

PEDIATRIC EMERGENCY MEDICINE PRACTICE

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Pediatric Ingestions: Emergency Department Management

Abstract

Pediatric ingestions present a common challenge for emergency clinicians. Each year, more than 50,000 children aged < 5 years present to emergency departments with concern for unintentional medication exposure, and nearly half of all calls to poison centers are for children aged < 6 years. Ingestion of magnetic objects and button batteries has also become an increasing source of morbidity and mortality. Although fatal pediatric ingestions are rare, the prescription medications most responsible for injury and fatality in children include opioids, sedative/hypnotics, and cardiovascular drugs. Evidence regarding the evaluation and management of common pediatric ingestions is comprised largely of case reports and retrospective studies. This issue provides a review of these studies as well as consensus guidelines addressing the initial resuscitation, diagnosis, and treatment of common pediatric ingestions. Also discussed are current recommendations for decontamination, administration of antidotes for specific toxins, and management of ingested foreign bodies.

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CME Objectives

Upon completion of this article, you should be able to:

1. Recognize pediatric ingestions with risk of rapid or delayed fatality.
2. Initiate appropriate resuscitation and decontamination for pediatric ingestions.
3. Utilize available antidotes and treatment modalities for pediatric ingestions.

Prior to beginning this activity, see "Physician CME Information" on the back page.

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Case Presentations

An 18-month-old girl is brought in to the ED by ambulance after her grandmother was unable to wake her from an unusually long nap. The grandmother reports that the child had not been ill that morning. After repeated questioning, she admits that the child was found earlier in the day holding her pillbox. She does not have the pillbox with her and does not remember the names of all of her medications. On examination, the child is breathing shallowly. In response to painful stimuli, the girl moans and withdraws, but does not open her eyes. The remainder of her physical examination is normal, without fever or evidence of trauma. The resident physician asks what initial testing should be performed. As the team applies monitor leads, obtains intravenous access, and administers oxygen to this lethargic toddler, you order a stat ECG and glucose level. As you prepare for possible intubation, you consider medications that could be fatal in a small dose, such as opioids, sedatives, cardiac drugs, and hypoglycemic agents. Could ingestion of a small amount of the grandmother's medication be fatal in this toddler? Is it appropriate to give activated charcoal at this time?

A 3-year-old boy is referred to the ED by his pediatrician. He arrives with an x-ray that was taken earlier in the day. The parents state that the child came to them holding his throat and saying that he had swallowed something, although they are not sure what it was. Soon afterward, he refused to eat and they took him to his doctor. On examination, the patient is afebrile, with normal vital signs, and no respiratory distress. His oropharynx and lungs are clear. You wonder what you should look for on the previous imaging. Should you obtain further radiographic studies? Is a surgical consultation indicated? Can he be safely discharged for observation at home?

A 15-year-old adolescent girl is brought in by her family for a possible suicide attempt. The patient's friend received a text in which the patient reported taking "a whole bottle of pain pills." The family reports that an old bottle of acetaminophen with hydrocodone that was in the bathroom cabinet is now empty. The patient does not know exactly how many pills she took or at what time, but says that it was just after sending that text, which you see from her phone, was 4 hours ago. She is tearful and tired, but answers questions appropriately, and her physical examination is normal. Are there any specific drug levels that should be checked and, if so, when? Should you give naloxone, activated charcoal, or N-acetylcysteine? When can the patient be medically cleared for transfer to a psychiatric facility?

Introduction

Each year in the United States, more than 50,000 children aged < 5 years present to emergency departments (EDs) with concern for unintentional medication exposure.¹ In 2013, United States poison control centers received reports of 1,341,862 expo-

sure in patients aged < 20 years, which accounted for 61.33% of all exposures.² Pediatric exposures demonstrate a bimodal pattern, with unintentional exposures in young children and exposures that are more likely to be intentional in adolescents.² Although the number of pediatric exposures is large, fatal pediatric ingestions are rare. Children aged < 6 years account for only 1.8% of all toxicologic fatalities reported to United States poison control centers, and patients aged < 20 years account for 6.1%.² According to the most recent annual report of the American Association of Poison Control Centers, the most common pediatric ingestions reported to the National Poison Data System include cosmetics/personal care products (13.8%), household cleaning substances (10.4%), analgesics (9.8%), foreign bodies/toys/miscellaneous (6.9%), and topical preparations (6.1%).² Prescription medications most responsible for injury and fatality in children are opioids, sedative/hypnotics, and cardiovascular drugs.³ Certain medications and household substances are known for a high risk of fatality upon ingestion, even if only a small amount is ingested by a small child.⁴ (See **Table 1, page 2.**) In addition, ingestions of magnetic objects and button batteries have become an increasing source of morbidity and mortality.⁵

Ingestion cases pose several challenges to the emergency clinician. Even when a potentially toxic ingestion has been reported, the exact agent, formulation, quantity, or time of ingestion may be unknown. More often, occult ingestion is only one item on an extensive list of differential diagnoses for a critically ill child who presents with altered mental status, respiratory distress, cardiovascular instability, or metabolic derangement. Although physical examination findings and information gleaned by electrocardiographic, laboratory, and radiologic testing may suggest a specific ingestion, timely identification of many substances remains unavailable. In addition to these diagnostic challenges, the management of many ingested agents is controversial and remains the subject of further study and evolving recommendations. Fortunately, many resources are available to clinicians, providing general guidelines as well as individual recommendations. (See **Table 2, page 2.**)

This review presents an evidence-based approach to common pediatric ingestions, with a focus on initial ED stabilization, diagnosis, and management of a selection of the most common and hazardous ingestions, including foreign bodies and medications that may be fatal to children in small doses.

Critical Appraisal Of The Literature

A literature search was performed in PubMed using the search terms *pediatric toxicology epidemiology, poison control, prehospital, toxidrome, electrocardiography, urine drug screen, ipecac, activated charcoal, whole-bowel*

irrigation, hemodialysis, magnet, battery, acetaminophen, salicylate, anticholinergic, cholinergic, alcohol, digoxin, calcium-channel blocker, beta blocker, and lipid emulsion. A search of the Cochrane Library for pediatric ingestion resulted in 5 relevant randomized controlled trials. References cited in review articles were further evaluated. A total of 304 articles were reviewed, 112 of which have been included here. Guidelines released by the American Academy of Pediatrics (AAP), the American Academy of Clinical Toxicology (AACT), and the European Association of Poisons Centres and Clinical Toxicologists (EAPCCT) were reviewed.

Literature regarding pediatric ingestions is largely

Table 1. Medications Known For A High Risk Of Fatality

Medication	Mechanism of Action and Clinical Presentation
Antiarrhythmics	Antiarrhythmic medications block cardiac conduction and may result in dysrhythmia in overdose situations.
Antimalarials	Drugs such as chloroquine, hydroxychloroquine, and quinine act as Class 1A antiarrhythmics to block sodium channels. Additional effects include respiratory and CNS depression.
Antipsychotics	Some antipsychotic medications (such as thioridazine) possess sodium-channel blockade properties, and many can result in CNS depression and hypotension.
Beta blockers	Beta blocker toxicity can result in bradycardia, hyperkalemia, hypotension, and CNS depression.
Calcium-channel blockers	Calcium-channel blocker toxicity can result in bradycardia, hypotension, and hyperglycemia.
Camphor	Camphor is present in many over-the-counter topical preparations, and camphor toxicity includes gastrointestinal symptoms, altered mental status, and seizure.
Clonidine	Alpha-2 receptor stimulation causes transient hypertension, followed by hypotension and bradycardia. Additional effects include respiratory and CNS depression.
Opioids	Opioid medications and other opioid agonists (such as loperamide and buprenorphine) may result in severe respiratory and CNS depression.
Methyl salicylate	Oil of wintergreen and other topical products are highly concentrated salicylates that can cause respiratory alkalosis and metabolic acidosis in overdose.
Sulfonylureas	Sulfonylurea medications can result in hypoglycemia.
Tricyclic antidepressants	Tricyclic antidepressant medications block sodium channels and also have anticholinergic properties.

Abbreviation: CNS, central nervous system.

comprised of case reports, case series, and retrospective studies. Several large retrospective studies have compared treatment modalities for safety and efficacy, and a few randomized controlled trials have evaluated newer treatment modalities. Clinical guidelines are based on expert consensus as well as the available literature, and many have been updated recently to reflect greater emphasis on evidence-based medicine. Data are available through the National Poison Data System, a repository of all calls to United States poison control centers, and the National Electronic Injury Surveillance System, a United States Consumer Product Safety Commission database of ED visits.⁶

Etiology And Pathophysiology

The normal developmental characteristics of children, including frequent hand-to-mouth behavior and increasing mobility and exploration, contribute to ingestions by pediatric patients. Morbidity and mortality resulting from these ingestions are functions of the degree of toxicity and amount of the ingested agent. These issues continue to be addressed by public health initiatives that have included child-resistant and blister packaging for medications, packaging and flavoring of household products to discourage consumption, and warnings about small objects that may easily be swallowed or aspirated by young children. Retrospective studies of these interventions have shown mixed results. Some interventions, such as child-resistant packaging, decreased accidental ingestions, while other interventions, such as embittering antifreeze, did not.⁷ The pathophysiology of common pediatric ingestions will be discussed in subsequent sections addressing the diagnosis and management of specific toxic agents.

Differential Diagnosis

When an ingested substance is unknown, appropriate diagnosis and management begins with a focused history and physical examination, and careful observation for abnormal vital signs and signs and symptoms of common toxidromes, as presented in Table 3. (See page 4.)

Table 2. Resources Available To Clinicians For Management Of Overdose

Resource	Contact Information
American Association of Poison Control Centers	http://www.aapcc.org/ 1-800-222-1222
Battery Ingestion Hotline	http://www.poison.org/battery/ 202-625-3333
Pill Identification: National Library of Medicine	http://pillbox.nlm.nih.gov/pillimage/search.php
World Health Organization Directory of Poison Centers	http://www.who.int/gho/phe/chemical_safety/poisons_centres/en/

Table 3. Differential Diagnosis For Common Toxidromes

Toxidrome or Signs and Symptoms	Drugs/Toxins
Anticholinergic toxidrome • Hyperpyrexia • Mydriasis • Tachycardia • Dry skin • Ileus • Urinary retention • Altered mental status	• Antihistamines • Carbamazepine • Cyclobenzaprine • Jimson weed • Tricyclic antidepressants
Cholinergic toxidrome • Diaphoresis • Bronchorrhea • Increased lacrimation • Salivation • Emesis • Diarrhea • Urination	• Organophosphates • Carbamates • Anticholinesterase inhibitors
Bradycardia with hypotension	• Beta blockers • Calcium-channel blockers • Clonidine • Digoxin
Hyperglycemia	• Alpha agonists • Beta agonists • Calcium-channel blockers • Colchicine • Methylxanthines
Hypoglycemia	• Ethanol • Insulin • Sulfonylureas
Opioid toxidrome • Miosis • Hypothermia • Hypotension • Respiratory depression • Ileus	• Natural opioids (eg, morphine and derivatives) • Synthetic opioids (eg, fentanyl) • Semisynthetic opioids (eg, hydromorphone, hydrocodone) • Methadone • Buprenorphine
Sedation	• Anticonvulsants • Antipsychotics • Barbiturates • Benzodiazepines • Ethanol • Opioids
Seizure	• Anticholinergic agents • Bupropion • Hypoglycemic agents • Isoniazid • Sympathomimetic agents
Sympathomimetic toxidrome • Hyperpyrexia • Mydriasis • Tachycardia • Hypertension • Diaphoresis • Altered mental status • Seizure	• Amphetamines • Cocaine • Ephedrine • Phenethylamines (eg, Class 2C drugs) • Synthetic cathinones (eg, "bath salts")

Prehospital Care

When a dangerous ingestion is suspected, families may contact their physician or poison control center, go directly to the ED, or call emergency medical services (EMS). Poison control centers will refer patients to the nearest ED, if indicated, and instruct them to bring any pill fragments, containers, or substance samples with them. EMS personnel should similarly collect these items at the scene, whenever possible.

Timely arrival to the ED is critical for resuscitation, decontamination, and antidote administration. A retrospective study of 1 ED found that prearrival communication from the poison control center decreased time to receipt of activated charcoal by 12 minutes.⁸ The same data set was used to evaluate the time to receipt of activated charcoal for patients transported by EMS versus other transportation, and no difference was found.⁹

Initial evaluation by EMS personnel includes assessment of airway, breathing, circulation, and neurological status. Although protocols vary by region, most EMS units can monitor cardiac rhythm, oxygen saturation, and glucose level, and may provide oxygen, obtain intravascular access, and administer certain medications. Several retrospective studies and 1 pilot study of 36 patients have evaluated the acceptability, use, timeliness, and complications of administration of activated charcoal by EMS personnel.¹⁰ Although charcoal administration by EMS decreased time to decontamination, it may expose patients with nontoxic ingestions to unnecessary therapy or risk aspiration in patients who may later become sedated.¹⁰ The same benefits and risks have been noted in studies of activated charcoal use in the home, which is not currently recommended by the AAP.¹¹ In cases of opioid overdose, naloxone may be administered by EMS or by laypeople in cities with programs that train potential bystanders in the intranasal administration of the medication.¹²

Emergency Department Evaluation

Initial Stabilization

Pediatric patients who present in critical condition secondary to a toxic ingestion require immediate stabilization according to the Pediatric Advanced Life Support guidelines.¹³ As for any patient requiring resuscitation, evaluation and treatment should begin with an orderly progression through primary and secondary surveys, with a focused history and rapid bedside testing. Airway and respiratory status may be compromised, requiring emergent intubation. The airway may be obstructed by an inhaled object or by increased secretions (in the case of cholinergic ingestion). Early endotracheal intubation may be necessary in cases of caustic ingestion with potential

for rapid development of airway damage.¹⁴ Respiratory status may be compromised by neurologic deterioration, sedation, metabolic derangement, or seizure. Cardiovascular instability secondary to toxic ingestion may include abnormal heart rate or rhythm, as well as hypotension or hypertension, signs that may aid in identification of the responsible agent. Stabilizing treatment may include administration of oxygen, dextrose, intravascular fluid, inotropic agents, antidysrhythmic agents, or antihypertensive agents.¹³

History

The initial history should include information regarding allergies, medications, past medical history, and events leading up to the presentation. A focused history regarding the ingestion of a medication or other substance should include the name, strength, formulation (ie, enteric-coated or extended release), quantity, and time of ingestion. Several online resources are available to assist in identifying unknown pills, including a website hosted by the National Institutes of Health (<http://pillbox.nlm.nih.gov/pillimage/search.php>). Supporting information includes the original and remaining amounts of medication, which may be corroborated by the prescription date and reported medication compliance. Over-the-counter medications may be combination products requiring close examination of the label for active ingredients. Information regarding active ingredients of household substances may also require further investigation of the manufacturer's website.

Details regarding the circumstances of a pediatric ingestion may include remaining substances found in the child's hands or mouth. Suspected but unwitnessed ingestions require questioning regarding the child's medications and other medications or substances in the home, including those brought in by visitors or those at other places where the child spends time. Careful interviewing with regard to suspected child abuse and neglect should be performed. Additional history regarding mental health and suicidal ideation is indicated if intentional ingestion is suspected.

Initial Testing

Immediate testing should include a blood glucose level to identify hypoglycemia or hyperglycemia. Retrospective studies have repeatedly shown that hypoglycemia is seen in sulfonylurea or alcohol ingestion, while hyperglycemia is seen in calcium-channel blocker ingestion.¹⁵ An electrocardiogram (ECG) is indicated to evaluate for dysrhythmia and for prolongation of the QRS complex or the QTc interval, as these abnormalities require rapid intervention and may be clues to identifying an unknown toxicologic agent. A blood-gas panel is helpful to identify disturbances in acid-base balance and elec-

trolyte levels in an ill-appearing patient, although a retrospective study of 595 patients showed no change in management for the 34 who had a blood-gas panel.¹⁶ Pregnancy testing in adolescent female patients is considered good practice and is supported by a retrospective study of nearly 5 million deliveries in which 0.4 per 1000 pregnant women required hospitalization for attempted suicide, with 86% of those attempts by toxic ingestion.¹⁷

Physical Examination

Following initial stabilization and immediate bedside testing, a complete physical examination should be performed, with special attention given to details that may suggest a particular toxidrome. (See **Table 3, page 4.**) Vital signs should be compared to normal ranges by age. Elevated temperature may be secondary to ingestion of salicylates, sympathomimetic or anticholinergic substances, or due to an infectious process. Tachycardia is concerning for sympathomimetic or anticholinergic ingestion, as are dehydration or sepsis. Bradycardia with hypotension suggests ingestion of a beta blocker, calcium-channel blocker, digoxin, or clonidine. Blood pressure may be elevated due to sympathomimetic agents. Respiratory rate may be elevated in sympathomimetic or aspirin ingestion and decreased secondary to narcotic or sedative ingestion.

Thorough examination of the pediatric patient can be aided by parental assistance, allowing the child to be on the parent's lap. The more anxiety-provoking aspects of the examination (such as the oropharynx and ears) should be performed at the conclusion of the examination. Some toxic ingestions, such as ethanol, methylsalicylates, and camphor, may be identified by their odor. Examination of the skin may reveal the hot and dry characteristics of anticholinergic poisoning or diaphoresis in cholinergic poisoning. Bruising or petechiae should raise concern for nonaccidental trauma or an infectious disease. Examination of the head, neck, and mouth includes evaluation for damage from caustic substances.

The cardiac examination should include consideration of rate and rhythm. Respiratory examination should include rate and depth of spontaneous breaths. Abdominal examination may reveal decreased bowel sounds with anticholinergic poisoning. Intussusception should also be considered when examining the abdomen, as children may present primarily with altered mental status that can be mistaken for a case of toxic ingestion. Neurologic examination should include examination of the pupils prior to administration of any medications. Dilated (mydriatic) pupils suggest sympathomimetic or anticholinergic exposure, while constricted (miotic) pupils suggest exposure to narcotics, alpha-1 antagonists, or alpha-2 agonists. Tremulousness may be secondary to hypoglycemia, hypocalcemia, or

lithium ingestion. Seizure may be secondary to electrolyte derangement or to ingestion of bupropion, isoniazid, antihistamine, or antiepileptic drugs.¹⁸ Although assessment of mental status in a young child is challenging, subtle sedation or confusion may be apparent to the caregiver.

Diagnostic Studies

Chemistry And Osmolality

A complete metabolic panel and serum osmolality may be helpful in evaluating for the presence of an anion gap or osmolal gap in an ill-appearing patient with suspected ingestion.¹⁹ The anion gap is estimated by subtracting chloride and bicarbonate levels from the sodium level. Etiologies of an increased gap are represented by the mnemonic MUDPILES. (See Table 4.) Osmolal gap is the difference between measured osmolality and calculated osmolality, which includes sodium, blood urea nitrogen, and glucose levels. An elevated gap may be due to the presence of alcohols, sugars, or other osmotically active substances. Usefulness of the osmolal gap is complicated by various formulas, a wide range of normal baseline values, and an inability to detect substances or metabolites that are not osmotically active.

Table 4. Anion Gap And Osmolal Gap Calculations

Formula	Normal Range	Etiologies of Changes in Levels
Anion Gap		
$\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$	3 to 11	- M: methanol, metformin - U: uremia - D: diabetic ketoacidosis - P: propylene glycol, paraldehyde - I: isoniazid, iron - L: lactate - E: ethylene glycol - S: salicylate
Osmolal Gap		
Measured Osm – Calculated Osm $[\text{Calc Osm}^* = 2(\text{Na}^+) + \text{BUN}/2.8 + \text{Glc}/18]$	-10 to +15	- Ethanol - Methanol - Ethylene glycol - Isopropyl alcohol - Propylene glycol - Acetone - Sugars: mannitol, sorbitol, glycerol

*Formula given in non-SI units: Na, mmol/L; Glc, mg/dL; BUN, mg/dL. Abbreviations: BUN, blood urea nitrogen; Glc, glucose; Na, sodium; SI, International System of Units.

Specific Medication/Substance Levels

Acetaminophen

Acetaminophen levels should be obtained for known or suspected ingestions, as patients may be asymptomatic but at risk for fulminant hepatic failure.²⁰ Acetaminophen is often present in combination products, and has contributed to increasing morbidity and mortality as general use of these products increases.²¹ For known or suspected ingestions of acetaminophen, a serum level should be sent 4 hours following the ingestion or immediately upon presentation if the ingestion time is unknown.

Salicylate

Some controversy surrounds the utility of salicylate levels, as patients are generally symptomatic and the yield of positive results is low in unsuspected cases.²² However, the tachypnea and hyperpnea seen in salicylate ingestion may be masked if there is a mixed ingestion with opioids. In addition, history and symptoms may be difficult to assess in pediatric or suicidal patients, leading to justification for checking this level in seemingly asymptomatic patients. Numerous case reports describe patients with non-specific presentations and unknown ingestions who were found to have elevated salicylate levels.¹⁶ Some recommend sending salicylate levels only in cases of altered mental status and elevated anion gap.²⁰ Because of these controversies, obtaining salicylate levels should be strongly considered.

Other Substances

With medications that form bezoars (such as salicylates or carbamazepine), serum levels will reflect erratic absorption and should be repeated until a definitive downward trend is observed. Blood ethanol level may be helpful in explaining an osmolal gap or in cases of obtundation without known ingestion. Serum iron level is helpful in cases of suspected iron ingestion. Availability and timeliness of other specific drug levels varies by institution. Although this information may be of diagnostic interest, it is unlikely to change management in the ED.

Electrocardiography

In cases of pediatric ingestion, ECGs are commonly used to evaluate critically ill patients, to monitor for abnormalities in stable patients with potentially cardiotoxic ingestions, and as a screening tool for unknown ingestions. Bradycardia may result from ingestion of beta blockers, calcium-channel blockers, digoxin, alpha-2 agonists (clonidine, tizanidine), or cholinergic agents. Characteristic ECG findings of digoxin overdose include first-degree heart block, sagging ST segment, and hyperkalemic changes. Tachycardia may result from anticholinergic agents and sympathomimetics. Drugs that block fast sodium channels (including carbamazepine, propranolol)

lol, Class I antiarrhythmics, and some local anesthetics) widen the QRS interval, while drugs that block potassium efflux channels (including anticholinergic drugs and Class III antiarrhythmics such as amiodarone) prolong the QTc interval.²³ Tricyclic antidepressants have both sodium-channel blockade and anticholinergic properties, resulting in tachycardia, QRS widening, and QTc prolongation.²⁴

The utility of ECG as a diagnostic test is limited by age-based and individual variations, as well as by low sensitivity and specificity. Although abnormal ECG findings in symptomatic patients may require intervention, slight ECG abnormalities in stable patients may not be clinically significant, and a normal ECG does not preclude adverse events. A retrospective study of tricyclic antidepressant ingestion that evaluated QRS duration threshold to predict dysrhythmia or seizure and to determine disposition from the ED showed mixed results.²⁵ ECG has been proposed as a screening test for unknown or suspected ingestions, but a retrospective chart review did not show significant ECG changes in 73 pediatric patients with known ingestions who had ECG performed.¹⁶ Similarly, a retrospective chart review of 35 pediatric patients with known tricyclic antidepressant ingestion did not demonstrate significant differences in ECGs compared with control patients.²⁶ However, these small studies have limited power to detect rare events.

Radiographic Studies

Radiologic studies may be indicated when a radiopaque medication or foreign body will be visible on plain x-ray. Although some nonmetallic objects may be visible on x-ray, identification was not reliable in an in vitro study.²⁷ Strongly radiopaque medications include ferrous sulfate, calcium carbonate, and potassium chloride, while many other medications are only weakly radiopaque and may not be identified on x-ray due to formulation, size and position of the medication, or interpretive skill of the clinician.²⁸ Radiographs may identify ingested foreign bodies that have become lodged and require surgical removal. Suspected magnet ingestion requires plain x-ray to evaluate the number of magnets.²⁹ Radiographs should also be obtained to distinguish coins from button batteries, the latter of which are recognized by their double rim on anterior-posterior radiographs (see Figure 1) or by a step-off on lateral film.³⁰ Radiography is also helpful in evaluation and management of foreign bodies containing lead.³¹

Urine Toxicology Screen

Often, rapid drug screen tests include a small number of drugs of abuse because they were initially developed for use in pre-employment screening. Emergency clinicians must be aware of which substances are included in their institution's urine

drug screen and the cut-off thresholds. These tests are known to have many common false-positive and false-negative results as well as known cross-reactivity.³² Comprehensive urine toxicology screens include additional substances, but may be of limited availability, and the results are not available in a timely fashion. A urine sample may aid in the diagnosis of ethylene glycol ingestion, since fluorescein (which may be present as an additive in antifreeze products) is detectable with illumination from a Wood's lamp.³³ However, not all antifreeze products contain fluorescein, and 1 study of physicians' abilities to detect fluorescence showed poor sensitivity and specificity.³⁴

Studies of adult patients presenting with polysubstance overdose show discordance between reported or clinically suspected ingestions compared to their urine toxicology screens; nonetheless, very few of the screenings resulted in changed management.³⁵ Although ingestions by children are less likely to be intentional, polysubstance, or involve drugs of abuse, some clinicians argue for the value of making a diagnosis using a comprehensive drug screen. In a 1981 study, 51% of confirmed poisonings in children were identified by comprehensive evaluation.³⁶ However, the expense of the test and conse-

Figure 1. Ingested Button Battery With Double Rim At Thoracic Inlet



Image courtesy of Stacy Tarango, MD.

quences of inaccurate results must be considered. In a retrospective study of 62 drug screens in children, patient management was changed in only 3 cases by the addition of a social work evaluation, as the caregivers subsequently confessed their child's exposure to cocaine or opioids after a positive result.¹⁶ A previous study showed similar results, with only 7 of 234 positive comprehensive urine toxicology screens resulting from an unsuspected exposure, with none changing management.³⁷

Treatment

Foreign Body Removal

Ingested foreign bodies generally pass without need for intervention. Objects may require removal if they become lodged at the level of the thoracic inlet, diaphragm, or pylorus.³⁸ Multiple magnets, or combinations of a magnet and another object (to which it may be attracted) require removal, as tissue trapped between the objects may necrose, leading to perforation and fistula formation.²⁹ Consultation with a surgical specialist is recommended.

Management guidelines regarding treatment of button battery removal are available through the National Button Battery Ingestion Hotline at (202) 625-3333 and are based on patient age, symptomatology, and battery size. **(See the Clinical Pathway on pages 10-11.)** Button batteries in the esophagus require immediate removal, as burns may occur within 2 hours of contact.³⁰ A button battery that has passed into the stomach in an asymptomatic patient without co-ingestion of a magnet may be observed at home with or without follow-up x-ray, depending on the size of the battery and the age of the patient. Older children with smaller battery ingestions may be observed at home with consideration of x-ray if the object is not noted in the stool.³⁰ Removal is indicated if follow-up radiography reveals that the battery has not passed the stomach or if symptoms develop, and specialist consultation is recommended.³⁰

Decontamination

Gastric Lavage

The AACT and the EAPCCT published a position paper in 1997, with updates in 2004 and 2013, concluding that gastric lavage should not be used routinely, if at all.³⁹ Although older literature on animal and human volunteer studies, as well as case reports, showed variable ability to remove toxins, the evidence for clinical utility is lacking and risks of the procedure, including aspiration and perforation, outweigh the benefits.³⁹

Ipecac

A randomized controlled trial of syrup of ipecac with activated charcoal versus charcoal alone showed that ipecac delayed charcoal administration, increased

vomiting of charcoal, and prolonged ED length of stay.⁴⁰ The AAP, the AACT, and the EAPCCT have published clinical guidelines recommending against the use of ipecac syrup to induce emesis.^{41,42}

Activated Charcoal

Activated charcoal serves to adsorb toxins and binds best to compounds that are organic, large, and poorly water soluble. Activated charcoal binds poorly and variably to alcohols, iron, lithium, acids, alkalis, electrolytes, arsenic, and other heavy metals.⁴³ Use of activated charcoal has declined in cases reported to poison centers, with only 1% of pediatric cases in 2012 receiving charcoal.² The most recent clinical guidelines by the AACT and the EAPCCT suggest that single-dose activated charcoal may be considered within 1 hour of ingestion of a life-threatening amount of a substance known to bind to charcoal.⁴⁴ Multidose activated charcoal interrupts enterohepatic reabsorption and has been shown to increase elimination of carbamazepine, dapsone, phenobarbital, quinine, and theophylline, although clinical benefit has not been proven for these ingestions and is even less clear for many other medications.⁴⁵ However, a randomized controlled trial of adult volunteers suggested efficacy of high-dose super-activated charcoal 3 hours after acetaminophen ingestion.⁴⁶

Aspiration of activated charcoal causes severe pneumonitis, so its use is contraindicated in patients who are unable to protect their airway unless the airway has been secured by intubation. Activated charcoal with a cathartic is not recommended in children due to increased risk of fluid and electrolyte imbalance.⁴⁵

The recommended dose is a charcoal-to-drug ratio of 10:1, or 1 to 2 g/kg to a maximum of 50 grams. It may be made more palatable by mixing it in food such as chocolate pudding or flavored syrup.

Whole-Bowel Irrigation

According to position papers from the AACT and the EAPCCT, whole-bowel irrigation should not be used routinely, but may be considered in potentially toxic ingestions of extended-release or enteric-coated medications, for substances that cannot be eliminated by other methods, and for patients who have ingested packets of illicit drugs.⁴⁷ This recommendation reflects studies showing decreased bioavailability of ingested substances, but without clear evidence for improved clinical outcomes. A retrospective review of ingestions that occurred between 2000 and 2010 in patients aged < 12 years and were reported to the California Poison Control System showed 176 cases of whole-bowel irrigation use, most commonly for ingestion of calcium-channel blockers, concentrated preparations of iron, and antidepressants.⁴⁸ Whole-bowel irrigation is contraindicated in patients with a compromised airway, intestinal obstruction, ileus, or bowel perforation. Dosing of polyethylene

glycol solution is 25 mL/kg/h up to 1 L/h, which often requires administration by nasogastric tube.

Hemodialysis

Hemodialysis serves to remove low-molecular-weight toxins that are water soluble, with a low volume of distribution, and are not highly bound by proteins. More recently, technological innovation is expanding the efficacy of hemodialysis for other toxins.^{49,50} The most common agents requiring dialysis are lithium, salicylates, and ethylene glycol. Additional agents well-suited to elimination by hemodialysis include methanol, valproic acid, isopropanol, and theophylline. Other agents, including ethanol, acetaminophen, carbamazepine, and metformin, are amenable to hemodialysis, but have other preferred treatment modalities. Agents with a high volume of distribution and extensive protein binding, such as digoxin, are not effectively dialyzed.⁴⁹ Patients for whom hemodialysis is expected require early transfer to an institution with this capability.

Treatment Of Specific Toxic Ingestions

Acetaminophen

Acetaminophen is a ubiquitous analgesic that is frequently present in combination products that have been linked to increasing fatality rates in adult ingestion cases.²¹ Acetaminophen toxicity occurs due to accumulation of N-acetyl-p-benzoquinone imine (NAPQI), a metabolite detoxified by glutathione. When glutathione stores are depleted, NAPQI causes hepatic centrilobular necrosis.⁵¹ Early symptoms of acetaminophen overdose (such as nausea, vomiting, and malaise) are nonspecific and progress to hepatomegaly, right upper quadrant pain, and, finally, hepatic failure that may be fatal or require liver transplantation.⁵²

Although the toxic dose of acetaminophen varies depending on chronicity of use, baseline hepatic function, concurrent medications, and other specific patient characteristics, acute doses > 150 mg/kg are considered toxic.⁵³ The classic nomogram developed by Rumack and Matthew provides thresholds for toxic levels, and begins 4 hours after ingestion with a level of 200 mcg/mL, although 150 mcg/mL is used routinely to avoid missed treatment opportunities.⁵⁴ (See Figure 2.) The Rumack-Matthew nomogram is only applicable in cases of acute acetaminophen overdose.²² Studies since that time have evaluated establishing levels prior to 4 hours, with variable results.⁵⁵

The antidote for acetaminophen toxicity, N-acetylcysteine (NAC), prevents toxicity by rapidly detoxifying NAPQI and is most efficacious if administered within 8 to 10 hours of acetaminophen ingestion, with some studies showing waning but persistent benefit up to 24 hours after ingestion and potential benefit via other mechanisms even beyond that time.^{56,57}

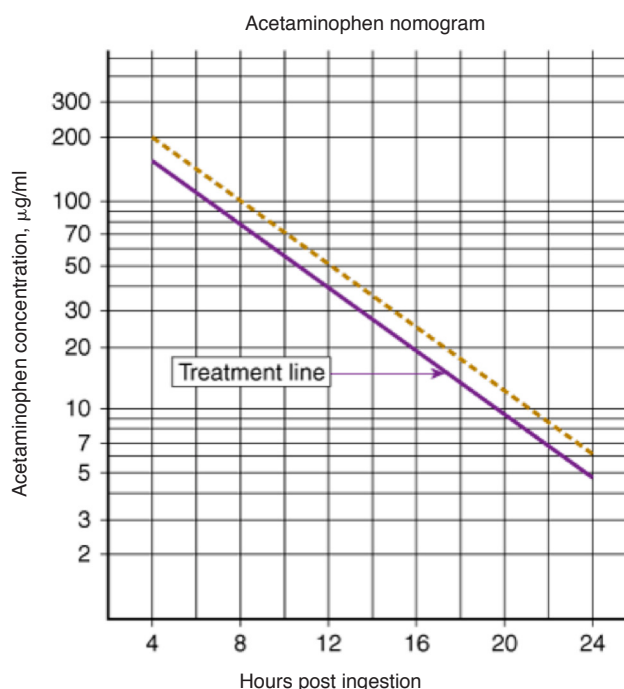
NAC may be given orally or intravenously, and numerous studies have compared the 2 routes as well as the optimal duration of therapy. The intravenous route is associated with anaphylactoid reactions and with hyponatremia if the concentration is not appropriate for small patients. Tolerance of oral administration may be improved by mixing NAC with flavored syrup and by administering antiemetic medication concurrently. Oral NAC is administered at a loading dose of 140 mg/kg, followed by 70 mg/kg every 4 hours for 17 doses, although shorter courses are possible.⁵⁸ Intravenous NAC is administered at 150 mg/kg over 1 hour, followed by 12.5 mg/kg/h for 4 hours, and then 6.25 mg/kg/h for 16 hours.⁵⁹

Levels of transaminases, prothrombin time (PT), international normalized ratio (INR), and acetaminophen should be checked prior to expected completion of therapy.⁶⁰ Intravenous NAC has been shown to increase PT in clinical reports and in in vitro studies, complicating interpretation of PT/INR values following therapy.⁶⁰ NAC may be discontinued when aspartate transaminase and alanine transaminase are both < 1000 units/L and trending downward, and when acetaminophen is undetectable. This may require a prolonged course in some situations.⁶¹

Alcohols

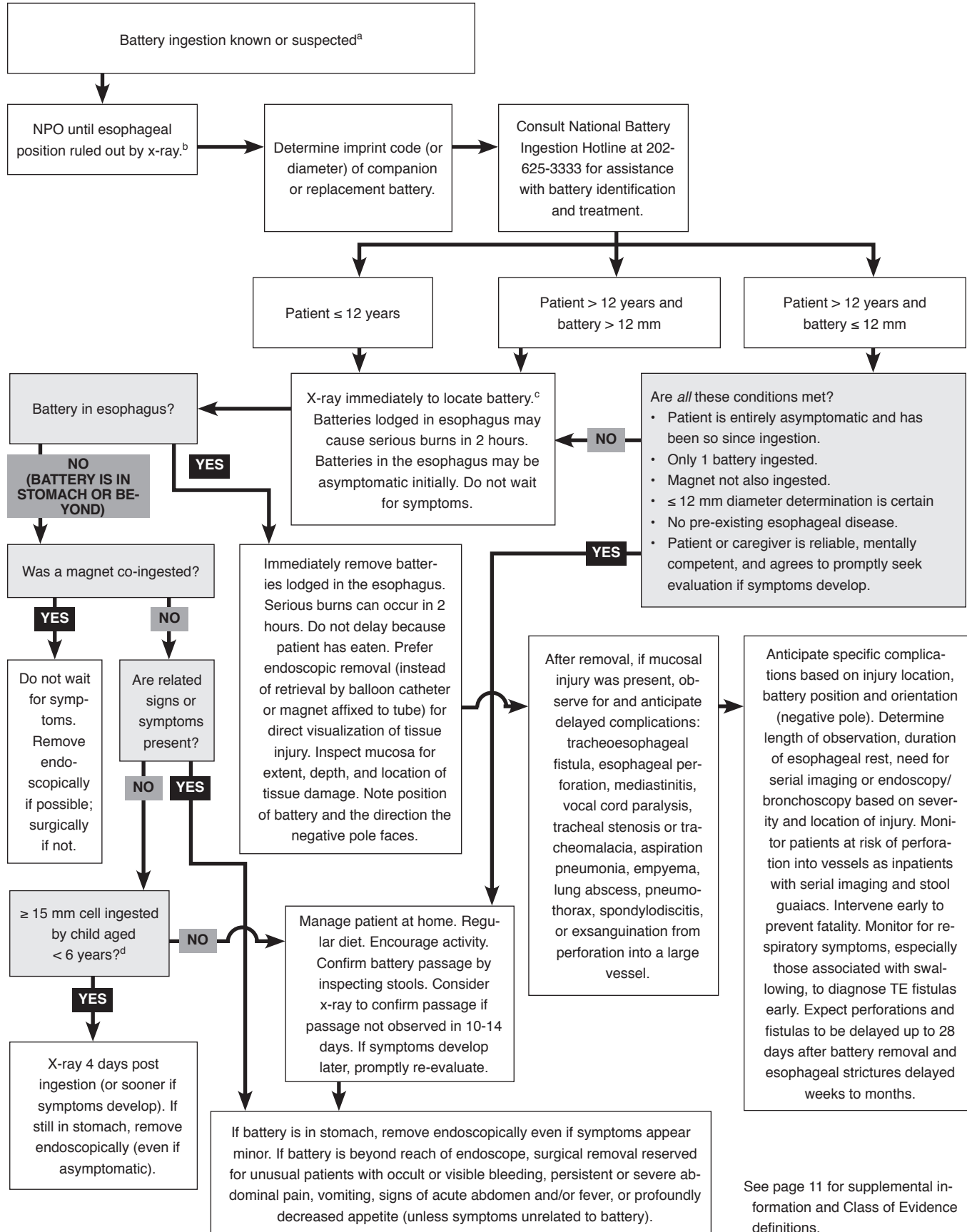
Ingestion of ethanol or isopropanol may result in respiratory and central nervous system depression.⁶² In small children, ethanol may also cause hypoglycemia. Methanol and ethylene glycol are toxic alcohols

Figure 2. 1981 Rumack-Matthew Nomogram



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Clinical Pathway For Management Of Battery Ingestions In Pediatric Patients



See page 11 for supplemental information and Class of Evidence definitions.

present in windshield wiper fluid and antifreeze, respectively, that are a risk to children and adolescents when imbibed accidentally or as an alternative to ethanol. Methanol is metabolized to acetaldehyde and then to formic acid, which causes a high anion gap metabolic acidosis, increased osmolal gap, and may lead to blindness and death, with toxicity noted at doses of 0.1 mL/kg of 100% methanol.⁶³ Ethylene glycol is toxic at 0.2 mL/kg and also causes high anion gap metabolic acidosis and an increased osmolal gap when it is metabolized to oxalates by alcohol dehydrogenase.⁶³ Methanol and ethylene glycol poisoning may be treated with fomepizole, a competitive inhibitor of alcohol dehydrogenase, and it has largely replaced ethanol as treatment for these ingestions.⁶⁴ Fomepizole induces a secondary metabolic process, which complicates dosing. It is given as a 15 mg/kg loading dose, followed by 10 mg/kg every 12 hours for 4 doses, and then 15 mg/kg every 12 hours until alcohol levels have decreased to safe levels. Hemodialysis is also a modality to eliminate these toxic alcohols, and fomepizole dosing must be adjusted in the setting of hemodialysis.⁶⁵

Anticholinergic Agents

Substances exhibiting anticholinergic qualities include tricyclic antidepressants, diphenhydramine, Jimson weed, atropine, scopolamine, and carbamazepine, among others. Anticholinergic symptoms include mydriasis; decreased urination, salivation, and sweating; hypoactive bowel sounds; tachycar-

dia; hyperpyrexia; delirium; and hallucinations. Physostigmine is an acetylcholinesterase inhibitor that crosses the blood-brain barrier and briefly alleviates anticholinergic toxicity, which may be of diagnostic utility.⁶⁶ However, it is associated with seizure, bronchospasm, and bradycardia, and so it is not routinely used and is contraindicated in cases of proconvulsant ingestion, abnormal ECG findings, or tricyclic antidepressant ingestion.

Although treatment for anticholinergic agent overdose is largely supportive, if physostigmine is utilized and the benefit is thought to outweigh the risk, the dose is 0.02 mg/kg intravenously to a maximum of 0.5 mg, and it should be administered slowly, over 5 to 10 minutes, with resuscitation equipment and atropine available.⁶⁷

Beta Blockers

Case reports and retrospective database studies have shown that beta blockade results in bradycardia, hypotension, and central nervous system depression.^{68,69} A prospective study of 208 patients with beta blocker exposure reported to poison control centers did not find any cases of serious toxicity, including hypoglycemia.⁷⁰ Treatment includes intravenous fluids, vasopressors, and supportive care. Glucagon is also used as an inotropic agent, although this remains controversial, as data are available only from animal studies and case series.⁷¹⁻⁷³

Glucagon may be given as a 50- to 150-mcg/kg intravenous bolus followed by a continuous infusion.⁶⁹

Clinical Pathway Supplemental Information

TIPS, PITFALLS, AND CAVEATS

- "3 Ns": Negative – Narrow – Necrotic. The negative battery pole, identified as the narrowest side on lateral x-ray, causes the most severe, necrotic injury. The negative battery pole is the side opposite the "+" and without the imprint.
- 20-mm lithium coin cell is most frequently involved in esophageal injuries; smaller cells lodge less frequently but may also cause serious injury or death.
- Definitive determination of the battery diameter prior to passage is unlikely in at least 40% of ingestions.
- Assume hearing aid batteries are < 12 mm.
- Manage ingestion of a hearing aid containing a battery as an ingestion of a small (≤ 12 mm) battery.
- Do not induce vomiting or give cathartics. Both are ineffective.
- Assays of blood or urine for mercury or other battery ingredients are unnecessary.

^aSuspect a battery ingestion in these situations:

1. "Coin" ingested: check AP x-ray for battery's double rim or halo effect and lateral view for step-off.
2. Symptomatic patient, no ingestion history. Consider battery ingestion if:
 - Airway obstruction or wheezing
 - Drooling
 - Vomiting
 - Chest discomfort
 - Difficulty swallowing, decreased appetite, refusal to eat
 - Coughing, choking, or gagging with eating or drinking

^bNPO. Anesthesia may be required for removal.

^cX-ray abdomen, esophagus, and neck. Batteries above the range of the x-ray have been missed. If battery in esophagus, obtain AP and lateral x-rays to determine orientation of negative pole. If ingestion suspected and no battery visualized on x-rays, check ears and nose.

^dIf battery diameter is unknown, estimate it from the x-ray, factoring out magnification (which tends to overestimate diameter).

Abbreviations: AP, anterior-posterior; NPO, nothing by mouth; TE, transesophageal.

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Hyperinsulinemia/euglycemia therapy, initially used for calcium-channel blocker toxicity, is a newer treatment modality. It appears to increase inotropy through a variety of mechanisms, including increased glucose uptake by myocardial cells,⁷⁴ and is administered as a high-dose insulin bolus of 1 U/kg intravenously, followed by 0.5 U/kg/h with concurrent glucose administration to maintain euglycemia.¹⁵

Calcium-Channel Blockers

Calcium-channel blockers are a group of antihypertensive agents that include amlodipine, verapamil, nifedipine, and diltiazem, among others. Although many pediatric patients remain asymptomatic after ingestion of calcium-channel blockers, toxicity in overdose includes hypotension, bradycardia, heart block, and hyperglycemia, according to large retrospective studies.⁷⁵⁻⁷⁷ Hyperglycemia is due to blockade of calcium channels involved in pancreatic insulin release and is a feature that differentiates this ingestion from the otherwise similar symptoms of beta-blocker toxicity.⁷⁸ Treatment modalities include intravenous fluids and vasopressors to support blood pressure, calcium, glucagon, and hyperinsulinemia/euglycemia.

Cholinergic Agents

Cholinergic agents include organophosphates and carbamates in pesticides and warfare agents, and anticholinesterase medications such as rivastigmine, donepezil, and galantamine, used in the treatment of Alzheimer disease. The cholinergic toxidrome results from the inhibition of cholinesterase enzymes, allowing excessive stimulation of cholinergic neurotransmission.⁷⁹ This toxidrome is often represented by the mnemonic devices SLUDGE (salivation, lacrimation, urination, diarrhea, gastrointestinal distress, emesis) and DUMBBELLSS (diarrhea, urination, miosis, bradycardia, bronchospasm, emesis, lacrimation, lethargy, salivation, seizures). Additionally, a retrospective study of adult patients showed a variety of cardiac manifestations, including dysrhythmia, ECG abnormalities, bradycardia or tachycardia, and hypertension or hypotension.⁸⁰

Patients must first be decontaminated, if necessary, to avoid contamination of healthcare personnel. Atropine should be given at a starting dose of 0.05 mg/kg intravenously over 3 minutes or intramuscularly, and this dose may be increased and repeated every 15 minutes as needed to dry respiratory secretions, since bronchorrhea and bronchospasm are the life-threatening symptoms of cholinergic poisoning.⁷⁹ Pralidoxime inhibits the aging of acetylcholinesterase so that the enzyme can be reactivated, although this depends on the properties of the poisoning agent and the time to pralidoxime administration. It is given as a bolus of 25 mg/kg to a maximum of 1 gram intravenously over 15 to 30 minutes and then as a continuous

infusion of 20 mg/kg.⁶³ Benzodiazepines should be administered for seizures.⁷⁹

Digoxin

Digoxin, a cardiac glycoside, augments myocardial contractility by inhibiting sodium/potassium-adenosine triphosphatase (Na^+/K^+ -ATPase), and has a narrow therapeutic window. Signs of digitalis toxicity include bradycardia, heart block, hypotension, and hyperkalemia, as well as nonspecific symptoms of headache, agitation, visual disturbances, nausea, and vomiting.⁸¹ An ECG and potassium level should be obtained initially and followed, as dysrhythmias and hyperkalemia are life-threatening. Classic ECG findings include first-degree heart block, sagging ST segments, and peaked T waves. Levels of digoxin may not reflect toxicity, given tissue redistribution and influence of baseline cardiac function and other medications. Multidose activated charcoal may be indicated.

The treatment for digoxin toxicity is digoxin immune Fab, an antibody fragment to digoxin.⁸² It is indicated for an ingestion of > 0.3 mg/kg, a serum level > 5 ng/mL, hyperkalemia, or rapid progression of toxicity.⁸¹ Dosing is calculated from a steady-state serum level 6 hours after ingestion, or from 80% of the amount ingested, to account for its incomplete absorption. One vial of 40 mg of digoxin immune Fab will bind 0.6 mg of digoxin, and the recommended dose is 80% of this calculated amount, given intravenously over 30 minutes.⁸³ If the ingestion amount is unknown, then the recommended dose is 10 to 20 vials of digoxin immune Fab intravenously.⁸⁴ Risks of digoxin immune Fab include allergic reaction to immunoglobulin and precipitation of hypokalemia.⁸⁵ Digoxin-Fab complexes are measured by the digoxin assay, so levels are no longer useful once digoxin immune Fab has been administered unless a free digoxin assay is available.⁸⁶ Bradycardia may be treated with atropine or cardiac pacing. Dysrhythmias, including automaticity and decreased conduction, may be treated with lidocaine, avoiding class IA or IC antiarrhythmics.

Iron

Iron is commonly present in the homes of toddlers, due to its use by pregnant and postpartum women, and it is toxic in doses as small as 40 mg/kg. Stages of iron poisoning progress from vomiting and gastrointestinal irritation to a relatively quiescent stage, and then to metabolic acidosis and shock, with potential renal and hepatic failure.⁸⁷ Treatment is largely based on consensus and in response to case reports and retrospective studies.^{88,89} Therapeutic modalities include supportive care, whole-bowel irrigation, iron chelation by deferoxamine, and exchange transfusion. Deferoxamine dosing is generally 15 mg/kg/h intravenously, although case reports describe both positive results and complications from higher doses.^{89,90}

Opioids

Opioid medications, including agents meant to combat addiction (such as methadone and buprenorphine), and the antidiarrheal loperamide, cause respiratory and central nervous system depression secondary to effects on the mu receptor.⁹¹ Naloxone is an opioid receptor antagonist and is dosed at 0.1 mg/kg intravenously up to 2 mg. The dose may be repeated every 2 to 3 minutes to a maximum of 10 mg. Naloxone's half-life is 30 to 100 minutes, and so a continuous infusion of two-thirds of the reversal dose (ie, 0.067 mg/kg/h if 0.1 mg/kg reversed symptoms) and titrated to effect, may be required to continue the reversal of long-acting opioids.⁹²

Salicylates

Salicylates uncouple oxidative phosphorylation, resulting in hyperthermia, lactic acidosis, respiratory alkalosis, and hypoglycemia.⁹³ Early symptoms of salicylism include tinnitus, hyperpnea or tachypnea, and gastrointestinal upset. Although serum salicylate levels are helpful, they do not reflect the total burden of salicylate and may be low in a patient with severe toxicity; therefore, management should be based upon clinical status.⁶⁵ Severe toxicity may resemble sepsis, with high temperature, altered mental status, and increased respiratory rate. Levels may also vacillate with prolonged and variable absorption from an aspirin bezoar, enteric formulations, or pylorospasm, as noted in numerous case reports as well as in vitro studies.^{93,94}

Decontamination modalities include multiple-dose activated charcoal and whole-bowel irrigation. Treatment of salicylate poisoning includes urinary alkalization, shown by experimental and clinical studies to increase elimination.^{93,95} This may be accomplished with 150 mEq sodium bicarbonate in 1000 mL of 5% dextrose and water with 40 mEq of potassium chloride per liter at 2 to 3 mL/kg/h to achieve urine output of 1 to 2 mL/kg/h, with urine pH > 7.5. Common dosing mistakes include omitting potassium or using 5% dextrose in saline, which will result in a hypertonic solution.⁹³ Indications for hemodialysis include severe acidosis, hypotension, end-organ damage, or neurologic impairment. A definitive salicylate level requiring hemodialysis in the absence of clinical indications is controversial and without strong evidence, but is often cited at 100 mg/dL and lower in cases of chronic exposure. Intubation may be unavoidable in some clinical scenarios, but will result in further decompensation when the patient loses the ability to hyperventilate.⁹⁶

Sedative-Hypnotics

Benzodiazepine drugs have sedative, hypnotic, and anticonvulsant properties due to their stimulation of gamma-aminobutyric acid-A (GABA_A) receptors. Retrospective studies demonstrate that ingestion by children results in ataxia, lethargy, coma, and respi-

ratory depression.⁹⁷ Flumazenil is a competitive inhibitor at the GABA_A receptor and may be used as a reversal agent at a dose of 0.01 mg/kg intravenously and repeated for 4 doses to a maximum of 1 mg total. The short half-life of flumazenil may require continued administration, but even a brief response may be of diagnostic value in a case of unknown or suspected ingestion in a sedated patient. Risks of flumazenil, such as precipitation of seizures in patients who use benzodiazepines chronically or who have ingested another proepileptic agent, may outweigh benefits. In pediatric patients who are more likely to present with acute ingestion and a known medical history, the benefit of diagnosis and reversal is greater and the risk of seizure lower than in adults.⁹⁸

Sulfonylureas

Sulfonylurea medications, such as glyburide and glipizide, stimulate the release of insulin to decrease glucose levels, with a peak effect within 2 to 6 hours and a duration of 12 to 24 hours.⁹⁹ Large retrospective studies show that hypoglycemia may be severe, prolonged, and with delayed onset in pediatric overdose.¹⁰⁰⁻¹⁰² Therefore, asymptomatic patients should be admitted and observed with frequent blood glucose evaluation and access to a regular diet.¹⁰³ Most experts recommend 16 to 24 hours of observation, including an overnight period, as a sleeping child may become hypoglycemic if not awakened to eat.⁹⁹ Treatment of hypoglycemia includes administration of dextrose, which may be given as a 5-mL/kg bolus of 10% dextrose via a peripheral intravenous line, followed by dextrose-containing maintenance fluid. Prophylactic dextrose is discouraged, as this may mask hypoglycemia.¹⁰⁰ Glucagon is not recommended due to its short half-life, rebound effect, and the possibility of inadequate pediatric glycogen stores to mobilize the substance. However, it may be useful if intravenous access has not been obtained, as it may be given intramuscularly or subcutaneously at a dose of 1 mg in adults, 0.5 mg in children, and 50 mcg/kg in infants.¹⁰² Octreotide is a long-acting somatostatin analog that inhibits insulin release and is recommended for hypoglycemia that is refractory to dextrose infusion.¹⁰¹ It may be given intravenously or subcutaneously at a dose of 1 to 1.5 mcg/kg to a maximum of 50 mcg, and repeated every 6 hours for a total of 4 doses, or infused continuously if needed.¹⁰⁴

A summary of emergency medications is provided in **Table 5, page 15**.

Special Populations

Children presenting with a toxic ingestion who are younger or older than the usual age for exploratory and mouthing behavior require additional consideration, as these may be cases of intentional ingestion.

Risk Management Pitfalls In Management Of Ingestions In Pediatric Patients

1. **"I didn't think the poison control center would be helpful."**
The American Association of Poison Control Centers provides recommendations from tremendous resources and experience. In addition to the benefits of better patient care and clinician education, the data you provide will be included in the National Poison Data System to further knowledge in the field.
2. **"I give activated charcoal to all patients with ingestions."**
Activated charcoal increases the risk of aspiration pneumonitis and is unlikely to be of benefit once the toxin has been absorbed. Routine use is no longer recommended unless a toxin shown to be bound by activated charcoal was ingested in the past hour by a patient to whom charcoal may be safely administered.
3. **"The parents didn't mention giving aspirin to their febrile child, so I didn't consider it."**
Symptoms of a toxic ingestion may be nonspecific, and an elevated temperature may be due to ingestion of salicylates, anticholinergic agents, or sympathomimetic agents, in addition to an infectious process. Always ask about use of over-the-counter medications and their ingredients.
4. **"The urine toxicology screen was negative, so ingestion is ruled out."**
Urine toxicology screen interpretations are limited by which drugs are included and at what threshold levels, in addition to false-negative and false-positive results.
5. **"He attempted suicide by taking ibuprofen. Why would we check for acetaminophen?"**
Polypharmacy is common in suicidal ingestions, and acetaminophen overdose may present without symptoms and lead to fulminant hepatic failure.
6. **"The ingested battery can wait until the surgical department opens in the morning."**
A battery lodged in the esophagus may cause necrotic damage within 2 hours and should be removed immediately to prevent perforation and fistula formation.
7. **"We'll give dextrose to prevent hypoglycemia after suspected sulfonylurea ingestion."**
Prophylactic dextrose will mask and possibly delay effects of sulfonylurea ingestion, confusing further management. Dextrose should only be administered as needed.
8. **"Naloxone reversed the effect of methadone ingestion, so the child can be discharged."**
Naloxone's half-life is less than that of methadone, and clinicians may expect recrudescence of central nervous system and respiratory depression, requiring additional antidote administration.
9. **"She became apneic after receiving lorazepam for her seizure, so we gave flumazenil."**
Flumazenil administration in a patient with seizure disorder or chronic benzodiazepine use may precipitate intractable seizures and is contraindicated.
10. **"The mother said that her 7-month-old baby got into this medication herself."**
Although most ingestions by young children are due to normal exploratory behavior, home safety and the possibility of abuse should be addressed by clinicians, especially in cases with an implausible history.

Numerous case reports have described apparent life-threatening events as a result of intentionally abusive or neglectful administration of medications, alcohol, or other toxic agents.^{105,106} A prospective study of patients with apparent life-threatening events demonstrated that 8.4% of patients with a comprehensive urine toxicology screen had a clinically significant result, including cough and cold preparations, that were not reported by the parent.¹⁰⁶ Consultation with social work and child protective services is required if abuse or neglect is suspected. Older children and teenagers presenting with toxic ingestions also require additional psychosocial intervention to screen for suicidality and to arrange for psychiatric care once they are medically stable.

Controversies And Cutting Edge

Marijuana Exposure

Increasing use of medical or recreational marijuana in the United States has raised concern for increased risk of ingestion by children. Numerous case reports and case series describe pediatric patients with altered mental status, ataxia, and respiratory insufficiency following cannabis ingestion.^{107,108} A retrospective study demonstrated increasing pediatric exposures as the number of states in which marijuana is legal has increased.¹⁰⁸ Given the risks of marijuana use without clear evidence for benefit, the AAP does not recommend use of medical marijuana in children and adolescents. Further study of cannabinoid use and toxicity and the development of guidelines or legislation to prevent accidental pediatric intoxication are likely forthcoming. Additional substances with increasing use and potential toxicity when ingested by children include liquid nicotine in e-cigarettes and synthetic cannabis products, such as "Spice" and "K2."

Intravenous Lipid Emulsion Therapy

A new treatment modality in toxicology is intravenous lipid emulsion (ILE) therapy, which has been used to treat toxicity from topical anesthetics by creating a "lipid sink." The mechanism continues to be elucidated.¹⁰⁹ Data are largely limited to numerous positive case reports, but a small randomized controlled trial of ILE in healthy volunteers did not show a difference in subjective findings or electroencephalogram results after lidocaine administration and infusion of lipids or saline.¹¹⁰ Case reports have described ILE as a therapy for ingestion of beta blockers, calcium-channel blockers, and antidepressants. Complications of ILE therapy include pancreatitis, laboratory interference, and a case report of lipemia precluding use of renal replacement therapy.¹¹¹ Dosing regimens vary and include a 1.5 mL/kg bolus of 20% lipid emulsion, which may be repeated after 5 minutes or followed by a constant

infusion of 0.25 mL/kg/min. ILE is contraindicated for patients with an egg or soy allergy.

Disposition

Disposition is contingent on clinical status as well as the expected course for a given toxicologic ingestion. Children requiring critical care or patients at risk for deterioration due to dysrhythmia, apnea, or seizure require admission to the intensive care unit for management and monitoring. Asymptomatic patients may require observation in the hospital if

Table 5. Emergency Medications For Overdose

Medication	Dose
Activated charcoal	• 1-2 g/kg PO or NG, max 50 gram
Atropine	• 0.05 mg/kg IV over 3 minutes or IM, repeated every 15-30 minutes to dry secretions
Calcium	• Calcium chloride: 10-20 mg/kg IV, given slowly* • Calcium gluconate: 10-20 mg/kg IV, given slowly
Dextrose	• 5 mL/kg IV of 10% dextrose
Deferoxamine	• 15 mg/kg/h IV
Flumazenil	• 0.01 mg/kg IV, may be repeated for 4 doses, max 1 mg total
Fomepizole	• 15 mg/kg IV, then 10 mg/kg q12h for 4 doses, then 15 mg/kg q12h
Glucagon	• For hypoglycemia: weight < 20 kg: 0.5 mg, weight > 20 kg: 1 mg IV, IM, or SC • For beta-blocker toxicity: 50-150 mcg/kg IV
Hyperinsulinemia/euglycemia therapy	• 1 unit/kg IV insulin bolus, followed by 0.5 units/kg/h with concurrent glucose administration
N-acetylcysteine	• PO: 140 mg/kg, then 70 mg/kg every 4 hours for 17 doses • IV: 150 mg/kg over 1 hour, then 12.5 mg/kg/h for 4 hours, then 6.25 mg/kg/h for 16 hours
Naloxone	• 0.1 mg/kg IV or IM, max 2 mg, repeated every 2-3 minutes to a maximum of 10 mg, followed by a continuous infusion of two-thirds of the reversal dose
Octreotide	• 1-1.5 mcg/kg SC or IV to a max of 50 mcg, repeated every 6 hours for a total of 4 doses, or continuous infusion
Polyethylene glycol	• 25 mL/kg/h PO or NG, max 1 L/h
Pralidoxime	• 25 mg/kg IV over 15-30 minutes, max 1 g IV or 2 g IM, followed by continuous infusion of 10-20 mg/kg/h
Sodium bicarbonate	• 0.5-1 mEq/kg IV, given slowly

*Calcium chloride has more elemental calcium and a higher risk of extravasation complications when given peripherally.

Abbreviations: IM, intramuscular; IV, intravenous; NG, nasogastric; PO, by mouth; q12h, every 12 hours; SC, subcutaneous.

the ingested agent has delayed effects secondary to a long-acting formulation. Patients discharged to home require a responsible caregiver, access to follow-up care, and detailed return precautions.

Summary

Ingestions by pediatric patients are common and include a broad range of substances ranging from foreign bodies to household products to medications. While very few of these ingestions are fatal, some substances are highly toxic to a small child, even in small doses. Management of these patients in the ED may require resuscitation, decontamination, and administration of antidotes. Additional consideration is required for agents with delayed toxicity and for patients whose ingestion may be the result of abuse or suicidal intention.

Time- And Cost-Effective Strategies

- **Contact a poison control center for recommendations.** By fielding calls from the community, poison control centers save time and resources by preventing unnecessary medical evaluation when patients may be observed at home. This claim is supported by studies showing an association between decreased call rates and increased ED visits, by studies surveying people who called poison control regarding their alternative plans, and by natural experiments in which poison control center resources became unavailable.^{100,101,112} A study evaluating the differences in morbidity and mortality with their associated costs for system models with and without regional poison control centers found cost savings and improved outcomes, although their conclusion is based upon estimations by experts rather than empirical data.¹¹³
- **Limit unnecessary laboratory studies.** In otherwise healthy and asymptomatic children, blood tests such as a complete blood count or metabolic panel are likely to be normal and unlikely to change management. Extensive drug screens or levels of specific medications are rarely available promptly for clinical decision making. Laboratory investigation should be based on symptoms, known complications of the specific ingestion, and levels of drugs such as acetaminophen or salicylate that may present with nonspecific or absent symptoms, although even these studies are of low yield.^{15,16}

Case Conclusions

The toddler's glucose level returned at 35 mg/dL and you estimated the child's weight at 10 kg, so you administered 50 mL of 10% dextrose. The child's mental status immediately improved, so you continued a dextrose infusion, contacted the poison control center, and requested PICU admission for further glucose monitoring and possible octreotide therapy. Given the unknown time of ingestion and the risk of recurrent hypoglycemia with sedation, you did not administer activated charcoal.

Upon examining the previous x-ray of the 3-year-old with suspected foreign body ingestion, you noted an object 20 mm in diameter with a double rim at the level of the thoracic inlet. You consulted the surgical service while repeating the x-ray to confirm that what was likely a button battery had remained lodged in the esophagus. The child was taken immediately to the operating room for endoscopic removal.

For your patient who ingested acetaminophen with hydrocodone in a suicide attempt, you ordered laboratory testing including acetaminophen level, transaminases, coagulation studies, and a pregnancy test. When her acetaminophen level result returned at 180 mcg/mL, you contacted the poison control center and began oral N-acetylcysteine. She did not receive naloxone or activated charcoal and was admitted for a full course of N-acetylcysteine and follow-up laboratory testing prior to transfer for psychological services.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study are included in bold type following the references, where available. The most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

1. Burt AJ, Ballesteros MF, Budnitz DS. Nonfatal, unintentional medication exposures among young children – United States, 2001–2003. *MMWR Morb Mortal Wkly Rep*. 2006; 55:1-5. **(Database review)**
2. Mowry JB, Spyker DA, Cantilena LR Jr, et al. 2012 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 30th annual report. *Clin Toxicol (Phila)*. 2013;51(10):949-1229. **(Database review)**
3. Bond GR, Woodward RW, Ho M. The growing impact of pediatric pharmaceutical poisoning. *J Pediatr*. 2012;160(2):265-270. **(Database review)**
4. Bar-Oz B, Levichek Z, Koren G. Medications that can be fatal

for a toddler with one tablet or teaspoonful: a 2004 update. *Paediatr Drugs*. 2004;6(2):123-126. **(Review)**

5. Sharpe SJ, Rochette LM, Smith GA. Pediatric battery-related emergency department visits in the United States, 1990-2009. *Pediatrics*. 2012;129(6):1111-1117. **(Database review)**
6. United States Consumer Product Safety Commission. Poison prevention packaging: a guide for healthcare professionals. 2005. **(Guidelines)**
7. Rodgers GB. The safety effects of child-resistant packaging for oral prescription drugs. Two decades of experience. *JAMA*. 1996;275(21):1661-1665. **(Population study)**
8. Tuuri RE, Wright JL, He J, et al. Does prearrival communication from a poison center to an emergency department decrease time to activated charcoal for pediatric poisoning? *Pediatr Emerg Care*. 2011;27(11):1045-1051. **(Retrospective study; 351 patients)**
9. Tuuri RE, Ryan LM, He J, et al. Does emergency medical services transport for pediatric ingestion decrease time to activated charcoal? *Prehosp Emerg Care*. 2009;13(3):295-303. **(Retrospective study; 351 patients)**
10. Crockett R, Krishel SJ, Manoguerra A, et al. Prehospital use of activated charcoal: a pilot study. *J Emerg Med*. 1996;14(3):335-338. **(Retrospective study; 36 patients)**
11. Spiller HA, Rodgers GC Jr. Evaluation of administration of activated charcoal in the home. *Pediatrics*. 2001;108(6):E100. **(Prospective study; 138 patients)**
12. Doe-Simkins M, Walley AY, Epstein A, et al. Saved by the nose: bystander-administered intranasal naloxone hydrochloride for opioid overdose. *Am J Public Health*. 2009;99(5):788-791. **(Prospective study; 385 subjects)**
13. 2005 American Heart Association (AHA) guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) of pediatric and neonatal patients: pediatric advanced life support. *Pediatrics*. 2006;117(5):e1005-e1028. **(Guidelines)**
14. Cowan D, Ho B, Sykes KJ, et al. Pediatric oral burns: a ten-year review of patient characteristics, etiologies and treatment outcomes. *Int J Pediatr Otorhinolaryngol*. 2013;77(8):1325-1328. **(Retrospective study; 75 subjects)**
15. Lheureux PE, Zahir S, Gris M, et al. Bench-to-bedside review: hyperinsulinaemia/euglycaemia therapy in the management of overdose of calcium-channel blockers. *Crit Care*. 2006;10(3):212. **(Review)**
16. Wang GS, Deakyn S, Bajaj L, et al. The limited utility of screening laboratory tests and electrocardiograms in the management of unintentional asymptomatic pediatric ingestions. *J Emerg Med*. 2013;45(1):34-38. **(Retrospective chart review; 595 pediatric patients)**
17. Gandhi SG, Gilbert WM, McElvy SS, et al. Maternal and neonatal outcomes after attempted suicide. *Obstet Gynecol*. 2006;107(5):984-990. **(Retrospective study; 4,833,286 patients)**
18. Finkelstein Y, Hutson JR, Freedman SB, et al. Drug-induced seizures in children and adolescents presenting for emergency care: current and emerging trends. *Clin Toxicol (Phila)*. 2013;51(8):761-766. **(Retrospective study; 142 patients)**
19. Chabali R. Diagnostic use of anion and osmolal gaps in pediatric emergency medicine. *Pediatr Emerg Care*. 1997;13(3):204-210. **(Review and case reports, 2 patients)**
20. Sporer KA, Khayam-Bashi H. Acetaminophen and salicylate serum levels in patients with suicidal ingestion or altered mental status. *Am J Emerg Med*. 1996;14(5):443-446. **(Retrospective study; 1820 subjects)**
21. Doyon S, Klein-Schwartz W, Lee S, et al. Fatalities involving acetaminophen combination products reported to United States poison centers. *Clin Toxicol (Phila)*. 2013;51(10):941-948.

(Retrospective study; 337 patients)

22. Wu AH, McKay C, Broussard LA, et al. National Academy of Clinical Biochemistry Laboratory Medicine practice guidelines: recommendations for the use of laboratory tests to support poisoned patients who present to the emergency department. *Clin Chem*. 2003;49(3):357-379. **(Guidelines)**
23. Kolecki PF, Curry SC. Poisoning by sodium channel blocking agents. *Crit Care Clin*. 1997;13(4):829-848. **(Review)**
24. Harrigan RA, Brady WJ. ECG abnormalities in tricyclic antidepressant ingestion. *Am J Emerg Med*. 1999;17(4):387-393. **(Review)**
25. Foulke GE, Albertson TE. QRS interval in tricyclic antidepressant overdose: inaccuracy as a toxicity indicator in emergency settings. *Ann Emerg Med*. 1987;16(2):160-163. **(Retrospective study; 102 patients)**
26. Berkovitch M, Matsui D, Fogelman R, et al. Assessment of the terminal 40-millisecond QRS vector in children with a history of tricyclic antidepressant ingestion. *Pediatr Emerg Care*. 1995;11(2):75-77. **(Retrospective study; 35 patients)**
27. Saps M, Rosen JM, Ecanow J. X-ray detection of ingested non-metallic foreign bodies. *World J Clin Pediatr*. 2014;3(2):14-18. **(Observational study)**
28. Savitt DL, Hawkins HH, Roberts JR. The radiopacity of ingested medications. *Ann Emerg Med*. 1987;16(3):331-339. **(Observational study)**
29. Agbo C, Lee L, Chiang V, et al. Magnet-related injury rates in children: a single hospital experience. *J Pediatr Gastroenterol Nutr*. 2013;57(1):14-17. **(Retrospective study; 112 patients)**
30. Litovitz T, Whitaker N, Clark L, et al. Emerging battery-ingestion hazard: clinical implications. *Pediatrics*. 2010;125(6):1168-1177. **(Database review)**
31. Clifton JC 2nd, Sigg T, Burda AM, et al. Acute pediatric lead poisoning: combined whole bowel irrigation, succimer therapy, and endoscopic removal of ingested lead pellets. *Pediatr Emerg Care*. 2002;18(3):200-202. **(Case report)**
32. George S, Braithwaite RA. A preliminary evaluation of five rapid detection kits for on site drugs of abuse screening. *Addiction*. 1995;90(2):227-232. **(Observational study)**
33. Winter ML, Ellis MD, Snodgrass WR. Urine fluorescence using a Wood's lamp to detect the antifreeze additive sodium fluorescein: a qualitative adjunctive test in suspected ethylene glycol ingestions. *Ann Emerg Med*. 1990;19(6):663-667. **(Observational study; 12 samples)**
34. Wallace KL, Suchard JR, Curry SC, et al. Diagnostic use of physicians' detection of urine fluorescence in a simulated ingestion of sodium fluorescein-containing antifreeze. *Ann Emerg Med*. 2001;38(1):49-54. **(Observational study; 30 samples)**
35. Brett AS. Implications of discordance between clinical impression and toxicology analysis in drug overdose. *Arch Intern Med*. 1988;148(2):437-441. **(Retrospective study; 209 patients)**
36. Flanagan RJ, Huggett A, Saynor DA, et al. Value of toxicological investigation in the diagnosis of acute drug poisoning in children. *Lancet*. 1981;2(8248):682-685. **(Retrospective study; 287 patients)**
37. Belson MG, Simon HK. Utility of comprehensive toxicologic screens in children. *Am J Emerg Med*. 1999;17(3):221-224. **(Retrospective study; 463 subjects)**
38. Wahbeh G, Wyllie R, Kay M. Foreign body ingestion in infants and children: location, location, location. *Clin Pediatr (Phila)*. 2002;41(9):633-640. **(Review)**
39. Benson BE, Hoppu K, Troutman WG, et al. Position paper update: gastric lavage for gastrointestinal decontamination. *Clin Toxicol (Phila)*. 2013;51(3):140-146. **(Guidelines)**
40. Kornberg AE, Dolgin J. Pediatric ingestions: charcoal alone

- versus ipecac and charcoal. *Ann Emerg Med.* 1991;20(6):648-651. **(Randomized controlled trial; 70 patients)**
41. Krenzelok EP, McGuigan M, Lheur P. Position statement: ipecac syrup. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. *J Toxicol Clin Toxicol.* 1997;35(7):699-709. **(Guidelines)**
 42. Hojer J, Troutman WG, Hoppu K, et al. Position paper update: ipecac syrup for gastrointestinal decontamination. *Clin Toxicol (Phila).* 2013;51(3):134-139. **(Systematic literature review)**
 43. Olson KR. Activated charcoal for acute poisoning: one toxicologist's journey. *J Med Toxicol.* 2010;6(2):190-198. **(Review)**
 44. Chyka PA, Seger D, Krenzelok EP, et al. Position paper: Single-dose activated charcoal. *Clin Toxicol (Phila).* 2005;43(2):61-87. **(Guidelines)**
 45. Position statement and practice guidelines on the use of multi-dose activated charcoal in the treatment of acute poisoning. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. *J Toxicol Clin Toxicol.* 1999;37(6):731-751. **(Guidelines)**
 46. Sato RL, Wong JJ, Sumida SM, et al. Efficacy of superactivated charcoal administered late (3 hours) after acetaminophen overdose. *Am J Emerg Med.* 2003;21(3):189-191. **(Randomized controlled trial; 46 patients)**
 47. Thanacoody R, Caravati EM, Troutman B, et al. Position paper update: Whole bowel irrigation for gastrointestinal decontamination of overdose patients. *Clin Toxicol (Phila).* 2015;53(1):5-12. **(Guidelines)**
 48. Lo JC, Ubaldo C, Cantrell FL. A retrospective review of whole bowel irrigation in pediatric patients. *Clin Toxicol.* 2012;50(5):414-417. **(Retrospective study; 176 patients)**
 49. Fertel BS, Nelson LS, Goldfarb DS. Extracorporeal removal techniques for the poisoned patient: a review for the intensivist. *J Intensive Care Med.* 2010;25(3):139-148. **(Review)**
 50. Holubek WJ, Hoffman RS, Goldfarb DS, et al. Use of hemodialysis and hemoperfusion in poisoned patients. *Kidney Int.* 2008;74(10):1327-1334. **(Retrospective study; 19,351 patients)**
 51. Prescott LF. Hepatotoxicity of mild analgesics. *Br J Clin Pharmacol.* 1980;10 Suppl 2:373S-379S. **(Review)**
 52. Rivera-Penera T, Gugig R, Davis J, et al. Outcome of acetaminophen overdose in pediatric patients and factors contributing to hepatotoxicity. *J Pediatr.* 1997;130(2):300-304. **(Retrospective study; 73 patients)**
 53. Burillo-Putze G, Mintegui S, Munne P. Changes in pediatric toxic dose of acetaminophen. *Am J Emerg Med.* 2004;22(4):323. **(Editorial)**
 54. Rumack BH, Matthew H. Acetaminophen poisoning and toxicity. *Pediatrics.* 1975;55(6):871-876. **(Review)**
 55. Froberg BA, King KJ, Kurera TD, et al. Negative predictive value of acetaminophen concentrations within four hours of ingestion. *Acad Emerg Med.* 2013;20(10):1072-1075. **(Prospective study; 83 patients)**
 56. Harrison PM, Wendon JA, Gimson AE, et al. Improvement by acetylcysteine of hemodynamics and oxygen transport in fulminant hepatic failure. *N Engl J Med.* 1991;324(26):1852-1857. **(Case series; 12 patients)**
 57. Keays R, Harrison PM, Wendon JA, et al. Intravenous acetylcysteine in paracetamol induced fulminant hepatic failure: a prospective controlled trial. *BMJ.* 1991;303(6809):1026-1029. **(Prospective randomized controlled trial; 50 patients)**
 58. Smilkstein MJ, Knapp GL, Kulig KW, et al. Efficacy of oral N-acetylcysteine in the treatment of acetaminophen overdose. Analysis of the national multicenter study (1976 to 1985). *N Engl J Med.* 1988;319(24):1557-1562. **(Retrospective study; 2540 subjects)**
 59. Prescott LF, Illingworth RN, Critchley JA, et al. Intravenous N-acetylcysteine: the treatment of choice for paracetamol poisoning. *Br Med J.* 1979;2(6198):1097-1100. **(Retrospective study; 100 patients)**
 60. Pizon AF, Jang DH, Wang HE. The in vitro effect of N-acetylcysteine on prothrombin time in plasma samples from healthy subjects. *Acad Emerg Med.* 2011;18(4):351-354. **(In vitro study)**
 61. Wang GS, Monte A, Bagdure D, et al. Hepatic failure despite early acetylcysteine following large acetaminophen-diphenhydramine overdose. *Pediatrics.* 2011;127(4):e1077-e1080. **(Case report)**
 62. Slaughter RJ, Mason RW, Beasley DM, et al. Isopropanol poisoning. *Clin Toxicol (Phila).* 2014;52(5):470-478. **(Review)**
 63. White ML, Liebelt EL. Update on antidotes for pediatric poisoning. *Pediatr Emerg Care.* 2006;22(11):740-746. **(Review)**
 64. Baud FJ, Galliot M, Astier A, et al. Treatment of ethylene glycol poisoning with intravenous 4-methylpyrazole. *N Engl J Med.* 1988;319(2):97-100. **(Case report)**
 65. Darracq MA, Cantrell FL. Hemodialysis and extracorporeal removal after pediatric and adolescent poisoning reported to a state poison center. *J Emerg Med.* 2013;44(6):1101-1107. **(Database study; 90 patients)**
 66. Frascogna N. Physostigmine: is there a role for this antidote in pediatric poisonings? *Curr Opin Pediatr.* 2007;19(2):201-205. **(Review)**
 67. Shannon M. Toxicology reviews: physostigmine. *Pediatr Emerg Care.* 1998;14(3):224-226. **(Review)**
 68. Lifshitz M, Zucker N, Zalzstein E. Acute dilated cardiomyopathy and central nervous system toxicity following propranolol intoxication. *Pediatr Emerg Care.* 1999;15(4):262-263. **(Case report)**
 69. Shepherd G. Treatment of poisoning caused by beta-adrenergic and calcium-channel blockers. *Am J Health Syst Pharm.* 2006;63(19):1828-1835. **(Review)**
 70. Love JN, Howell JM, Klein-Schwartz W, et al. Lack of toxicity from pediatric beta-blocker exposures. *Hum Exp Toxicol.* 2006;25(6):341-346. **(Prospective cohort study; 208 patients)**
 71. Smith RC, Wilkinson J, Hull RL. Glucagon for propranolol overdose. *JAMA.* 1985;254(17):2412. **(Case report)**
 72. Bindon MJ, Barlotta K. Glucagon treatment for bradycardia and a junctional rhythm caused by excessive beta-blockade. *Resuscitation.* 2009;80(11):1327. **(Case report)**
 73. Love JN, Howell JM. Glucagon therapy in the treatment of symptomatic bradycardia. *Ann Emerg Med.* 1997;29(1):181-183. **(Case series)**
 74. Engebretsen KM, Kaczmarek KM, Morgan J, et al. High-dose insulin therapy in beta-blocker and calcium channel-blocker poisoning. *Clin Toxicol (Phila).* 2011;49(4):277-283. **(Review)**
 75. Ramoska EA, Spiller HA, Myers A. Calcium channel blocker toxicity. *Ann Emerg Med.* 1990;19(6):649-653. **(Retrospective, study; 91 patients)**
 76. Truitt CA, Brooks DE, Dommer P, et al. Outcomes of unintentional beta-blocker or calcium channel blocker overdoses: a retrospective review of poison center data. *J Med Toxicol.* 2012;8(2):135-139. **(Database study)**
 77. Belson MG, Gorman SE, Sullivan K, et al. Calcium channel blocker ingestions in children. *Am J Emerg Med.* 2000;18(5):581-586. **(Retrospective review)**
 78. Levine M, Boyer EW, Pozner CN, et al. Assessment of hyperglycemia after calcium channel blocker overdoses involving diltiazem or verapamil. *Crit Care Med.* 2007;35(9):2071-2075. **(Retrospective study, 40 patients)**
 79. Jokanovic M. Medical treatment of acute poisoning with organophosphorus and carbamate pesticides. *Toxicol Lett.* 2009;190(2):107-115. **(Review)**
 80. Saadeh AM, Farsakh NA, al-Ali MK. Cardiac manifestations

- of acute carbamate and organophosphate poisoning. *Heart*. 1997;77(5):461-464. **(Retrospective study; 46 patients)**
81. Thacker D, Sharma J. Digoxin toxicity. *Clin Pediatr (Phila)*. 2007;46(3):276-279. **(Review)**
 82. Woolf AD, Wenger TL, Smith TW, et al. Results of multi-center studies of digoxin-specific antibody fragments in managing digitalis intoxication in the pediatric population. *Am J Emerg Med*. 1991;9(2 Suppl 1):16-20. **(Retrospective study; 57 patients)**
 83. Fazio A. Fab fragments in the treatment of digoxin overdose: pediatric considerations. *South Med J*. 1987;80(12):1553-1556. **(Review)**
 84. Jang DH, Spyres MB, Fox L, et al. Toxin-induced cardiovascular failure. *Emerg Med Clin N Am*. 2014;32(1):79-102. **(Review)**
 85. Woolf AD, Wenger T, Smith TW, et al. The use of digoxin-specific Fab fragments for severe digitalis intoxication in children. *N Engl J Med*. 1992;326(26):1739-1744. **(Retrospective study; 29 patients)**
 86. Kaufman J, Leikin J, Kendzierski D, et al. Use of digoxin Fab immune fragments in a seven-day-old infant. *Pediatr Emerg Care*. 1990;6(2):118-121. **(Case report)**
 87. Reynolds LG. Diagnosis and management of acute iron poisoning. *Baillieres Clin Haematol*. 1989;2(2):423-433. **(Review)**
 88. Sankar J, Shukla A, Khurana R, et al. Near fatal iron intoxication managed conservatively. *BMJ Case Rep*. 2013;2013. **(Case report)**
 89. Chang TP, Rangan C. Iron poisoning: a literature-based review of epidemiology, diagnosis, and management. *Pediatr Emerg Care*. 2011;27(10):978-985. **(Review)**
 90. Madiwale T, Liebelt E. Iron: not a benign therapeutic drug. *Curr Opin Pediatr*. 2006;18(2):174-179. **(Review)**
 91. Geib AJ, Babu K, Ewald MB, et al. Adverse effects in children after unintentional buprenorphine exposure. *Pediatrics*. 2006;118(4):1746-1751. **(Case series; 5 patients)**
 92. Goldfrank L, Weisman RS, Errick JK, et al. A dosing nomogram for continuous infusion intravenous naloxone. *Ann Emerg Med*. 1986;15(5):566-570. **(Prospective study; 11 patients)**
 93. O'Malley GF. Emergency department management of the salicylate-poisoned patient. *Emerg Med Clin North Am*. 2007;25(2):333-346. **(Review)**
 94. Bogacz K, Caldron P. Enteric-coated aspirin bezoar: elevation of serum salicylate level by barium study. Case report and review of medical management. *Am J Med*. 1987;83(4):783-786. **(Case report and in vitro study)**
 95. Proudfoot AT, Krenzelok EP, Vale JA. Position paper on urine alkalization. *J Toxicol Clin Toxicol*. 2004;42(1):1-26. **(Guidelines)**
 96. Stolbach AI, Hoffman RS, Nelson LS. Mechanical ventilation was associated with acidemia in a case series of salicylate-poisoned patients. *Acad Emerg Med*. 2008;15(9):866-869. **(Retrospective study; 7 patients)**
 97. Wiley CC, Wiley JF 2nd. Pediatric benzodiazepine ingestion resulting in hospitalization. *J Toxicol Clin Toxicol*. 1998;36(3):227-231. **(Retrospective study; 46 patients)**
 98. Kreshak AA, Tomaszewski CA, Clark RF, et al. Flumazenil administration in poisoned pediatric patients. *Pediatr Emerg Care*. 2012;28(5):448-450. **(Retrospective study; 83 patients)**
 99. Harrigan RA, Nathan MS, Beattie P. Oral agents for the treatment of type 2 diabetes mellitus: pharmacology, toxicity, and treatment. *Ann Emerg Med*. 2001;38(1):68-78. **(Review)**
 100. Calello DP, Kelly A, Osterhoudt KC. Case files of the Medical Toxicology Fellowship Training Program at the Children's Hospital of Philadelphia: a pediatric exploratory sulfonylurea ingestion. *J Med Toxicol*. 2006;2(1):19-24. **(Case report)**
 101. Glatstein M, Garcia-Bournissen F, Scolnik D, et al. Sulfonylurea intoxication at a tertiary care paediatric hospital. *Can J Clin Pharmacol*. 2010;17(1):e51-56. **(Case series; 10 patients)**
 102. Pollack CV Jr. Utility of glucagon in the emergency department. *J Emerg Med*. 1993;11(2):195-205. **(Review)**
 103. Levine M, Ruha AM, Lovecchio F, et al. Hypoglycemia after accidental pediatric sulfonylurea ingestions. *Pediatr Emerg Care*. 2011;27(9):846-849. **(Retrospective case series; 93 patients)**
 104. Glatstein M, Scolnik D, Bentur Y. Octreotide for the treatment of sulfonylurea poisoning. *Clin Toxicol (Phila)*. 2012;50(9):795-804. **(Meta-analysis)**
 105. Hickson GB, Altemeier WA, Martin ED, et al. Parental administration of chemical agents: a cause of apparent life-threatening events. *Pediatrics*. 1989;83(5):772-776. **(Case series; 9 patients)**
 106. Pitetti RD, Whitman E, Zaylor A. Accidental and nonaccidental poisonings as a cause of apparent life-threatening events in infants. *Pediatrics*. 2008;122(2):e359-e362. **(Prospective study; 596 patients)**
 107. Macnab A, Anderson E, Susak L. Ingestion of cannabis: a cause of coma in children. *Pediatr Emerg Care*. 1989;5(4):238-239. **(Case series; 6 patients)**
 108. Wang GS, Roosevelt G, Le Lait MC, et al. Association of unintentional pediatric exposures with decriminalization of marijuana in the United States. *Ann Emerg Med*. 2014;63(6):684-689. **(Retrospective study; 985 patients)**
 109. Lokajova J, Holopainen JM, Wiedmer SK. Comparison of lipid sinks in sequestering common intoxicating drugs. *J Sep Sci*. 2012;35(22):3106-3112. **(In vitro study)**
 110. French D, Armenian P, Ruan W, et al. Serum verapamil concentrations before and after Intralipid(R) therapy during treatment of an overdose. *Clin Toxicol (Phila)*. 2011;49(4):340-344. **(Case report)**
 111. Rodriguez B, Wilhelm A, Kokko KE. Lipid emulsion use precluding renal replacement therapy. *J Emerg Med*. 2014;47(6):635-637. **(Case report)**
 112. Kearney TE, Olson KR, Bero LA, et al. Health care cost effects of public use of a regional poison control center. *West J Med*. 1995;162(6):499-504. **(Cost-effectiveness analysis)**
 113. Harrison DL, Draugalis JR, Slack MK, et al. Cost-effectiveness of regional poison control centers. *Arch Intern Med*. 1996;156(22):2601-2608. **(Cost-effectiveness analysis)**

CME Questions



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1. Ingestion of which of the following agents may cause hyperglycemia in addition to bradycardia with hypotension?
 - a. Digoxin
 - b. Propranolol
 - c. Verapamil
 - d. Clonidine
2. A 3-year-old presents to the ED after spending the day at her grandparents' home, where butyropion, glyburide, and isoniazid were found on the coffee table. Which symptom could be caused by any of these medications?
 - a. Dysrhythmia
 - b. Hypoglycemia
 - c. Increased drooling
 - d. Seizure
3. Which ECG abnormality may be noted in tricyclic antidepressant ingestion?
 - a. Bradycardia
 - b. Tachycardia
 - c. Peaked T wave
 - d. First-degree heart block
4. Which of the following is the recommended management of a button battery noted on x-ray to be in the stomach of an asymptomatic 5-year-old after ingestion?
 - a. Immediate endoscopic removal
 - b. Removal within 2 hours
 - c. Computed tomography scan to definitively locate the battery
 - d. Repeat x-ray in 4 days
5. A 3-year-old was found playing with a desk toy of small magnets, and 2 objects are noted in the stomach on x-ray. What is the recommended management?
 - a. Endoscopic or surgical removal
 - b. Admit for overnight observation
 - c. Repeat x-ray in 4 days
 - d. Monitor stool for passage at home
6. Which of the following cases is most suitable for oral administration of activated charcoal?
 - a. Antifreeze ingestion 30 minutes previously
 - b. Amlodipine ingestion 30 minutes previously
 - c. Glipizide ingestion 2 hours previously
 - d. Methadone ingestion 2 hours previously
7. Which toxin is least likely to be eliminated by hemodialysis?
 - a. Ethylene glycol
 - b. Digoxin
 - c. Aspirin
 - d. Lithium
8. A family arrives to the ED 2 hours after realizing that they have inadvertently given their toddler an overdose of acetaminophen. The child is otherwise healthy and had not received any other acetaminophen recently. Given the amount remaining in the newly opened bottle, you calculate that the child has received approximately 130 mg/kg. What is the best next step?
 - a. Obtain an acetaminophen level 4 hours following the ingestion.
 - b. Begin oral N-acetylcysteine immediately.
 - c. Begin intravenous N-acetylcysteine immediately.
 - d. Discharge the child to home if the acetaminophen level, aspartate aminotransferase, alanine transaminase, and INR are normal now.
9. A hallucinating teenager is brought in by EMS, along with a sample of the leaves found at the scene, which you identify as Jimson weed. What are the expected signs of this toxidrome?
 - a. Flushed, warm skin, tachycardia, decreased bowel sounds
 - b. Cool, clammy skin; shallow respirations; miosis
 - c. Profuse sweating, respiratory distress, diarrhea
 - d. Diaphoretic, warm skin, hypertension, mydriasis
10. For the patient in question 9, which of the following is the best first step in management?
 - a. Place a nasogastric tube and administer activated charcoal.
 - b. Obtain a urine drug screen.
 - c. Obtain an ECG.
 - d. Give physostigmine 0.02 mg/kg intravenously.

Coming Soon In *Pediatric Emergency Medicine Practice*

Pediatric Congenital Heart Disease: Recognizing And Managing The Undiagnosed

Congenital heart disease (CHD) is the most common form of all congenital malformations of the heart, and, despite advances in prenatal and newborn screening, it may present undiagnosed to the emergency department (ED). Signs and symptoms of CHD are variable and often nonspecific, making treatment challenging. Patient presentations can range from life-threatening shock or cyanosis in a neonate to respiratory distress or failure to thrive in infants. Advances in surgical techniques have improved short- and long-term survival of infants and children with CHD, but these children are at risk for a variety of complications related to their underlying or surgical anatomy and physiology. This review focuses on the recognition and initial management of undiagnosed CHD presenting to the ED and touches on considerations for postoperative infants and children with complex CHD.

Risk Management Pitfalls In Congenital Heart Disease

1. **"This neonate had normal prenatal care, including a prenatal ultrasound, so CHD has been ruled out. There must be another cause for his shock."**

While prenatal ultrasound has advanced significantly over recent decades, only about one-third of all CHD and 57% to 85% of critical CHD is detected before birth. Normal prenatal care and screening ultrasound do not exclude the possibility of significant CHD.

2. **"I don't hear a murmur or a gallop, so this isn't CHD."**

The absence of abnormal heart sounds does not preclude underlying structural disease. A murmur requires turbulent blood flow across a defect, usually from a significant pressure gradient. In the first days of life, high pulmonary vascular resistance can minimize left-to-right shunting across a large atrial or ventricular septal defect, and a murmur may not be detected prior to discharge from the nursery.

3. **"This is the fourth bad case of bronchiolitis I've had this shift! She's getting worse despite IV fluids, so I'll just admit her and try nebulized epinephrine."**

CHD presenting with congestive heart failure can mimic common viral illness such as bronchiolitis, and, during epidemics, it is easy to overlook heart disease as a cause of respiratory distress in an infant. Worsening of clinical condition with usual treatment (such as intravenous fluids for presumed dehydration) in bronchiolitis should alert you to the possibility of CHF, for which diuretics are first-line therapy. A brain natriuretic peptide and chest x-ray may help in these circumstances.

4. **"Although this 1-week-old infant is in shock, we can't get an echocardiogram and I'm not sure what is going on, so I don't want to start prostaglandin E1 until we have more information. I'll just fluid resuscitate...."**

In the critically ill neonate presenting with shock, prostaglandin E1 can be life-saving and should be empirically initiated if there is no response to an initial 10 mL/kg bolus of intravenous fluid. Careful monitoring of clinical response is all that is needed and the infusion can be stopped if the clinical condition worsens.

Carbon Monoxide Poisoning In Children: Early Intervention In The Emergency Department

Carbon monoxide (CO) poisoning is the leading cause of poisoning from toxic gas in the United States. Approximately 5000 children present to the emergency department (ED) annually with unintentional CO poisoning. CO is tasteless, odorless, and colorless. It binds hemoglobin with strong affinity and decreases the oxygen-carrying capacity of the blood. CO also binds other iron moieties, activating pathways that lead to cell death. Children may be more vulnerable to CO poisoning because of their increased metabolic demand and inability to vocalize symptoms or recognize a dangerous exposure. Mild CO poisoning may present as viral symptoms with the absence of fever. In children, headache, nausea, and vomiting are the most common presenting symptoms. The most common symptom in infants with severe CO poisoning is consciousness disturbance. The clinician must remain alert for comorbidities or intoxicants. The mainstay of treatment for CO poisoning is 100% oxygen. There are no randomized controlled trials of hyperbaric oxygen therapy in pediatric patients, and its use remains controversial; however, it may have benefit in preventing delayed neurologic sequelae. This review addresses the initial diagnosis and management of CO poisoning in children.

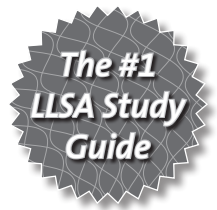
Time- And Cost-Effective Strategies

- Noninvasive CO detection with pulse CO-oximetry may decrease the time to diagnosis of CO poisoning and referral to hyperbaric oxygen therapy, though its cost-effectiveness has yet to be studied. In 1 study, patients screened with pulse CO-oximetry were referred to a hyperbaric oxygen therapy facility 1 hour sooner than those not screened with pulse CO-oximetry, which may save personnel time and resources in the referring ED.

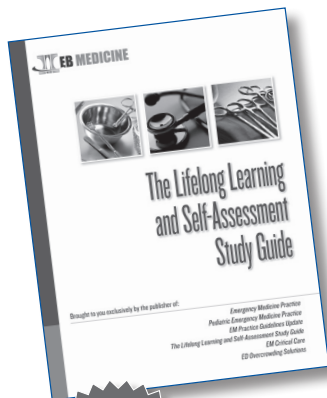
Risk management caveat: All CO blood levels obtained from noninvasive pulse CO-oximeters must be confirmed with a laboratory blood sample, as the precision of these devices is 3% at 1 standard deviation, varies in patient populations, and is not validated in children.

- Oxygen should be applied immediately in all cases of suspected CO poisoning even before having a definitive CO blood level. Since oxygen is the mainstay of treatment and is cheap and generally harmless, providing oxygen early will decrease a patient's length of stay, improve symptoms sooner, and possibly defer an inpatient admission for continued therapy.

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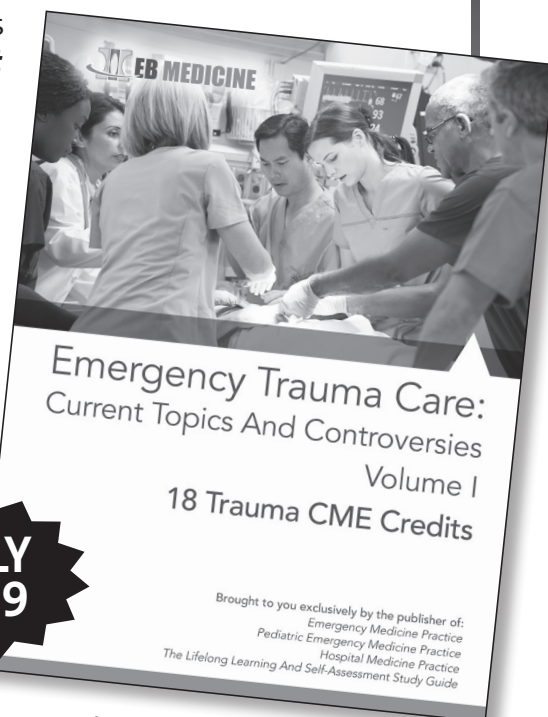
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Goals: Upon completion of this activity, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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