ACUTE INGESTIONS

GOALS
- WHY IMPORTANT?
- WHAT TO DO?
- WHAT IS GOING ON?
- DECONTAMINATION OR NOT?
- IS THERE AN ANTIDOTE?

WHY IS THIS IMPORTANT?
- 2-5 million overdoses a year in the US, 5-10% all ED visits, 55 adult ICU admissions
- 1-2% mortality rate in hospitalized patients

WHAT TO DO? CALL POISON CONTROL, BE SYSTEMATIC!!!
- ABC’s: airway protection, ACLS as needed
- Quick exam: vitals, mental status (up or down), pupils, cardiac monitor and ekg
- History: usually unreliable or unattainable, try to elicit type and time of ingestion, ask family, friends, witnesses and paramedics for information.
- PE: to assist you in identification of toxidrome
  - General: diaphoresis, lacrimation, salivation,
  - Pulm: bronchoconstriction, crackles
  - GI/GU: incontinence, diarrhea, urinary retention, bowel sounds,
  - Skin: dry, flushed, sweaty
  - MS: rigidity, myoclonus, choreoathetosis,
  - CNS: nystagmus, seizures, tremor, reflexes, clonus
- LABS/TESTS:
  - electrolytes, LFT’s, ABG, ASA and tylenol levels are most useful.
  - Urine toxicology rapid immunoassay screen for drugs of abuse (screen takes 1 hour, confirmation 6 hours) not too helpful in initial evaluation.
  - Serum send out screens for 100’s of drugs (takes 2 days, needs lab medicine resident approval), not helpful initial evaluation, however, may be instructive in unclear and complicated cases not fitting any of the below toxidromes.

WHAT IS GOING ON? TRY TO IDENTIFY THE TOXIDROME

<table>
<thead>
<tr>
<th>TOXIDROME</th>
<th>CLINICAL SYNDROME</th>
<th>AGENTS</th>
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<tbody>
<tr>
<td>Sympathomimetic</td>
<td>Agitation, paranoia, mydriasis, hyperthermia, tachycardia, hypertension, tachypnea, tremors, seizures, diaphoresis, seizures</td>
<td>Cocaine, amphetamines, ephedrine, phenylpropanolamine, theophylline, caffeine, beta-agonist, thyroid hormone</td>
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<td>Anticholinergic</td>
<td>Agitation, paranoia, delirium, hallucinations, coma, mydriasis, hyperthermia, tachycardia, tachypnea, hypertension, dry flushed skin, dry mucous membranes, decreased bowel sounds, urinary retention, myoclonus, choreoathetosis</td>
<td>Antihistamines, TCA’s, anti parkinson agents, phenothiazines, atropine, scopolamine, antispasmodics, cyclobenzaprine,</td>
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<td>Hallucinogenic</td>
<td>Agitation, hallucinations,</td>
<td>PCP, LSD, MDMA, marijuana</td>
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<tr>
<td>Condition</td>
<td>Symptoms</td>
<td>Causes</td>
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<td>Sedative/hypnotic</td>
<td>CNS depression, stupor, coma, miosis, hypothermia, bradycardia, hypotension, hypopnea, hyporeflexia</td>
<td>BDZ, barbiturates, alcohol, muscle relaxants, GHB,</td>
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<tr>
<td>Opiod</td>
<td>Lethargy, stupor, coma, miosis, hypotension, bradycardia, hypothermia, hypopnea, hyporeflexia</td>
<td>Heroin, opiates</td>
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<td>Cholinergic</td>
<td>Confusion, coma, miosis, bradycardia, hyper or hypotension, tachy or hypopnea, salivation, lacrimation, urinary and fecal incontinence, diarrhea, emesis, diaphoresis, bronchoconstriction, seizures</td>
<td>Organophosphates, nicotine, pilocarpine, pyridostigmine, bethanocol,</td>
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<td>Sympatholytic syndrome</td>
<td>Confusion, lethargy, bradycardia, hypotension, nausea, vomiting, AV block, prolong QT, wide QRS, sinus bradycardia, ventricular arrhythmia, seizures, ataxia</td>
<td>Alpha-blockers, beta-blockers, CCB, digitalis, TCA’s, antiarrhythmics.</td>
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<td>Serotonin syndrome</td>
<td>Agitation, confusion, coma, mydriasis, hyperthermia, tachycardia, BP lability tremor, myoclonus, hyper reflexia, clonus, flushing, diarrhea</td>
<td>Usually combination of MAOI’s, TCA’s, SSRI’s, ergots, reserpine, trazadone, buspar, effexor, atypical antipsychotics.</td>
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<td>Neuroleptic malignant syndrome</td>
<td>Agitation, confusion, coma, hyperthermia, BP labile, lead pipe rigidity, dyskinesia, dystonia, rhabdo, SIRS</td>
<td>Antipsychotics, atypical antipsychotics.</td>
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**DECONTAMINATION OR NOT?** May decrease absorption, may cause more harm than good, no good clinical evidence that outcomes are improved.

- Ipecac syrup: natural alkaloid, potent emetic agent, emesis eliminates poison
  - Evidence: NOT GOOD, experimental models reported variable recovery of ingested material, controlled volunteer studies reported 25 % decrease in absorption if administered within 30 minutes of ingestion, small clinical trials reported no improvement in patient outcome.
- Gastric lavage: mechanical cleansing of poison with about 5 liters of tap water or normal saline
  - Evidence: NOT GOOD, controlled volunteer studies reported 25 % decrease in absorption if done within 30 minutes, large clinical trials reported no improvement in clinical outcomes, associated with increased risks of aspiration and ICU admission.
- Activated charcoal: single dose of highly porous carbon, with high affinity for binding compounds with molecular weight of 100-100 Daltons, thus potential to bind poison, administer 25-50 grams in 120-240 ml of water.
  - Evidence: BETTER, BUT NOT GOOD, controlled volunteer studies reported 69 % decrease absorption if administered within 30 minutes, one clinical trial reported no improvement in clinical outcomes, rare toxicity. Common practice.
- Cathartics: osmotic retention of fluid in bowel stimulates motility and enhances expulsion.
  - Evidence: ABSOLUTELY NONE, associated with significant electrolyte abnormalities.
- Whole bowel irrigation: bowel purging with polyethylene glycol solution
• Evidence: NOT GOOD, three controlled human trials reported a 67% decrease in absorption when administered within 4 hours, no clinical trials. Case reports suggest efficacy in ingestions of SR and EC medications, lead, iron, arsenic, lithium, and foreign bodies.

IS THERE AN ANTIDOTE? Usually not
• Naloxone for opiate overdose: 0.2-4.0 mg IV or IM, titrate to effect, may precipitate withdrawal symptoms, don’t hesitate.
• Flumazenil for BDZ overdose: 0.2 mg IV, NTE 3 mg, do not administer to patients with chronic BDZ, or on stimulants or TCA’s because it will precipitate life threatening withdrawal and seizures.
• Glucagon for BB overdose: 5-10mg IV over 2 minutes, followed by infusion at 5/hr.
• Calcium gluconate for CCB & BB overdose: 1 amp IV bolus over 2 minutes.
• Digibond for digoxin overdose: 10-15 vials IV over 30 minutes or bolus during arrest, indicated with severe life threatening arrhythmias or hyperkalemia.
• Physostigmine for anticholinergic overdose: 1-2 mg slow IV infusion over 3-5 minutes, indicated use in patients with uncontrolled agitation or delirium, do NOT use in patients with any conduction abnormalities may precipitate asystole, watch closely for cholinergic symptoms.
• Atropine for cholinergic overdose; 2-4 mg IV, titrate as needed, until bronchial secretions dry up, no effect on mental status.
• Sodium bicarbonate for TCA overdose: 1-2 mg/kg IV bolus or infusion for widened QRS, maintain pH about 7.45.

NO ANTIDOTE, WHAT DO YOU DO?
• SUPPORTIVE THERAPY AS NEEDED, CLOSE OBSERVATION WITH MONITORING
• Forced elimination of poisons???
  • Multiple doses of activated charcoal: may aid to eliminate previously absorbed toxins that are secreted in the gut or bile, NO evidence, however case reports have seen increased elimination with ingestions of carbamezapine, dapsone, phenobarbital, theophylline, quinine.
  • Forced diuresis with alkalinization: ionize acidic poisons and speed up their transit time in the renal tubule, NO evidence that this is superior to alkalinization alone in salicylate toxicity.
  • Hemodialysis: reasonable for poisons that are small in size, long half-life, low protein bound, small volumes of distribution, indicated in severe poisoning from barbituates, chloral hydrate, ethanol, ethylene glycol, lithium, methanol, procainimide, theophylline, salicylates,

Burns, M. General approach to drug intoxication” Up to Date 2002.