اصول کلی برخورد با بیماران مسموم

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GENERAL MANAGEMENT OF POISONED PATIENTS

- Poisoning is a worldwide problem that consumes substantial healthcare resources and causes many premature deaths.
- This increase has been ascribed to increasing prescription rates and aging of the baby-boom population.
- 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.
- 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.

GENERAL MANAGEMENT OF POISONED PATIENTS

- Exposures occur most commonly by ingestion; other routes include:
- Inhalation
- Insufflation
- Cutaneous
- Mucous membrane exposure
- Injection

The criteria used to determine whether the exposure is nontoxic are as follows:

- (1) an unintentional exposure to a clearly identified single substance
- (2) an estimate of the dose is known
- (3) a recognized information source

GENERAL MANAGEMENT OF POISONED PATIENTS

- **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%.10**

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.**

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. Utilization of extracorporeal cardiac and respiratory assist devices until organ toxicity resolves may be lifesaving**

RESUSCITATION

Airway Breathing Circulation

- **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.**

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	

ANTIDOTES

- **Stabilization of airway, breathing, and circulation allows further assessment of blood glucose concentration, temperature, and conscious state.**

- **Although the proper use of antidotes 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	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

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.
- Supplemental oxygen, thiamine, glucose, and naloxone are often administered empirically as a cocktail in cases of altered mental status.
- Metabolic, infective, and surgical (intracranial injury) causes of altered mental status should be considered.

CARDIAC ARRHYTHMIAS

- **In general, anti dysrhythmic drugs are not first-line treatment for toxin-induced dysrhythmias, as most anti dysrhythmic drugs have pro dysrhythmic and negative inotropic properties.**

- **Most toxin-induced dysrhythmias respond to correction of hypoxia, metabolic/acid-base abnormalities, and administration of an antidote (digoxin Fab).**

- **Sodium bicarbonate is administered for sodium channel–blocker toxicity with cardiovascular complications, such as wide QRS complex tachy dysrhythmias. Ventricular tachy dysrhythmias 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).**
- **A small study provided evidence for the safety of levetiracetam for treatment of toxin-induced seizures .**
- **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 theoretical disadvantages, including anticholinergic and extrapyramidal effect .
- First generation antipsychotics, such as haloperidol have been associated with QT interval prolongation and cardiac dysrhythmias; however, the incidence of adverse effects appears to be very low.

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 (cyproheptadine19), and neuromuscular malignant syndrome (bromocriptine20).
- Drug-induced coma with subsequent immobility and environmental exposure or inherent drug toxicity (opioids, phenothiazines, ethanol) may produce hypothermia. A core temperature

NALOXONE

- Naloxone 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) suggest possible opioid exposure.
- Naloxone is titrated to clinical effect using bolus doses, 0.1 to 0.4 milligram. Large initial bolus doses may precipitate vomiting and aspiration .
- Miosis is an unreliable indicator of naloxone's adequate clinical effect, as some opioids do not affect pupil size.
- 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.

IV LIPID EMULSION

- IV lipid emulsion has been postulated to 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.

- Does not support IV lipid emulsion as first-line treatment of life-threatening toxicity associated with other drugs, including amitriptyline, calcium channel antagonists, non–lipid-soluble beta-receptor antagonists, cocaine diphenhydramine, lamotrigine, bupropion, malathion, and cocaine.

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

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**

- **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	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

TOXIDROMES

Substances belonging to a particular pharmaceutical or chemical class often produce a cluster of symptoms and signs, or “toxidrome” , 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 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**

DIAGNOSTIC TESTING

- **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 .**

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

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

DIAGNOSTIC TESTING

- Toxicologic screening tests of urine can be done in a central laboratory or performed with point-of-care assays.
- Cross-reactivity is common and some drugs within the class may not be detected .
-
- Dilute urine can make it difficult to detect low levels. Because some drugs are present in urine for an extended period of time, the positive test may not be related to the current clinical condition.
- 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

TABLE 176-6 Standard Enzyme-Immunoassay Urine Drug Screens

Class (drugs typically tested)	Drugs not in this class possibly detected (false positives)*	Drugs in this class possibly not detected (false negatives)	Comments
Amphetamine and Methamphetamine	Amantadine **Bupropion** Chlorpromazine Desipramine **Dimethylamylamine** **Ephedrine** Fluoxetine Isoxsuprine **Labetalol** **Metformin** Phentermine Phenylephrine Phenylpropanolamine Promethazine **Pseudoephedrine** **Ranitidine** **Selegiline** Thioridazine Trazodone Trimethobenzamide Trimipramine	Methylenedioxymethylamphetamine [MDMA]	Detectable up to 48 h after single use
Barbiturates	Ibuprofen Naproxen	N/A	Detectable up to: Short-acting: 24 h Long-acting: 21 d
Benzodiazepines (oxazepam, nordiazepam)	**Oxaprozin** **Sertraline**	Alprazolam Clonazepam Flunitrazepam Lorazepam Triazolam	Detectable up to: Short-acting: 3 days Long-acting: 30 d
Cannabinoids (delta-9-tetracannabinol-9-carboxylic acid)	**Efavirenz** Ibuprofen Naproxen Pantoprazole Promethazine	Nabilone Synthetic cannabinoids (e.g., "K2" or "spice")	Detectable up to: Single use: 3 d Moderate use: 5–7 d Daily use: 10–15 d Long-term use: > 30 d
Cocaine (benzoylecgonine)	-	-	Detectable 2–4 d after single use
Methadone	Chlorpromazine Clomipramine **Diphenhydramine** **Doxylamine** Ibuprofen Quetiapine Thioridazine **Verapamil**	N/A	Detectable up to 3 d
Opiates (morphine, 6-acetylmorphine)	Dextromethorphan Diphenhydramine Fluoroquinolones (ciprofloxacin, levofloxacin, **ofloxacin**) Poppy seed and oil **Rifampin** Quinine	Buprenorphine Fentanyl Meperidine Methadone Oxycodone Oxymorphone Tramadol	Detectable 2–4 d depending on specific drug
Phencyclidine	Dextroamphetamine **Dextromethorphan** **Diphenhydramine** Doxylamine Ibuprofen Ketamine Lamotrigine Meperidine **Methylendioxyprovalerone** Thioridazine **Tramadol** **Venlafaxine**	N/A	Detectable up to 8 d after single use
Tricyclic Antidepressants	**Carbamazepine** **Cyclobenzaprine** Cyproheptadine Diphenhydramine Hydroxyzine Oxcarbazepine Promethazine **Quetiapine** Thioridazine	Clomipramine	Detectable 2–7 days

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 (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 .**

Gastric econtamination may be considered in individual patients after a threequestion risk-benefit analysis:

- **(1) Is this exposure likely to cause significant toxicity?**
- **(2) Is GI decontamination likely to change clinical outcome?**
- **(3) Is it possible that GI decontamination will cause more harm than good ?**

TABLE 176-7 Indications, Contraindications, and Complications of GI Decontamination Procedures

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
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
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

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.

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

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 in the majority of poisoned patients, and the procedure has numerous complications**
- **gastric lavage may be considered in cases of ingestion of a lifethreatening amount of poison within the previous hour where institution of supportive care and antidotal therapy would not ensure full recovery.**

- **Adherence to best-practiceprinciples can minimize complications from orogastric lavage .**

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.

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.
- 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 GI motility (anticholinergics) or after massive ingestion of a substance associated with bezoar formation (salicylates).
- Activated charcoal can be administered to intubated patients using an orogastric or nasogastric tube.

WHOLE-BOWEL IRRIGATIONA

- Polyethylene glycol can be administered orally to cooperative, awake patients, but consider formal airway control if consciousness is likely to deteriorate
- Nonsurgical treatment of asymptomatic body drug packers using whole-bowel irrigation is increasingly common.
- An antiemetic such as the prokinetic agent metoclopramide may be required to control polyethylene glycol–induced gastric distention and vomiting.
- The end point of whole-bowel irrigation treatment is clear rectal effluent and imaging demonstrating absence of foreign bodies

MULTIDOSE ACTIVATED CHARCOAL

- **Multi dose activated charcoal increases elimination of toxins with entero enteric, enterohepatic, or entero gastric 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.**

- **Multi dose activated charcoal may also adsorb residual intraluminal toxins; this is more likely for substances slowing gastric motility or forming bezoars .**

TABLE 176-9	Indications, Contraindications, and Complications of Enhanced Elimination Procedures
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)

EXTRACORPOREAL REMOVAL

- **Extracorporeal removal techniques, including :**
- **hemodialysis**
- **hemoperfusion**
- **continuous renal replacement therapies**

These procedures require a critical care setting, are expensive and invasive, are not always available, and have complications.

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.

TABLE 176-11	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:** 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

URINARY ALKALINIZATION

- **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.**
- **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.**
- **Although urinary acidification can enhance the elimination of weak bases including amphetamines and phencyclidine, associated risks (rhabdomyolysis) outweigh potential benefit.**

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 blockers, selective norepinephrine reuptake inhibitors (tramadol, venlafaxine), and newer antipsychotics .**

DISPOSITION

- 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.