# Chapter 176: General Management of Poisoned Patients

Shaun Greene

## INTRODUCTION

Poisoning is a worldwide problem that consumes substantial health care resources and causes many premature deaths. The burden of serious poisoning is carried by the developing world[1,2]; however, poisoning-related morbidity and mortality is also a significant public health concern in the developed world.[3,4,5,6,7,8,9]

Unintentional poisoning deaths in the United States are increasing, especially as a result of prescription analgesics. This increase has been ascribed to increasing prescription rates and aging of the baby-boom population.[10,11,12] U.S. poison control centers documented 2.38 million human exposures in 2010, with 1146 associated deaths.[13] Prevention is the key to reducing unintentional poisoning deaths. Pharmacists can that ensure medications are labeled correctly, anticipate potential drug interactions, and educate patients to use medications safely. To prevent pediatric deaths from poisoning, parents have the responsibility to ensure that poisons are placed in childproof, labeled containers stored in adult-only accessible nonfood storage areas. Teachers and healthcare providers can provide age-appropriate education to children about the dangers of poisons. After an exposure, poison control centers staffed by highly trained individuals can provide customized advice to healthcare providers and the public. Poison control centers also participate in prevention, education, and toxico-surveillance activities.

Exposures occur most commonly by ingestion; other routes include inhalation, insufflation, cutaneous and mucous membrane exposure, and injection. Some exposures have minimal risk. The criteria used to determine whether the exposure is nontoxic are: (1) an unintentional exposure to a clearly identified single substance, (2) where an estimate of dose is known, and (3) a recognized information source (e.g., a poison control center) confirms the substance as nontoxic in the reported dose. Asymptomatic patients with nontoxic exposures may be discharged after a short period of observation, providing they have access to further consultation and a safe discharge destination.

Serious clinical effects occur in <5% of acutely poisoned patients presenting to developed-world hospitals, and in-hospital mortality rates are <1%.[9,13]

# RESUSCITATION

Resuscitation is the first priority in any poisoned patient. After resuscitation, a structured risk assessment is used to identify patients who may benefit from an antidote, decontamination, or enhanced elimination techniques. Most patients only require provision of good supportive care during a period of observation in an appropriate environment.

Treatment of cardiac arrest in poisoned patients follows Advanced Cardiac Life Support guidelines with the addition of interventions potentially beneficial in toxin-induced cardiac arrest (**Table 176-1**).[14] Prolonged resuscitation is generally indicated, as patients are often young with minimal preexisting organ dysfunction. Utilization of extracorporeal cardiac and respiratory assist devices until organ toxicity resolves may be life-saving.

TABLE 176-1

**Potential Interventions in Toxin-Induced Cardiac Arrest**[15]

| **Toxin or Toxin/Drug Class** | **Intervention** |
|---|---|
| Toxins with a specific antidote (examples)<br>Digoxin<br>Organophosphates<br>Envenomation | Antidote<br>Digoxin Fab<br>Atropine<br>Antivenom |
| Sodium channel blocker or wide-complex tachycardia | Sodium bicarbonate |
| Calcium channel blocker or β-blocker | High-dose insulin |
| Local anesthetic agents<br>Lipophilic cardiotoxins | IV lipid emulsion |
| **Other Therapies to Consider** | |
| Cardiac pacing<br>Intra-aortic balloon pump<br>Extracorporeal membrane oxygenation | |

Stabilization of airway, breathing, and circulation represents initial priorities. Compromised airway patency or reduced respiratory drive may lead to inadequate ventilation; provision of a mechanical airway and assisted ventilation is vital in these circumstances. IV crystalloid bolus (10 to 20 mL/kg) is first-line treatment

of hypotension. Since most patients without toxin-induced fluid loss are generally not fluid depleted, avoid administration of excess fluid. Persisting hypotension despite an adequate volume infusion may respond to a specific antidote. Otherwise, cautious administration of an inotropic agent is indicated. Inotrope choice is guided by knowledge of the toxin's toxicodynamic properties and assessment of circulatory status (e.g., cardiac pump failure versus vasodilatory shock).

## ANTIDOTES

Stabilization of airway, breathing, and circulation allows further assessment of blood glucose concentration, temperature, and conscious state. Although the proper use of antidotes (**Table 176-2**) is important, only a few are indicated before cardiopulmonary stabilization (e.g., naloxone for opiate toxicity, cyanide antidotes for cyanide toxicity, and atropine for organophosphate poisoning).

TABLE 176-2

**Common Antidotes Used in Resuscitation of the Acutely Poisoned Patient**

| Antidote | Pediatric Dose | Adult Dose | Indication |
|---|---|---|---|
| **Calcium chloride 10%**<br>27.2 milligrams/mL elemental Ca | 0.2–0.25 mL/kg IV | 10 mL IV | Calcium channel antagonists |
| **Calcium gluconate 10%**<br>9 milligrams/mL elemental Ca | 0.6–0.8 mL/kg IV | 10–30 mL IV | Hypermagnesemia<br>Hypocalcemia |
| **Cyanide antidote kit**<br>Amyl nitrite | Not typically used | 1 ampule $O_2$ chamber of ventilation bag 30 s on/30 s off | Cyanide<br>Hydrogen sulfide (use only sodium nitrite) |
| Sodium nitrite (3% solution) | 0.33 mL/kg IV | 10 mL IV | Cyanide |
| Sodium thiosulfate (25% solution) | 1.65 mL/kg IV | 50 mL IV | Cyanide |
| **Dextrose (glucose)** | 0.5 gram/kg IV | 1 gram/kg IV | Insulin<br>Oral hypoglycemics |
| **Digoxin Fab**<br>Acute toxicity | 1–2 vials IV | 5–10 vials | Digoxin and other cardioactive steroids |
| **Flumazenil** | 0.01 milligram/kg IV | 0.2 milligram IV | Benzodiazepines |

| Antidote | Pediatric Dose | Adult Dose | Indication |
|---|---|---|---|
| **Glucagon** | 50–150 micrograms/kg IV | 3–10 milligrams IV | Calcium channel blockers<br>β-Blockers |
| **Hydroxocobalamin** | 70 milligrams/kg IV (maximum 5 grams). Can be repeated up to 3 times. Administer with sodium thiosulfate. | | Cyanide<br>Nitroprusside |
| **IV lipid emulsion 20%** | 1.5 mL/kg IV bolus over 1 min (may be repeated two times at 5-min intervals), followed by 0.25 mL/kg per minute | 100-mL IV bolus over 1 min, followed by 400 mL IV over 20 min | Local anesthetic toxicity<br>Rescue therapy for lipophilic cardiotoxins |
| **Methylene blue** | 1–2 milligrams/kg IV<br>Neonates: 0.3–1.0 milligram/kg IV | 1–2 milligrams/kg IV | Oxidizing toxins (e.g., nitrites, benzocaine, sulfonamides) |
| **Naloxone** | As much as required<br>Start: 0.01 milligram IV | As much as required<br>Start: 0.1–0.4 milligram IV | Opioids<br>Clonidine |
| **Pyridoxine** | Gram for gram if amount isoniazid ingested is known | Isoniazid<br>*Gyromitra esculenta*<br>Hydrazine | |
| | 70 milligrams/kg IV (maximum 5 grams) | | 5 grams IV |
| **Sodium bicarbonate** | 1–2 mEq/kg IV bolus followed by 2 mEq/kg per h IV infusion | | Sodium channel blockers<br>Urinary alkalinization |
| **Thiamine** | 5–10 milligrams IV | 100 milligrams IV | Wernicke's syndrome<br>Wet beriberi |

## HYPOGLYCEMIA

Treat hypoglycemia with IV dextrose (glucose). Patients at risk of Wernicke's encephalopathy also require thiamine, but do not require that it be administered before the dextrose.[15] Altered mental status when hypoglycemia cannot be excluded is an indication for IV dextrose. Supplemental oxygen, thiamine, glucose, and naloxone are often administered empirically as a cocktail in cases of altered mental status. Although relatively safe and affordable in the developed world, this approach may not be cost-effective in developing countries. The decision to administer an antidote should be made after a rapid collateral history is obtained and targeted examination completed. Altered mental status not responding to an antidote or not consistent with exposure history requires further investigation. Metabolic, infective, and surgical (e.g., intracranial injury) causes of altered mental status should be considered.

### CARDIAC ARRHYTHMIAS

In general, antiarrhythmic drugs are not first-line treatment for toxin-induced arrhythmias, as most antiarrhythmic drugs have proarrhythmic and negative inotropic properties. Most toxin-induced arrhythmias respond to correction of hypoxia, metabolic/acid–base abnormalities, and administration of an antidote (e.g., digoxin Fab). Sodium bicarbonate is administered for sodium-channel blocker toxicity with cardiovascular complications, such as wide QRS complex tachyarrhythmias. Ventricular tachyarrhythmias may respond to overdrive pacing.

### SEIZURES

Drug-induced seizures are treated with titrated doses of IV benzodiazepines, with the exception that isoniazid-induced seizures require pyridoxine. Metabolic disorders, such as hypoglycemia and hyponatremia, can also produce seizures and should be rapidly excluded. Barbiturates are second-line agents for benzodiazepine-resistant seizures (once isoniazid-induced seizures are excluded). **There is no role for phenytoin in the treatment of toxin-induced seizures; it has neither theoretical nor proven efficacy, and may worsen toxicity.**[16]

### AGITATION

Agitation is treated with titrated doses of benzodiazepines. Large doses may be required and are appropriate in monitored settings where advanced airway interventions are available if required. Although antipsychotic agents are often used as second-line agents for toxin-induced agitation, they have theoretical disadvantages, including anticholinergic and extrapyramidal effects.[17] Droperidol has been associated (rarely) with QT interval prolongation and cardiac arrhythmias.

### HYPERTHERMIA AND HYPOTHERMIA

Patients with core temperatures of >39°C (>102.2°F) require aggressive active cooling measures to prevent complications such as rhabdomyolysis, organ failure, and disseminated intravascular coagulation. Sedation, neuromuscular paralysis, and intubation are required if active measures are ineffective. Several toxidromes

associated with hyperthermia are treated with specific pharmaceutical agents: sympathomimetic (benzodiazepines), serotonin (cyproheptadine[18]), and neuromuscular malignant syndrome (bromocriptine[19]).

Drug-induced coma with subsequent immobility and environmental exposure or inherent drug toxicity (opioids, phenothiazines, ethanol) may produce hypothermia. A core temperature <32°C (<90°F) is an indication for active rewarming.

### NALOXONE

Naloxone is a nontoxic, diagnostic, and therapeutic antidote. It is a competitive opioid antagonist administered IV, IM, or intranasally[20] to reverse opioid-induced deleterious hypoventilation. Naloxone can be used as a diagnostic agent when history and/or examination findings (respiratory rate of <12 breaths/min is a predictor of response to naloxone) suggest possible opioid exposure. Naloxone is titrated to clinical effect using bolus doses, typically 0.1 to 0.4 milligrams. Large initial bolus doses may precipitate vomiting and aspiration, acute opioid withdrawal, or an uncooperative, agitated patient. Miosis is an unreliable indicator of naloxone's adequate clinical effect, as some opioids do not affect pupil size. Doses are titrated to achieve desirable ventilation and conscious state (adequate respiratory rate, normal arterial oxygen saturations on room air, and verbal or motor response to voice). Although naloxone may reverse the effects of opioids for 20 to 60 minutes, the effect of many opioids will outlast this time frame with possible return of respiratory depression. Patients should be observed for 2 to 3 hours after administration of IV naloxone.

### INTRAVENOUS LIPID EMULSION

Animal studies demonstrate the potential for IV lipid emulsion to act as an antidote for lipophilic toxins. Provision of an intravascular "lipid sink" is postulated as the predominant mechanism, as sequestration of lipophilic toxins prevents target receptor interaction. Human case reports indicate that IV lipid emulsion may provide benefit in cases of potentially life-threatening toxicity from a local anesthetic agent, haloperidol, tricyclic antidepressant, lipophilic β-blocker, or calcium channel blocker.[21] **Currently, IV lipid emulsion can be considered in life-threatening cardiotoxicity caused by lipophilic cardiotoxins that is resistant to conventional therapies.**

## RISK ASSESSMENT

Following initial resuscitation and stabilization, a risk assessment is performed to predict course of clinical toxicity, interventions required, and patient disposition. Risk assessment is formulated using history, examination, and ancillary test results. Acute poisoning is a dynamic process; therefore, risk assessment may change with time and requires ongoing review.

### HISTORY

Patients may not provide a clear history due to psychiatric illness, clinical effects of exposure, and fear of arrest or repercussions from family or friends. Information including identity of substances, doses, and route of exposure is crucial in formulating a risk assessment. Obtain collateral information from family, friends, previous medical records, and usual healthcare provider. Prehospital emergency services can provide information regarding empty medication containers or the scene environment (smells, particular materials or substances present). If possible, obtain knowledge of hobbies, occupation, presence of a suicide note, and recent changes in patient behavior.

## EXAMINATION

A systematic physical examination can yield important clues to the nature and potential severity of an exposure (**Table 176-3**). Examine the skin folds, body cavities if appropriate, and clothing for retained tablets or substances.

TABLE 176-3

**Examination of the Poisoned Patient**

| Organ System | Examination | Example of Finding (Possible Significance) |
|---|---|---|
| General appearance | General demeanor and dress<br>Signs of injury<br>Odors<br>Mental state<br>Nutritional state<br>Temperature | Unkempt (psychiatric illness)<br>Scalp hematoma (intracranial injury)<br>Malnourished (IV drug use, HIV infection)<br>Smell of bitter almonds (cyanide toxicity) |
| Central nervous | Conscious state<br>Pupil size and reactivity<br>Eye movements<br>Cerebellar function/gait | Miosis (opioids, organophosphates, phenothiazines, clonidine intoxication)<br>Nystagmus/ataxia (anticonvulsant and ethanol toxicity) |
| Cardiovascular | Heart rate/blood pressure<br>Cardiac auscultation | Murmur (endocarditis/IV drug abuse) |
| Respiratory | Oxygen saturation<br>Respiratory rate<br>Chest auscultation | Fever/crepitations/hypoxia (aspiration pneumonia)<br>Bronchorrhea/crepitations/hypoxia (organophosphate toxicity) |
| Gastrointestinal | Oropharynx<br>Abdomen<br>Bladder | Urinary retention (anticholinergic toxicity)<br>Oral cavity burns (corrosive ingestion)<br>Hypersalivation (cholinergic toxidrome) |
| Peripheral nervous | Reflexes<br>Tone<br>Fasciculations<br>Tremor<br>Clonus | Tremor/fasciculations (lithium toxicity)<br>"Lead pipe" rigidity (neuromuscular malignant syndrome)<br>Clonus/hyperreflexia (serotonin toxicity) |

| Organ System | Examination | Example of Finding (Possible Significance) |
|---|---|---|
| Dermal/peripheral | Bruising<br>Cyanosis<br>Flushing<br>Dry/moist skin<br>Injection sites<br>Bullae | Bruising (coagulopathy, trauma, coma)<br>Flushing /warm, dry skin (anticholinergic toxicity)<br>Warm, moist skin (sympathomimetic toxicity)<br>Bullae (prolonged coma, barbiturates) |

## TOXIDROMES

Substances belonging to a particular pharmaceutical/chemical class often produce a cluster of symptoms and signs, or "toxidrome" (Table 176-4), enabling the identification of potential toxins when a clear history is unavailable.

TABLE 176-4

**Common Toxidromes**

| Toxidrome | Examples of Agents | Examination Findings (most common in bold) |
|---|---|---|
| Anticholinergic | Atropine, *Datura* spp., antihistamines, antipsychotics | **Altered mental status, mydriasis, dry flushed skin, urinary retention, decreased bowel sounds, hyperthermia, dry mucous membranes**<br>Seizures, arrhythmias, rhabdomyolysis |
| Cholinergic | Organophosphate and carbamate insecticides<br>Chemical warfare agents (Sarin, VX) | **Salivation, lacrimation, diaphoresis, vomiting, urination, defecation, bronchorrhea, muscle fasciculations, weakness**<br>Miosis/mydriasis, bradycardia, seizures |
| Ethanolic | Ethanol | **Central nervous system depression, ataxia, dysarthria, odor of ethanol** |
| Extrapyramidal | Risperidone, haloperidol, phenothiazines | **Dystonia, torticollis, muscle rigidity**<br>Choreoathetosis, hyperreflexia, seizures |
| Hallucinogenic | Phencyclidine<br>Psilocybin, mescaline<br>Lysergic acid diethylamide | **Hallucinations, dysphoria, anxiety**<br>Nausea, sympathomimetic signs |
| Hypoglycemic | Sulfonylureas<br>Insulin | **Altered mental status, diaphoresis, tachycardia, hypertension**<br>Dysarthria, behavioral change, seizures |
| Neuromuscular malignant | Antipsychotics | **Severe muscle rigidity, hyperpyrexia, altered mental status**<br>Autonomic instability, diaphoresis, mutism, incontinence |
| Opioid | Codeine<br>Heroin<br>Morphine | **Miosis, respiratory depression, central nervous system depression**<br>Hypothermia, bradycardia |

| Toxidrome | Examples of Agents | Examination Findings (most common in bold) |
|---|---|---|
| Salicylate | Aspirin<br>Oil of Wintergreen (methyl salicylate) | **Altered mental status, respiratory alkalosis, metabolic acidosis, tinnitus, tachypnea, tachycardia, diaphoresis, nausea, vomiting**<br>Hyperpyrexia (low grade) |
| Sedative/hypnotic | Benzodiazepines<br>Barbiturates | **Central nervous system depression, ataxia, dysarthria**<br>Bradycardia, respiratory depression |
| Serotonin | SSRIs<br>MAOIs<br>Tricyclic antidepressants<br>Amphetamines<br>Fentanyl<br>St. John's wort | **Altered mental status, hyperreflexia and hypertonia (>lower limbs), clonus, tachycardia, diaphoresis**<br>Hypertension, flushing, tremor |
| Sympathomimetic | Amphetamines<br>Cocaine<br>Cathinones | **Agitation, tachycardia, hypertension, hyperpyrexia, diaphoresis**<br>Seizures, acute coronary syndrome |

*Abbreviations:* MAOI = monoamine oxidase inhibitor; SSRI = selective serotonin reuptake inhibitor.

## DIAGNOSTIC TESTING

A serum acetaminophen concentration is a routine screening test in poisoned patients. Early acetaminophen poisoning is often asymptomatic and does not have a readily identifiable toxidrome at the time when antidotal treatment is most efficacious. Acetaminophen screening is especially important in patients presenting with altered mental status or a self-harm ingestion, for whom an accurate history may not be available.

An electrocardiogram is a useful test to detect cardiac conduction abnormalities and identify patients at increased risk of toxin-induced adverse cardiovascular events.[22]

Measurement of drug or toxin concentrations in body fluids is not required in most poisonings, but in some exposures, measurement of serum drug concentrations does influence management (**Table 176-5**).

TABLE 176-5

**Drug Concentrations That May Assist Patient Assessment or Management**

| | |
|---|---|
| Acetaminophen | Methanol |
| Carbamazepine | Methotrexate |
| Carbon monoxide | Paraquat |
| Digoxin | Phenobarbital |
| Ethanol | Phenytoin |
| Ethylene glycol | Salicylate |
| Iron | Theophylline |
| Lithium | Valproic acid |
| Methemoglobin | |

Toxicologic screening tests of the urine and/or blood can be done in a central laboratory or performed with point-of-care drug screening assays.[23] However, the results seldom directly influence patient management, and toxicology screening has limitations (**Table 176-6**).

TABLE 176-6

**Limitations of Toxicologic Drug Screening Assays**

| | |
|---|---|
| **Nonspecific** | Most tests use enzyme-immunoassays that only detect *typical* drugs within a class: opioids, amphetamines, benzodiazepines, cannabinoids, cocaine, barbiturates.<br>Amphetamine screens do not detect methylenedioxymethamphetamine.<br>Opioid screens do not detect meperidine.<br>Benzodiazepine screens do not detect flunitrazepam. |
| **Time frame** | Drugs may be detected days to weeks after exposure. A positive test may not account for current clinical findings. |
| **Cross-reactivity** | Carbamazepine, cyproheptadine, and chlorpromazine test positive for tricyclic antidepressants.<br>Selegiline, methylphenidate, and pseudoephedrine test positive for amphetamines. |
| **Noninclusive** | A negative drug screen does not exclude a rare exposure. |
| **Sampling error** | Assay may be negative if dilute urine is tested. |

Toxicologic screening may be appropriate for medicolegal reasons, especially in pediatric cases when inappropriate drug administration or nonaccidental injury is suspected. A positive urine drug screen for an illicit substance is an indication to involve local child protection services.

# DECONTAMINATION

Decontamination is required for toxic exposures affecting large dermal areas. Healthcare providers wearing personal protective equipment (if indicated) or observing universal precautions (gown, gloves, eye protection) should assist with undressing and washing the patient using copious amounts of water. Contaminated clothing is collected, bagged, and properly disposed. Decontamination ideally occurs in a separate area adjacent to the ED, minimizing cross-contamination.

### OCULAR DECONTAMINATION

Eye exposures may require local anesthetic (e.g., 0.5% tetracaine) instillation and lid retractors to facilitate copious irrigation with crystalloid solution. Alkalis produce greater injury than acids due to deep tissue penetration via liquefaction so that prolonged irrigation (1 to 2 hours) may be required. Ten minutes after irrigation (allowing equilibration of crystalloid and conjunctival sac pHs), conjunctival sac pH is tested. Irrigation continues until pH is <7.4. Ophthalmologic consultation is indicated for all ocular alkali injuries.

### GASTROINTESTINAL DECONTAMINATION

Gastric decontamination is not a routine part of poisoned-patient management; there is minimal evidence demonstrating positive benefit, and there are associated complications (**Table 176-7**). Gastric decontamination may be considered in individual patients after a three-question risk-benefit analysis: (1) Is this exposure likely to cause significant toxicity? (2) Is gastrointestinal decontamination likely to change clinical outcome? (3) Is it possible that gastrointestinal decontamination will cause more harm than good?[24]

TABLE 176-7

**Indications, Contraindications, and Complications of Gastrointestinal Decontamination Procedures**

| **Orogastric Lavage** | |
|---|---|
| Indications | Rarely indicated<br>Consider for recent (<1 hour) ingestion of life-threatening amount of a toxin for which there is no effective treatment once absorbed |
| Contraindications | Corrosive/hydrocarbon ingestion<br>Supportive care/antidote likely to lead to recovery<br>Unprotected airway<br>Unstable, requiring further resuscitation (hypotension, seizures) |
| Complications | Aspiration pneumonia/hypoxia<br>Water intoxication<br>Hypothermia<br>Laryngospasm<br>Mechanical injury to gastrointestinal tract<br>Time consuming, resulting in delay instituting other definitive care |
| **Activated Charcoal** | **Adults 50 grams orally, children 1 gram/kg orally** |
| Indications | Ingestion within the previous hour of a toxic substance known to be adsorbed by activated charcoal, where the benefits of administration are judged to outweigh the risks |
| Contraindications | Nontoxic ingestion<br>Toxin not adsorbed by activated charcoal<br>Recovery will occur without administration of activate charcoal<br>Unprotected airway<br>Corrosive ingestion<br>Possibility of upper gastrointestinal perforation |
| Complications | Vomiting<br>Aspiration of the activated charcoal<br>Impaired absorption of orally administered antidotes |

| **Orogastric Lavage** | |
|---|---|
| **Whole-Bowel Irrigation** | **Polyethylene glycol 2 L/h in adults, children 25 mL/kg per hour (maximum 2 L/h)** |
| Indications (potential) | Iron ingestion >60 milligrams/kg with opacities on abdominal radiograph<br>Life-threatening ingestion of diltiazem or verapamil<br>Body packers or stuffers<br>Slow-release potassium ingestion<br>Lead ingestion (including paint flakes containing lead)<br>Symptomatic arsenic trioxide ingestion<br>Life-threatening ingestions of lithium |
| Contraindications | Unprotected airway<br>Gastrointestinal perforation, obstruction or ileus, hemorrhage<br>Intractable vomiting<br>Cardiovascular instability |
| Complications | Nausea, vomiting<br>Pulmonary aspiration<br>Time consuming; possible delay instituting other definitive care |

#### Emesis

Traditionally, ipecac syrup was administered to induce vomiting, theoretically emptying the stomach of poisons. No published evidence supports the induction of emesis, and adverse outcomes associated with emesis are documented.[25] The American Association of Poison Control Centers guideline[25] comments that ipecac may be used in rare circumstances in remote locations, but this recommendation has been questioned.[26] There is no role for the induction of emesis in the ED.[27]

#### Orogastric Lavage

Once a widely practiced intervention, attempted removal of ingested toxin from the stomach by aspiration of fluid placed via an orogastric tube is now rarely indicated. No published evidence demonstrates that orogastric lavage changes outcome, and the procedure has numerous complications.[28] Gastric lavage may be considered in cases of ingestion of a life-threatening amount of poison within the previous hour where institution of supportive care and antidotal therapy would not ensure full recovery. When orogastric lavage is performed in a resuscitation area:

Ensure a protected airway if consciousness level is reduced.

Use a 36 to 40F-gauge orogastric tube (22 to 24F in children).

Position the patient on the left side with the head down 20 degrees.

Pass lubricated tube down the esophagus a distance equal to that between chin and xiphoid process.

Confirm tube position by insufflation of air.

Gently lavage with 200 mL (10 mL/kg in children) of warm tap water.

Continue until returned fluid is clear.

Consider administration of activated charcoal via orogastric tube before removal.

#### Single-Dose Activated Charcoal

Super-heating carbonaceous material produces activated charcoal, a highly porous substance, which is suspended in solution and given PO as a slurry. Toxins within the gastrointestinal lumen are adsorbed onto the activated charcoal and carried through the gastrointestinal tract, limiting absorption.[29] Activated charcoal does not effectively adsorb metals, corrosives, and alcohols. The decision to give activated charcoal requires individual patient risk assessment and is not considered routine management.

Activated charcoal may be effective when given >60 minutes after ingestion of substances known to slow gastrointestinal motility (e.g., anticholinergics)[30] or after massive ingestion of a substance associated with bezoar formation (e.g., salicylates). Activated charcoal mixed with ice cream improves palatability for children. Activated charcoal can be administered to intubated patients using an orogastric or nasogastric tube. There are insufficient published data supporting the routine use of a cathartic agent added to activated charcoal.[31]

#### Whole-Bowel Irrigation

Polyethylene glycol is an osmotically balanced electrolyte solution. Administration in large quantities mechanically forces substances through the gastrointestinal tract, limiting toxin absorption.[32] Polyethylene glycol can be administered orally to cooperative, awake patients, but consider formal airway control if consciousness is likely to deteriorate. Minimize risk of pulmonary aspiration during whole-bowel irrigation by patient positioning (head up 30 degrees), ensuring bowel sounds are present during fluid administration, 1:1 nursing with suctioning of the oral cavity during infusion, and utilization of cuffed endotracheal tubes.

Evidence supporting whole-bowel irrigation is limited to volunteer studies and case reports[32] from which potential indications have been developed (**Table 176-7**). Nonsurgical treatment of asymptomatic body drug packers using whole-bowel irrigation is increasingly common, although no randomized clinical trials exist.[33]

An antiemetic such as the prokinetic agent metoclopramide may be required to control polyethylene glycol–induced gastric distension and vomiting. The endpoint of whole-bowel irrigation treatment is clear rectal effluent and imaging demonstrating absence of foreign bodies.

# ENHANCED ELIMINATION

### MULTIDOSE ACTIVATED CHARCOAL

Multidose activated charcoal increases elimination of toxins with enteroenteric, enterohepatic, or enterogastric recirculation. Lipophilic drugs with low volume of distribution, protein binding, and molecular weight may pass down a concentration gradient between intravascular space and activated charcoal in the gut lumen. Multidose activated charcoal may also adsorb residual intraluminal toxins; this is more likely for substances slowing gastric motility or forming bezoars (**Table 176-8**). Although animal studies, volunteer studies, case reports, and case series demonstrate increased elimination rates (in some cases comparable to those of hemodialysis or charcoal hemoperfusion) of carbamazepine, dapsone, phenobarbital, quinine, and theophylline, there is limited evidence that multidose activated charcoal changes clinical outcome.[34]

TABLE 176-8

**Indications, Contraindications, and Complications of Enhanced Elimination Procedures**

| **Multidose Activated Charcoal** | **Initial dose: 50 grams (1 gram/kg children), repeat dose 25 grams (0.5 gram/kg children) every 2 hours** |
|---|---|
| Indications | Carbamazepine coma (reduces duration of coma)<br>Phenobarbital coma (reduces duration of coma)<br>Dapsone toxicity with significant methemoglobinemia<br>Quinine overdose<br>Theophylline overdose if hemodialysis/hemoperfusion unavailable |
| Contraindications | Unprotected airway<br>Bowel obstruction<br>Caution in ingestions resulting in reduced gastrointestinal motility |
| Complications | Vomiting<br>Pulmonary aspiration<br>Constipation<br>Charcoal bezoar, bowel obstruction/perforation |
| **Urinary Alkalinization** | |
| Indications | Moderate to severe salicylate toxicity not meeting criteria for hemodialysis<br>Phenobarbital (multidose activated charcoal superior)<br>Chlorophenoxy herbicides (2-4-dichlorophenoxyacetic acid and mecoprop): requires high urine flow rate 600 mL/h to be effective<br>Chlorpropamide: supportive care/IV dextrose normally sufficient |
| Contraindications | Preexisting fluid overload<br>Renal impairment<br>Uncorrected hypokalemia |
| Complications | Hypokalemia<br>Volume overload<br>Alkalemia<br>Hypocalcemia (usually mild) |

Multidose activated charcoal may be administered by an orogastric or nasogastric tube to intubated patients. Regular aspiration of stomach contents helps avoid gastric distension. Multidose activated charcoal should not be given when bowel sounds are absent. Continued requirement for further multidose activated charcoal should be reviewed regularly during therapy.

### URINARY ALKALINIZATION

Alkaline urine favors ionization of acidotic drugs within renal tubules, preventing resorption of the ionized drug back across the renal tubular epithelium and enhancing elimination through the urine.[35] Urinary alkalinization is most effective for weak acids primarily eliminated by the renal tract that are also readily filtered at the glomerulus and have small volumes of distribution (**Table 176-8**). Hypokalemia will reduce the effectiveness of urinary alkalinization. The primary indication for urinary alkalinization is moderate to severe salicylate toxicity when criteria for hemodialysis have not been met. Urinary alkalinization for adult patients can be instituted as follows:

Correct any existing hypokalemia.

Administer a 1 to 2 mEq/kg IV sodium bicarbonate bolus.

Infuse 100 mEq of sodium bicarbonate mixed with 1 L of D5W at 250 mL/h.

20 mEq of potassium chloride may be added to the solution to maintain normokalemia.

Monitor serum potassium and bicarbonate every 2 to 4 hours to detect hypokalemia or excessive serum alkalinization.

Check urine pH regularly (every 15 to 30 minutes), aiming for a pH of 7.5 to 8.5.

A further IV bolus of 1 mEq/kg of sodium bicarbonate may be necessary if sufficient alkalinization of the urine is not achieved.

Although urinary acidification can enhance the elimination of weak bases including amphetamines and phencyclidine, associated risks (e.g., rhabdomyolysis) outweigh potential benefit. Forced diuresis has no indication for any poisoning, with the exception of chlorophenoxy herbicides (see chapter 201, Pesticides).

### EXTRACORPOREAL REMOVAL

Extracorporeal removal techniques, including hemodialysis, hemoperfusion, and continuous renal replacement therapies, have limited indications in poisoned patients (**Table 176-9**). These procedures require a critical care setting, are expensive and invasive, are not always available, and have complications. Extracorporeal removal techniques were utilized in less than 0.1% of cases reported to U.S. poison control centers in 2010.[13]

TABLE 176-9

**Indications, Contraindications, and Complications of Extracorporeal Removal Techniques**

| | | |
|---|---|---|
| **Hemodialysis** | **Movement of solute down a concentration gradient across a semipermeable membrane** | |
| Toxin requirements | Low volume of distribution, low protein binding, low endogenous clearance, low molecular weight | |
| Indications | **Life-threatening poisoning by:**<br>Lithium<br>Metformin lactic acidosis<br>Phenobarbital<br>Salicylates<br>Valproic acid | Methanol/ethylene glycol<br>Metformin-induced lactic acidosis<br>Potassium salts<br>Theophylline |
| Contraindications | Hemodynamic instability<br>Infants (generally) | Poor vascular access<br>Significant coagulopathy |
| **Hemoperfusion** | **Movement of toxin from blood, plasma, or plasma proteins onto a bed of activated charcoal (or other adsorbent)** | |
| Toxin requirements | Low volume of distribution, low endogenous clearance, bound by activated charcoal | |
| Indications | **Life-threatening poisoning caused by:**<br>Theophylline (high-flux hemodialysis is an alternative)<br>Carbamazepine (multidose activated charcoal or high-efficiency hemodialysis also effective)<br>Paraquat (theoretical benefit only if instituted early after exposure) | |
| Contraindications | Hemodynamic instability<br>Infants (generally)<br>Poor vascular access | Significant coagulopathy<br>Toxin not bound to activated charcoal |
| **Continuous Renal Replacement Therapies** | **Movement of toxin and solute across a semipermeable membrane in response to hydrostatic gradient. Can be combined with dialysis.** | |

| **Hemodialysis** | **Movement of solute down a concentration gradient across a semipermeable membrane** |
|---|---|
| Indications (potential) | Life-threatening ingestions of toxins when hemodialysis or hemoperfusion is indicated, but is unavailable, or hemodynamic instability precludes their utilization |
| Contraindications | Hemodialysis or hemoperfusion is available<br>Poor vascular access<br>Significant coagulopathy |
| **Complications of Extracorporeal Removal Techniques** | |
| Fluid/metabolic disruption<br>Removal of antidotes<br>Limited availability | Limited by hypotension (not continuous renal replacement therapy)<br>Infection/bleeding at catheter site<br>Intracranial hemorrhage secondary to anticoagulation |

A toxin must possess a number of properties to be effectively removed by an extracorporeal technique in a clinically meaningful timeframe: low volume of distribution (<1.0 L/kg), low molecular weight (<500 Da), relatively low protein binding, and low endogenous clearance.[36] In general, extracorporeal removal must improve endogenous clearance rate by >30% to be clinically beneficial. Hemoperfusion uses a charcoal (or other adsorbent) filter, which comes into direct contact with blood, partially overcoming molecular weight and protein-binding limitations.

Continuous renal replacement therapies (including venovenous hemofiltration and venovenous hemodiafiltration) are widely available and easily instituted in most hospitals. However, there is sparse evidence demonstrating any benefit in poisoning, primarily due to slow clearance rates.[36] A patient who requires extracorporeal removal should undergo hemodialysis or hemoperfusion, if available. Continuous renal replacement therapy can be used if hemodialysis or hemoperfusion is unavailable or will not be tolerated (e.g., due to hypotension).[36] Extracorporeal removal techniques including high-flux hemodialysis are constantly evolving, so discussion with an intensivist or nephrologist may be beneficial when this approach is considered.

## DISPOSITION

Planning for patient disposition from the ED should be part of initial risk assessment. Admission is indicated if the patient has persistent and/or severe toxic effects or will require a prolonged course of treatment. In most cases, a 6-hour observation period is sufficient to exclude the development of serious toxicity. Onset of

clinical toxicity can be delayed after a number of exposures, including (but not limited to) modified-release preparations of calcium channel antagonists, selective norepinephrine reuptake inhibitors (tramadol, venlafaxine), and newer antipsychotics (amisulpride); hence a period of extended observation is indicated. In the developed world, toxicity will resolve within 24 hours in most poisoned patients requiring noncritical care inpatient management, and so these patients can be efficiently and safely managed in a toxicology or short- stay ward, if available. Patients who have deliberately self-poisoned require appropriate mental health assessment before disposition.

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## USEFUL WEB RESOURCES

American Association of Poison Control Centers—http://www.aapcc.org/DNN/

National Pesticide Information Center—http://npic.orst.edu/index.html

Agency for Toxic Substance and Disease Registry—http://www.atsdr.cdc.gov