

Pediatric Toxicology

Specialized Approach to the Poisoned Child

Diane P. Calello, MD^{a,b,c,*}, Fred M. Henretig, MD^d

KEYWORDS

- Detergent pods • ECG • Hemodialysis • Pediatric • Poisoning • Salicylate
- Supportive care

KEY POINTS

- Pediatric poison exposures most commonly occur in children 1 to 5 years of age and are exploratory in nature. In recent years, incidence and morbidity of these exposures have been increasing.
- Child abuse by poisoning should be considered when the patient is outside this age range, when multiple substances are involved, with recurrent episodes, and when the history is inconsistent with clinical picture.
- Because of inherent differences in physiology and pharmacokinetics, certain substances are more dangerous to young children than would be expected based on adult experience.
- Supportive care and adherence to resuscitation principles are the cornerstone of treatment in the poisoned child.
- The administration of antidotes and use of enhanced elimination techniques have specific implications in the young pediatric patient.
- Pediatric poison fatalities, although rare compared with adult statistics, are in many cases inherently preventable and involve the same substances year after year.
- Newer poison hazards include magnetic foreign bodies, laundry detergent pods, and button batteries. Continued toxicosurveillance is essential for awareness of emerging dangers.

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^a New Jersey Poison Information and Education System, Rutgers, the State University of New Jersey, New Brunswick, NJ, USA; ^b Department of Emergency Medicine, Morristown Medical Center, 100 Madison Avenue, Morristown, NJ 07960, USA; ^c Emergency Medical Associates Research Foundation, Parsippany, NJ, USA; ^d Section of Clinical Toxicology, Division of Emergency Medicine, The Poison Control Center, The Children's Hospital of Philadelphia, Perelman School of Medicine, University of Pennsylvania, 34th Street and Civic Center Boulevard, Philadelphia, PA, USA

* Corresponding author. Department of Emergency Medicine, Morristown Medical Center, 100 Madison Avenue, Morristown, NJ 07960.

E-mail address: dianepcalello@yahoo.com

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INTRODUCTION

A child is rushed into the emergency department (ED) by anxious parents after the witnessed ingestion of a household product or medication. Such a scenario unfolds nearly 90,000 times per year in the United States,¹ yet it remains a uniquely compelling event for all the actors involved: patient, family, and medical staff. In the most dramatic of cases, the child's life depends on the ED staff's ability to rapidly recognize the poisoning, institute life support, and provide definitive initial treatment. For most such visits, the family returns home within a few hours, after a period of benign observation, with perhaps a few laboratory tests obtained, or a dose of charcoal administered. Parents might even be advised that next time a quick call to the regional poison control center (PCC) would have obviated the ED visit in the first place. However, in every case, it is likely that the patient and family bear some lasting impression of their ED experience.² Young children fear strangers (especially physicians) and are made uncomfortable by even the prospect of the most minor medical interventions. Parents are the natural protectors and sources of comfort for their children when sick, and yet, in the ED setting they often feel obligated to serve in a quasi-professional helping role. In the context of childhood poisoning, they may also feel considerable anxiety about their child's outcome and guilt for allowing the incident to have occurred. In essence, they too are patients. Emergency providers (EPs) may appropriately dread having to draw blood, insert an intravenous (IV) line, or place a nasogastric (NG) tube into a screaming toddler, and would gladly omit such interventions if they were medically unnecessary. In the rare context of the critically ill poisoned child, EPs also welcome the knowledge and confidence to initiate potentially lifesaving treatment appropriately. This article therefore attempts to guide EPs confronted with the wide spectrum of pediatric exposures to potentially toxic substances, with a focus on exploratory ingestions in young children and selected toxins that have proved to be particularly dangerous in this age group. In addition, some attention is given to special pediatric topics, including particularly poisons that are deadly in small dose; child abuse by poisoning; pediatric medication errors; approach to the well-appearing child who may have ingested a toxic substance; and new (or resurgent) toxic household products and medication formulations.

CAUSE, EPIDEMIOLOGY, AND PREVENTION

Children may be poisoned by numerous mechanisms, including ingestion, inhalation, dermal contact, envenomation, and transplacental exposure. The focus of this article is on the most common of these mechanisms: ingestion. The ingestion of a nonfood, potentially poisonous substance by a young child typically represents a complex interplay of child-related, substance-related, and environmental factors.³ The term accidental ingestion was formerly used to describe these common events⁴⁻⁶ but has fallen out of favor and is now replaced by inadvertent, unintentional, or perhaps most properly, exploratory ingestion.⁷ This usage emphasizes the modern injury model that views injuries as predictable events based on several critical factors, not unlike the infectious disease model, with a victim (or host), agent (or microbe), and a conducive environment, as elucidated by Haddon in 1980.⁸ Typical poisoning victims are between 1 and 5 years of age, at a developmental stage that allows mobility and expression of normal exploratory behavior, yet too young to learn what is dangerous.⁹ They tend to be more hyperactive and impulsive, and more pica prone.^{5,10,11} Some agents are more likely to be ingested, either because of ease of access or attractiveness to the youngster.¹² A classic example was adult-intended iron tablets that simulated candy, were small, smooth-coated and easy for toddlers to swallow intact, were

available over the counter, and were typically prescribed to pregnant or postpartum women, who often have an older toddler-aged child in the home. As a result, acute iron poisoning had been one of the leading causes of childhood poisoning mortality until 1997, when the US Food and Drug Administration (FDA) required most iron preparations to be blister packaged (the regulation was subsequently suspended, but many manufacturers have voluntarily continued this practice, and pediatric iron-related morbidity has since remained low).¹³ A recent example of a new product that has proved enticing, and dangerous, to children is laundry detergent pods.^{14,15} Certain environmental changes or stresses are also highly poisoning-prone, including the arrival of a new baby, moving to a new home or apartment, parental illness or disability, and grandparent caretaking or visiting.^{16–19} The concordance of two or three such factors likely further increases the probability of exploratory ingestion.

Given the propensity to exploratory ingesting, many physicians (and parents) swear that “kids will eat anything.” The number of childhood ingestions are compelling, and the scope of drugs and nonpharmaceutical agents involved in childhood poisoning is broad. The American Association of Poison Control Centers (AAPCC) National Poison Data System (NPDS) data reveal an average of more than 1.2 million exposures per year in children younger than 6 years between 2009 and 2011, the 3 most recent years for which tabulated data were available.^{20–22} These data typically represent more than 50% of all poison-related calls to the nation’s PCCs. **Table 1** summarizes the most commonly ingested agents reported in 2011. Among these agents are cosmetics and personal care products, noncorrosive cleaners, and plants, all with a low likelihood of causing serious effects. An excellent effort to stratify the litany of pediatric exposures into those with real toxicologic hazard potential, based on frequency of occurrence and inherent toxicity of particular agents (hazard factor), was published in 1992.²³ For pharmaceuticals, the most hazardous agents at that time were iron supplements, antidepressants, cardiovascular agents, and salicylates (of these, iron in particular has diminished as a threat through decreased accessibility, as noted earlier). Additional hazardous drugs included opioids, anticonvulsants, chloroquine,

Substance Category	Percent of Total Substances^a
Cosmetics and personal care products	14
Analgesics	10
Cleaning products	9
Foreign bodies, toys, and so forth	7
Topical preparations	7
Vitamins	4
Antihistamines	4
Pesticides	3
Cold and cough medications	3
Antimicrobials	3
Gastrointestinal medications	3
Plants	3

^a Rounded to nearest integer.

Data from Bronstein AC, Spyker DA, Cantilena LR Jr, et al. 2011 annual report of the American Association of Poison Control Centers’ National Poison Data System (NPDS): 29th annual report. Clin Toxicol 2012;50:911–1164.

isoniazid, theophylline, oral hypoglycemics, and diphenoxylate/atropine. The most hazardous nonpharmaceutical household products were hydrocarbons, pesticides, alcohols/glycols, drain and oven cleaners, and gun bluing agents that contain selenious acid; several of these remain highly hazardous today. Button batteries would now also rank near the top of the list of nonpharmaceutical hazards.²⁴ These and several new threats are discussed in more detail later.

Of those 3.6 million childhood exposures reported in the United States from 2009 to 2011, more than 2500 developed a life-threatening illness, and 109 died of exposure-related effects.^{20–22} Yet, these summary data represent a vast improvement in morbidity and mortality from a half-century ago, when 300 to 500 childhood poisoning deaths per year were routine. Pediatricians, public health authorities, and consumer advocates rightfully take great pride in this evolution, believed largely caused by the widespread use of child-resistant packaging for many medications and hazardous household products after passage of the Poison Prevention Packaging Act in 1970.²⁵ Conceptualizing the accidental poisoning of the 1950s in the modern injury model has allowed for substantial inroads in poison prevention efforts, primarily by attacking the toxic agent via decreased accessibility through regulation with child-resistant packaging and household product reformulation to less toxic forms. Additional decreases in childhood morbidity are undoubtedly caused by the poison center movement and advances in emergency and hospital-based care for the poisoned patient.

Despite these enormous gains, recent data suggest a disturbing trend since 2000 that pediatric ingestions, and in particular, related ED visits and hospital admissions are increasing again.^{26–28} Analysis of AAPCC data from 2001 to 2008 determined that pharmaceutical exposures and related ED visits increased significantly, with parallel increases in injuries and hospital admissions.²⁶ The agents most often involved in serious exposures were prescription medications, particularly oral hypoglycemics, opioid analgesics, sedative/hypnotics, and cardiovascular drugs. It was postulated that the best explanation for this disturbing trend was the general increase in such potent medications in current use, and thus in the environment of young children. This hypothesis was tested by researchers who compared AAPCC data for pediatric exposures with data from the National Ambulatory Medical Care Surveys for adult-intended prescriptions written for 2000 to 2009.²⁸ A striking association of these variables was found, particularly for children 0 to 5 years old, and again, for opioid analgesics, oral hypoglycemics and cardiovascular medications. Thus, challenges remain to further decrease pediatric toxic exposures, and new efforts are being addressed, including so-called next-generation safety packaging, which limits flow rate of liquid medications, or use of a blister packet within a traditional child-resistant container for pill-form medications.²⁹

Two etiologic considerations deserve special comment: malicious poisoning in young children and pediatric medication errors. Child abuse by poisoning is uncommon, occurring in only 0.007% to 0.02% of pediatric poisonings reported to the AAPCC.^{30,31} However, the frequency may be higher when hospital-based cases are analyzed. One investigation determined that 13% of ED and in-patient pediatric poisonings resulted in consultation to their hospital's child abuse team, and 4% were referred to the regional child protective services agency (although many of these were for concern of poor supervision, neglect or exposure to illicit substances, rather than truly malicious intent).³² Such cases might be especially suspected in poisoned children younger than 1 year, or between 5 and 11 or so years old (eg, preadolescent), and when the history is inconsistent or otherwise arouses clinician discomfort. Additional risk factors include previous history of poisoning or siblings who were poisoned; massive overdose; ingestion of multiple agents (unless perhaps the child was found

with an open pill minder or equivalent); exposure to illicit drugs; unusual poisonings from common household substances such as salt, pepper, and even water; and evidence of other forms of child abuse or neglect.^{33–35} The morbidity of such cases tends to be higher,^{30,31} and if child abuse is suspected, these patients require prompt reporting to child protective services, meticulous documentation of clinical and laboratory findings, and careful attention to chain-of-custody procedures for handling of toxicology specimens.

Drug toxicity in young children may also be the result of iatrogenic or parental medication error.³⁵ Children may be more prone to these errors because of several factors, including the inevitable necessity of calculating weight-based or age-based dosing, and the fact that they cannot speak for themselves regarding allergy history or early symptoms of an adverse event. Moreover, medically complex children in hospital settings may be at increased risk.^{36,37} A not uncommon scenario is a 10-fold overdose caused by calculation error of a mg/kg dose. Alternatively, compounding of medications lacking a standardized pediatric formulation presents the opportunity for errors in the compounded concentration or on administration of alternative concentrations. Furthermore, potentially toxic medications with multiple pediatric oral suspension concentrations exist, such as verapamil, atenolol, carvedilol, labetalol, propranolol, and tacrolimus.³⁸ The frequency and morbidity from pediatric medication errors are considerable.³⁹ They account for as many as 6% of all exposures in young children, and for 12% of poisoning deaths in this age group.³⁵ Prevention strategies include computerized order entry systems, unit-based clinical pharmacists, and enhanced efforts at communication among health care team providers.^{40,41}

PEDIATRIC PATHOPHYSIOLOGIC CONSIDERATIONS

Pediatric patients respond differently to poisoning than adults, the reasons for which extend beyond their comparatively smaller size. Myriad differences in the child's anatomy and physiology affect vulnerability to toxic exposures. In addition, developmental changes in drug disposition and effect render some agents unusually toxic in the very young child.

Dermal absorption is clearly increased in children, who have a higher body surface area/weight ratio, increased skin perfusion, and increased skin hydration.⁴² There is greater potential for toxicity from dermal exposures and greater susceptibility to dehydration and insensible losses. Absorption by inhalation is also a particular pediatric vulnerability; the increased respiratory rate and minute ventilation of young children deliver a higher dose in a shorter time for many airborne toxins. The most common of these toxins is carbon monoxide, in which a group of exposed persons have varying degrees of symptom severity, the most severe of which are often found in the smallest child.

Because of a higher metabolic rate and decreased reserve, children are more sensitive to hypoxia and respiratory failure. Increased reliance on the diaphragm and limited capacity of other accessory muscles lead to the abdominal breathing so often seen in young children with respiratory distress, and an increased tendency to fatigue and respiratory failure. This situation can affect a child's resilience to a direct respiratory toxin (such as an aspirated hydrocarbon), as well as the ability to compensate for acid-base disturbances. As a result, children may be more acidemic at initial presentation with salicylism and may have more severe acidemia with other clinical scenarios, such as toxic alcohol poisoning. An additional metabolic vulnerability is a relative lack of glycogen stores, which significantly increases the likelihood of fasting hypoglycemia from ethanol, β -receptor antagonists, and other agents altering glucose homeostasis.

Children have more limited cardiovascular reserve in response to stress. Cardiac output is heavily reliant on heart rate, with limited capacity to augment stroke volume. However, increased adrenergic tone allows for maintenance of normal blood pressure until the advanced stages of shock. Thus, a child in impending circulatory failure may appear deceptively stable, with a normal blood pressure, and tachycardia as a lone vital sign abnormality. When a drug is ingested that alters this fragile balance, a precipitous decline may ensue. For example, drugs inducing bradycardia such as calcium channel antagonists or organophosphorus pesticides may precipitate circulatory arrest in very small doses.

Although a detailed discussion of pediatric pharmacokinetics and pharmacodynamics is beyond the scope of this review, it is becoming increasingly clear that the manner in which a given drug is absorbed, distributed, metabolized, and excreted changes considerably throughout childhood.⁴³ Various neurotransmitter receptors and ion channels also undergo maturation in this period. These developmental alterations in drug distribution and response may explain the long-observed phenomenon of agents that cause specific toxicity only in young children. Several opioid receptor agonists or their structural isomers cause enhanced central nervous system (CNS) and respiratory depression in children, including dextromethorphan cough syrups, clonidine, diphenoxylate antidiarrheals, codeine, and buprenorphine.^{44–47} Young infants are more prone to paradoxical reactions to benzodiazepines⁴⁸ and increased tendency to QT_c prolongation with sotalol and other prodysrhythmic drugs.⁴⁹

EMERGENCY MANAGEMENT OVERVIEW

Despite the relative infrequency of serious clinical toxicity resulting from most common pediatric exploratory ingestions, as noted earlier, some become seriously ill. Thus, it remains incumbent for EPs to recognize and treat poisoned children.

Readily available recent literature offers excellent summaries of the general approach to the poisoned patient.^{50–53} Little modification is necessary in expanding these overviews to focus on the pediatric situation.^{54–56} Several comments are offered that represent our experience and method of conceptualizing this approach, particularly as it applies to the child who is critically ill or at risk for precipitous decline. This suggested approach offers an updated improvement of the senior author's previous effort in this regard 20 years ago.³

Severe poisoning in a young child may be considered analogous to the modern multiple trauma model and approached in a similar manner.⁵⁷ A previously well child is potentially injured in multiple organ systems, with a great variance in the degree of (chemical) injury at each site. There is often a brief window of opportunity for emergency medical services personnel and EPs to make dramatic interventions that prove life-saving. Prompt and thorough evaluation of life-threatening conditions accompanied by sequential immediate intervention (or primary survey) allows for a more detailed secondary evaluation and detoxification phase (secondary survey). This approach is summarized in [Table 2](#). EPs are well versed in this paradigm, and only a few comments are here annotated.

Life Support

The initial phase of management includes attention to the traditional ABCs (airway, breathing, circulation) well known to the EP, with some toxicologic expansion to ABCD₃EF. Additional Ds in this mnemonic stand for disability assessment (eg, brief neurologic examination, such as a level of consciousness, pupillary size, and reactivity), empirical drug therapy (especially oxygen, dextrose, and naloxone), and initial

Table 2	
Emergency management of the poisoned child	
Phase	Actions and Considerations
Initial Life Support Phase (ABCD₃EF)	
Airway	Emphasis on protection in obtunded child Possible compromise in caustic exposures
Breathing	Adequate oxygenation and ventilation
Circulation	Close monitoring of vital signs, capillary perfusion Early IV access
Disability	Level of consciousness Pupillary size, reactivity
Drugs	Dextrose (\pm rapid bedside testing) Oxygen Naloxone Other ACLS medications as needed
Decontamination	Ocular: copious saline lavage Skin: remove contaminated clothes, copious water, then soap and water GI: consider options (often none)
Electrocardiogram	Rate and rhythm QRS width, QTc length Terminal R wave in lead AVR
Fever	Core temperature check for hyperthermia Emergent cooling as needed
Evaluation, Decontamination, and Supportive Care Phase	
History	Brief, focused Known toxin Estimate amount, elapsed time, early symptoms, home treatment, PMH? Suspected but unknown toxin, consider if Acute onset of illness; age 1–5 y PMH of pica, ingestions Current household stressors Multiorgan system dysfunction Puzzling clinical picture New medication access Suspicious HPI, PMH, or FH for child abuse Institute hospital protocols Consider expanded laboratory testing with chain-of-custody procedures
Physical Examination	Vital signs, pulse oximetry (with core temperature) Level of consciousness, neuromuscular status Eyes: pupillary size and reactivity, extraocular movements, nystagmus Mouth: corrosive lesions, odors on breath, hydration of mucous membranes Cardiovascular: rate, rhythm, capillary perfusion Respiratory: rate, chest excursion, air entry, auscultatory signs GI: tenderness, bowel sounds Skin: color, bullae, burns, autonomic signs (eg, diaphoretic, flushed, dry) Odors: breath, clothing, vomitus

(continued on next page)

Phase	Actions and Considerations
Laboratory (individualize)	CBC, co-oximetry ABG or VBG, \pm serum osmolarity Chest radiograph, abdominal radiograph Electrolytes, BUN, creatinine, glucose, calcium, magnesium, liver function tests Rapid overdose toxicology screen Quantitative toxicology tests (especially acetaminophen, salicylate, ethanol) Comprehensive toxicology testing at reference laboratory
Assessment of severity and diagnosis	Clinical findings (see Table 3 toxidromes) Laboratory and ECG abnormalities
Specific detoxification and continued supportive care	Reassess ABCD ₃ EF (always) Consider GI decontamination options Antidotal therapy, as indicated Enhance elimination, as indicated Supportive care (in every case!)

Abbreviations: ABG, arterial blood gas; ACLS, advanced cardiac life support; BUN, blood urea nitrogen; CBC, complete blood count; ECG, electrocardiogram; FH, family history; GI, gastrointestinal; HPI, history of present illness; PMH, past medical history; VBG, venous blood gas.

decontamination, with urgent emphasis on ocular and dermal decontamination and consideration of gastrointestinal decontamination options. E is added to address a more detailed electrocardiogram evaluation, and an F reminds the practitioner to check core temperature, which may be critically increased (hyperthermia) in many intoxications.

As noted earlier, the poisoned child shows the same precariousness of airway and respiratory function that complicates infectious (eg, croup, epiglottitis) and other CNS-depressed (eg, cranial injury) states. Seriously poisoned children often pass rapidly from obtundation with minimally impaired respiration to deep coma and apnea, and even those with seemingly normal respiratory drive may suffer airway obstruction because of narrow airway caliber, copious secretions, and depressed airway protective reflexes. Patients may vomit or be selected to undergo NG tube administration of activated charcoal (AC), which poses aspiration risks. Blood gas analysis may help in assessing ventilatory status, but in our view, EPs should usually rely on clinical judgment and maintain a low threshold for endotracheal intubation for definitive airway protection and to ensure adequate ventilation in the significantly obtunded, poisoned child. This approach allows for an orderly, if urgent, elective intubation, and obviates the chaos of a precipitous pediatric arrest.

Similarly, any symptomatic poisoned child deserves early assessment of cardiac rate and rhythm (including a 12-lead electrocardiogram [ECG]), blood pressure and capillary perfusion, and rapid attainment of IV access. The poisoned child in cardiac arrest or with severe hemodynamic compromise requires an approach that generally follows established American Heart Association guidelines for pediatric advanced life support.⁵⁸ Occasional exceptions to this rule include the early use of sodium bicarbonate in advanced cyclic antidepressant (or other sodium channel blocking agent) toxicity or additional specific antidotal therapy for other cardiotoxic drugs, such as digitalis antibodies for severe digoxin overdose, glucagon for β -adrenergic blocker (BB) toxicity, and calcium and insulin/glucose therapy for severe calcium channel

blocker (CCB) toxicity.^{59,60} For the child who has not arrested, but is in shock, the initial management usually begins with IV crystalloid fluids (eg, 20 mL/kg bolus, repeated and titrated to clinical effect), again followed by specific antidotes if such are appropriate. Cautious use of inotropes is warranted for persistent shock after circulatory filling has been achieved. Such severe cases should prompt seeking emergent toxicology advice from an in-house consultant or a call to the PCC.

The use of empirical drug therapy in obtunded young children who are poisoned, or potentially so, is similar to that in adults, with the exception of the routine use of thiamine. Although pediatricians and emergency physicians are usually timely in their consideration and use of dextrose, they may occasionally omit a trial of naloxone in toddlers. However, many opioids are available to toddlers in the form of prescription analgesics (which increased in the past decade, as noted earlier), antidiarrheal preparations, cough medicines, or illicit drugs, as well as the partially naloxone-responsive antihypertensive agent clonidine.⁶¹ All potentially poisoned obtunded children deserve a trial of naloxone. Toddlers who are deeply obtunded or apneic may be treated immediately with relatively high doses, by adult standards, with little fear of precipitating withdrawal; we routinely initiate therapy with 0.1 mg/kg (or 1–2 mg) IV.⁶² This therapy may be repeated when necessary if opioid toxicity is highly suspected, especially for agents such as methadone, fentanyl, buprenorphine, and clonidine.

As mentioned earlier, dextrose administration is a potentially critical intervention, and should be considered early in the approach to the comatose or seizing child. A rapid bedside test for blood glucose may be useful if it is clearly in the normal range, but one should be wary of relying on a borderline reading. In addition to coma or seizures, patients with hypoglycemia may show an atypical neuropsychiatric picture, with aphasia, slurred speech, and focal neurologic signs. Hypoglycemia is frequently seen after ethanol ingestions in toddlers (as opposed to adults),⁶³ as well as in ingestions of oral hypoglycemics, and occasionally with β -blocker and salicylate intoxication. The initial dose is 0.5 g/kg dextrose, which is provided as a 25% solution (2 mL/kg) in toddlers or as a 10% solution (5 mL/kg) in infants in order to minimize osmotic shifts from the typical 50% adult solution.

Additional advanced life support medications and anticonvulsants are used as needed. Dysrhythmias caused by poisonings are often the result of sodium channel or potassium channel blockade and may be worsened by traditional antiarrhythmic drugs. The former are often effectively treated with sodium bicarbonate and the latter by magnesium infusion or override pacing. Toxin-induced seizures tend to respond best to benzodiazepine therapy, titrated to effect. A barbiturate is often a preferred second-line agent. Phenytoin is relatively ineffective for almost all toxin-induced seizures. Blood glucose should be checked in all seizing patients. Pyridoxine is a specific antidote for isoniazid-induced seizures. The occurrence of toxin-induced dysrhythmias or seizures, especially if refractory to initial therapy, should again suggest the potential value of an emergent toxicology consult or call to the regional PCC.

Rarely, a young poisoned child might manifest extreme hyperthermia. This complication may occur after overdose of several classes of drugs, including sympathomimetics, anticholinergics, salicylates, and other uncouplers of oxidative phosphorylation, as well as in the context of the specific drug-induced hyperthermic syndromes, including malignant hyperthermia, serotonin syndrome, and neuroleptic malignant syndrome.⁶⁴ One additional hyperthermic scenario, that of alcohol or sedative/hypnotic withdrawal, is highly unlikely in a toddler presenting to the ED. Treatment consists of high-dose benzodiazepine administration (with ventilatory support as necessary) in most such cases, and rapid external cooling with consideration for neuromuscular paralysis. Specific antidotes (eg, bromocriptine for neuroleptic malignant syndrome; cyproheptadine for

serotonin syndrome) might be of value and should be considered after toxicology consultation.

Within a few minutes of presentation, the poisoned child should be carefully assessed by the ABCD₃EF approach, and life-support interventions should be initiated as appropriate. Patients with significantly altered mental status should be considered for airway intubation, have IV access, and undergo empirical trials (or relevant rapid bedside testing) of oxygen, naloxone, and glucose. Additional advanced life-support medications such as anticonvulsants or antiarrhythmic agents, and cooling interventions for hyperthermic patients, should be instituted as necessary. Decontamination options should be considered.

Evaluation, decontamination, and supportive care

History A brief, focused history should be obtained as soon as the life-support phase has been completed. In the child with a known or suspected exposure, the usual questions regarding what, when, and how much was ingested are asked. However, young poisoned children often do not present to the ED with a clear history of toxin exposure, but rather with an acute illness of questionable origin. Features highly suggestive of occult poisoning in such cases include patient-related factors, such as age 1 to 5 years; history of pica-prone behavior; acute onset; multiple organ system dysfunction; altered sensorium; and any puzzling clinical picture.⁵⁷ Family and social history factors may also be helpful. Have any environmental stressors occurred, as noted earlier? Was the child visiting a grandparent's home, or vice versa, allowing the introduction of new medications into the household in a context that might be less child-proofed? Are siblings or parents ill or taking newly prescribed medications, such as the pregnant or postpartum mother with the nearly universal prescription of iron supplementation? Holiday gatherings, with numerous relatives of all ages visiting and a general lessening of parental availability to supervise toddlers, are also high-risk occasions, as are recent moves in residence with boxes full of medications and household products often temporarily on the floor. As mentioned earlier, a history that is inconsistent, or a concerning past medical or family history, might suggest malicious poisoning.

Physical examination The usual features on physical examination of any poisoned patient should be sought in the young child. A careful reassessment of vital signs and capillary perfusion should be performed, including measurement of core temperature. The examination should focus on central and autonomic nervous system findings, pupillary size and reactivity, and any obvious abnormalities of the skin, mucous membranes, and cardiorespiratory or gastrointestinal tracts. Characteristic odors of the breath or clothing ought to be sought. The classic constellations of clinical findings (toxidromes) seen in many categories of poisoning (eg, opioids, sympathomimetics, cholinergics, and anticholinergics)⁶⁵ are just as characteristic in young children as in adults when appropriate adjustment is made for age-corrected vital signs and baseline developmental status.^{61,66} Several of the more common toxidromes are outlined in [Table 3](#). As noted earlier, examination findings suggestive of child abuse or neglect might raise the possibility of malicious poisoning.

Laboratory and ECG evaluation The same issues regarding both rapid overdose toxicology panels and quantitative drug levels apply to toddlers as well as to adolescents or adults and are not commented on in detail. Toxicology screens have limited value in the emergency management of most poisoned patients.^{61,65,67} This observation is particularly true for the toddler with a witnessed ingestion of a single agent. In the

Toxidrome	Examples	Significant Clinical Findings
Anticholinergic	Atropine Antihistamines Cyclic antidepressants	VS: ↑T, ↑HR, ↑BP (↓BP, dysrhythmias with CAs) CNS: delirium, coma, seizures Eyes: mydriasis (sluggishly reactive), blurred vision Skin: flushed, hot, dry Misc.: ileus, urinary retention
Cholinergic	Organophosphorus and carbamate pesticides Military nerve agents	VS: ↑ or ↓HR, ↑RR (with pulmonary effects) CNS: confusion/drowsiness to coma, seizures Eyes: miosis, blurry vision, lacrimation Skin: diaphoresis Misc.: SLUDGE; bronchorrhea, bronchospasm, pulmonary edema; muscle fasciculations, weakness to paralysis
Sympathomimetic	ADHD medications Amphetamines Cathinones Cocaine	VS: ↑T, ↑HR, ↑BP CNS: agitation, delirium, psychosis Eyes: mydriasis (normally reactive) Skin: diaphoresis Misc.: tremor, myoclonus
Opioid	Prescription analgesics Antitussives Antidiarrheals Antihypertensives (clonidine) ADHD medication	VS: ↓T, ↓HR, ↓BP, ↓RR CNS: euphoria to coma Eyes: miosis (pinpoint pupils) Skin: normal Misc.: hyporeflexia

Abbreviations: ↑, increased; ↓, decreased; ADHD, attention-deficit/hyperactivity disorder; BP, blood pressure; CA, cyclic antidepressants; HR, heart rate; Misc., miscellaneous; RR, respiratory rate; SLUDGE, salivation, lacrimation, urination, defecation, gastric cramping, emesis; T, temperature; VS, vital signs.

patient with an unknown ingestion, the toxicology screen may be of some value (especially for forensic purposes if child abuse is suspected), but routine chemistries, blood gas analysis (\pm co-oximetry), and serum osmolality (to evaluate pH disturbances, anion, and osmolal gaps) are more helpful in case management. A quantitative acetaminophen level is often indicated for the adolescent with an intentional overdose, because this may be an unreported or unrecognized coingestant. Routine screening for unreported acetaminophen ingestion is not usually indicated in small children.

Several clinically important drugs that are commonly ingested by toddlers, which can produce coma or disturbed cardiovascular function, and for which the usual toxicology screen is negative, are clonidine, digoxin, CCBs, and BBs, and iron.⁶⁸ However, each of these has characteristic clinical, ECG, or routine laboratory abnormality patterns. Clonidine resembles an opioid overdose, with variable response to naloxone, and a seemingly disproportionate degree of hypotension and bradycardia. Iron toxicity may produce marked vomiting, diarrhea, and hypotension, with an anion gap metabolic acidosis, hyperglycemia, and leukocytosis; CCBs and BBs often present with a history of drug availability from family members, especially grandparents, with combined bradycardia and hypotension, whereas digoxin more typically

manifests sinus bradycardia with typical ECG findings, and hyperkalemia and ventricular dysrhythmias in severe cases.

A closer ECG examination is indicated during this evaluation and supportive care phase. Numerous drugs and toxins are capable of causing subtle ECG abnormalities, which provide clues to diagnosis and represent pathophysiologic changes that contribute to hemodynamic instability and dysrhythmia potential.⁶⁵ In particular, many drug classes result in sodium channel blockade (resulting in hypotension and propensity to ventricular tachycardia or fibrillation) or potassium efflux channel blockade, resulting in potential torsades de pointes. These conditions are manifested by lengthened QRS and QTc durations, respectively. Sodium channel blocking agents may also cause a significant rightward axis deviation in the terminal 40 milliseconds of the QRS complex, noted particularly with a significant terminal upright R wave in lead AVR, which is not typically present in normal children beyond the neonatal period. Common examples of sodium channel blockers include cyclic antidepressants, carbamazepine, chloroquine and hydroxychloroquine, class Ia and Ic antiarrhythmics, and diphenhydramine. A similar list of potassium channel blocking agents includes several nonsedating antihistamines, phenothiazines and butyrophenones, other antipsychotics, some serotonin selective antidepressants, such as citalopram and escitalopram, some macrolide and quinolone antibiotics, and again, class Ic antiarrhythmics.

Assessment For the child with a known exposure, a careful clinical evaluation, and at times, additional laboratory input and ECG interpretation, allow the emergency physician to formulate an assessment of the potential severity of the intoxication. In the context of an occult poisoning, the same approach should allow an educated guess as to the likely agent or class of agents responsible for the child's condition. In either case, the practitioner may at this point consider further input from the PCC or a local toxicology consultant, for assistance in the management of those children exposed to the more exotic substances or who are more critically ill.

Specific detoxification issues Children with significant ocular or dermal contamination need rapid topical decontamination, as appropriate for any aged patient based on substance and clinical criteria. Gastrointestinal decontamination recommendations have evolved considerably over the past decade and are similar for children and adults.^{57,69} Overall, most poisoned patients are managed safely and effectively in the ED without any gastrointestinal decontamination. Gastric emptying with syrup of ipecac is no longer recommended for in-home or hospital use. Gastric lavage is rarely indicated except for high-lethality ingestions in patients presenting within 30 to 60 minutes and is technically more difficult and complication prone in small children.

Similarly, single-dose AC administration is no longer a routine ED intervention but may be considered for patients who present soon after ingestion of agents that bind to AC, for whom supportive care or antidotal therapy may not be sufficient to prevent serious toxicity. AC is contraindicated for ingestions of caustics and hydrocarbons, because systemic toxicity is less consequential than direct mucosal injury or pulmonary aspiration risk, respectively. When elected, the pediatric dose is typically 1 g/kg, or an average of 10 to 15 g for toddlers. Many children swallow this amount, or close to it, when it is mixed in a fruit-flavored beverage and offered by mouth, especially if a cup with a plastic top and straw can be used to mitigate the unpleasant appearance. AC administration, especially by NG tube, results in vomiting in about 20% of children, so is relatively contraindicated in obtunded patients without previous airway protection.⁷⁰ In addition, NG tube use adds the potential for the life-threatening complication of inadvertent tracheal placement in a struggling child. We rarely use an

NG tube for administration of AC in toddlers, except in the most highly lethal overdoses, and in such cases, confirmation of gastric placement is crucial before its use, as well as serious consideration of previous endotracheal intubation for relative airway protection.

Of the several substances not well adsorbed to AC but ingestion of which is potentially mitigated by gastrointestinal decontamination, only iron is commonly of clinical importance in young children. Most children vomit profusely after a significant iron exposure but may still benefit from abdominal radiography to evaluate for remaining iron pills, fragments, or concretions. If present, whole bowel irrigation (WBI) with a polyethylene glycol balanced electrolyte solution, warrants consideration. WBI in toddlers does typically require NG tube placement, and is administered at a rate of 250 to 500 mL/h until the rectal effluent is clear, usually within 3 to 4 hours. WBI may also be used for the uncommon ingestion of various medications in patch formulations, the child who ingests illicit drug packets or vials, the young patient found to have large amounts of lead paint chips in the gastrointestinal tract, or large overdoses of significantly toxic medications, especially if in extended-release formulation (such a scenario is uncommon in toddlers; exceptions include several CCBs and β -blockers).

Antidotal therapy Although most poisonings are managed optimally with supportive care alone, specific antidotal therapy is warranted in select cases. The indications for and choice of antidotes in children are similar to those in adults, with some additional considerations.^{51,71} Like many newer or limited-use therapies, pediatric experience is often limited, and the pediatric indication is off label. The potential for medication errors is high, because these are uncommonly administered medications that require weight-based dosing and diluent volume. Some medications, such as calcium salts and ethanol infusions, require large-bore IV access for continued administration, which is technically difficult to obtain in a small child. Nevertheless, many essential antidotes have shown a wide safety margin and in situations in which specific antidotal therapy may be lifesaving should not be withheld. **Table 4** highlights those antidotes with an occasional but critical role in pediatric toxicology management, a few of which deserve special mention.

The rapid administration of atropine can be vital to survival from organophosphorus pesticide or nerve agent poisoning, and pralidoxime likely plays a consequential adjunctive role in severe cases. For this reason, adult-dose autoinjectors are widely stocked by emergency medical services squads, which deliver higher doses than recommended for a small child.⁷² Reduced-dose pediatric atropine autoinjectors may be available, and adult-dose kits can be easily modified to provide a reduced pralidoxime dose.⁷³ However, safety data from asymptomatic children inadvertently given adult atropine autoinjectors showed anticholinergic symptoms but no serious effects,⁷⁴ and in the event of severe nerve agent or organophosphorus pesticide poisoning, the doses needed may be higher than anticipated.⁷² In this scenario, the therapeutic benefit of treating a child with a higher but immediately available dose exceeds the risk of toxicity.

N-acetylcysteine (NAC) for acetaminophen toxicity was FDA approved in an IV formulation in 2004. The development of a specific IV formulation has simplified the management of many acetaminophen-poisoned patients, eliminating issues of odor, unpalatability, and noncompliance associated with the enteral formulation. However, several pediatric therapeutic errors have been reported, with inappropriate dosing and diluent volume, which highlight the perils of pediatric antidote administration. Children receiving the adult diluent volume in error have developed hyponatremia and seizures.⁷⁵ Overdosage of NAC itself in the IV formulation may be fatal in young

Drug or Toxin	Antidote
Acetaminophen	<i>N</i> -acetylcysteine
Benzodiazepines	Flumazenil
β-Adrenergic antagonists (β-blockers)	Glucagon ^a
CCBs	Calcium ^a High-dose insulin euglycemia (insulin and glucose) ^a IV lipid emulsion ^a
Coumadin (and similar rodenticides)	Vitamin K ₁
Cyanide	Hydroxocobalamin (preferred) Sodium nitrite and sodium thiosulfate
Digoxin	Digoxin immune Fab
Ethylene glycol	Fomepizole (preferred) Ethanol ^a
Iron	Deferoxamine
Isoniazid	Pyridoxine
Lead	British anti-Lewisite CaNa ₂ EDTA (versenate) Succimer (dimercaptosuccinic acid)
Methanol	Fomepizole (preferred) Ethanol ^a
Methemoglobinemia	Methylene blue
Opioids	Naloxone
Organophosphorus insecticides and nerve agents	Atropine Pralidoxime
Sulfonylureas	Dextrose Octreotide ^a
Rattlesnake (and other crotalid) envenomations	Crotalidae polyvalent immune Fab
Tricyclic antidepressants	Sodium bicarbonate IV lipid emulsion ^a

^a Without specific FDA approval for this indication.

children.^{76,77} Meticulous adherence to prescribing information and consultation with a toxicologist are advised to ensure appropriate administration.

In patients with toxic alcohol poisoning, alcohol dehydrogenase inhibition is the mainstay of therapy to prevent toxic metabolite formation, with attendant organ injury and metabolic acidosis. This treatment is most commonly accomplished by the administration of fomepizole, which has shown efficacy and safety in the pediatric population.⁷⁸ Before the development of fomepizole, IV or oral ethanol administration achieved the same enzyme inhibitory effects and is still used in areas where fomepizole is not available. IV ethanol administration can be technically complicated, requiring central venous access and careful attention to dose titration, mental status depression, and the potential for fasting hypoglycemia. Although young children are more prone to hypoglycemia and CNS depression in the context of ethanol poisoning, therapeutic ethanol administration may have fewer adverse effects. One study of 60 methanol-poisoned children treated with IV or oral ethanol⁷⁹ reported no symptomatic hypoglycemia or significant CNS depression, which likely reflects the impact of close monitoring.

The cyanide antidotes are essential to survival in the event of poisoning from inhalation of fire smoke or other sources. Historically, cyanide antidotes available in the United States have included amyl nitrite, sodium nitrite, and sodium thiosulfate. However, nitrites, which generate methemoglobin to form nontoxic excretable cyanomethemoglobin, pose substantial risk for pediatric use. An increased proportion of fetal hemoglobin and decreased activity of methemoglobin reductase both engender high concentrations of methemoglobin with nitrite therapy.^{80,81} In inhalation of fire smoke, the additive effects of methemoglobinemia, carboxyhemoglobinemia, and hypoxemia can overwhelm a child's already reduced respiratory and metabolic reserve. As a result, only sodium thiosulfate has traditionally been recommended for pediatric cyanide poisoning caused by smoke inhalation. In 2006, the vitamin B₁₂ precursor hydroxocobalamin gained FDA approval in IV formulation for this indication and has long been used elsewhere with efficacy that seems comparable with if not superior to sodium nitrite combined with thiosulfate.⁸² It is therefore the preferred agent if available for treatment of cyanide toxicity. Pediatric safety data are limited but reassuring with both hydroxocobalamin and sodium thiosulfate.⁸³ Both agents should be considered appropriate in the context of pediatric cyanide poisoning, for which expeditious antidote administration is vital.

Resuscitative IV lipid emulsion has been the focus of much investigation since its successful use first with local anesthetic toxicity and then with other poisonings causing cardiovascular collapse.⁸⁴ Clear indications are still evolving, but it seems to be an effective therapy for severe poisoning caused by certain lipophilic drugs, including bupropion, calcium channel antagonists, and tricyclic antidepressants.⁸⁵ Reported adverse effects include pancreatitis, fat embolus, acute respiratory distress syndrome (ARDS), and digit amputation.^{86,87} Although the rate of these events is unknown, and pediatric data are even more scant, it seems a reasonable option for refractory cardiovascular collapse.

Enhanced elimination The ability to enhance toxin elimination in specific cases may be a critical adjunct to therapy after several important poisonings. Urinary alkalization is a mainstay of therapy for moderate to severe salicylate intoxication.⁸⁸ Multiple-dose AC (MDAC) has been shown to increase clearance of several agents, including barbiturates, salicylate, carbamazepine, and theophylline via intestinal dialysis.⁸⁹ However its use was associated with complications of repeated AC administration, such as vomiting, aspiration, and intestinal obstruction, and it is not clear that the apparent pharmacokinetic benefit confers improved clinical outcomes. In our experience, MDAC was most useful when mild to moderate theophylline intoxications were common in children, and occasionally staved off the need for hemodialysis (HD); because this is no longer the case, there are few absolute indications for MDAC in young children, although it might be considered in moderately severe salicylate or carbamazepine intoxications.

For patients in whom a highly toxic substance has been absorbed and achieves a significant serum concentration, extracorporeal toxin removal methods can prevent worsened organ injury, metabolic compromise, or organ system collapse. High-flux HD clears solutes and toxins from the blood by diffusion and convection across a semi-permeable membrane, and is the primary modality for expeditious toxin removal. Other methods such as charcoal hemoperfusion, exchange transfusion, plasmapheresis, and peritoneal dialysis have little role and are significantly less effective in both the amount and rate of toxin removal. Continuous renal replacement therapies, such as continuous venovenohemofiltration, also have slower clearance rates and are indicated only for the hemodynamically unstable patient who cannot tolerate acute HD.

Acute HD should be considered for poison removal if: (1) there is clinical benefit to faster removal than would be expected from endogenous clearance, (2) there is a clear relationship between serum concentrations and toxicity, and (3) the toxin itself can be removed in significant amounts. Highly dialyzable toxins generally have low molecular weight, are not significantly protein bound, and have low volumes of distribution. The classic examples of these toxins are the toxins for which HD is most often used: salicylates, toxic alcohols, lithium, and theophylline. Other toxins in which HD achieves some removal and may confer some benefit include valproic acid, barbiturates, and methotrexate. HD may also be lifesaving to reverse metabolic derangements and electrolyte disturbances without appreciable toxin clearance, as in the case of metformin-associated lactic acidosis.⁹⁰

The use of acute HD in pediatrics is common for chronic and acute renal insufficiency, and it can be safely performed in conjunction with an experienced nephrologist. Adverse events include those associated with central venous access (insertion trauma, infection, anticoagulation), as well as electrolyte disturbances and hemodynamic instability. In very young infants, volume considerations may require specific small-volume tubing, specialized priming solutions, and close monitoring of the amount of fluid removed to prevent hypotension.⁹¹ Despite these technical challenges, most pediatric tertiary-care centers are capable of performing HD, even in the neonate. It should be used without hesitation in the critically ill child in urgent need of toxin removal, even if transport to such a center is necessitated.

Supportive care In 1994, it was opined that the “most important aspect of managing poisoned children remains meticulous attention to detail in both routine and intensive supportive care.”³ We hold the same opinion today.⁵⁷ This treatment includes close observation of vital signs, cardiac monitoring, and pulse oximetry. Respect for the precipitous nature of respiratory failure in children has already been mentioned. Careful monitoring of fluid and electrolyte balance and responsive adjustment of fluid therapy is especially important in young children, whose large body surface area/mass ratio and immature renal function put them at increased risk of fluid overload or dehydration. Some intoxications warrant frequent serial drug levels (eg, salicylate, lithium, digoxin), and others necessitate close monitoring of organ system function (eg, liver function tests after toxic acetaminophen exposure). Much of this ongoing supportive care takes place after the child is admitted, but can be initiated in the ED, and with long boarding times, may need to be maintained for several hours by EPs. Severely poisoned children are most likely to receive optimal definitive care in specialized centers with experienced pediatric critical care staff and access to toxicology consultation.

THE WELL-APPEARING CHILD WITH POISON EXPOSURE

In contrast to the patient with overt signs of poisoning, the asymptomatic child with a feared or presumed exposure poses a different set of challenges. The nature of exploratory ingestions often entails an unsupervised period when a drug or chemical was accessible and unwitnessed ingestion may have occurred. Many of the substances involved in exposures to children younger than 6 years are nontoxic,^{20–22,57} and many of these cases if called to the regional poison center are not referred to a health care facility.^{20–22} However, once the patient presents to the ED, the emergency physician is tasked with evaluating the significance of the exposure.

A detailed history is most important, including the timing, nature, and estimated amount of the feared exposure. An exploratory ingestion generally can be expected to involve a few pills, or a small volume of an unpalatable liquid. More appealing

liquids, chewable or dissolving tablets, and longer unsupervised periods may allow for ingestion of larger amounts. Important circumstantial evidence may include number and type of missing pills, residue in the child's mouth or on clothing, presence of coughing, gagging, or emesis after ingestion, and what the child states occurred, if the child is sufficiently verbal. Questions regarding medications in the home should include all potential exposures, not just those medications that the caretaker believes to be the likely exposure.

The child without clinical symptoms or signs of poisoning after a reasonable period of observation can be safely discharged in most cases, with a few caveats. First, the ingestion should be either of inconsequential amount, or a substance of inconsequential toxicity. Second, an observation period must sufficiently account for the pharmacokinetics of the presumed exposure (eg, formulation, absorption time, onset time to clinical effects, coingestants). The circumstances of ingestion need raise no red flags for suspicious circumstances, as detailed earlier.³⁴ Adequate follow-up must be in place. Consultation with a regional poison center may be helpful in determining the need for observation and appropriateness of discharge, and may provide follow-up by telephone as needed.

DEADLY IN SMALL DOSES: PERSISTENT PERILS AND EMERGING EXPOSURES

Although many substances ingested by young children may be nontoxic, certain exposures warrant extreme caution for potentially fatal effects in small doses.^{23,92,93} It is advisable in these cases to presume the worst-case scenario in terms of amount and type of toxin ingested, and admit children for observation. This advice is especially true in the case of sustained-release preparations of highly dangerous pharmaceuticals. **Box 1** lists some of these most hazardous exposures.

New hazards are ever emerging of which the emergency physician needs to be aware. The first of these hazards involves foreign body ingestions with propensity for severe tissue damage. Button batteries, long known to require urgent endoscopic removal if lodged in the esophagus, have been associated with an increased number of exsanguination deaths from aortoesophageal fistula formation. This condition seems to be largely caused by increased availability of the 20-mm lithium disk battery. Often, the child is evaluated several times before the final ED visit, in which the child presents with massive hematemesis, shock, or asystole. In several cases, there is no known history of battery ingestion, which is then discovered post mortem.²⁴

Other dangerous foreign bodies include small magnet toys, which can attract one another in the intestine, causing bowel obstruction and necrosis. The same risk is posed by expanding foam toys and flower fertilizer pellets, which increase in size on water exposure. Laundry detergent pods, which have entered the US consumer market over the past few years, are an enticing, colorful, compact package of highly concentrated detergent enclosed in a thin membrane, which dissolves in the presence of moisture. Shortly after they became available, cases of pediatric exposures began to appear, in which even a mouthful of the pod caused oral and aerodigestive tract burns, aspiration, respiratory distress, and CNS depression.^{14,15} Although similar in appearance to dishwashing detergent packets, they seem to cause more severe clinical effect, and caution is advised in treating these children, who may develop toxicity in the hours after exposure. **Box 2** indicates several other pediatric exposures that have been reported in recent years.

In 2010, as a cooperative effort to the annual report published from NPDS, a specific review of pediatric poisoning fatalities was instituted to advance the detection of trends, prevention targets, and sentinel events in these most tragic cases.^{36,37}

Box 1**Drugs and chemicals that may be fatal in small doses**

Alcohols
 Antidysrhythmics
 Antimalarials
 Benzocaine
 Beta-receptor antagonists
 Button batteries^a
 Calcium channel antagonists
 Clonidine and other imidazolines
 Cyclic antidepressants
 Hydrocarbons, petroleum distillates
 Laundry detergent pods^a
 Lomotil (diphenoxylate/atropine)
 Magnetic or expanding foreign bodies^a
 Organophosphorus pesticides
 Opioids and opiates
 Salicylates (methylsalicylate)
 Sulfonylurea oral hypoglycemics

^a Indicates new or worsening potentially fatal hazards.

Box 2**Illustrative cases of poisonings in young children^a**

Substance

Alcohol (infant)⁹⁴
 Alcohol (toddler)⁶³
 Benzocaine (methemoglobinemia)⁹⁵
 Carbamazepine (child abuse)³³
 Clonidine⁶¹
 Lamp oil (hydrocarbon)⁹⁶
 Laundry detergent pods¹⁵
 Mercury⁹⁷
 Opioids⁶²
 Sertraline⁶⁴

^a These and additional cases are accessible in the "Pick your poison" section in *Pediatric Emergency Care*.

Although pediatric poisoning fatalities comprise only a few poison-related deaths each year, a closer evaluation of exposure circumstances shows that many are distinctly preventable. First, opioids continue to be among the most common responsible substances, with a disproportionate number of methadone and buprenorphine cases, highlighting the still unrelenting risk to these youngest victims of the opioid abuse epidemic. Therapeutic errors persistently appear, most of which involve medically complex children in health care facilities. Torch fuel and other hydrocarbon ingestions continue to rank among the most prevalent fatalities, often in the face of immediate and optimal airway management, ventilatory support, and exhaustive ICU care. There have been several exploratory ingestions of refrigerated medications, including liquid methadone, and several cases in which toddlers ingested their own antidysrhythmics, with rapidly ensuing cardiac arrest. Because of frequent use throughout the day by multiple members of the household, a refrigerator is more difficult to secure than a single medicine cabinet. This observation should promote caution to prescribers, who may elect an alternative medication not requiring refrigeration, a closer look at the true necessity of refrigerated storage for these compounds, and improved anticipatory guidance for families with these medications in the home.

SUMMARY

Pediatric poisoning cases require knowledge on the part of EPs of all the critical management principles for poisoned patients but also of where important differences lie in the epidemiology, toxicology, and optimal therapy for poisoned children compared with their adult counterparts. The circumstances of the exposure, the impact on the child and family, the physiologic response to poisoning, and the implications for evaluation and management all present unique considerations, which merit a specialized approach. This article provides a framework for this practice and shows the need for ongoing vigilance to remain current with evolving pediatric hazards and advances in diagnosis, treatment, and prevention.

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