

# Approach to Poisonings and Ingestions

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## Objectives

Upon completion of this self-study module, you should be able to:

1. List the key elements of history and physical exam in an overdose patient
2. Explain the uses and limits of laboratory testing in the setting of an overdose
3. Recall the types of decontamination used in poisonings and specific contraindications for their use
4. Recognize the classic toxidromes including anticholinergic, sympathomimetic, cholinergic, and opioid poisoning

## Case Study

A 19-year-old college student is brought in to the emergency department via ambulance after her friends found her confused and appear intoxicated. Her friend tells you that the patient was in a fight with her girlfriend and that she was really upset. They found an open bottle of prescription pills but they are unsure of how many she took or what they are. Her vital signs are: BP = 110/80, P = 140, RR = 38, T = 98.7F, O<sub>2</sub> sat = 98% on room air. On exam she is responsive to noxious stimuli but does not open her eyes spontaneously. Moves all extremities but is unable to cooperate with your exam. She has dry mucous membranes. Pupils are dilated and sluggishly reactive. Heart is tachycardic but regular.

## Introductions

An estimated 5 million accidental and intentional poisonings that occur in the US each year, though the true incidence is unknown due to under diagnosis and underreporting.

Patients who have been poisoned may present with a wide array of symptoms or complaints. Presentations may include the asymptomatic patient claiming to have taken an overdose to patients with altered mental status or patients who are hemodynamically unstable or apneic. Additionally, poisoned patients may present in stable condition and rapidly deteriorate. Many of

the clinical courses are dependant upon the ingested substance. The purpose of this article is to provide a general approach to poisonings.

## Initial Actions

As you're walking toward her room, what actions should you take upon arriving?

- Assess the primary survey
- Place patient on a monitor and obtain IV access
- Order basic laboratory tests and ECG
- Consider an order for a safety companion/sitter and/or standard suicide precautions, if appropriate

## Primary Survey

As with all patients, the initial survey begins with the ABC's and DE's. If the patient is unstable, then the history and physical must be performed while simultaneously performing resuscitation. It is important to get collateral history if available from family, friends, and/or EMS personnel or those at the scene. An AMPLE history, while performing the Primary Survey, includes Allergies, Medications (including herbals, alternatives, 'internet' and over-the-counter meds), Past Medical History, Last Meal and Events leading up to presentation.

Depending on the ingestion, a poisoned patient may present anywhere on the spectrum from completely awake to profoundly obtunded. An unresponsive patient has lost their **Airway** reflexes and is at risk for airway obstruction as well as aspiration. Faced with an unresponsive patient with a history of overdose, a quick run-through of a few reversible possibilities while preparing to control the patient's airway is crucial. These reversible causes have come to be known as the "coma cocktail." In this setting, the following should be considered:

- Hypoxia: Place on 100% O<sub>2</sub> non-rebreather (also useful prior to intubation)
- Hypoglycemia: obtain a point of care fingerstick blood glucose, IV D50 if less than 60
- Opioids: administer Naloxone 0.4 to 2mg IV to reverse opioids . Patients with difficult IV access may also have Naloxone given by intramuscular (IM), subcutaneous (SC), endotracheal, intralingual, submental and nasal routes.
- With an unresponsive patient, if these measures do not reverse the patient's profound compromised mental status, proceed with intubation to control the **Airway**.
- Once the airway is secured, attention may turn to **Breathing**. Many toxins can affect the respiratory status and cause a variety of symptoms including frank respiratory failure, hypoxia, flash pulmonary edema, and/or bronchospasm. These should all be treated with standard therapies and, in some cases, specific antidotes.
- **Circulation** may be compromised as well. Blood pressure, peripheral pulses, skin assessment with capillary refill, color and presence or absence of diaphoresis are all elements of assessing circulation and may be addressed with IV fluid bolus if compromised.

A multitude of toxins can affect hemodynamics including heart rate and blood pressure (hyper or hypotension) as well as cardiac rhythm and intervals. Each of these symptoms can give a clue as to which toxin may have been ingested. Agents such as sympathomimetics and anticholinergics induce tachycardia, while agents such as beta blockers, calcium channel blockers, digoxin and clonidine often cause bradycardia; drugs such as tricyclic antidepressants (TCAs) can cause QRS widening, and many cause QT prolongation leading to lethal arrhythmia.

Use Glasgow Coma Score (GCS) or Alert Verbal Painful Unresponsive (AVPU) scale for a quick assessment of level of consciousness and neurologic **Disability**: (A=alert, V=responds to verbal stimuli, P=responds to painful stimuli, U=unresponsive).

Finally, **Exposure** includes determination of any other injuries, sources of ongoing exposure – including clothing which may be saturated with the toxin, or other potentially dangerous delivery devices such as needles or pipes.

If the patient is completely asymptomatic, there is time to obtain a more detailed history and perform a complete physical exam.

## Secondary Survey

### Key Historical Data

In all cases, these are some of the highest yield questions to ask:

- What was ingested? What else might have been ingested?
- How much was ingested? How was it ingested (injected, inhaled, snorted, swallowed)?
- When was the ingestion and over how long?
- Why? (accidental or intentional)?

To answer these questions, the physician can ask the patient, family, friends and EMS. Pill bottles can be of great help if available, understanding that reliable and accurate information may be difficult to obtain. The patient may purposely try to mislead the physician, or the patient may accidentally mislabel the agent ingested. For instance, some laypeople call any over-the-counter pain reliever by a single name regardless of actual name and drug class. When the circumstances of the overdose are not clear, intentional overdose should be suspected and suicide precautions instituted until more information is available.

### Key Elements of Physical Exam

On the physical exam, there are several things to pay close attention to in addition to the standard exam:

- Vital Signs
- Mental status (agitated, confused, or somnolent?)
- Pupils (fully dilated or pinpoint?)
- Skin color (Pale or flushed?)

- Track marks (evidence of skin popping?)
- Presence of sweat (dry or diaphoretic? The toxicologic handshake refers to checking a patient's armpit for degree or lack of sweat)
- Bladder size (Use point of care ultrasonography to assess for urinary retention. A full bladder may explain the patient's level of agitation)

Some poisonings cause tachycardia (sympathomimetics, anticholinergics), bradycardia, apnea, hypotension, etc. Some toxins also cause mydriasis or miosis, flushed skin, sweating, diaphoresis, or lack of sweat when expected. These signs offer clues to the diagnosis. Occasionally, there will be mixed findings if the intoxication involves more than one class of these toxins.

Though she initially appeared intoxicated, she deteriorates during your exam. She becomes unresponsive to painful stimuli. You immediately proceeded to control her airway, confirm tube placement, and finish your exam. Her pupils are 5mm and reactive. Her skin is not flushed. She is tachycardic with regular beats, and the rest of her exam is relatively unremarkable. *There are no signs of trauma.*

## Toxidromes

While looking for clues to aide in the diagnosis, there are a few classic “doorway diagnoses” where “toxicologic syndromes” are easily recognizable. These classic presentations are called Toxidromes (see below).

### Anticholinergic

A young college student is brought in by his friends after being found confused at a party. His friends tell you that he was just dumped by his girlfriend. His vital signs are as follows: HR 122, RR rate 18, BP 120/80, Temp 100.8F, O2 Sat 98% on room air. He is mumbling and picking at his clothes. On exam, his pupils are 8mm and his skin is flushed, although he is not sweating. You noted his bladder is full.

This is the classic anticholinergic syndrome:

- Mad as a hatter (Altered mental status)
- Blind as a bat (mydriasis/dilated pupils)
- Hot as a hare (or hell or Hades)
- Red as a beat
- Dry as a bone

Patients with an anticholinergic toxidrome may present with some or all of these findings. Possible toxins with anticholinergic properties include the following:

- TCA
- Antihistamines
- Overactive bladder medication

Treatment is mostly supportive. Please see specific therapies for TCA ingestions

## Cholinergic

A migrant worker is found wandering on a deserted road. She is confused, sweating and wheezing. You notice that she has been incontinent. Her vitals are as follows: HR 36, RR rate 24, BP 100/68, Temp 98F, O2 Sat 96% on room air.

This is the classic mnemonic SLUDGE:

- Salivation
- Lacrimation
- Urination
- Diaphoresis and defecation
- Gastrointestinal upset
- Excessive bradycardia or tachycardia (muscarinic or nicotinic)

Another mnemonic is DUMBELLS:

- Diarrhea
- Urination
- Miosis/Muscle weakness
- Bronchorrhea
- Bradycardia
- Emesis
- Lacrimation
- Salivation/Sweating

Sources of cholinergic poisoning include organophosphate poisoning (pesticides) and nerve agents

Treatment: Atropine, pralidoxime, decontaminate

## Sympathomimetic

A young college student is brought in by EMS after becoming combative at a concert. He is very agitated with an altered mental status and requires restraints. His vitals are as follows: HR 138, RR 24, BP 154/92, Temp 101.2, O2 Sat 98% on room air. Physical Exam reveals mydriasis, flushed skin, sweating and agitation.

This is the classic sympathomimetic syndrome with a fight or flight response:

- Tachycardia
- Hypertension
- Mydriasis
- Diaphoresis

- Hyperthermia
- Agitation

Sources include nonprescription sympathomimetic agents including the over-the-counter cold agents (containing ephedrine), street drugs (e.g., cocaine, amphetamines, methamphetamine), dietary supplements (ephedra), and illicit designer drugs (e.g., 3,4-methylenedioxy methamphetamine also known as MDMA or “ecstasy”)

Treatment involves sedation, hydration, and treatment of complications such as rhabdomyolysis and hyperthermia.

## Opioid

Friends bring in a young high school student who is apneic and unresponsive. They tell you they were just ‘partying a little’ and their friend collapsed. Her vitals are as follows: HR 128, RR 4, BP 100/70, Temp 98F and O2 Sat 82% on room air. On physical exam, she is unresponsive with pinpoint pupils.

Classic signs:

- Apnea
- Hypoxia
- Miosis
- Unresponsiveness
- Flash pulmonary edema (rare)

Treatment: These patients are usually apneic and may appear to require intubation. Administration of Naloxone can reverse the apnea and obviate the need for intubation.

**A word on Naloxone.** The Naloxone may wear off before the opioid depending on type (e.g. methadone or extended release formulations) so the patient must NOT be discharged without a period of observation. Administration of Naloxone may cause the patient to go into opioid withdrawal, so be prepared for a very violent and combative response and use only the dose necessary to return a normal respiratory rate.

## Diagnostic Testing

Toxicology is one of the last areas of medicine where a keen mind and good diagnostic skills are still imperative without the benefit of advanced testing and technology to provide answers.

Nonetheless, there are still important tests to perform:

- EKG: may show changes in QRS or QT intervals, elevated terminal R wave or the last 40 msec in lead aVR, dysrhythmias, or other findings that can help diagnose the toxin and help with treatment.
- Urine Drug Screen (UDS): often of limited value in the acute setting. It is often not quantitative and usually only tests for a few toxins. Common drugs tested include

amphetamines, barbiturates, benzodiazepines, cocaine, opiates, phencyclidine (PCP), cannabis or tetrahydrocannabinol (THC), and methadone metabolite.

- Ethanol levels: may provide clues on the patient's mental status. It is important to highlight that ethanol levels and toxicology screens should be taken with a grain of salt. Vigilance to other causes of alterations in mental status must be ruled out, especially in the setting of poisoning.
- Acetaminophen level: One of the most important substances to screen for is an acetaminophen level. Unlike other toxic ingestions, acute acetaminophen overdose can initially present without symptoms and can be easily missed if not specifically tested.
- Salicylate level: One of the more common overdoses historically, many preparations contain significant salicylate concentration and lead to accidental toxicity. In addition, patients often do not view salicylates as potentially toxic, since they are readily available as over-the-counter preparations.
- Electrolytes: Many toxins will produce abnormalities in electrolytes
- Drug Levels (specific to medication)
- Other levels that provide surrogate markers for toxicity depending on situation: ammonia levels in valproic acid toxicity, lactic acidosis in setting of volatile ingestions and metformin overdose

Anion Gap Metabolic Acidosis: There are several causes of an anion gap metabolic acidosis, and many of them involve poisonings. The mnemonic **CAT-MUDPILES** is a helpful list of differential diagnosis for high anion gap metabolic acidosis:

- Carbon monoxide/Cyanide
- Aminoglycosides
- Theophylline/Toluene (glue sniffing)
- Methanol
- Uremia
- DKA or AKA
- Paraldehyde
- Iron, Isoniazid
- Lactate (many causes including carbon monoxide, cyanide, metformin, etc)
- Ethylene Glycol
- Salicylates

## So How Do I Make the Diagnosis

With suspected overdoses in the emergency department, there are rarely perfect tests to provide absolute answers. Also, some drugs remain in the urine after a week and may not be a reliable source of the true cause of a toxidrome. Always obtain an acetaminophen level and other levels as well depending specific drug levels as dictated by history. There are certain poisonings where the presence of an anion gap metabolic acidosis is key to helping diagnose the cause (see **CAT-MUDPILES** above).

Focus on key elements of history – if possible, obtain what agent was ingested, when this occurred, and how much was ingested and over how long. Management changes for example when an entire bottle of acetaminophen is ingested in one sitting vs. over the course of the day.

Look for clues in the physical exam – look at vital sign derangements, pupil size, skin color and moisture, and overall mental status.

Always check for pregnancy test in women of child bearing age.

Use accessory data for collateral information. Though it may not reveal the answer, use the toxicology screen as well as electrolytes to aide in the diagnostic workup.

Be vigilant.

If the patient is asymptomatic for 4 – 6 hours and the work up is negative, they may not have taken a toxic level of ingestion. If the patient has findings, the provider must use all available information gathered to arrive at the most likely diagnosis and disposition the patient appropriately.

She does not appear to have a clear toxidrome.

Her EKG shows sinus tachycardia with normal intervals.

You send a full set of labs including an ABG, CBC, complete metabolic panel, and a tox screen including ethanol level, acetaminophen level and salicylate. You also remembered to check her pregnancy status.

## Treatment

As with all patients, treatment begins with the ABC's. The treatment of poisoned patients includes removing them from the toxin and decontaminating them when possible.

### Decontamination Methods

- Activated Charcoal
- Whole Bowel Irrigation
- Gastric Lavage (rare)

Activated Charcoal is given orally to absorb toxins that are present in the GI tract. It is most efficacious if given within the first four hours post ingestion but may still work beyond that point. Consult with your local toxicologist for guidance. Toxins bind to the charcoal and are excreted without being digested. Charcoal does not bind metals (such as iron), alcohols or hydrocarbons. It should be avoided in patients with somnolence as they run the risk of aspiration.

Whole bowel irrigation involves the administration of an osmotically balanced polyethylene glycol electrolyte solution (like Go Lytely) to flush the bowel to prevent the absorption of

ingested toxins. It is used in cases where charcoal is not effective, with certain sustained release products, and in cases of street drug packet ingestions (body packers).

Gastric lavage is rarely used and carries significant risks with questionable benefit. In some cases, however, such as recently ingest lethal doses or an intubated overdose following a known recent ingestion, the benefits may outweigh the risks and warrant consideration. Lavage involves the application of a very large bore (36 – 40 French) orogastric tube and then flushing the stomach with aliquots of water ideally to obtain pill fragments.

Note: Many years ago, there was an agent called syrup of Ipecac that was universally promoted as a decontamination method. This agent *should no longer* be used. It is not effective in removing ingested toxins and has side effects that may cause lethargy further delaying the administration and/or reducing the effectiveness of other superior decontamination methods and treatments.

## Disposition

Many patients with potential ingestions may be observed for six hours and then dispositioned home or to a psychiatric treatment facility if clinically asymptomatic, provided the ingestion is not an extended release preparation. It is important to contact the state Poison Control Center with every overdose or toxic exposure. They may provide clinical assistance, but they also record toxic exposures and maintain a database for public health concerns. For example, when children's acetaminophen was first marketed, it had the taste and appearance of candy – hence many accidental toxic ingestions occurred. When Poison Control Centers noted this spike in childhood exposures, it led to legislative requirements for preparations to be sold in packaging that was difficult for small children to access, as well as being sold in smaller, non-toxic amounts.

## Other Specific Poisonings

### Acetaminophen

Acetaminophen (APAP) overdose is one of the most common and dangerous poisonings in the US. Acetaminophen is available in many formulations. Although an acute overdose may cause symptoms, patients may present completely asymptomatic even after a lethal ingestion. Therefore, it is imperative that an acetaminophen level is checked on ALL overdose patients as treatment is readily available and potentially lifesaving. There are four main stages of an acute APAP overdose. Typical symptoms usually involve nausea, vomiting in the first two stages. The lethal dose of APAP is 150mg/kg. In an acute overdose, APAP is metabolized to NAPQI which combines with glutathione and is excreted. When liver stores of glutathione are exhausted, NAPQI causes hepatic toxicity. The toxic level of acetaminophen can be measured on the Rumack-Matthew nomogram and the toxic plasma level at four hours is 150. In addition to decontamination with repeated doses of activated charcoal, the antidote N-acetylcysteine (NAC/Mucomyst) should be administered if indicated by the nomogram. Note that the Rumack-Matthew nomogram is for acute ingestion. Ingestions over hours or unclear point of

ingestion makes the Rumack nomogram less helpful. Concomitant screening of liver function tests may aid in your decision for initiating and completing the NAC treatment.

### Aspirin/Salicylates

Unlike the Rumack-Matthew nomogram for acetaminophen, the Done nomogram associated with aspirin ingestions is typically not used to determine toxicity and treatment. Patients with an acute overdose of aspirin are usually quite ill-appearing; they may be tachypneic (secondary to metabolic acidosis), vomiting, confused, and sometimes febrile. The toxic effects are complex and involve an uncoupling of oxidative phosphorylation. This causes a profoundly elevated anion gap metabolic acidosis. The general approach to aspirin overdose is the management of the airway, gastric decontamination, the administration of sodium bicarbonate, and sometimes hemodialysis.

### Tricyclic Antidepressants (TCA)

TCAs have historically been some of the most dangerous agents ingested in an overdose situation. In addition to their anticholinergic properties, TCAs cause a direct alpha-adrenergic blockade, inhibition of norepinephrine and serotonin reuptake, and blockade of fast sodium channels in myocardial cells. This can lead to tachycardia, prolongation of the QRS complex, dysrhythmias, and cardiovascular collapse. They may also induce protracted seizures.

Disposition of TCA overdose includes close monitoring for a period of at least six to eight hours in the asymptomatic patient. In patients with altered mental status and a potential TCA overdose who are tachycardic and shows signs of widening of the QRS (over 100msec), administration of sodium bicarbonate provides both a diagnostic and a therapeutic benefit. Evaluation of the QRS immediately after IV sodium bicarbonate will typically narrow QRS complex. The height of the terminal R wave on lead aVR, however, will not be affected, and is present in patients taking therapeutic doses of TCA. It can also be a normal variant. Seizures may be treated with benzodiazepines but may be intractable until administration of sodium bicarbonate. Newer recommendations for lipid emulsion therapy exist in the treatment of severe toxicity.

### Alcohols

While any alcohol consumed in great quantities can be dangerous, there are three major alcohols that are considered “toxic”. These “toxic” alcohols include isopropanol, methanol, and ethylene glycol. Isopropyl alcohol is found in many solvents, mouthwashes, and rubbing alcohols. Methanol is found in windshield wiper fluid. Ethylene Glycol is typically found in antifreeze. Patients who have ingested any of these agents may appear to be intoxicated or even comatose. It is important to obtain a metabolic panel on these patients. Isopropanol will NOT cause significant metabolic acidosis, while methanol and ethylene glycol both cause a profoundly elevated anion gap metabolic acidosis. Additional diagnostic measures may include the application of a wood’s lamp to the urine of a patient with a suspected ethylene glycol ingestion; commercial antifreeze has an additive that will fluoresce. The urine can also be examined for the presence of calcium oxalate crystals. Osmolal gap will help clue in other volatile ingestions.

Isopropanol is usually not life threatening and can be managed with supportive care. In rare instances hemodialysis may be required. Methanol and ethylene glycol, on the other hand, are more lethal and should be aggressively treated as soon as suspected. Methanol is metabolized to formaldehyde, and ethylene glycol is broken down into oxalate. All alcohols are metabolized by alcohol dehydrogenase (ADH). Therefore, the initial treatment for methanol and ethylene glycol involves the blockade of ADH. This can be accomplished by either simple ethanol or fomepizole. In addition, removal of the toxin may be necessary by hemodialysis. Sodium bicarbonate and glucose may also be necessary. For chronic ethanol drinkers, consider high dose thiamine as this may be the cause of their elevated lactic acidosis.