IN THE NAME OF GOD

General Management of Poisoned Patients

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TUMS

Poisoning related morbidity and mortality are also a

significant public health concern in the developed world.

Exposures occur most commonly by:

Ingestion

Inhalation

Insufflation

cutaneous and mucous membrane exposure,

Injection

The criteria used to determine nontoxic:

(1) an unintentional exposure to a clearly identified single substance

(2) an estimate of the dose is known

(3) a recognized information source (e.g., a poison control center) confirms the substance as nontoxic in the reported dose.

ASSESSMENT

-History

-Examination

-Ancillary test results

Acute poisoning is a dynamic process; therefore, risk assessment may change with time and requires ongoing review.

HISTORY

-Identity of substances, doses, and route of exposure

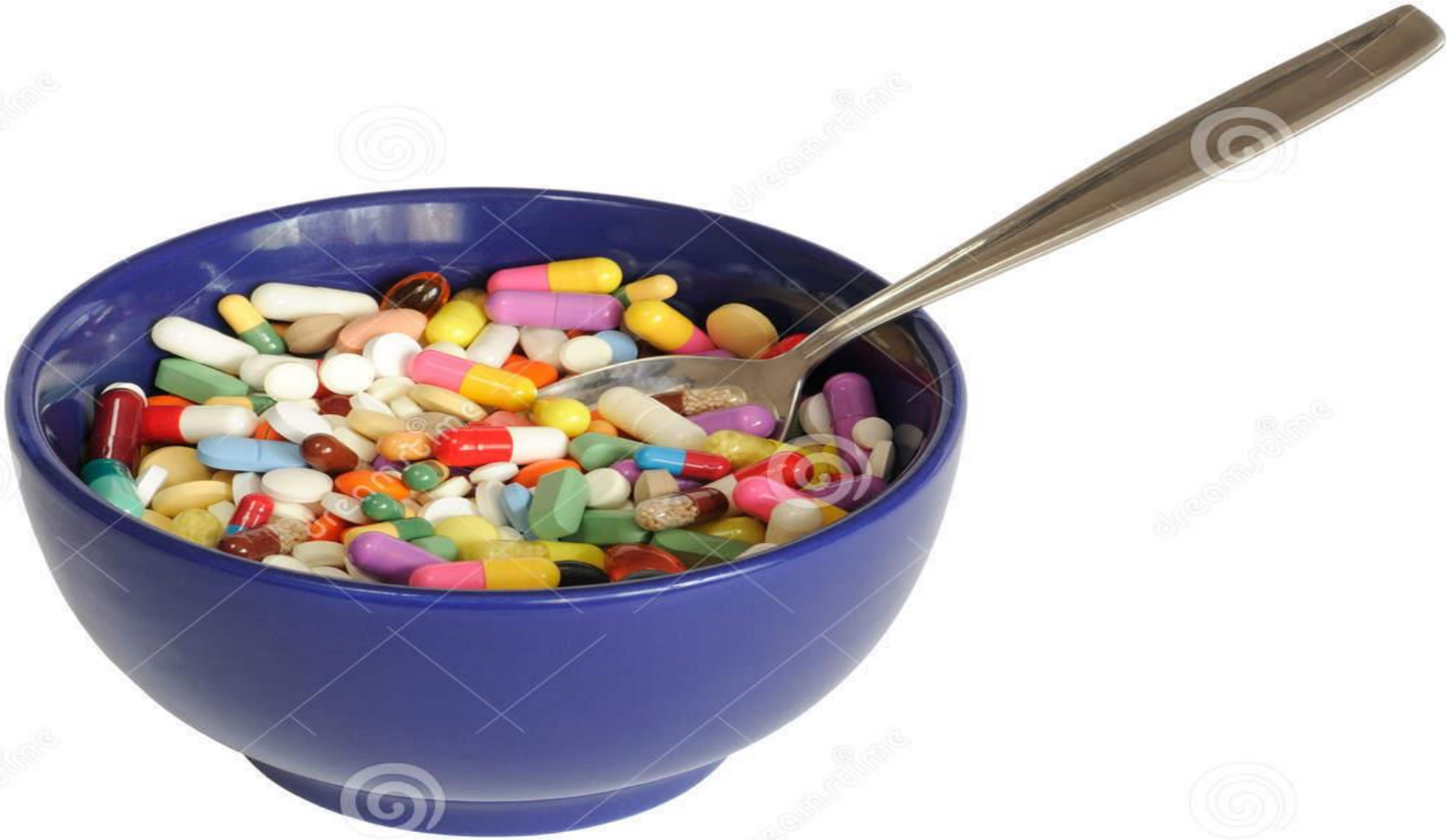
-Obtain collateral information from family, friends, previous medical records, and usual healthcare provider.

-Prehospital emergency services can provide information regarding empty medication containers or the scene environment (smells, particular materials or substances present).

EXAMINATION

Organ System	Examination	Example of Finding (Possible Significance)
General	Mental state and dress Signs of injury Odors Nutritional state Vital signs	Unkempt (psychiatric illness) Scalp hematoma (intracranial injury) Malnourished (IV drug use, human immunodeficiency virus infection) Smell of bitter almonds (cyanide toxicity)
CNS	Conscious state Pupil size and reactivity Eye movements Cerebellar function/gait	Miosis (opioids, organophos-phates, phenothiazines, clonidine intoxication) Nystagmus/ataxia (anticonvulsant and ethanol toxicity)
Cardiovascular	Heart rate/blood pressure Cardiac auscultation	Murmur (endocarditis/IV drug use)
Respiratory	Oxygen saturation Respiratory rate Chest auscultation	Fever/crepitations/hypoxia (aspiration pneumonia) Bronchorrhea/crepitations/hypoxia (organophosphate toxicity)

GI	Oropharynx Abdomen Bladder	Urinary retention (anticholinergic toxicity) Oral cavity burns (corrosive ingestion) Hypersalivation (cholinergic toxidrome)
Peripheral nervous	Reflexes Tone Fasciculations Tremor Clonus	Tremor/fasciculations (lithium toxicity) "Lead pipe" rigidity (neuromuscular malignant syndrome) Clonus/hyperreflexia (serotonin toxicity)
Dermal/peripheral	Bruising Cyanosis Flushing Dry/moist skin Injection sites Bullae	Bruising (coagulopathy, trauma, coma) Flushing/warm, dry skin (anticholinergic toxicity) Warm, moist skin (sympathomimetic toxicity) Bullae (prolonged coma, barbiturates)



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 Seizures, arrhythmias, rhabdomyolysis
Cholinergic	Organophosphate and carbamate insecticides Chemical warfare agents (sarin, VX)	Salivation, lacrimation, diaphoresis, vomiting, urination, defecation, bronchorrhea, muscle fasciculations, weakness Miosis/mydriasis, bradycardia, seizures
Ethanolic	Ethanol	CNS depression, ataxia, dysarthria, odor of ethanol
Extrapyramidal	Risperidone, haloperidol, phenothiazines	Dystonia, torticollis, muscle rigidity Choreoathetosis, hyperreflexia, seizures
Hallucinogenic	Phencyclidine Psilocybin, mescaline Lysergic acid diethylamide	Hallucinations, dysphoria, anxiety Nausea, sympathomimetic signs
Hypoglycemic	Sulfonylureas, insulin	Altered mental status, diaphoresis, tachycardia, hypertension Dysarthria, behavioral change, seizures
Neuromuscular malignant	Antipsychotics	Lead-pipe muscle rigidity, bradyreflexia, hyperpyrexia, altered mental status Autonomic instability, diaphoresis, mutism, incontinence
Opioid	Codeine, heroin, morphine	Miosis, respiratory depression, CNS depression Hypothermia, bradycardia

Salicylate	Aspirin Oil of wintergreen (methyl salicylate)	Altered mental status, respiratory alkalosis, metabolic acidosis, tinnitus, tachypnea, tachycardia, diaphoresis, nausea, vomiting Hyperpyrexia (low grade)
Sedative/hypnotic	Benzodiazepines Barbiturates	CNS depression, ataxia, dysarthria Bradycardia, respiratory depression
Serotonin	SSRIs MAOIs Tricyclic antidepressants Amphetamines Fentanyl St. John's wort	Altered mental status, hyperreflexia and hypertonia (>lower limbs), clonus, tachycardia, diaphoresis Hypertension, flushing, tremor
Sympathomimetic	Amphetamines Cocaine Cathinones	Agitation, tachycardia, hypertension, hyperpyrexia, diaphoresis Seizures, acute coronary syndrome

DIAGNOSTIC TESTING

A serum acetaminophen concentration is a routine screening test in poisoned patients because 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 ECG is a useful test to detect cardiac conduction abnormalities and identify patients at increased risk of toxin-induced adverse cardiovascular events.

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

some drugs are present in urine for an extended period of time, the positive test may not be related to the current clinical condition.

Urine drug screen results seldom influence patient management in most adult overdoses and poisoning

TABLE 176-5 Drug Serum Measurements That May Assist Patient Assessment or Management

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

RESUSCITATION

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

Prolonged resuscitation is generally indicated, as patients are often young with minimal preexisting organ dysfunction.

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.

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.

Altered mental status when hypoglycemia cannot be excluded is an indication for IV dextrose.

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

Antidysrhythmic drugs are not first-line treatment for toxin-induced dysrhythmias, as most antidysrhythmic drugs have prodysrhythmic and negative inotropic properties.

Most toxin-induced dysrhythmias 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 tachydysrhythmias.

Ventricular tachydysrhythmias 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.

Propofol and 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 it may worsen toxicity

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 anticholinergic and extrapyramidal effects.

First generation antipsychotics, such as haloperidol have been associated with QT-interval prolongation and cardiac dysrhythmias.

HYPERTHERMIA AND HYPOTHERMIA

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), and neuromuscular malignant syndrome (bromocriptine).

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 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 milligram.

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).

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.

IV LIPID EMULSION

Provide an intravascular “lipid sink,” sequestrating lipophilic toxins and preventing target receptor interaction.

IV lipid emulsion should be used as part of management of cardiac arrest in bupivacaine toxicity.

IV lipid emulsion therapy may cause fat deposition in extracorporeal membrane oxygenation circuits and increase blood clot formation.

IV lipid emulsion can be considered as a potential rescue therapy in life-threatening cardiotoxicity caused by lipophilic cardiotoxins that is resistant to conventional therapies.

TABLE 176-2 Common Antidotes Used in Resuscitation of the Acutely Poisoned Patient

Antidote	Initial Pediatric Dose*	Initial Adult Dose*	Indication
Calcium chloride 10% 27.2 milligrams/mL elemental Ca	0.15 mL/kg IV	10 mL IV	Calcium channel blockers
Calcium gluconate 10% 9 milligrams/mL elemental Ca	0.5–0.45 mL/kg IV	10–30 mL IV	Hypermagnesemia Calcium channel blockers
Cyanide antidote kit			
Amyl nitrite	Not typically used	Crack vial and inhale over 30 seconds, or place in chamber of ventilation bag and use 30 s on/30 s off	Cyanide
Sodium nitrite (3% solution)	Dosed according to hemoglobin level. If unknown, assume hemoglobin level is 12 g/dL (120 g/L) and dose with 0.33 mL/kg IV	10 mL IV	Cyanide Hydrogen sulfide (use only sodium nitrite)
Sodium thiosulfate (25% solution)	1.65 mL/kg IV	50 mL IV	Cyanide
Dextrose (glucose)	0.5–1.09 gram/kg IV	1 gram/kg IV	Insulin Oral hypoglycemics
Digoxin Fab Acute toxicity	5–10 vials IV	10 vials	Digoxin and other cardioactive steroids
Flumazenil	0.01 milligram/kg IV	0.2 milligram IV	Benzodiazepines

Glucagon	30 micrograms/kg IV over 1–2 min for CCB toxicity and 30–150 micrograms/kg IV over 1–2 min for BB toxicity	5 milligrams IV	Calcium channel blockers Beta-blockers
Hydroxocobalamin	70 milligrams/kg (maximum 5 grams) IV over 15 min	5 grams IV over 15 min	Cyanide Nitroprusside
IV lipid emulsion 20%	1.5 mL/kg IV bolus over 1 min (may be repeated 2 times at 5-min intervals), followed by 0.25 mL/kg per min IV infusion for 20 min	100-mL IV bolus over 1 min (may be repeated 2 times at 5-min intervals), followed by 18 mL/min IV infusion for 20 min	Local anesthetic systemic toxicity Rescue therapy for lipophilic cardiotoxins
Methylene blue	1 milligram/kg IV Neonates: 0.3–1.0 milligram/kg IV	1 milligram/kg IV	Oxidizing toxins (e.g., nitrites, benzocaine, sulfonamides)
Naloxone	As much as required Start: 0.01 milligram IV	As much as required Start: 0.1–0.4 milligram IV	Opioids Clonidine
Pyridoxine	Gram for gram if amount of isoniazid ingested is known, otherwise:		Isoniazid
	70 milligrams/kg IV (maximum 5 grams)	5 grams IV	
Sodium bicarbonate	1–2 mEq/kg IV over 1–2 min followed by 0.3 mEq/kg per hour IV infusion		Sodium channel blockers Urinary alkalinization
Thiamine	5–10 milligrams IV	100 milligrams IV	Wernicke's syndrome Wet beriberi

TABLE 176-1 Potential Interventions in Toxin-Induced Cardiac Arrest[11]

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

DECONTAMINATION

OCULAR DECONTAMINATION

Eye exposures 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 pH), conjunctival sac pH is tested.

Irrigation continues until pH is between 7.2 and 7.4. Ophthalmologic consultation is indicated for all ocular alkali injuries.

GI DECONTAMINATION

Gastric decontamination is not a routine part of poisoned patient management; there is minimal evidence demonstrating positive benefit, and there are associated complications

There is no role for the induction of emesis in the ED in the poisoned patient

Orogastric Lavage

Indications	Rarely indicated Consider for recent (<1 h) ingestion of life-threatening amount of a toxin for which there is no effective treatment once absorbed
Contraindications	Corrosive/hydrocarbon ingestion Supportive care/antidote likely to lead to recovery Unprotected airway Unstable, requiring further resuscitation (hypotension, seizures)
Complications	Aspiration pneumonia/hypoxia Water intoxication Hypothermia Laryngospasm Mechanical injury to GI tract Time consuming, resulting in delay instituting other definitive care

TABLE 176-8 Principles to Minimize Complications From Orogastric Lavage

- Ensure a protected airway if consciousness level is reduced.
- Use a 36F- to 40F-gauge orogastric tube (22F 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, allowing drainage after each aliquot.
- Continue until returned fluid is clear.
- Consider administration of activated charcoal via orogastric tube before removal.

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 Life-threatening ingestion of diltiazem or verapamil Body packers or stuffers Slow-release potassium ingestion Lead ingestion (including paint flakes containing lead) Symptomatic arsenic trioxide ingestion Life-threatening ingestions of lithium
Contraindications	Unprotected airway GI perforation, obstruction or ileus, hemorrhage Intractable vomiting Cardiovascular instability
Complications	Nausea, vomiting Pulmonary aspiration Time consuming; possible delay instituting other definitive care

ENHANCED ELIMINATION

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 Toxin not adsorbed by activated charcoal Recovery will occur without administration of activated charcoal Unprotected airway Corrosive ingestion Possibility of upper GI perforation
Complications	Vomiting Aspiration of the activated charcoal Impaired absorption of orally administered antidotes

Multidose Activated Charcoal	Initial dose: 50 grams (1 gram/kg children), repeat dose of 25 grams (0.5 gram/kg children) every 2 hours
Indications	Carbamazepine coma (reduces duration of coma) Phenobarbital coma (reduces duration of coma) Dapsone toxicity with significant methemoglobinemia Quinine overdose Theophylline overdose if hemodialysis/hemoperfusion unavailable
Contraindications	Unprotected airway Bowel obstruction Caution in ingestions resulting in reduced GI motility
Complications	Vomiting Pulmonary aspiration Constipation Charcoal bezoar, bowel obstruction/perforation

Urinary Alkalinization	
Indications	Moderate to severe salicylate toxicity not meeting criteria for hemodialysis Phenobarbital (multidose activated charcoal superior) Chlorophenoxy herbicides (2-4-dichlorophenoxyacetic acid and mecoprop): requires high urine flow rate of 600 mL/h to be effective Chlorpropamide: supportive care/IV dextrose normally sufficient
Contraindications	Preexisting fluid overload Renal impairment Uncorrected hypokalemia
Complications	Hypokalemia Volume overload Alkalemia Hypocalcemia (usually mild)

TABLE 176-10 Protocol for Urinary Alkalinization in Adults With Normal Renal Function

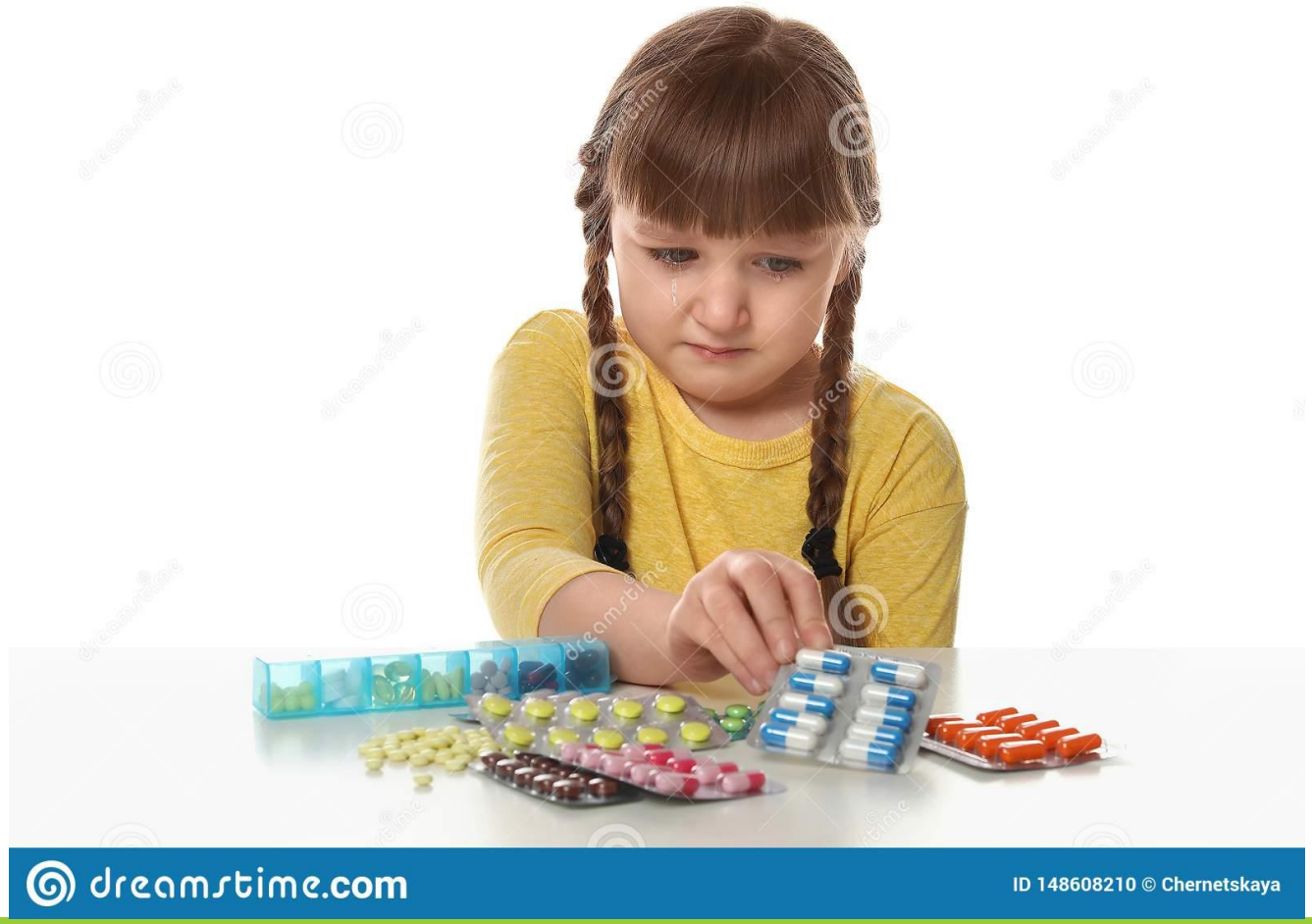
- Correct existing hypokalemia.
- Administer a 1 to 2 mEq/kg IV sodium bicarbonate bolus.
- Infuse 100 mEq of sodium bicarbonate mixed with 1 L of D_5W at 250 mL/h.
- 20 mEq of potassium chloride may be added to the solution to maintain normokalemia.
- Monitor serum potassium and bicarbonate every 2–4 h to detect hypokalemia or excessive serum alkalinization.
- Check urine pH regularly (every 15–30 min); goal is a pH of 7.5–8.5.
- A further IV bolus of 1 mEq/kg of sodium bicarbonate may be necessary if sufficient alkalinization of the urine is not achieved.

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:** Lithium Phenobarbital Salicylates Valproic acid Methanol/ethylene glycol Metformin-induced lactic acidosis Potassium salts Theophylline
Contraindications	Hemodynamic instability Infants (generally) Poor vascular access 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:** Theophylline (high-flux hemodialysis is an alternative) Carbamazepine (multidose activated charcoal or high-efficiency hemodialysis also effective) Paraquat (theoretical benefit only if instituted early after exposure)
Contraindications	Hemodynamic instability Infants (generally) Poor vascular access Significant coagulopathy 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.
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 Poor vascular access Significant coagulopathy
Complications of Extracorporeal Removal Techniques	
Fluid/metabolic disruption Removal of antidotes Limited availability	Limited by hypotension (not continuous renal replacement therapy) Infection/bleeding at catheter site Intracranial hemorrhage secondary to anticoagulation

PREVENTION



Prevention is the key to reducing unintentional poisoning deaths.

Pharmacists can ensure that medications are labeled correctly, anticipate potential drug interactions, and educate patients to use medications safely.

Parents have the responsibility to ensure that poisons are placed in childproof, labeled containers stored in adult only accessible nonfood storage areas to reduce pediatric exposures.

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 toxicosurveillance activities.

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 exposures to modified-release preparations of calcium channel blockers, selective norepinephrine reuptake inhibitors (tramadol, venlafaxine), and newer antipsychotics (amisulpride); a period of extended observation is indicated.

Patients who have deliberately self-poisoned require appropriate mental health assessment before disposition.

THANK YOU FOR YOUR ATTENTION