following exposure, should be observed until the risk for further deterioration has passed. Extended observation or admission may be prudent for the select group of products deemed highly toxic for young patients, and for the products with potential for delayed manifestations.

## **Summary**

Millions of children ingest household products and medications yearly. The continuous proliferation of new products and pharmaceutical agents makes it difficult for physicians to maintain a current command of toxicologic information. Multiple sources, including poison control centers, can provide information; however, EPs must be familiar with several agents that are either significant for their frequency or for their disproportionate potential for morbidity and mortality in pediatric patients. With this

select group of intoxicants, physicians must anticipate cardiovascular and pulmonary instability and rapid changes in central nervous system functioning. Appropriate supportive care requires monitoring of the following: vital signs, level of consciousness, airway control, ventilation and circulatory support, body temperature, urine output, and acid base balance. Once these concerns are addressed, prevention of further absorption, enhancing a product's elimination, and treatment with specific antidotes may enhance supportive care. Care is also likely to be enhanced if the EP recognizes the inherent differences (medically and socially) between adults and children of various ages. Definitive emergency care is completed only after the provision of a developmentally oriented preventive strategy.

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