Toxicology (General overview)

Drug overdoses are common and complex because they are usually poly-drug exposures and rarely fit the textbook description and treatment of toxidromes; further to this, the patients are unreliable in divulging specifics of intoxication requiring the use of collateral sources

ANESTHETIC CONSIDERATIONS:

- Potentially life threatening emergency even in situation of normal appearing patient
- Considerations of concomitant trauma, suicide attempt, pediatric / geriatric patient
- Resuscitation priorities (ABCs etc.)
- Identification of drugs / toxidromes in face of potential poly-drug overdose
- Management including ICU disposition

ANESTHETIC GOALS:

- Management of ABCs
- Prevention & treatment of life-threatening complications
- Specific antidote therapy if applicable

HISTORY

- AMPLE, collateral history = key (EMS, family / friends), what / how much / where / when, pharmanet search, previous visits, psych history

PHYSICAL

- ABCs (see below under optimization for management)

INVESTIGATIONS

- **Labs**
  - CBC
  - lytes, BUN, Cr for AG & renal failure
  - PTT, INR
  - ABG (including CO-Hb)
  - Glucose
  - Blood toxicology: EtOH, ASA, acetaminophen
  - Urine for toxicology & myoglobin
- **Imaging**
  - ECG for arrythmia
  - CXR for aspiration or edema
  - AXR for opaque substances

OPTIMIZATION

- **Management = ATLS Approach**
- **1° Survey:**
  - A = assess adequacy & patency + C-spine precautions
    - Common problems: vomitus, tongue obstruction
  - B = O_2 & support PRN, focused respiratory exam
    - Common problems: ventilatory failure, aspiration, hypoxemia, bronchospasm
  - C = 2 large IVs, vitals & monitor, support PRN
    - Common problems: hTN, arrythmias
    - Initiate ACLS protocol if needed w/ following caveats:
      - Hemodynamically significant bradycardias:
        - Atropine is rarely helpful except for anticholinesterase agents
        - Pacing is often effective (transcutaneous better than transvenous since transvenous is assoc. w/ more ventricular arrhythmias)
        - Vasopressors w/ beta activity are indicated for atropine / pacing resistant bradycardias
        - Isoproterenol is usually harmful in drug-induced bradycardia as it usually aggravates ventricular arrhythmias (EXCEPT in massive beta-blocker OD)
        - Dig-Fab-Ab fragments are extremely useful in digoxin OD
      - Hemodynamically significant tachycardias
        - Benzodiazepines are great especially for sympathetic overdrive
        - Physostigmine should be used in PURE anticholinergic OD only, not in mixed TCAs
        - Beta blockers should not be used in sympathetic overdrive poisonings where unopposed alpha can occur (i.e. cocaine)
        - Hypertonic saline & alkalinization is useful in poisonings w/ wide-complex conduction impairments (i.e. Na channel blockade seen in TCAs & cocaine)
          - Goal = pH 7.5 using repeated boluses of 1-2 mEq/kg (or respiratory alkalosis acutely until bicarbonate working)
        - Avoid adenosine or synchronized cardioversion as drug-induced tachycardias are usually resistant & refractory
        - Avoid CCBs in patients w/ questionable perfusion as shock may be worsened
        - Torsades in drug OD is treated using correction of hypoxia, hypokalemia & Mg, pacing at 100-120, & isoproterenol
        - For VT / VF, cardioversion is used and drug treatment is even less useful than in normal VT / VF arrest

        - Drug induced HTN:
            - Often short-lived and self-terminating
            - Consider benzodiazepines & SNP & avoid beta-blockers
    - D = GCS, evidence of dystonia or rigidity
        - Common problems: agitation, seizures, coma, hypoglycemia
        - Universal cocktail:
            - Thiamine 100 mg IV
            - D50W ½ amp IV
            - Naloxone 0.4 mg IV
            - DO NOT give flumazenil since it is potentially more hazardous than benzodiazepine intoxication
    - E = expose and evaluate temperature
        - NG & Foley
        - Common problems: needle-stick injury from drug paraphernalia, hypothermia, hyperthermia, undigested pill fragments, myoglobinuria
- **History:** as above
- **Initial investigations:** as above
- **2° Survey**:
    - Head-to-toe examination (fingers and tubes in every orifice)
    - Physiologic excitation:
        - CNS stimulation and increased HR, BP, RR and depth, and temperature
        - Anticholinergics, sympathomimetic, central hallucinogenic agents or drug withdrawal
    - Physiologic depression:
        - CNS depression and decreased HR, BP, RR and depth, and temperature
        - Cholinergic (parasympathomimetics), sympatholytic, opiate, or sedative-hypnotic, alcohol
    - Mixed physiologic effects:
        - Poly-drug ODs or following exposure to certain metabolic poisons (hypoglycemic agents, salicylates, cyanide), membrane-active agents (volatiles, antiarrhythmics, local anesthetics), heavy metals (iron, arsenic, mercury, lead), or agents w multiple action (TCAs)
    - Consider:
        - Alter absorption:
            - Charcoal:
                - 1 g/kg up to 50 g (q2-4h until passes charcoal stool)
                - Doesn't work for Li, Fe, EtOH
                - Potential complications: aspiration, ileus
            - Gastric lavage:
                - No evidence that lavage changes outcome
                - Suggest limit use to < 1 hour since ingestion & lethal drug amount
            - Don't give emetics
        - Alter metabolism & distribution
        - Enhance elimination: HD, plasmapheresis, HBO
    - Specific management for toxins (see below)
    - Toxicology, nephro, & ICU consults

PATHOPHYSIOLOGY

- 10% of all trauma mortalities are d/t fatal poisonings = 3rd most lethal injury
- Poisons are very common but are rarely fatal (7 in 1000 poisonings)
- Table of Toxidromes:

Toxidromes

Toxidrome	Mental status	Pupils	Vital signs	Other manifestations	Examples of toxic agents
Sympathomimetic	Hyperalert, agitation, hallucinations, paranoia	Mydriasis	Hyperthermia, tachycardia, hypertension, widened pulse pressure, tachypnea, hyperpnea	Diaphoresis, tremors, hyperreflexia, seizures	Cocaine, amphetamines, ephedrine, pseudoephedrine, phenylpropanolamine, theophylline, caffeine
Anticholinergic	Hypervigilance, agitation, hallucinations, delirium with mumbling speech, coma	Mydriasis	Hyperthermia, tachycardia, hypertension, tachypnea	Dry flushed skin, dry mucous membranes, decreased bowel sounds, urinary retention, myoclonus, choreoathetosis, picking behavior, seizures (rare)	Antihistamines, tricyclic antidepressants, cyclobenzaprine, orphenadrine, antiparkinson agents, antispasmodics, phenothiazines, atropine, scopolamine, belladonna alkaloids (eg, Jimson Weed)
Hallucinogenic	Hallucinations, perceptual distortions, depersonalization, synesthesia, agitation	Mydriasis (usually)	Hyperthermia, tachycardia, hypertension, tachypnea	Nystagmus	Phencyclidine, LSD, mescaline, psilocybin, designer amphetamines (eg, MDMA, MDEA)
Opioid	CNS depression, coma	Miosis	Hypothermia, bradycardia, hypotension, hypopnea, bradypnea	Hyporeflexia, pulmonary edema, needle marks	Opiates (eg, heroin, morphine, methadone, oxycodone, hydromorphone), diphenoxylate
Sedative-hypnotic	CNS depression, confusion, stupor, coma	Miosis (usually)	Hypothermia, bradycardia, hypotension, hypopnea, bradypnea	Hyporeflexia	Benzodiazepines, barbiturates, carisoprodol, meprobamate, glutethimide, alcohols, zolpidem
Cholinergic	Confusion, coma	Miosis	Bradycardia, hypertension or hypotension, tachypnea or bradypnea	Salivation, urinary and fecal incontinence, diarrhea, emesis, diaphoresis, lacrimation, GI cramps, bronchoconstriction, muscle fasciculations and weakness, seizures	Organophosphate and carbamate insecticides, nerve agents, nicotine, pilocarpine, physostigmine, edrophonium, bethanechol, urecholine
Serotonin syndrome	Confusion, agitation, coma	Mydriasis	Hyperthermia, tachycardia, hypertension, tachypnea	Tremor, myoclonus, hyperreflexia, clonus, diaphoresis, flushing, trismus, rigidity, diarrhea	MAOIs alone or with: SSRIs, meperidine, dextromethorphan, TCAs, L-tryptophan
Tricyclic antidepressant	Confusion, agitation, coma	Mydriasis	Hyperthermia, tachycardia, hypertension then hypotension, hypopnea	Seizures, myoclonus, choreoathetosis, cardiac arrhythmias and conduction disturbances	Amitriptyline, nortriptyline, imipramine, clomipramine, desipramine, doxepin

- Lab geek dreams:
  - Anion gap = Na - ($Cl + HC0_3$)
    - Normal = 12
    - Not a sensitive marker of organic acid accumulation (i.e. lactic acidosis)
  - AMUDPILES:
    - Alcoholic ketoacidosis
    - Methanol
    - Uremia
    - DKA
    - Paraldehyde / phenformin
    - INH / iron
    - Lactic acidosis
    - Ethylene glycol
    - ASA (Salicylates)
    - CO, CN
    - Toluene
  - Osmolal gap = measured plasma osmolality - calculated plasma osmolality (2Na +BUN + glucose)
    - Normal < 10
    - If gap > 10, ? unmeasured osmoles
      - Alcohols: ethanol, methanol, ethylene glycol, isopropyl alcohol
      - Mannitol, sorbitol, glycerol
      - Acetone
      - Paraldehyde

SPECIFIC POISONINGS

- **Acetaminophen:**
  - Background:
    - Acetaminophen itself is nontoxic, but hepatic metabolism leads to formation of a toxic metabolite, N-acetyl-benzoquinoneimine (NAPQI)
    - The liver metabolizes more than 90% of acetaminophen to glucuronide and sulfate conjugates, which are eliminated in the urine
    - Hepatotoxicity is the result of formation of the reactive and toxic metabolite NAPQI by the cytochrome P-450 system
      - Normally, glutathione binds NAPQI and leads to excretion of nontoxic mercapturate conjugates
      - Production of NAPQI by the cytochrome P-450 system is the cause of liver toxicity
      - Toxicity is increased by malnutrition, EtOH abusers & those taking isoniazid
    - Renal toxicity occurs less commonly but by the same mechanism as hepatotoxicity
    - Symptoms are often masked by acute symptoms of other OD drugs
    - Max doses:
      - The maximum daily dose of acetaminophen is 4 g in adults and 90 mg/kg in children
      - A single ingestion of 7.5 g in an adult or more than 150 mg/kg in a child is a potentially toxic dose

- Features = 4 Phases:
  - Phase 1: nonspecific and include pallor, malaise, vomiting, and diaphoresis
  - Phase 2:
    - RUQ pain
    - Tachycardia and hTN may occur w/ volume loss
  - Phase 3: jaundice, GI bleeding, abdominal pain, and encephalopathy
  - Phase 4: resolution of physical findings or death
- Treatment:
  - Lab guided:
    - Nomogram 4 h post-ingestion
    - Follow LFTs
  - NAC:
    - PO: 140 mg/kg load, then 70 mg/kg q4h over 72 h
    - IV: 150 mg/kg in 200 cc D5W over 15 min, then 50 mg/kg in 500 cc D5W over 4 h, followed by 100 mg/kg in 1 L of D5W over 16 h

- **Amphetamines:**
  - Background:
    - Activate SNS via CNS, increase peripheral catecholamine release, inhibit reuptake of catecholamines, & inhibit MAO
    - Results in increased cortical alertness w/ associated appetite suppression and decreased need for sleep
    - Group of drugs each of which has variable central & peripheral effects on the SNS
    - Low therapeutic index
    - Chronic use results in depletion of body stores of catecholamines
  - Features:
    - CNS: anxiety, psychotic state, progressive irritability (hyperactivity, hyperreflexia, seizures)
    - CVS: HTN, tachycardia, dysrhythmias, diaphoresis, dehydration
    - GI: decreased motility, cachexia
    - Metabolic: lactic acidosis, ketosis
    - Hyperthermia
    - Pulmonary HTN in > 3/12
  - Treatment:
    - Consider gastric lavage +/- activated charcoal
    - Benzodiazepines
    - Alkalinization of urine promotes elimination
    - Direct acting vasopressors in shock or withdrawal
- **Barbiturates:**
  - Background:
    - Rule of thumb: toxic dose = 5-10x hypnotic dosage
    - Cross-tolerance to depressant effects of anesthetics in chronic users
    - Acute ingestion decreases MAC
    - Induces hepatic enzymes so may alter other drug pharmacology
    - Acute withdrawal can be life-threatening d/t ANS dysfunction
  - Features:
    - CNS depression via GABA & sympatholysis + decreased contractility = anesthetized patient
    - Hypothermia
    - W/D: seizures, anxiety, MSK tremors, hyperreflexia, hyperthermia, diaphoresis, tachycardia, orthostatic hTN, CV collapse
  - Treatment:
    - Supportive
    - Titrated barbiturate for withdrawal
- **Benzodiazepines:**
  - Background:
    - Mechanism of action = CNS depression via GABA & poorly defined depression of spinal reflexes & reticular activating system
    - Less likely to produce respiratory depression than barbiturates
    - However, combinations of benzodiazepines with other CNS depressants may be lethal
  - Features:
    - CNS: lethargy, slurred speech, ataxia, coma
    - Resp: respiratory arrest
    - Hypothermia
  - Tx:
    - Supportive
    - Flumazenil 0.2 mg IV is controversial d/t concomitant drug toxicities:
      - Induces seizures in TCA concomitant overdose
      - Acute withdrawal in benzodiazepine addicted patient = seizures & ANS dysfunction
      - Re-sedation can occur since short half-life
- **Cocaine = see Cocaine Seminar**
- **Digoxin:**
  - Background:
    - Cardiac glycosides inhibit the Na-K-ATPase and decreases the transmembrane Na gradient as well as the driving force of the Na-Ca transporter (thus, Ca accumulates in the cell)

- Features:
  - Non-specific: fatigue, blurred vision, disturbed color perception, anorexia, N/V/D, abdominal pain
  - CNS: H/A, dizziness, confusion, delirium and hallucinations
  - Cardiac: bradycardia, occasional hypotension
  - EKG:
    - ST segment scooping
    - Ventricular ectopy (PVCs)
    - Accelerated junctional and bidirectional VT
  - Serum potassium:
    - Acute toxicity: hyperkalemia is a predictor or morbidity and mortality
    - Reflects the degree of poisoning of Na-K-ATPase
    - In chronic toxicity, hypokalemia potentiates toxicity by decreasing Na-K-ATPase activity and thus it should be corrected immediately
- Treatment:
  - Supportive
  - Symptomatic bradycardias should be treated w/ atropine:
    - Avoid transvenous pacing as this may precipitate arrhythmias
    - Beta-agonists (e.g. isoproterenol) should also be avoided
  - GI decontamination w/ activated charcoal
  - Hyperkalemia:
    - Treat in the usual fashion
    - Do not administer Ca as this can exacerbate intracellular hypercalcemia
  - Digibind (digoxin specific Fab fragments) indications:
    - Hemodynamic instability
    - Life-threatening arrhythmias
    - Severe bradycardia
    - Serum K > 5.0 in the setting of acute overdose
    - Plasma digoxin concentration > 13 nmol/L
    - Ingestion of > 10 mg of digoxin in adults (> 4mg in children)
    - Presence of a digoxin toxic rhythm in the setting of elevated digoxin level

- **Hallucinogens:**
  - Background:
    - Includes LSD, PCP
    - Analgesic and pulmonary depressant effects of opioids prolonged
    - Anesthesia & surgery may provoke panic attacks or bad trip
  - Features = rarely produces seizures and apnea
  - Treatment = supportive care
- **Marijuana:**
  - Background:
    - THC binds to anandamide receptors in the brain
    - Effects of THC last 2-3 hours
    - Increases SNS activity & decreases PNS activity
    - Potentiate opioid-induced ventilatory depression
  - Features:
    - Has stimulant, sedative & hallucinogenic actions depending on dose and timing since ingestion
    - Increased resting HR & orthostatic hTN
    - Munchies
    - Drowsiness
  - Tx:
    - Supportive & reassurance
    - Titrated adjunctive benzodiazepines may be needed
- **Methanol / ethylene glycol:**
  - Background:
    - Three alcohols can lead to fatal intoxication:
      - Methanol: leads to increased osmolar gap and increased anion gap:
        - Wood alcohol (CH_3OH) is found in varnish & de-icing solutions
      - Ethylene glycol: leads to increased osmolar gap and increased anion gap:
        - Found in anti-freeze & solvents
      - Isopropyl alcohol: leads to increased osmolar gap but normal anion gap (due to metabolism to acetone which is neutral and does not contribute to AG)
        - Rapidly absorbed
    - Metabolism:
      - Both methanol and ethylene glycol are metabolized by alcohol dehydrogenase
        - Methanol is metabolized to formaldehyde
        - Ethylene glycol is metabolized to various toxic compounds including glycolic acid and oxalic acid
          - Glycolic acid is toxic to tubules
          - Oxalic acid precipitates in tissues

        - EtOH is preferentially metabolized by alcohol dehydrogenase and thus prevents accumulation of toxic metabolites in methanol or ethylene glycol poisonings
    - Isopropyl alcohol is itself toxic but its metabolites are benign
- Features:
    - Methanol presents w/ primary CNS findings: weakness, headaches, nausea, decreased vision which can lead to blindness, coma and death
        - Specific eye findings include mydriasis, decreased light reflex and hyperemia of optic disk
    - Ethylene glycol:
        - CNS: drunkenness, coma
        - Pulmonary edema
        - Renal failure w/ Ca-oxalate crystals
        - Can perform Wood's lamp examination (UV light) of patient or urine which may reveal fluorescence of antifreeze solution
    - Isopropyl alcohol:
        - Ataxia and lethargy which can progress to coma
        - Ketonuria or ketonemia with acetone on breath
- Treatment:
    - Methanol & ethylene glycol:
        - Consider gastric lavage
        - Sodium bicarbonate to correct metabolic acidosis
        - Fomepizole is a competitive inhibitor of alcohol dehydrogenase:
            - 15 mg/kg over 30 minutes, 10 mg/kg q12h x 48 h, then 15 mg/kg q12h and keep administering until ethylene glycol concentration falls below 20 mg/dL
            - Must increase dosing to q4h if concomitant hemodialysis
        - Intravenous ethanol (do not use if fomepizole being used)
        - Hemodialysis:
            - High plasma level of methanol (> 20 mg/dL) or ethylene glycol (> 15 mmol/L)
            - Metabolic acidosis
            - Mental status changes
    - Isopropyl alcohol: supportive + hemodialysis

- **Opioids:**
    - Background:
        - Multiple receptors w/ various effects:
            - Euphoria = Mu_1
            - Bradycardia + Hypothermia = Mu_1
            - Sedation = Kappa
            - Diuresis = Kappa
            - Dysphoria = Kappa
            - N/V = Delta
            - Retention = Mu_1 + Delta
            - Resp Depression = Mu_2 + Delta
            - Dependance = Mu_2 + Delta
            - Constipation = Mu_2 + Delta (little)
    - Features:
        - As above
        - Cachexia d/t to chronic drug habit and lack of nutritional support
        - Decreased anesthetic requirements in acute opioid administration
        - Tendency for perioperative hTN & inadequate intravascular volume
        - Adrenocortical insufficiency
        - Opioid tolerance & decreased pain tolerance
    - Treatment:
        - Maintain baseline opioid levels peri-hospitalization
        - Naloxone 0.2-0.4 mg IV in acute life-threatening OD but be prepared for combative pissed off patient
- **Organophosphates:**
    - Background:
        - Organophosphates (insecticides, nerve agents – sarin, soman) and carbamates are potent acetylcholinesterase inhibitors
        - These can be rapidly absorbed cutaneously, enterally or via inhalation
        - Carbamates differ from organophosphates in that they are transient inhibitors, thus lead to shorter duration of symptoms (< 48 h)
        - Results in increased ACh levels causing cholinergic crisis which can have nicotinic and muscarinic effects
    - Features:
        - Think of increased PNS tone
        - Muscarinic Effects
            - SLUDGE / BBB: salivation, lacrimation, urination, defecation, GI upset, emesis, bronchorrhea, bronchospasm, bradycardia
            - Also get miosis, diaphoresis, hypotension
        - Nicotinic Effects
            - Fasciculations, muscle weakness, paralysis
        - CNS effects:
            - Acute: CNS depression, lethargy, excitability, seizures and coma
            - Delayed neurotoxicity: organophosphorous agent induced delayed neuropathy (OPAIDN)

        - MSK weakness can occur up to 4 days post ingestion causing death by apnea
    - Treatment:
        - Health care workers should maintain careful precautions against contacting the skin or clothes of contaminated patients to prevent serious illness d/t secondary contamination
        - Supportive
        - Volume
        - Atropine:
            - 2-5 mg IV for adults
            - 50 mcg/kg for children
            - Dose should be doubled q3-5 minutes until pulmonary muscarinic signs are alleviated (respiratory secretions, bronchoconstriction)
            - Do not rely on tachycardia and mydriasis as appropriate end-points
        - Pralidoxime:
            - Binds to nicotinic receptors as well as muscarinic receptors
            - Do not administer w/out atropine
            - 30 mg/kg IV bolus over 30 minutes
        - Benzodiazepines:
            - Should be used if seizures and also decreased neurocognitive defects
        - Decontamination: flood cutaneous exposure w/ dilute bleach
- **Salicylate:**
    - Background:
        - ASA directly stimulates the medullary respiratory center causing respiratory alkalosis w/ significant hyperventilation
            - Results in a respiratory alkalosis
        - Salicylates also cause uncoupling of oxidative phosphorylation, which leads to decreased ATP production, increased oxygen consumption, increased carbon dioxide production, and increased heat production
            - Accumulation of organic acids, including pyruvate, lactate, and acetoacetate, causing anion gap metabolic acidosis
            - AGMA increases levels of H-salicylate, the basic, non-ionized form of salicylate which can readily cross membranes (i.e. BBB & CNS cells thereby increasing neurotoxicity)
            - Thus, need to maintain alkalemic milieu which traps the salicylate and prevents CNS toxicity
        - Increased metabolic rate, pyrexia, tachypnea, and vomiting lead to fluid loss and dehydration
            - Because of significant water losses, hyponatremia might not be present; however, hypokalemia is prominent
        - Diagnosis is by ABG & plasma salicylate level:
            - 6-hour post-ingestion level (in acute, single ingestion only) can be applied to the Done nomogram to assist in predicting the level of toxicity
    - Features:
        - Vital signs: fever, increased RR & HR
        - CNS: tinnitus, vertigo, altered mental status, coma
        - Resp: tachypnea, ALI (non-cardiogenic pulmonary edema)
        - CVS: dysrhythmias (PVCs, VT / VF), hTN and shock
        - GI: abdominal pain, vomiting (+/- blood), diarrhea
        - Acid-base disturbance
    - Treatment:
        - Avoid intubation:
            - These patients hyperventilate and maintain a respiratory alkalosis on their own
            - Difficult to maintain degree of minute ventilation on mechanical ventilation
        - Volume resuscitation
        - Supplemental glucose:
            - ASA results in decreased CNS glucose levels despite normal peripheral glucose levels
            - Thus, any patient w/ altered LOC should be given peripheral glucose
        - Sodium bicarbonate:
            - Target pH 7.5-7.55
            - Urine pH > 7.5
        - Hypokalemia treatment
        - Hemodialysis
- **TCA:**
    - Background:
        - Low toxicity threshold
        - Block reuptake of centrally and peripheral acting neurotransmitters (NE, serotonin):
            - Anticholinergic effects: muscarinic ACh receptor blockade
            - Alpha-adrenergic blockade induces vasodilation
            - Slowing Na^+ channel in myocardium: quinidine effect
            - Direct myocardial toxicity: combo of catecholamine depletion & alpha-adrenergic blockade can produce profound CVS dysfunction
    - Features usually combo of 3:
        - TCA poisoning should be suspected in any patient w/ lethargy, coma, or seizures accompanied by prolonged QRS
        - Anticholinergic effects: altered mental status (agitation, confusion, lethargy), resting sinus tachycardia, dry mucous membranes, mydriasis, fever

        - CVS: HTN (early and transient, should not be Tx), tachycardia, orthostatic hTN, arrhythmias = wide complex tachycardias (every part of the ECG gets wider)
        - CNS: coma, seizures, myoclonic twitches / tremor, hyperreflexia
        - Resp: hypoventilation resulting from CNS depression, pulmonary edema in severe cases
        - GI: decreased motility
    - Tx:
        - Consider gastric lavage as there is delayed gastric emptying
        - Alkalosis by hyperventilation or bicarb (1-2 mEq/kg then 3 amps in 1 L D5W to make 150 mEq/L - infuse at 100 cc/hr) to pH > 7.45
        - Seizure control - use benzodiazepines; if refractory, then paralyze patient to prevent hyperthermic death
        - Volume replacement: aim for U/O > 2 mL/h - shock may be difficult to treat - there are anecdotal reports of severe, refractory shock patients being saved by CPB
        - Pacing is useful for bradycardias, AV block, or overdrive of Torsades
        - Physostigmine, used historically, should NOT be used as it is associated w/ increasing cardiac death rate
        - There is no evidence that lidocaine is useful in treatment of cardiac conduction disturbances in TCA overdose

- **Beta Blockers**
    - Essentials:
        - Bradycardia, hypotension, coma, history of beta blocker ingestion or availability
    - Clinical Presentation
        - Cardiovascular - bradycardia, conduction delays, decreased contractility with systemic hypotension
        - Central nervous system - coma, seizures
        - Pulmonary - bronchospasm (unusual)
        - Metabolic - acidosis, hypoglycemia (rare)
    - Lab Tests:
        - 12 lead ECG (bradycardia, heart block)
        - Lytes, BUN, Cr, glucose, and acid base status to help direct supportive care
        - Specific drug levels can help confirm exposure but do not correlate with symptoms / alter management
    - Treatment:
        - The goal of therapy is to restore perfusion to critical organ systems by improving myocardial contractility or increasing heart rate, or both (resuscitation)
        - Gastrointestinal decontamination with 1 g/kg activated charcoal
        - No proven means of enhanced elimination
        - Atropine is safe but rarely effective
        - Note: glucagon makes everyone vomit, therefore consider definitive airway prior to glucagon
    - Treatment essentials of β-adrenergic receptor antagonist toxicity
        - Glucagon bolus (3.5-5 mg) followed by infusion (1-5 mg/hr)
        - Epinephrine infusion (1 µg/kg/min) and titrate or isoproterenol infusion (2 µg/min) and titrate
        - Milrinone infusion (0.5 µg/kg/min) and titrate
        - Electrical pacing
        - Consider aortic balloon pump
    - Treatment endpoints:
        - HR > 60 bpm, SBP > 90
        - Evidence of adequate end organ perfusion (improved LOC, U/O 1 mL/kg/h)
    - Monitoring / disposition:
        - Continuous ECG monitoring, serial 12 lead ECG, serial physical examinations, Foley
        - A. line +/- PAC for patients in cardiogenic shock
        - Patients presenting in shock, altered mental status, conduction delays or not meeting treatment endpoints should be monitored in a HDU
        - Asymptomatic patients with history of overdose can be safely discharged after 10 h monitoring, provided they remain asymptomatic and have normal vitals and ECG
    - Sequelae:
        - Patients surviving beta blocker overdose recover without obvious long-term complications
- **Calcium Channel Blockers**
    - Essentials:
        - Bradycardia, hypotension
        - Metabolic acidosis with hyperglycemia
        - History of Ca blocker ingestion or availability, history of HTN or heart disease
    - Clinical Presentation
        - Bradycardia with hypotension
        - Altered mental status (patients may have clear sensorium early in clinical course)
        - Metabolic acidosis with hyperglycemia
        - Sinus arrest on electrocardiogram
        - Refractory shock
            - Suspect in patients with a history of hypertension or cardiac disease having access to calcium channel blockers
    - Lab Tests:
        - 12 lead ECG (bradycardia, heart block)
        - Lytes, BUN, Cr (serum calcium and K should be serially monitored → hypokalemia frequently seen but no prognostic value, hyperkalemia suggests severe cellular poisoning and marked negative inotropy can be expected)
        - Serum digoxin level if concomitant digoxin toxicity suspected, specific CCB levels don't correlate with symptoms

        - ABG to guide O_2 and ventilatory therapy
        - ECHO – assess LV function
    - Treatment:
        - Three treatment objectives: 1) providing supportive care, 2) decreasing drug absorption and 3) augmenting myocardial function
        - Gastrointestinal decontamination with 1 g/kg activated charcoal
        - No proven means of enhanced elimination
        - Atropine is safe but rarely effective
    - Treatment of calcium channel antagonist toxicity
        - Ensure airway protection and adequate ventilation and oxygenation, preferably via endotracheal intubation and mechanical ventilation
        - Insert a 7 F central venous "cordis" catheter
            - Administer a 10 to 20 mL/kg normal saline injection
        - Keep arterial pH > 7.30 with hyperventilation and potassium level < 5.0 mEq/L (particularly if considering an insulin infusion)
        - Begin electrical pacing for heart rate below 40 beats per minute with shock
        - Administer 10 to 20 mg/kg of 1% calcium chloride solution
            - If favorable response, begin infusion at 20 mg/kg/h (monitor ionized calcium, not to exceed two times normal baseline level)
        - Bolus inject 0.05 to 0.2 mg/kg glucagon
            - If beneficial, begin 0.1 mg/kg/h infusion and titrate
        - Consider beginning insulin / dextrose bolus / infusion at 1.0 U/kg IV bolus, followed by 1.0 U/kg/h infusion with 40 mL 50% dextrose/h
            - Monitor glucose hourly
            - Monitor potassium
        - For refractory hypotension
            - Begin dopamine infusion at 10 µg/kg/min; titrate infusion (consider isoproterenol / epinephrine)
            - Bolus milrinone and begin infusion
            - Consider intra-aortic balloon counter-pulsation treatment or extracorporeal cardiopulmonary bypass
    - Treatment endpoints:
        - HR > 60 bpm, SBP > 90
        - Evidence of adequate end organ perfusion (improved LOC, U/O 1 mL/kg/hr)
    - Monitoring / Disposition:
        - Continuous ECG monitoring, serial 12 lead ECG, serial physical examinations, Foley
        - A. line +/- PAC for patients in cardiogenic shock
        - Patients presenting in shock, altered mental status, conduction delays or not meeting treatment endpoints should be monitored in a HDU
        - Asymptomatic patients with history of overdose need to be monitored for 24 hours as many CCB overdoses associated with long acting preparations
    - Sequelae:
        - Patients surviving CCB overdose recover without obvious long-term complications
- **Lithium**
    - Essentials:
        - Mild / moderate toxic symptoms: tremor, hyperreflexia, drowsiness, clonus
        - Major toxic symptoms: confusion, ataxia, stupor, coma, seizures
        - History of lithium therapy of ingestion
        - Li level greater than 1.2 mEq/L
    - Clinical Presentation
        - Gastrointestinal
            - Minor / persistent - nausea and vomiting, diarrhea, abdominal discomfort
            - Acute - nausea and vomiting, diarrhea
            - Chronic - anorexia, nausea and vomiting
        - Neurologic
            - Minor / persistent - fine tremor, muscle weakness, fatigue
            - Acute - may be asymptomatic; can develop tremors, rigidity, clonus, fasciculations, hyperreflexia, lethargy, confusion, coma, seizures
            - Chronic - muscle weakness, tremor, apathy, dysarthria, muscle rigidity, fasciculations, choreoathetosis, vertigo, pseudotumor cerebri, tinnitus, ataxia, blurred vision, confusion, seizures, coma
        - Cardiovascular
            - Minor / persistent - sinus node dysfunction, T wave changes, U waves
            - Acute - hypotension
            - Chronic - hypotension, sinus node dysfunction, T wave changes, prolonged QTc, myocarditis, dysrhythmias
        - Renal
            - Minor / persistent - polyuria, mild thirst
            - Chronic - nephrogenic diabetes insipidus, interstitial nephritis, distal tubular acidosis, acute renal failure (rare)
        - Endocrine
            - Minor / persistent - thyroid goiter, hypothyroidism
            - Chronic - thyroid goiter, hypothyroidism, hyperthyroidism, hypercalcemia, hyperglycemia, weight gain, sexual dysfunction
        - Hematologic
            - Minor / persistent - leukocytosis
            - Chronic - leukocytosis, aplastic anemia

        - Dermal
            - Chronic - psoriasis, angioedema, acne
    - Lab Tests:
        - Li level (normal range 0.6-1.2 mEq/L) low therapeutic to toxic ratio
        - 12 lead ECG
        - Lytes, BUN, Cr (note high lithium level may decrease or cause absent anion gap since it is unmeasured cation)
        - Red blood cell Li level correlates with neurotoxicity but not available in all hospital labs
    - Treatment:
        - Gastrointestinal decontamination should be performed for most acute ingestions
        - Gastric lavage if done in 1-2 hours may be efficacious
        - Activated charcoal does not absorb Lithium but should be administered for possible co-ingestants
        - Li is water soluble, poorly protein bound, small ion with small Vd, it can be eliminated by HD more rapidly than renal elimination
    - Treatment essentials of lithium toxicity
        - Gastrointestinal decontamination
            - Lavage with a large-bore tube (adults: 36–42 Fr; children: 24–32 Fr) for ingestions within 1 to 2 hours of presentation
            - Whole-bowel irrigation with PEG solution (adults: 500 mL–2 L/h; children 20 mL/kg/h) for ingestions of sustained-release preparations
        - General support
            - Intravenous isotonic crystalloid to correct dehydration and maintain urinary output of 1–2 mL/kg/h
            - Dopamine, 2 µg/kg/min, intravenously for enhanced diuresis (if necessary) after adequate hydration
        - Enhanced elimination
            - Hemodialysis
                - If presence of severe neurologic findings: altered mental status, ataxia, seizures, coma
                - If presence of elevated lithium levels and renal failure
                - If development of any symptoms in acute or acute-on-chronic overdose
            - CAVHD and CVVHD (can be considered in hemodynamically unstable patients)
    - Treatment Endpoints:
        - Cessation of neurologic symptoms (seizures, coma etc.)
        - Normal Li level
    - Monitoring / disposition:
        - ICU for all patients with severe neurologic effects
        - Asymptomatic patients with history of overdose and Li < 2 can be safely transferred to Psych after 24 h monitoring
    - Sequelae:
        - Neurotoxic effects after chronic poisoning last days to weeks and may result in permanent sequelae
        - Dysarthria, muscle rigidity, hyperreflexia, hypertonia and short term memory deficits have been described

REFERENCES

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- Goldfranks Toxicologic Emergencies
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- ATLS Manual
- Ford: Clinical Toxicology, 1st ed., 2001

DRUGS AND ANTIDOTE DOSAGES

Drug	Indication	Preparation	Dose (Intravenous unless otherwise stated)	
			Pediatric	Adult
N-Acetylcysteine (Mucomyst)	Acetaminophen	4, 10, 30, 100 mL vials of 20% solution	140 mg/kg PO loading dose, then 70 mg/kg q 4 hr	140 mg/kg PO loading dose, then 70 mg/kg q 4 hr
Adenosine (Adenocard)	Supraventricular tachydysrhythmias	3 mg/mL	50 µg/kg; can increase by 50 µg/kg to max of 250 µg/kg	6 mg, repeat 12 mg × 2 prn
Antivenin, Crotalidae Polyvalent	Crotalid envenomation	Vial lyophilized serum	Mild symptoms: 5–10 vials	Mild symptoms: 5–10 vials
			Moderate: 10–20 vials	Moderate: 10–20 vials
			Severe: 20–40+ vials	Severe: 20–40+ vials
At the time of this writing, the FDA is reviewing Savage Labs' application for Crotab, a Fab fragment antivenin. However, this agent is not currently FDA-approved.				

Antivenin, *Latrodectus mactans*	Black widow spider bite	6000 U vial	1 vial in 50-mL NS over 15 min	1 vial in 50 mL NS over 15 min
Antivenin, *Micrurus fulvius*	North American coral snake envenomation	Vial lyophilized serum	3–5 vials in 250–500 mL NS; may repeat prn	3–5 vials in 250–500 mL NS; may repeat prn
Atropine	Organophosphate poisoning	0.05, 0.2, 0.5, 1.0, 1.2 mg/mL	0.05 mg/kg q 5–10 min until excessive bronchial secretions terminate; can give continuous infusion of 0.02–0.08 mg/kg/hr	2–4 mg q 5–10 min until excessive bronchial secretions terminate; can give continuous infusion of 0.02–0.08 mg/kg/hr
	CPR, asystole, complete heart block		0.01–0.03 mg/kg q 2–5 min IV or ET (minimum dose 0.1 mg, maximum dose 1.0 mg)	0.5–1.0 mg q 2–5 min IV or ET; maximum dose 3 mg
	Bronchospasm		0.05 mg/kg with 2.5 mL NS nebulized (0.25–1.0 mg/dose)	2–3 mg in 2.5 mL NS nebulized
Bretylium	Ventricular tachydysrhythmias	50 mg/mL	Bolus 5 mg/kg initially, then 5–10 mg/kg boluses q 15–30 min (maximum 30 mg/kg); can infuse at 1–2 mg/min	Bolus 5 mg/kg initially, then 5–10 mg/kg boluses q 15–30 min (maximum 30 mg/kg); can infuse at 1–2 mg/min
Calcium chloride	Hyperkalemia, hypocalcemia	1 g/10 mL (13.6 mEq)	20 mg (0.2 mL)/kg/dose q 10 min; monitor serum calcium if multiple doses given	25–50 mg (2–5 mL)/kg/dose q 10 min prn; monitor serum calcium if multiple doses given
	Calcium channel blocker overdose		0.2–0.25 mg/kg/dose q 10 min until response seen	1 g (10 mL) q 10 min or infusion of 20–50 mg/kg/hr until response seen
Calcium gluconate	Hydrofluoric acid burns	1 g/10 mL (4.65 mEq)	*Topical:* 3.5 g in 5 oz of water-soluble jelly	*Topical:* 3.5 g in 5 oz of water-soluble jelly
			Subcutaneous/intradermal 0.5 mL injected per cm^2 of burned skin	*Subcutaneous/ intradermal:* 0.5 mL injected per cm^2 of burned skin
			Intra-arterial infusion: not studied	*Intra-arterial infusion:* 10 mL in 40 mL D_5W over 4 hr
	Hyperkalemia, hypocalcemia		50–125 mg (0.5–1.25 mL)/kg/dose; monitor serum calcium if multiple doses given	0.5–1.0 g (5–10 mL)/dose q 10 min prn; monitor serum calcium if multiple doses given
Charcoal, activated	GI decontamination; ineffective for metals, hydrocarbons, iron, lithium, caustics	12.5 g, 15 g, 25 g, 30 g, 50 g as powder or liquid, with or without sorbitol	1 g/kg PO or NG	1 g/kg PO or NG; maximum dose 100 g
Cyanide Antidote Kit	Cyanide	1. Amyl nitrite, 0.3 mL pearl	1. Crush pearl and inhale over 30 sec; then	1. Crush pearl and inhale over 30 sec; then
		2. Sodium nitrite, 300 mg/10 mL	2. See chart	2. 10 mL over 10 min; then
		3. Sodium thiosulfate, 12.5 g/50 mL	3. 1.65 mL/kg of 25% solution	3. 50 mL over 10 min

Deferoxamine mesylate (Desferal)	Iron toxicity	500-mg vial	15 mg/kg/hr; Duration:	15 mg/kg/hr; Duration:
			Mild-moderate toxicity: 6–12 hr, then reassess	*Mild-moderate toxicity:* 6–12 hr, then reassess
			Severe toxicity: 24 hr, 12 hr off, 12 hr on, then reassess	*Severe toxicity:* 24 hr, 12 hr off, 12 hr on, then reassess
Dextrose	Hypoglycemia, hyperkalemia	0.5 g/mL (D_{50})	*Neonates:* bolus 2 mL/kg $D_{10}W$, then infusion of 6–8 mg/kg/min	1–2 ampules of $D_{50}W$ (25 g); repeat prn
		0.25 g/mL (D_{25})	*Children:* bolus 0.5–1 g/kg/dose $D_{25}W$, repeat prn	
Diazepam	Status epilepticus	5 mg/mL	0.2–0.3 mg/kg q 2–5 min prn; maximum dose 10 mg. Monitor respiratory status	5–10 mg/dose q 2–5 min prn. Monitor respiratory status
Diazoxide (Hyperstat)	Hypoglycemia	15 mg/mL ampule, 50-mg capsule, 50 mg/mL oral suspension	*Neonates/infants:* 8–15 mg/kg/day PO divided q 8–12 hr	3–8 mg/kg/day PO divided q 8–12 hr
			Children: 3–8 mg/kg/day PO divided q 8–12 hr	
Digoxin-specific antibody fragments (Digibind)	Digoxin, digitoxin toxicity	38-mg vial	If digoxin dose/level unknown, or for plant source/toad venom: Pt <20 kg: 1 vial	If digoxin dose/level unknown, or for plant source/toad venom: 20 vials for acute toxicity; 6 vials for chronic toxicity
	Oleander, foxglove, cardiac genins (toad venom) toxicity			
Dimercaprol (BAL)	Lead	100 mg/mL	3–5 mg/kg IM q 4–12 hr until symptoms resolve	3–5 mg/kg IM q 4–12 hr until symptoms resolve
	Arsenic			
	Mercury			
Diphenhydramine hydrochloride (Benadryl)	Anaphylaxis	50 mg/mL ampule, 12.5 mg/5 mL elixir	1.25 mg/kg/dose IV/IM/PO q.i.d.; maximum dose 300 mg/day	25–50 mg IV/IM/PO q 4–8 hr; maximum dose 400 mg/day
	Dystonia associated with neuroleptic drugs	25-, 50-mg tablet, capsule		
DMSA, Succimer (Chemet)	Arsenic	100-mg capsule	10 mg/kg PO q 8 h × 5 days, then 10 mg/kg q 12 hr × 14 days; repeat prn after 2-wk drug hiatus	10 mg/kg PO q 8 hr × 5 days, then 10 mg/kg q 12 hr × 14 days; repeat prn after 2-wk drug hiatus
	Lead			
	Mercury			
Dobutamine	Cardiogenic shock	250 mg/vial	2.5–15 µg/kg/min; maximum of 40 µg/kg/min	2.5–15 µg/kg/min; maximum of 40 µg/kg/min
Dopamine	Shock	40, 80, 160 mg/mL	*Renal effects:* 1–5 µg/kg/min	*Renal effects:* 1–5 µg/kg/min

			Renal, beta-adrenergic effects: 5–15 µg/kg/min	*Renal, beta-adrenergic effects:* 5–15 µg/kg/min
			Alpha-adrenergic effects: >15 µg/kg/min	*Alpha-adrenergic effects:* >15 µg/kg/min
Edetate calcium disodium (EDTA)	Lead	200 mg/mL	50–75 mg/kg/24 hr IM/IV in 3–6 divided doses for 5 days; may repeat course after 2-day hiatus	50–75 mg/kg/24 hr IM/IV in 3–6 divided doses for 5 days; may repeat course after 2-day hiatus
Epinephrine	Cardiopulmonary arrest	1:10,000	0.1 mL/kg IV/ET q 5 min	10 mL (1 mg) IV/ET q 5 min
	Anaphylaxis	1:1000	0.01 mL/kg SC/IM; maximum of 0.5 mL/dose	0.2–0.5 mL SC/IM
	Asthma	1:1000	0.01 mL/kg SC q 20 min; maximum of 0.3 mL/dose; maximum total dose 1 mL	0.3 mL SC q 20 min; maximum total dose 1 mL
Ethanol (ethyl alcohol)	Ethylene glycol	95% ampule	1 mL/kg diluted to 10% over 1 hr, then 0.15 mL/kg/hr to target blood ethanol level 100–150 mg/dL	1 mL/kg diluted to 10% over 1 hr, then 0.15 mL/kg/hr to target blood ethanol level 100–150 mg/dL
	Methanol			
Flumazenil (Romazicon)	Benzodiazepines	0.1 mg/mL	0.01 mg/kg q 1 min to total of 1 mg	0.2 mg over 30 sec q 1 min to total of 5 mg
				Reversal of conscious sedation: 0.2 mg over 15 sec; repeat q 1 min to total of 1 mg
Fomepizole (4-MP) (Antizol)	Ethylene glycol	1 g/mL vials	*Initial dose:* 15 mg/kg	*Initial dose:* 15 mg/kg
	Methanol		*Next 4 doses:* 10 mg/kg	*Next 4 doses:* 10 mg/kg
			Subsequent doses: 15 mg/kg until ethylene glycol/methanol levels are below 20 mg/dL	*Subsequent doses:* 15 mg/kg until ethylene glycol/methanol levels are below 20 mg/dL
			Mix in 100 mL D_5W or NS; administer q 12 hr unless patient on hemodialysis	Mix in 100 mL D_5W or NS; administer q 12 hr unless patient on hemodialysis
			Hemodialysis: give q 4 hr	*Hemodialysis*: give q 4 hr
Glucagon	Beta-adrenergic receptor antagonists	1-mg, 10-mg vials	50–150 µg/kg bolus, then 50 µg/kg/hr infusion	3.5–5 mg (50–150 µg/kg) bolus, then 1–5 mg/hr infusion (higher infusion doses have been used)
	Calcium channel antagonists			
	Hypoglycemia		*Neonates:* 0.3 mg/kg/dose IV/IM/SC; maximum 1 mg	1 mg IV/IM/SC; may repeat in 20 min
			Children: 0.025–1 mg/kg/dose IV/IM/SC; maximum 1 mg/dose; may repeat in 20 min	
Ipecac Syrup	GI decontamination (prehospital)	30-mL bottles	10–15 mL PO	30 mL PO; rarely indicated
Leucovorin calcium (Wellcovorin)	Methanol	3 mg, 50 mg, 100 mg vials; 5-, 15-, 25-mg tablets	1–2 mg/kg q 4 hr × 24 hr up to 50 mg/dose	1–2 mg/kg q 4 hr × 24 hr up to 50 mg/dose
	Methotrexate		1–2 × methotrexate dose q 3–6 hr × 3 days or until methotrexate level is less than 1×10^{-8} mmol/L	1–2 × methotrexate dose q 3–6 hr × 3 days or until methotrexate level is less than 1×10^{-8} mmol/L
Lidocaine	Ventricular tachydysrhythmias	10, 20, 40, 200 mg/mL	1 mg/kg, then 20–50 µg/kg/min	1 mg/kg, then 1–4 mg/min
Magnesium citrate	Cathartic	300-mL bottle	Rarely indicated	150–300 mL PO
Magnesium sulfate	Hypomagnesemia	100, 125, 500 mg/mL	25–50 mg/kg q 4–6 hr; monitor serum levels to avoid hypermagnesemia	5 g in 1 L D_5W or D_5NS over 3 hr or 1 g IM q 6 hr; monitor serum levels to avoid hypermagnesemia
Methylene blue	Methemoglobinemia	10 mg/mL (1%)	1–2 mg/kg (0.1–0.2 mL/kg of 1% solution) over 5 min; may repeat to total of 7 mg/kg (0.7 mL/kg)	1–2 mg/kg (0.1–0.2 mL/kg of 1% solution)

				over 5 min; may repeat to total of 7 mg/kg (0.7 mL/kg)
Metoclopramide (Reglan)	Antiemetic to facilitate oral antidote administration	5 mg/mL vial	0.1 mg/kg given 30 min before oral antidote dose	1–2 mg/kg given 30 min before oral antidote dose
Midazolam (Versed)	Status epilepticus	1 mg/mL, 5 mg/mL	150 µg/kg initially, then 1–2 µg/kg/min; can increase by 1–2 µg/kg/min every 5–15 min until complete control of seizures	200 µg/kg initially, then 1–10 µg/kg/min until complete control of seizures
Naloxone hydrochloride (Narcan)	Opioid analgesics	0.02, 0.4, 1 mg/mL	*Neonate:* 0.01–0.03 mg/kg/dose	0.4–2.0 mg/dose
			Child: 0.4–2.0 mg/dose	
Octreotide acetate (Sandostatin)	Hypoglycemia	0.05, 0.1, 0.2, 0.5, 1 mg/mL	*Neonate:* 5 µg/kg/dose q 6–8 hr, up to 40 µg/kg/24 hr divided q 4–8 hr; or continuous SC infusion of 5–10 µg/kg/24 hr	50 µg SC q 12 hr
			Child: 1–10 µg/kg/24 hr divided q 12–24 hr IV/SC; maximum of 1500 µg/24 hr	
Ondansetron (Zofran)	Antiemetic to facilitate oral antidote administration	2 mg/mL	0.15 mg/kg/dose 30 min before oral antidote dose; maximum 8 mg/dose	0.15 mg/kg/dose 30 min before oral antidote dose; maximum 8 mg/dose
Physostigmine sulfate	Anticholinergic toxicity	1 mg/mL	0.02 mg/kg up to 2 mg over 5 min; maximum rate 0.5 mg/min	1–2 mg over 5 min
Polyethylene glycol–electrolyte solution (GoLYTELY, Colyte)	Whole-bowel irrigation for lithium, iron, metals, delayed-release drugs, body stuffers and packers	4 L container of powder	15–25 ml/kg/hr PO/NG	500–2000 mL/hr PO/NG; start at 500 mL/hr and increase rate as tolerated to 2000 mL/hr
Pralidoxime (2-PAM) (Protopam)	Organophosphates	1 g/20 mL	25–50 mg/kg initially, then infusion of 9–19 mg/kg/hr	1–2 g initially, then infusion of 500 mg/hr
Protamine sulfate	Heparin	10 mg/mL	1 mg/100 U of heparin given	1 mg/100 U of heparin given
Pyridoxine hydrochloride	Isoniazid	100 mg/mL	1 g per gram of isoniazid or 5 g if amount unknown; repeat prn	1 g per gram of isoniazid or 5 g if amount unknown; repeat prn
	Monomethylhydrazine-containing mushrooms		25 mg/kg, maximum 5 g; repeat prn	25 mg/kg, maximum 5 g; repeat prn
	Ethylene glycol		50 mg q 6 hr	50 mg q 6 hr
Sodium bicarbonate	Cyclic antidepressants	50 mEq/50 mL	1–2 mEq/kg bolus prn QRS interval > 0.13–0.14 sec, hypotension	1–2 mEq/kg bolus prn QRS interval > 0.13–0.14 sec, hypotension
Sodium polystyrene sulfonate (Kayexalate)	Hyperkalemia	3.75 g/tsp powder	1 g/kg/dose PO q 6 hr or as enema q 2–6 hr	15 g PO or 30–50 g as enema q 6 hr
		15 g/60 mL suspension		
Sorbitol	Cathartic	35% or 70% solution	4.3 mL/kg of 35% solution; however, is rarely indicated	1–2 mL/kg 70% solution PO
Thiamine	Ethylene glycol	100 mg/mL	10–25 mg IV/IM q day	100 mg IV/IM q day
	Thiamine deficiency		10–25 mg IV/IM q day	5–30 mg IV/IM t.i.d.
Verapamil (Isoptin)	Supraventricular tachydysrhythmias	2.5 mg/mL	0.1–0.2 mg/kg (max 5 mg), repeat q 10–30 min prn	5–10 mg, may repeat in 15–30 min
Vitamin K_1 (phytonadione)	Anticoagulant rodenticides, warfarin	2, 10 mg/mL	2.5–10 mg SC or PO; doses up to 100 mg/day in divided doses may be required for a prolonged time	5–10 mg SC or PO; doses up to 200 mg/day in divided doses may be required for a prolonged time
		5-mg tablet		

ET, endotracheal tube; NG, nasograstric; NS, 0.9% NaCl; PO, by mouth; SC, subcutaneous.