

The Approach to the Poisoned Patient

by [Brit Long](#)

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This article will discuss the initial assessment, identification of toxidromes, and stabilization of patients suspected of toxic exposures. This discussion is by no means to be considered comprehensive, as Toxicology is a vast subject that cannot be quickly covered in depth. When in doubt, in the US, always call [1-800-222-1222](tel:1-800-222-1222) to speak to your regional poison control center and obtain directed advice.

General Approach

Most toxicologic exposures involve ingestions or localized chemical/biologic exposures involving single individuals with limited risk to medical personnel. In the uncommon event of an exposure which could be transmitted to emergency personnel, be sure to perform **decontamination FIRST and OUTSIDE of the Emergency Department** in order to not contaminate your personnel or life-saving equipment. At a minimum, the readily available PPE (cap, gown, gloves, mask, and eye protection) should be worn by all personnel in order to prevent accidental exposure.

As with all unstable patients, the initial assessment (once safe) begins with the ABCs. A detailed history and physical examination is key. Knowledge of medications, medical problems, and potential ingestions or exposures are very important historical facts in order to narrow down the list of potential toxic agents.

The physical exam should be comprehensive with special attention directed at finding evidence of a specific toxidrome. Also be **wary of anchoring on the diagnosis of toxin exposure**: don't forget to keep trauma, CNS infection, and the myriad of other causes of altered mental status on the differential.

Also don't forget the generally harmless "quick fix" medications that can rapidly reverse altered mental status in a previously comatose patient – naloxone and dextrose. Generally, patients will not be harmed with the indiscriminate provision of sugar and opioid reversal... though care should be taken in the chronic opioid abuser: lower doses are probably better such as 0.04-0.1mg IV at a time.

There are many, many reasons why drugs become toxic so remember that not all patients did something nefarious... many things affect drug clearance/protein binding/metabolism including underlying renal disease, hepatic dysfunction, dietary changes, iatrogenic, etc.

Initial orders

Now is not the time to be frugal. Full laboratory panels should be drawn, especially **electrolytes, serum osmolality, hepatic function, coags, urine beta HCG, aspirin, tylenol, ethanol**, and any other specific levels based on your history and physical exam.

An **EKG** is a must in all unknown toxic exposures, as interval derangements and electrocardiographic clues to underlying toxicities are very common. Imaging with a chest Xray can be helpful in assessing for pulmonary edema, pill fragments, or other radiopaque objects in the chest or abdomen.

GI Decontamination

There are several methods of GI decontamination for toxic ingestions; some are very useful/beneficial while others can be harmful.

Forced Emesis - Generally never indicated, as "natural" emesis is just as good as forcing expulsion of gastric contents.

Gastric Lavage

- Lavage with a large bore >36 French tube (not just NGT suction with a narrow tube) in order to empty the stomach of toxic contents. Awake patients should be lavaged in left lateral decubitus position to prevent aspiration and facilitate more complete gastric emptying.
- Controversial but thought to be potentially helpful if performed within 4 hours of ingestion; preferred if initiated within one hour of ingestion.
- **Indicated if the airway is protected, removal of toxin is feasible (within a reasonable time frame), and will be beneficial if even a small amount is removed.**
- Contraindicated for caustic ingestions, large contents unlikely to be removed by lavage, unprotected airway, or timeframe when toxin has probably moved out of the stomach.

Activated Charcoal (AC)

- Binds toxins; **not indicated for caustics, heavy metals such as lithium, lead, zinc and iron, toxic alcohols, hydrocarbons, and small molecules like sodium, chloride, etc.**
- Adult dose for unknown exposure is 60 - 90 grams, kids 1g/kg; best if can obtain a ratio of 10:1 of AC:toxin.
- No clear timeframe for AC: definite benefit within one hour, suggested benefit within 4 hours; generally no harm in giving for any timeframe if no contraindications exist especially for large ingestions or sustained release preparations.
- Contraindicated if absence of gut motility, perforation, risk of aspiration or if endoscopy will be needed; aspirated AC can cause severe pneumonitis.

Whole Bowel Irrigation (WBI)

- Instillation of up to 2L per hour (25mL/kg/h for children) of polyethylene glycol solution orally (or via NGT) **until the rectal effluent is clear.**
- Can be used concurrently with AC but may actually compete with toxin for binding sites on AC.
- **Especially useful for body packers and stuffers.**

Caustic Ingestions

Acids – Proton donators, cause injury with pH < 3, hydrogen ions desiccate mucosal cells and cause development of an eschar (**coagulative necrosis**) that prevents deep penetration.

- Can lead to metabolic acidosis with systemic absorption of acids.
- Toilet bowl cleaners, hydrofluoric acid, etc.

Alkalis – Proton acceptors, cause injury with pH > 11, hydroxide ions penetrate tissue surfaces and cause **liquefactive necrosis** until neutralized. Extent of injury is dependent on duration of contact, volume, pH, concentration, penetrating ability of the substance and the TAR (titratable acid or alkaline reserve) – basically the amount of neutralizing substance required to bring the substance to physiologic pH, the higher the TAR the more damaging the substance.

- Most household cleaning agents are alkali – ammonium hydroxide (Windex), sodium hypochlorite (bleach), oven cleaners, Drano, detergents etc.

Initial symptoms can be misleading: all patients with stridor or oral lesions require early EGD (within 12-24 hours) in order to accurately diagnose the extent of injury and decrease the risk of iatrogenic perforation.

- Combination of multiple symptoms such as drooling, emesis, and chest pain will also likely have high-grade lesions and will need early EGD.
- No visible lesions does not mean there is not a high-grade lesion in the esophagus or lower: clinical history and physical exam should guide further investigation.

Initial management should be for decontamination of the patient's skin and oropharynx as necessary, aggressive control of the airway by direct visualization (consider fiberoptics), and caution with paralytics in severe burns as this may distort airway anatomy with loss of muscular tone.

- Consider IV decadron for airway edema.

Gastric decontamination is generally contraindicated unless very early presentation of large volume toxic exposure or with certain high-risk substances as guided by poison control.

- Can consider NGT suction for the above if present within 30 minutes; after that there is a very high risk of iatrogenic perforation with NGT so placement should be under direct visualization with EGD.
- Initial dilution of liquid caustic ingestions with milk or water may be beneficial but should be discussed with poison control first.

Most patients will require EGD for diagnosis. In the rare patient with late presentation and suspicion of perforation, esophagogram and CT of chest/abdomen are indicated. All high-grade lesions/perforations will require **surgical consultation**.

All button batteries lodged in the esophagus require emergent endoscopic removal to prevent perforation

Body Packers and Stuffers

Body stuffer - Spontaneous ingestion of poorly packaged drugs, for instance swallowing a bag of contraband just prior to arrest.

- Likely will not require whole bowel irrigation, usually admitted for observation for 24 hours though some suggest 6 hour observation period and then discharge if no evidence of toxidrome.

Body packer - "Drug mule," a carefully planned ingestion of presumably carefully packaged illicit drugs.

- If asymptomatic but known packer, can CT scan to quantify packets, or just give AC and WBI until several clear stools without packets.
- If second CT scan at this point is negative, then they are clear (if they remain asymptomatic).
- If known or suspected cocaine packing and the patient is symptomatic, i.e. sympathomimetic toxidrome; highly likely one of the packets has burst or is leaking, which is an indication for emergent surgery.

General considerations for hemodialysis

- Toxin must be very **small particles** (able to cross the membrane).
- Toxin will produce harm if not removed.
- Volume of distribution should be **small** (1L/kg) indicating most of the toxin is in serum.
- Toxin should **not be highly protein bound** (some notable exceptions include aspirin and valproic acid which are almost entirely protein bound at therapeutic levels, but at toxic levels they saturate protein binding sites and the remainder is in serum and therefore dialyzable).
- Toxin is unable to be cleared by the body (renal failure, hepatic failure, etc.).

Toxidromes

Anticholinergic - The old mnemonic rules supreme here - blind as a bat (**mydriasis**), mad as a hatter (**altered mental status**), hot as Hades (**hyperthermic**), red as a beet (**flushing**), dry as a bone (**no sweating**), the bowel and bladder increase their tone (**urinary retention, decreased bowel sounds**), and the heart runs alone (**tachycardia**).

- Commonly described symptoms
 - Lilliputian hallucinations (picking at unseen small objects on the body), "pleasantly altered."
 - Synesthesia - crossed sensory stimuli such as "I can taste the music."
- Common offending agents (many)
 - Over-the-counter medicines such as antihistamines
 - Synthetic cannabinoids like spice
 - Antipsychotics, antidepressants, antiparkinsonian drugs, antiemetics (phenothiazines), muscle relaxants (cyclobenzaprine)
- *Differentiate from sympathomimetic toxidrome by:*
 - **Dry skin**
 - **Mydriasis with limited or absent pupillary response to light**

- In anticholinergic toxidrome there is inhibition of cholinergic input to the ciliary apparatus of the eye; therefore, pupillary response to light will be limited or absent, whereas the opposite is true in the sympathomimetic toxidrome.

***For treatment myths and pearls, please see prior

post: <http://www.emdocs.net/physostigmine-for-management-of-anticholinergic-toxidrome/>***

Sympathomimetic

Agents – Cocaine, MDMA (ecstasy), ephedrine, methamphetamine, khat, etc.

Toxidrome – Hypertension, tachycardia, diaphoresis, mydriasis, hyperthermia, CNS excitation and delirium.

- Differentiate from anticholinergic toxidrome by **diaphoresis and mydriasis with brisk pupillary response.**

Treatment – Benzodiazepines are the mainstay of treatment in the patient suspected of a sympathomimetic ingestion/toxicity. Benzodiazepines restore inhibitory balance to the CNS to help prevent the tremendous sympathetic outflow stimulated by these agents. Life-threatening hyperthermia may also occur; **aggressive cooling measures and benzodiazepine** administration are keys to early treatment.

- For refractory hypertension, consider phentolamine (pure alpha blocker).
- Be wary of mixed alpha/beta antagonist drugs such as labetalol as the alpha:beta ratio is very much in favor of beta blockade, approx. 1:7 ratio. Efficient beta blockade of beta-2 receptors will worsen vasoconstriction, causing nearly unopposed alpha-agonism by the original toxic agent leading to worsening hypertension.
- Beware dysrhythmias: SVT is common and sodium channel blockade often leads to wide complex tachycardia that may degenerate into non-perfusing rhythms.
 - SVT unresponsive to benzodiazepines and cooling can be treated with calcium channel blockade.
 - Wide complex tachyarrhythmia, especially in cocaine toxicity, should be treated with empiric bicarbonate bolus and ACLS measures.

Opioids

Agents: Long/short acting opioids, heroin, methadone, buprenorphine, etc.

Toxidrome – Drowsy, hypoventilation, hypotension, apnea, miosis, decreased bowel sounds.

Treatment – Largely supportive (airway support, fluids, vasopressors), if acute overdose can use naloxone in higher doses (0.4-2mg IV).

- Caution in the chronic opioid dependent patient or opioid abuser as may precipitate withdrawal, also the patient will become agitated and combative if completely reversed immediately so should start with lower doses of repeat aliquots of 0.04-0.2mg IV.
- Generally the goal is to find the amount required to reverse the respiratory depression and allow spontaneous respiration: the total dose given to reach this goal should be multiplied by 2/3, and this amount given as a drip per hour. Obviously the patient needs to be monitored but in a strict opioid overdose without other factors, reversal of respiratory depression is the most important step.

Sedative/Hypnotic

Agents: Barbiturates, benzodiazepines, alcohol, GHB, sleep aids, zolpidem, buspirone.

Toxidrome - Drowsy, slurred speech, nystagmus, hypotension, ataxia, coma, respiratory depression.

Treatment is supportive, intubation as necessary for airway control, fluids/vasopressors for hypotension. Few specific antidotes, flumazenil is antidote for benzodiazepine overdose but should almost never be used... unless it is a known iatrogenic overdose of a pure benzodiazepine without any possible stimulant medication in a person that is not a chronic user of benzodiazepines nor has a history of seizures. Otherwise, may cause seizures refractory to benzodiazepine administration.

Please see <http://www.emdocs.net/wp-content/uploads/2014/10/Flumazenil-Bodford-.pdf> for further details.

Cholinergic

Agents – Organophosphate and carbamate pesticides, nerve agents; mechanism is poisoning of acetyl cholinesterase at ganglionic and neuromuscular junctions leading to increased acetylcholine neurotransmitter stimulation, with both muscarinic and nicotinic receptor stimulation effects.

Toxidrome – DUMB BELLS (**D**iarrrhea/**D**iaphoresis, **U**rination, **M**iosis, **B**radycardia, **B**ronchorrhea, **E**mesis, **L**acrimation, **L**ow BP, **S**alivation).

- Killer B's (from muscarinic stimulation) – **Bradycardia, Bronchorrhea, Bronchospasm.**
- Will also get nicotinic stimulation effects such as **fasciculations, tetany, paralysis** and increased sympathetic ganglionic stimulation which may result in paradoxical **tachycardia and hypertension early.**
- Seizures are common in overdose.

Treatment – These patients will commonly need **prehospital decontamination**, DO NOT bring into the ED until they have been adequately decontaminated. The most common cause of death is **airway compromise** so early securing of the airway is paramount.

- **Atropine in high doses of 2-4mg IV at a time, keep giving until oral secretions are dry.**
- Pralidoxime (2-PAM) in order to reverse acetyl cholinesterase inhibition. This must be given early before enzyme “aging.”
- Benzodiazepines for seizures and agitation.

References / Further Reading

- Rosen's Emergency Medicine - Concepts and Clinical Practice. 8th Edition.
- Goldfrank's Toxicologic Emergencies 2002.
- An intensive review course in clinical toxicology. New York City Poison Control Center and Bellevue Hospital Center Course Syllabus March 13 and 14; 2014.