EMERGENCY MEDICINE PRACTICE

AN EVIDENCE-BASED APPROACH TO EMERGENCY MEDICINE

Toxicology Update: A Rational Approach To Managing The Poisoned Patient

"What is it that is not a poison?
All things are poison and nothing is without poison.
It is the dose only that makes a thing not a poison."
—Paracelsus (1493-1541),
the Renaissance "Father of Toxicology," in his Third Defense.[1]

TOXIC overdose can present with a great variety of clinical symptoms—from minor presentations such as nausea and vomiting, to the calamitous, including altered mental status, seizures, cardiac dysrhythmias, hypotension, and respiratory depression. In the patient with altered mental status, there may be few clues to diagnosis at the time of initial assessment and management. The diagnosis may be complicated by the ingestion of multiple drugs.

The clinical course of a poisoned patient depends largely on the specific toxicity of the agent and the quality of care delivered within the first several hours. Fortunately, in most instances, the drug or toxin can be quickly identified by a careful history, a directed physical examination, and commonly available laboratory tests. Attempts to identify the poison should, of course, never delay life-saving supportive care. Once the patient has been stabilized, the physician needs to consider how to minimize the bioavailability of toxin not yet absorbed, which antidotes (if any) to administer, and whether other measures to enhance elimination are necessary.[2]

Clinical Practice Guidelines And Systematic Reviews

There are several published position statements,[3-7] practice guidelines, and consensus statements[8] regarding the management of overdoses. However, the majority of the toxicology literature is based on retrospective case series analysis or isolated case reports (Class III evidence) with isolated animal / bench research. Investigations regarding gastric decontamination either involve adult

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CME Objectives

Upon completing this article, you should be able to:
1. form a differential diagnosis after presentation of clinical scenarios involving toxicology;
2. discuss the management (including antidote use) of a variety of specific poisonings;
3. identify the utility of laboratory data and other aids for diagnosis of poisoned patients;
4. determine by critical evaluation the validity of previously rigid standards of care in toxicology; and
5. evaluate the role of various poisoning treatment modalities using evidence-based medicine.

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See "Physician CME Information" on back page.

volunteers taking sub-toxic amounts who receive decontamination at a set post-ingestion time, or involve mildly to moderately poisoned patients, excluding those with significant overdoses. Very few children have been included in these trials.

Well-controlled, randomized, human trials with adequate sample sizes are infrequent. The conflicting literature regarding the utility of hyperbaric oxygen (HBO) therapy in carbon monoxide (CO) poisoning is one example where the absolute conviction of various experts is outweighed only by the paucity of good data.[9]

The American Association of Poison Control Centers provides annual reports based on data from Regional Poison Centers.[10] However, poison center data may not accurately reflect current reality. Under-representation may occur in the case of lethal agents, because the majority of poisoning deaths never arrive at hospitals (instead, they become cases for the medical examiner).[11] On the other hand, emergency physicians treat most minor ingestions without consulting a Poison Center. Well-designed forensic toxicology data are rare.

Epidemiology And Etiology

Descriptions of poisonings come to us from ancient times. (Some even come from the future. An investigation of the logs of the Enterprise obtained from the original "Star Trek" television series revealed that 35% of the episodes involved toxin-related incidents.[12] These toxic mishaps usually befell the unnamed crew member who had the misfortune to beam down with Captain Kirk.) In 1999, over 2.2 million human exposures to toxins were reported to the American Association of Poison Control Centers.[10] Over 75% were reported from the home, and 13% from a healthcare facility. Two-thirds of these exposures involved patients less than 20 years of age. The leading agents were cleaning substances (10%), followed by analgesics and cosmetics / personal care products. There were 873 poisoning fatalities. The leading fatal agents were analgesics, antidepressants, cardiovascular drugs, stimulants, and street drugs.

Poisoning is an important cause of non-traumatic cardiac arrest in children and young adults.[13-15] There are numerous case reports of survival after toxin-induced cardiac arrest, often as a result of antidote administration. However, arrest secondary to CO poisoning has an especially grim prognosis. One case series examined the outcome of 18 patients who arrested after CO poisoning and were initially resuscitated; all died despite administration of HBO.[16]

Pathophysiology

The Renaissance toxicologist Paracelsus opined that any substance could be poisonous depending on the dose and duration of exposure. Once the person is exposed to toxic substances, factors such as absorption, distribution, and elimination—or "toxicokinetics"—become important. In the overdose patient, toxicokinetic concepts help interpret the significance of urine or plasma drug levels. Toxicokinetics may also be used to predict the onset of symptoms and duration of toxicity.[17]

Differential Diagnosis

Toxins may cause fever, headache, and abdominal pain; indeed, any symptomatic patient can be a potential drug overdose. Altered mental status, GI complaints, cardiovascular compromise, and seizures can all be toxin-related. Some toxins produce subtle effects, such as the "flu-like" symptoms seen with CO poisoning, or cases of digitalis overdose, which may mimic intrinsic heart disease. Furthermore, when faced with any known overdose, the clinician should also consider agents that have similar effects or may be found on the same shelf in the bathroom cabinet. That is, with acetaminophen, think aspirin; with digitalis, add ß-blockers and calcium-channel antagonists to the list of suspects.

Prehospital Care

There are few well-controlled trials that examine the impact of prehospital care on the poisoned patient. It is taken for granted that medics should perform basic stabilization measures such as providing oxygen when needed, employing cardiac monitoring for those with unstable vital signs and cardiotoxic overdoses, and establishing venous access in those who may require fluids or life-saving medications. The exact indications for these measures, however, remain unstudied.

Ipecac has generally fallen from favor in the management of overdoses. It may delay the ultimate administration of charcoal and has the potential to cause aspiration in those with potential for seizures or depressed mentation.

At least two clinical trials have studied the efficacy of prehospital charcoal administration.[18,19] In the first, the average time from first encounter with paramedics to administration of activated charcoal was 5.0 minutes when given in the ambulance, compared to 51.4 minutes when charcoal was delayed until arrival in the ED.[18] In the second study, the median time to activated charcoal in the ED was 82 minutes, suggesting that prehospital charcoal administration could significantly shorten the time to GI decontamination. However, in the vast majority of cases, medics failed to start activated charcoal in the field when indicated.[19] (In fairness to the medics, it is likely that the combination of charcoal plus a careening ambulance could make the emesis scene from "The Exorcist" seem as refined as an afternoon tea.) Both studies focused on the timing of charcoal administration, and neither examined clinical outcomes.

In a patient with depressed mental status, paramedics should check the serum glucose and administer intravenous dextrose when necessary. Hypoglycemia and overdose are not mutually exclusive. An overdose of hypoglycemic agents will, of course, drop the blood sugar. Alcohol ingestion may also cause hypoglycemia, especially in children.[20]

Small doses of naloxone may be required if opiates

are highly suspected and the patient is hypoxic or suffering airway compromise. Naloxone can be given intravenously (IV), intramuscularly (IM), or subcutaneously (SC). In one study of nearly 200 patients with suspected opioid overdose, the time needed to resolve the respiratory depression was similar whether SC or IV naloxone was used, as the slower rate of absorption via the SC route was offset by the delay in establishing an IV.[21] When opioid overdose is suspected, rescuers should give up to 6 mg of naloxone before determining that narcosis is not the etiology of the coma.[22]

Not every patient with altered mental status may require naloxone. One study showed that several clinical findings predict which patients will respond to naloxone. Limiting administration to patients with respirations of 12 or less, those with mitotic pupils, and those with circumstantial evidence of opiate abuse (such as a syringe hanging from the patient's arm) could decrease the use of this drug by 75% to 90% without missing a significant number of naloxone responders.[23]

Flumazenil (Romazicon) should *not* be given empirically to the patient with somnolence. Life-threatening seizures may ensue if the patient has co-ingested a tricyclic or is benzodiazepine-dependent.[24,25]

Other prehospital interventions may occasionally be helpful. Benzodiazepines can control toxic-induced seizures. Prehospital IV sodium bicarbonate administration may be useful for known TCA overdoses if the patient has a wide QRS complex on the cardiac monitor.

Technology may provide novel approaches to field toxicology. In one study, EMTs used handheld CO detectors to measure household levels of carbon monoxide.[26] There were 264 residential CO readings obtained, and nine (3.4%) positive residential readings. However, all chief complaints were believed to be unrelated to CO toxicity.

The fact that a patient arrives by ambulance may accelerate the ED response. One study of 281 subjects showed that overdose patients transported by ambulance have a shorter time interval from ED arrival to gastrointestinal decontamination than patients arriving by other means.[27] However, this difference was largely related to more rapid gastric lavage—an intervention of questionable benefit (as described in subsequent sections).

ED Evaluation

"The surest poison is time."
—Ralph Waldo Emerson (1803-1882)[28]

History

Historical data should include the type of toxin or toxins, time of exposure (acute vs chronic), amount taken, and route of administration (e.g., ingestion, intravenous, inhalation). The timing of ingestion is very important in some overdoses. For example, management of acetaminophen overdose largely depends on how high the blood level is at a known interval post-ingestion. Time to emesis after a mushroom ingestion provides important clues to whether the patient has eaten a highly toxic fungus. Emesis that begins in less than six hours is good (no risk of liver failure); after six hours is bad (high risk of hepatotoxicity).[29]

Also inquire as to why the exposure occurred (accidental, suicide attempt, a search for euphoria, therapeutic misadventure, etc.). Also ask about prior suicide attempts or psychiatric history. Question the patient about all drugs taken, including prescription drugs, over-the-counter medications, vitamins, and herbal preparations. (See also **Table 1** for common drugs of abuse and their street names.) Drug interactions play an important role in poisoning. For example, a variety of agents can heighten the effects of cocaine. Some cocaine abusers co-ingest organophosphates to prolong the effects of cocaine and may develop combined cholinergic and sympathomimetic toxicity.[30] Serotonin syndrome, characterized by muscle rigidity, hyperthermia, diarrhea, and seizures, can occur when sympathomimetics

Table 1. Drugs Of Abuse And Their Street Names.

Marijuana
- Acapulco gold
- Bhang
- Doobie
- Ganja
- Grass
- Joint
- Mary Jane
- Pot
- Reefer
- Rope

Heroin
- Boy
- China white
- Dust
- Harry
- Horse
- Junk
- Monkey
- Smack
- Speed ball (with cocaine)
- Atom bomb (with marijuana)

Cocaine
- All-American drug
- Coke
- Crack
- Girl
- Mother of pearl
- Nose candy
- Peruvian powder
- Snow
- Toot
- White lady

Amphetamines
- Black beauties
- Cat (methcathinone)
- Crank
- Crystals
- Ecstasy
- Ice
- Love drug
- Meth
- Pep pills
- Smart drug (Ritalin)
- Speed
- Uppers
- XTC

PCP
- Angel dust
- Goon
- Hog
- Horse tranquilizer
- Sherman
- Tank
- Wickie stick (when combined with marijuana)

LSD
- Acid
- Blotters
- Microdots
- Paper acid
- Pyramids
- Window pane
- Zen

GHB
- Bioski
- Georgia home boy
- Grievous bodily harm
- Liquid G
- Liquid ecstasy
- Somatomax
- Cow growth hormone

are taken with a selective serotonin reuptake inhibitor.[31] Monoamine oxidase inhibitors (MAOIs) can provoke hypertensive crises in patients taking sympathomimetics—known as "agony after Ecstasy."[32]

Recognize that intoxicated patients can be unreliable historians, particularly if they are suicidal, psychotic, have altered mental status, or are under the influence of recreational drugs.[33-35] Patients who take overdoses lie. In one study, information obtained on admission was completely in accordance with the laboratory finding in less than a third of patients.[36] Fortunately, the development of serious symptoms (which occurred in approximately 20% of the patients) did not correlate with the incorrect historical information.

Information solicited from paramedics, police, family, and friends may prove helpful. Ask about the nature and progression of signs and symptoms. Although issues of confidentiality may arise when talking with family or friends, it is advisable to err on the side of acting in the patient's best interest. Paramedics or EMTs are especially good sources of information, since they may be able to furnish details such as the presence of empty pill bottles or drug paraphernalia at the scene. When the medics bring in empty or partially filled pill bottles, the physician or nurse may perform a "pill count." This traditional late-night ritual involves counting the number of pills left in the bottle and estimating the maximum number the patient may have taken based on the amount dispensed (as indicated on the label).

In some cases, it may be worthwhile to send someone back to the scene to look for clues or a suicide note. Further history can be obtained by consulting the patient's physician or through medical records. A call to the patient's pharmacy may provide important information regarding the type and amount of medications the patient had available. In cases of occupational exposure, obtain a description of the work environment and contact people at the site for relevant data.

If the toxin is known, specific questions are in order. For example, with hydrocarbons, did the patient have a coughing spell upon ingestion? With a caustic ingestion, did he or she drool or vomit? A history of bloody emesis is significant following iron ingestion, while the presence of seizures is an important historical factor in those with TCA overdose. If the patient was exposed to CO, ask whether he or she lost consciousness.

Physical Examination

In the emergency setting, patient stabilization will take precedence over the minutiae of a physical examination. However, once life-saving measures begin, the examination will provide direction to future management. Serial examinations are even more important to determine a prognostic trajectory.

A systematic approach to the examination of the poisoned patient is probably useful. Some literature suggests that using preformatted charts may improve data collection,[37] although their impact on patient outcomes is less clear.

Vital Signs

In many cases, the clinician may be able to deduce the class of drug or toxin taken simply by means of the patient's vital signs. Mnemonics and phrases may help narrow the differential diagnosis when the patient has abnormalities of heart and respiratory rate, body temperature, and blood pressure. (See **Table 2**.) Because rapid mouth breathing, dry mucous membranes, and agitation all produce unreliable oral temperatures, a rectal temperature may be necessary to confirm suspected hyperthermia (or hypothermia) in some patients.

The Eyes

Look carefully at the eyes of the poisoned patient. Pupillary size provides crucial information. (See **Table 3** on page 5.) Dilated pupils can occur with a number of toxins, most notably anticholinergics and sympathomimetics. Small pupils are most commonly seen in conjunction with

Table 2. Diagnosing Toxicity From Vital Signs.

Bradycardia (PACED)
- **P**ropranolol or other beta-blockers, poppies (opiates), propafenone, phenylpropanolamine
- **A**nticholinesterase drugs
- **C**lonidine, calcium-channel blockers
- **E**thanol or other alcohols
- **D**igoxin

Tachycardia (FAST)
- **F**ree base or other forms of cocaine
- **A**nticholinergics, antihistamines, amphetamines
- **S**ympathomimetics (cocaine, amphetamines), solvent abuse
- **T**heophylline

Hypothermia (COOLS)
- **C**arbon monoxide
- **O**piates
- **O**ral hypoglycemics, insulin
- **L**iquor
- **S**edative-hypnotics

Hyperthermia (NASA)
- **N**euroleptic malignant syndrome, nicotine
- **A**ntihistamines
- **S**alicylates, sympathomimetics
- **A**nticholinergics, antidepressants

Hypotension (CRASH)
- **C**lonidine, calcium-channel blockers
- **R**eserpine or other antihypertensive agents
- **A**ntidepressants, aminophylline
- **S**edative-hypnotics
- **H**eroin or other opiates

Hypertension (CT SCAN)
- **C**ocaine
- **T**hyroid supplements
- **S**ympathomimetics
- **C**affeine
- **A**nticholinergics, amphetamines
- **N**icotine

Rapid respiration (PANT)
- **P**CP, paraquat, pneumonitis (chemical)
- **A**SA and other salicylates
- **N**on-cardiogenic pulmonary edema
- **T**oxin-induced metabolic acidosis

Slow respiration (SLOW)
- **S**edative-hypnotics (including GHB)
- **L**iquor
- **O**piates, sedative-hypnotics
- **W**eed (marijuana)

depressed mental status in those with opioid toxicity. Small pupils in association with hypotension may indicate clonidine overdose.

Horizontal nystagmus occurs with lithium,[38] barbiturates, sedative-hypnotics,[39] and antiepileptic poisoning (most notably phenytoin [Dilantin] and carbamazepine [Tegretol]).[40] However, alcohol may be the most common offender. Vertical or rotary nystagmus is a finding peculiar to phencyclidine (PCP).[41] (This characteristic finding is even referred to as "Groucho eyes.")

Optic neuritis and vision loss may indicate advanced methanol poisoning.

Neurologic Examination

A systematic neurological evaluation is important, particularly with patients exhibiting altered mental status. In contrast to the patient with structural brain injury, the patient with a toxic-metabolic cause of coma may exhibit global or non-anatomic neurologic impairment. Toxicologic causes of coma rarely produce focal neurologic deficits. Focal findings, a prolonged comatose state, loss of midbrain pupillary function, or decerebrate or decorticate posturing should prompt the clinician to rule out an intracranial process.[42] Recognize, however, that massive barbiturate poisoning can cause profound neurologic depression[43] and can even mimic brain death. The often-quoted Glasgow Coma Scale, while useful in head trauma victims, has little prognostic value in the poisoned patient.[44,45]

Seizures are common in overdose, and the list of toxins that can induce convulsions is lengthy. (See **Table 4**.) Other general neurologic signs include muscle fasciculations (organophosphate poisoning), rigidity (tetanus and strychnine), tremors (lithium and methylxanthines), and dystonic posturing (neuroleptic agents). Listen closely to the patient's speech. Anticholinergic toxicity results in a characteristic mumbling, as if the patient is trying to quickly recite a haiku with a mouthful of marbles.

Skin Examination

A careful examination of the skin may provide critical data. Remove the patient's clothing. Note the color and temperature of the skin, as well as whether it is dry or diaphoretic. The absence of diaphoresis is an important clinical distinction between anticholinergic (dry) and sympathomimetic (wet) poisoning. Note any bites or wounds, as found with spider and snake envenomations. The presence of rash or bullae may also help provide a diagnosis. While uncommon, bullous lesions are typically located on dependent portions of the body such as between the fingers, knees, and axillae as a result of prolonged immobility. They may be associated with any sedative-hypnotic drug-induced coma but are classically described with barbiturate poisoning.[46] Needle tracks suggest parenteral opiate or cocaine abuse.

Look at the skin color. Cyanosis is seen with profound hypoxia or abnormal hemoglobins such as methemoglobin and sulfhemoglobin. These abnormal hemoglobins may be caused by a variety of toxins and generally produce a central cyanosis refractory to supplemental oxygen. Flushed, red skin can occur in a number of settings, including poisoning from anticholinergics, niacin, or boric acid as well as in disulfiram reactions, scombroid poisoning (histamine toxicity from certain improperly handled fish),[47] and Chinese restaurant syndrome (MSG toxicity).[48] (See **Table 5**.) One unusual color change—flecks of metallic paint

Table 3. Agents That Affect Pupil Size.

Miosis (COPS)
- **C**holinergics, clonidine
- **O**piates, organophosphates
- **P**henothiazines, pilocarpine
- **S**edative-hypnotics

Mydriasis (AAAS)
- **A**ntihistamines
- **A**ntidepressants
- **A**tropine and other anticholinergics
- **S**ympathomimetics

Table 4. Agents That Cause Seizures.

OTIS CAMPBELL*
- **O**rganophosphates
- **T**ricyclic antidepressants
- **I**soniazid, insulin
- **S**ympathomimetics
- **C**amphor, cocaine
- **A**mphetamines, anticholinergics
- **M**ethylxanthines (theophylline, caffeine)
- **P**hencyclidine (PCP)
- **B**enzodiazepine withdrawal, botanicals (water hemlock), GHB
- **E**thanol withdrawal
- **L**ithium, lidocaine
- **L**ead, lindane

*The "town drunk" on "The Andy Griffith Show."

Table 5. Agents That Cause Skin Signs.

Diaphoretic skin (SOAP)
- **S**ympathomimetics
- **O**rganophosphates
- **A**cetylsalicylic acid or other salicylates
- **P**hencyclidine

Dry skin
- Antihistamines, anticholinergics

Bullae
- Barbiturates and other sedative-hypnotics

Acneiform rash
- Bromides
- Chlorinated aromatic hydrocarbons

Flushed or red appearance
- Anticholinergics
- Disulfiram reaction
- Niacin
- Boric acid
- Scombroid poisoning
- Chinese restaurant syndrome
- Carbon monoxide (rare)
- Cyanide (rare)

Cyanosis
- Ergotamine
- Nitrates
- Nitrites
- Aniline dyes
- Phenazopyridine
- Dapsone
- Any agent causing hypoxemia, hypotension, or methemoglobinemia

around the nose and mouth—is sometimes seen in the moribund teen or young adult. These are the stigmata of "huffing" (inhaling hydrocarbons).

Oral burns and corrosives can produce significant chemical burns to the lips, tongue, and mucosa. Remember, however, that some patients who swallow liquid corrosives can have esophageal burns in the absence of oral burns (although they are likely to have drooling or dysphagia).[49]

Another bit of forensic evidence may be found on the patient's fingernails. Mees' lines, or transverse striate leukonychia, are classically associated with arsenic poisoning (but they may also occur with other causes of acute or chronic illness).[50]

Odors

Some poisons produce odors characteristic enough to suggest the diagnosis, such as oil of wintergreen (methylsalicylates) or garlic (organophosphate insecticides). Other smells may be subtle, like the bitter-almond scent associated with cyanide (missed by approximately 50% of the population).[51] Certain odors are overpowering. For example, sulfur dioxide and hydrogen sulfide are gaggingly reminiscent of rotten eggs. (See **Table 6**.)

Toxidromes

A collection of symptoms associated with certain classes of poisons is known as a toxic syndrome, or toxidrome. In patients with unknown overdoses, a toxidrome can assist in making a diagnosis and helps anticipate other symptoms. Cholinergics, anticholinergics, sympathomimetics, and narcotics all have characteristic toxidromes; withdrawal from many addictive agents will produce its own distinctive constellation of symptoms. (See **Table 7**.) The traditional description of the anticholinergic toxidrome, for example, is "hot as a hare, dry as a bone, red as a beet, blind as a bat, mad as a hatter." (Historically, "mad as a hatter" referred to occupational mercury poisoning in the felt hat industry.)

Toxidromes are most clinically useful when the patient has been exposed to a single drug. When multiple drugs have been ingested, conflicting effects may cloud the clinical picture. In addition, the onset of specific toxic complications can be delayed.[52] Toxidrome recognition can also improve the efficiency of drug screening when these findings are communicated to laboratory personnel.[53] (On the other hand, drug screening is of questionable utility in the patient with a classic toxidrome.)

"Sir, if you were my husband, I would poison your drink."
—Lady Aster to Winston Churchill

"Madam, if you were my wife, I would drink it."
—His reply

Table 6. Odors That Suggest A Diagnosis.

Odor	Possible source
Bitter almonds	Cyanide
Carrots	Cicutoxin (water hemlock)
Fruity	Diabetic ketoacidosis, isopropanol
Garlic	Organophosphates, arsenic, dimethyl sulfoxide (DMSO), selenium
Gasoline	Petroleum distillates
Mothballs	Naphthalene, camphor
Pears	Chloral hydrate
Pungent aromatic	Ethchlorvynol
Oil of wintergreen	Methylsalicylate
Rotten eggs	Sulfur dioxide, hydrogen sulfide
Peanut butter	Vacor (rodenticide)

Diagnostic Testing

Electrocardiogram

Frequent or continuous cardiac monitoring along with a 12-lead electrocardiogram is indicated following exposure to any potential cardiotoxin. Cardiac monitoring may be especially useful in poisoning due to sympathomimetic agents, cyclic antidepressants, digitalis, ß-blockers, calcium-channel antagonists, antihypertensive agents, arsenic, cyanide, and carbon monoxide.

The ECG may demonstrate conduction abnormalities such as blocks associated with digitalis or other cardioactive drugs and is essentially diagnostic for serious TCA overdose. The most consistent findings in TCA toxicity are QRS widening (greater than 0.10 seconds) and perhaps more importantly a rightward shift of the terminal 40 ms of the frontal plane QRS complex vector (the patient will have a terminal R

Table 7. Common Toxidromes.

Cholinergic (organophosphates) (DUMBELS)
- **D**iarrhea, diaphoresis
- **U**rination
- **M**iosis
- **B**radycardia, bronchosecretions
- **E**mesis
- **L**acrimation
- **S**alivation

Anticholinergic (antihistamines, TCAs)
- Hyperthermia (HOT as a hare, RED as a beet)
- Dry skin (DRY as a bone)
- Dilated pupils (BLIND as a bat)
- Delirium, hallucinations (MAD as a hatter)
- Tachycardia
- Urgency retention

Sympathomimetic (cocaine, amphetamines)
- Diaphoresis
- Mydriasis
- Tachycardia
- Hypertension
- Hyperthermia
- Seizures

Narcotic (heroin, methadone)
- Miosis
- Hypoventilation
- Coma
- Bradycardia
- Hypotension

Withdrawal (from alcohol, opioids, benzodiazepines, barbiturates, antihypertensives)
- Diarrhea
- Mydriasis
- Goose flesh
- Tachycardia
- Lacrimation
- Hypertension
- Yawning
- Cramps
- Hallucinations
- Seizures (with ETOH and benzodiazepine withdrawal)

wave in aVR).[54,55] (See **Figure 1** and **Figure 2**.) These findings best determine the need for intravenous bicarbonate.[56,57]

TCAs are not the only culprits in a person with a widened QRS. A number of other toxins may produce this finding, including cocaine, propoxyphene, antiarrhythmics, thioridazine (Mellaril), and quinine.

While conduction abnormalities are the most important toxin-related ECG findings, the cardiogram may demonstrate other significant findings as well. Cocaine or carbon monoxide may cause myocardial ischemia or infarction, detectable on the ECG.[58]

Because the ECG is so valuable in cases of tricyclic ingestion, some authorities recommend its routine use in the management of any known or suspected overdose. The utility and cost-effectiveness of this suggestion remain unknown.

Laboratory Tests

Routine Tests

Several simple, readily available laboratory tests may provide important diagnostic clues in the symptomatic overdose patient. These include measurements of electrolytes, blood urea nitrogen and creatinine, serum glucose, a measured bicarbonate level, and arterial blood gases. If the patient is a female of childbearing age, a pregnancy test is useful since these patients often overdose for suicidal or abortifacient reasons.[59]

Anion Gap

The finding of a wide gap metabolic acidosis can significantly narrow the differential diagnosis in an unknown overdose, as well as determine necessary therapy. To check for anion gap metabolic acidosis, calculate the anion gap using serum mEq/L measurements:

$$\mathbf{Na - (Cl + HCO_3)}$$

The normal range for an anion gap is 8-12 mEq/L. When a patient presents with an elevated anion gap (greater than 12), the mnemonic METALACID GAP can assist in identifying the toxic cause. (See **Table 8**.)

Delta Gap

Some patients may have mixed acid-base disorders. For this reason, some believe that knowledge of the dynamic relationship between the rise in anion gap and the fall in bicarbonate is important (i.e., *delta* AG – *delta* HCO_3).[52] If the patient has a metabolic acidosis and the delta gap is greater than +6, a secondary metabolic alkalosis is usually present because the rise in the anion gap is more than the fall in HCO_3. Conversely, if the delta gap is more negative than -6, suspect a concomitant hyperchloremic acidosis because the rise in the anion gap is less than the fall in HCO_3.[52] The clinical utility of routinely measuring a delta gap is unknown. This is because most mixed acid-base disorders in toxicology are clinically obvious (as when a patient ingests an acid-inducing toxin such as iron or aspirin and then begins to vomit).

Osmolar Gap

When a patient presents with an *unexplained* metabolic acidosis, measurement of the osmolar gap may be helpful. An elevated osmolar gap accompanied by anion gap acidosis should immediately suggest poisoning by methanol or ethylene glycol.

The osmolar gap is the difference between measured serum osmolality (most accurately determined by freezing-point depression) and the calculated serum osmolality (most

ECG Changes In Tricyclic Overdose*

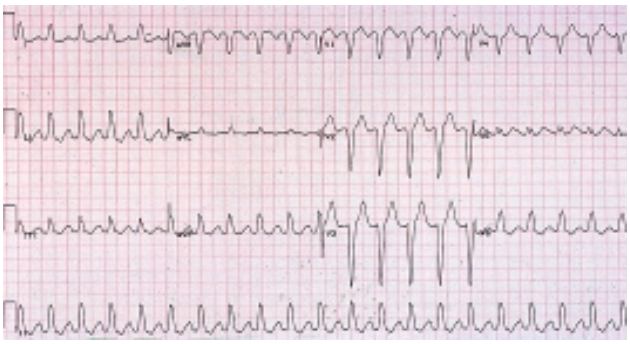


Figure 1. QRS Widening.

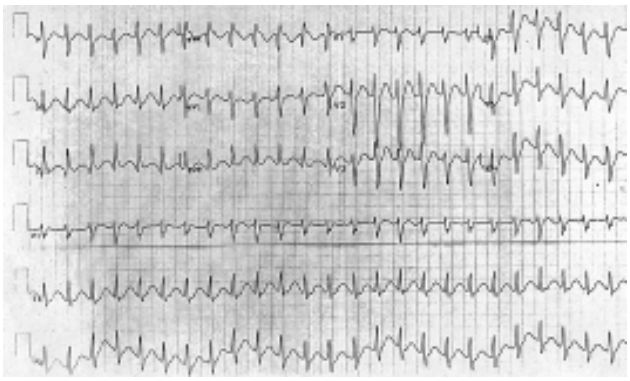


Figure 2. Prominent R Wave In Lead aVR.

*ECGs courtesy Dr. Russ Kerns.

Table 8. Agents Causing An Elevated Anion Gap.

METALACID GAP

Methanol, metformin
Ethylene glycol
Toluene
Alcoholic ketoacidosis
Lactic acidosis
Aminoglycosides, other uremic agents
Cyanide, carbon monoxide
Isoniazid, iron
Diabetic ketoacidosis
Generalized seizure-producing toxins
ASA or other salicylates
Paraldehyde, phenformin

commonly determined by the following formula):

$$2\,Na + \frac{Glucose}{18} + \frac{BUN}{2.8} + \frac{ETOH}{4.6}$$

If the patient has ingested methanol, ethylene glycol, or isopropanol, a modification of this formula can be used to estimate serum levels of these toxic alcohols. The formula can be modified by using the following values in place of ETOH (or in addition to the ETOH if the patient has consumed both alcohol and a toxic alcohol):[60]

Methanol	Ethylene glycol	Isopropanol
3.2	6.2	6.0

An osmolar gap of greater than 10 mOsm / kgH_2O has been arbitrarily defined as increased and indicates the presence of a low-molecular-weight, osmotically active substance in the serum.[60] The mnemonic "ME DIE" helps recall the major toxins that produce an increased osmolar gap. (See **Table 9**.)

While an increased gap may be helpful, a "normal gap" (i.e., < 10 mOsm) does not absolutely exclude osmotically active substances. Furthermore, depending on the extent of metabolism, and the time of ingestion, little parent compound may be present when a patient presents to the ED.[61,62]

Toxicology Screens

Toxicology screens come in two flavors—*qualitative* screens, which test for the presence of multiple drugs, and *quantitative* screens, which measure the level of a particular drug. In general, qualitative toxicology screens are less important than the patient history and clinical status, but quantitative levels of suspected substances, such as acetaminophen or aspirin, may be valuable in certain circumstances.

The utility of qualitative toxicology screens is limited by practical considerations. Laboratory turnaround time is often longer than the time course of an overdose. The drugs tested are by necessity restricted, as hospitals cannot support the cost of maintaining the procedures, instruments, training, and specialized labor needed to analyze every toxin on a 24-hour basis.[63] While most immunoassays are capable of detecting commonly abused drugs such as marijuana and cocaine, many common and dangerous substances are not routinely included, such as isoniazid, digitalis glycosides, calcium antagonists, ß-blockers, heavy metals, and pesticides. Therefore, *a negative screen does not rule out the possibility of poisoning.* On the other hand, the screen may detect some drugs that present in therapeutic amounts, such as opioids and benzodiazepines, even though they are not responsible for the presenting symptoms. Finally, technical limitations of the assay can cause either false-positive or false-negative results (although improvements over the past decade have rendered the tests increasingly more sensitive and specific).[63-66]

The toxicology screen may have little medical value if the specimens are collected too early or late for detection. In general, metabolites in the urine can be detected as long as 2-3 days (or longer) after exposure, compared with 6-12 hours in the blood. The analysis of gastric contents is not clinically useful and is usually reserved for forensic cases.

A comprehensive urine toxicology screen is labor-intensive and is intended to detect as many drugs as possible using common techniques. Usually included on the panel are the alcohols, sedative-hypnotics, barbiturates, benzodiazepines, anticonvulsants, antihistamines, antidepressants, antipsychotics, stimulants, opioids, cardiovascular drugs, oral hypoglycemics, and methylxanthines (caffeine, theophylline). Although comprehensive screening is unlikely to affect emergency management, the results may assist the admitting physician in evaluating the patient if the diagnosis remains unclear.[67]

The most germane question is: "Does a toxicology screen affect the management of a patient who has taken overdose?" In most studies that have looked at this question, the answer is no.

In one trial of over 400 ED patients, qualitative screens rarely affected management.[68] Other studies yield similar results.[69,70] While it is true that qualitative screens occasionally show unexpected findings or drugs in addition to those admitted by the patient, this information is rarely clinically relevant. In a study of 209 patients, unexpected toxicology findings led to changes in therapy in only three cases, and none of these changes appeared to have a major impact on outcome.[64]

Similar findings occur in children. Two studies show that qualitative urine drug screens provided minimal useful information and that unexpected findings on urine drug screening leading to changes in management were uncommon.[71,72]

Quantitative blood tests can be helpful when the emergency physician suspects intoxication with one of the following: acetaminophen, salicylate, theophylline, lithium, lead, iron, carbon monoxide, methemoglobin, toxic alcohols, anticonvulsants, and digoxin. In addition to the victim of smoke inhalation or intentional CO poisoning, consider a CO level in patients who have headaches and use stoves for heat or who live with someone who is similarly symptomatic.[73]

Acetaminophen Levels

A recurring issue regards the need for a routine acetaminophen (APAP) level in the overdose patient. In most dangerous poisonings, the patient is symptomatic by 4-6 hours. Acetaminophen toxicity is the one common poisoning where the patient may be asymptomatic at this time despite a potentially lethal ingestion. For this reason, some authorities suggest a routine quantitative

Table 9. Agents That Increase The Osmolar Gap.

ME DIE
- **M**ethanol
- **E**thylene glycol
- **D**iuretics (osmotic diuretics like mannitol)
- **I**sopropyl alcohol
- **E**thanol

serum acetaminophen level in the overdosed patient. On the other hand, most literature on the subject questions the need for this. One review looked at over 1800 patients with a history of suicidal ingestion or an altered mental status with a strong suspicion of ingestion. Universal screening for acetaminophen showed that 0.3% of suicidal ingestions had a potentially toxic APAP intoxication not suggested by history.[34] However, none of these patients required therapy with N-acetylcysteine. Another retrospective study from Hong Kong examined the clinical value of screening for acetaminophen in 294 Chinese patients with acute poisoning. Of the 208 patients with no suspected acetaminophen ingestion, four were found to have elevated but non-toxic plasma levels. In this population, the authors felt that routine screening of all patients with acute poisoning for toxic plasma acetaminophen concentrations was not indicated.[74]

This said, it still seems prudent to measure an acetaminophen level in patients who take an overdose during a suicide attempt. In the future, urine screens for acetaminophen may play an increasing role. In one study, a negative urine screen for acetaminophen obviated the need for a four-hour serum level.[75]

Urine Analysis

A urine ferric chloride test is one of the most useful urine tests in the poisoned patient. In this test, 1 mL of the patient's urine is added to 1 mL of the ferric chloride. A darkening of the solution (often purple) indicates the presence of salicylates[76] or phenothiazines. This test, however, does not confirm toxicity; electrolytes and a serum salicylate level are then indicated in the symptomatic patient.

Other urine tests may occasionally be helpful. Calcium oxalate crystals are considered pathognomonic for ethylene glycol poisoning. However, these are usually discovered late in the clinical course. They also may be absent in the urine early after ethylene glycol ingestion, or not detected at all if timely therapy has been instituted.[77] Years ago, some

Cost-Effective Strategies For Managing The Poisoned Patient

1. Avoid "shotgunning" laboratory data.
Ordering excessive laboratory tests on the overdose patient is commonplace. Consult the regional poison center or a comprehensive toxicologic database in order to narrow the scope of lab acquisitions. Every toxic ingestion does not require liver function tests, thyroid function tests, and an amylase.

Risk-Management Caveat: In the symptomatic patient with an unknown overdose, measurement of the anion gap may be useful.

2. Limit toxicology qualitative screening panels.
It is virtually a Pavlovian response to order a "tox screen" on the overdose patient—yet these screens rarely impact management. If the patient admits to using cocaine, don't get a test to confirm this. No one lies about using cocaine (although many may lie about *not* using it).

Risk-Management Caveat: Quantitative serum levels such as digoxin, theophylline, salicylate, acetaminophen, and carbon monoxide are useful when the physician has reason to suspect poisoning with these agents. Levels may help direct therapeutic interventions, such as antidote administration, and determine prognosis.

3. Avoid unnecessary use of expensive antidotes.
Although antidotes are the glamorous celebrities of toxicology, they are probably necessary in only 10%-15% of all cases in which they are used. The majority of patients will recover uneventfully with supportive care alone. Follow established criteria for antidotes such as digoxin-specific antibodies (Digibind) and N-acetylcysteine (NAC). Cavalier administration of antidotes when not indicated can result in dangerous clinical side effects, particularly if the offending agent is misdiagnosed.

Risk-Management Caveat: Antidotes can be lifesaving in the appropriate clinical circumstances.

4. Use a suitable amount of hospital resources.
The majority of poisoned patients may not require a full admission and may require less than 24 hours of observation. An ED observation unit is often adequate to monitor mildly symptomatic or asymptomatic poisonings. When a patient does require admission, consider the safety of observation on a general hospital ward. Admitting all poisoned patients to an ICU setting is an expensive and unnecessary practice. Clearing a stable patient medically for a psychiatric admission or admitting the patient to the medical floor with a "sitter" are viable, cost-effective options.

Risk-Management Caveat: Patients with potentially lethal poisoning, arrhythmias, unstable vital signs, or significantly altered mental status will require an ICU admission.

5. Provide easily accessible poison control information and support for regional poison treatment centers.
Free public access to poison information using a toll-free telephone number is not only cost-effective but a public health necessity. Accessible poison information also assists healthcare professionals in the early intervention of poisoned patients, potentially avoiding costly ICU admissions when definitive management is delayed.

The concept of regional poison treatment centers is similar to that of Level I trauma centers. Overdose patients would be bypassed to a tertiary care facility properly staffed to manage more complicated or unstable patients.[167] ▲

literature suggested using a Wood's lamp to detect urine fluorescence following possible ethylene glycol ingestion.[78] However, a more recent trial refutes the utility of this technique, citing numerous false-positive results.[79]

At times, a dipstick urinalysis is positive for hemoglobin, but the microscopic analysis reveals no red blood cells. This suggests either myoglobinuria from muscle breakdown or hemolysis possibly due to a toxin.

Urine color may also provide a diagnostic clue. For example, an orange to red-orange hue is seen with phenazopyridine, rifampin, deferoxamine, mercury, or chronic lead poisoning; pink with cephalosporin or ampicillin overdose; brown with chloroquine or carbon tetrachloride; and greenish-blue with copper sulfate or methylene blue. Finally, monitoring urinary pH can be important, particularly when instituting bicarbonate therapy for salicylate overdose.

Radiologic Studies

Abdominal Films (Plain Film Or KUB)

A KUB (kidney, ureter, and bladder) radiograph is most useful to visualize metals and drug-filled packets. In practice, the only prescription medication worth looking for on KUB is iron. A KUB may also be clinically useful in suspected body packers (drug smugglers). These professional "mules" differ from body stuffers, who quickly "swallow the evidence." Plain films of the abdomen are usually negative in stuffers[80] and should not be routine. In cases where a plain film demonstrates iron or drug packets, serial films may be used to monitor response to whole bowel irrigation.

Other substances that can occasionally be seen are recalled by the mnemonic "COINS": **C**hloral hydrate and cocaine packets; **O**piate packets; **I**ron and other heavy metals such as lead, arsenic, and mercury; **N**euroleptics; and **S**ustained-release or enteric-coated preparations, which may be only faintly visible.[81] (See **Table 10**.) In some cases, the vehicle in which the drug is contained, such as an enteric coating or latex, will be more radiopaque than the drug itself. For many slightly radiodense drugs such as neuroleptics and salicylates, visibility will be dependent on the time of ingestion. When a patient presents several hours after the ingestion, the radiograph is rarely useful.

Chest Films

Patients with tachypnea, coma, or obtundation should have radiographs to search for potential causes of hypoxemia, including chemical or aspiration pneumonitis, cardiogenic or non-cardiogenic pulmonary edema, and atelectasis. Drugs that can cause non-cardiogenic pulmonary edema can be remembered by the mnemonic "MOPS": **M**eprobamate and **m**ethadone; **O**pioids; **P**henobarbital and **p**henothiaxines; and **S**alicylates. (See **Table 11**.) Chest films are also useful for detecting pneumothorax or pneumomediastinum in patients smoking cocaine.

"Give a man a fish, and he can eat for a day.
But teach a man how to fish, and he'll be dead
of mercury poisoning inside of three years."
—Charlie Haas (1889-1964)[82]

Supportive Measures

ABCs

While the majority of patients with poisoning are awake and have stable vital signs, some may present unconscious, in shock, or actively seizing. The first priority is to stabilize the ABCs and manage life-threatening complications.

Clear the airway by repositioning the patient and institute suctioning if necessary. While antidotes such as naloxone and flumazenil may reverse respiratory depression, they should not be the first intervention in an apneic or severely bradypnic patient. Such individuals need respiratory assistance with a bag-valve mask until the antidote can be drawn up and administered.

Unlike patients with more classic cardiopulmonary disease states (like CHF, COPD, and asthma), where the progression of respiratory compromise is more predictable, the toxic patient may unexpectedly lose airway control. Therefore, aggressive airway management is paramount, as this inevitably has the greatest impact on outcome in these patients. In many cases, intubation, with supplemental oxygen or assisted ventilation, may be required. In one uncontrolled series looking at the airway management of poisoned patients, neuromuscular blockade with sedation provided better intubating conditions than sedation alone.[83] However, whether induction agents are necessary or useful if the patient has ingested a sedative-hypnotic agent remains unknown.

The patient's oxygenation status can be monitored with bedside pulse oximetry. However, in certain cases, the pulse oximetry may be falsely normal due to the effect of the poison. This is particularly true in poisonings that produce abnormal hemoglobin, such as carboxyhemoglobin or methemoglobin.[84] In these cases, obtain an arterial blood gas and measure the carboxyhemoglobin or methemoglobin

Table 10. Agents Visible On Abdominal Films.

COINS
- **C**hloral hydrate, cocaine packets, calcium
- **O**pium packets
- **I**ron, other heavy metals such as lead, arsenic, mercury
- **N**euroleptic agents
- **S**ustained-release or enteric coated agents

Table 11. Drugs Causing Pneumonitis Or Pulmonary Edema.

MOPS
- **M**eprobamate, methadone
- **O**piates, organophosphates
- **P**henobarbital, propoxyphene, phenothiazines
- **S**alicylates, smoke inhalation (including cocaine smoke), solvents

levels. Recall that pulse oximetry only reflects oxygen saturation and does not assess the patient's acid-base status or ventilation (as reflected by the PCO_2).

In symptomatic overdosage or exposures to a potentially dangerous substance, initial intravenous access is indicated. Consider placing an intravenous line even when the patient is apparently alert and stable if the suspected toxins can produce delayed symptoms (e.g., hypotension or seizures) that may make later intravenous access difficult.

Whether or not the patient is unconscious or hemodynamically compromised on arrival in the ED, continued absorption of the ingested drug or poison may lead to more serious intoxication during the next several hours. Keep the patient under close observation with frequent checks of alertness, oxygenation status, and determination of vital signs.

The Life-Threatening Overdose

Toxins can produce hypotension in a number of ways—by vasodilation, myocardial depression, and fluid losses from vomiting, diarrhea, or third spacing. Despite the individual mechanism, one of the first interventions in the hypotensive overdose patient (in the absence of profound arrhythmia) should be volume loading with normal saline or Ringer's lactate. However, when drug-induced shock remains refractory to fluid therapy, high-dose vasopressor therapy may be lifesaving.[22] If the patient still remains hypotensive, central hemodynamic monitoring should be instituted when feasible. An intra-aortic balloon pump (IABP) can be used in those whose shock cannot be reversed by any other means.[22]

Myocardial depression is common in poisoning with calcium-channel blockers and ß-blockers. Epinephrine, norepinephrine, and other catecholamine-type vasopressors are the drugs of choice in treating calcium-channel-blocker-induced shock.[22] Calcium chloride (1-3 g given by slow IV push) remains a secondary therapy in those whose hypotension does not resolve with catecholamines. Intravenous glucagon (2-5 mg IV; some suggest up to 10 mg) can successfully treat hypotension associated with ß-blocker and calcium-channel blocker overdose.[85,86] The bolus can be followed by a drip of 5-10 mg per hour, titrated to effect.

Toxins may also lead to dangerous arrhythmias. Many of the arrhythmias can be treated with standard ACLS protocols—with a few exceptions. One of the most important of these is the poisoned patient with a wide complex tachycardia. This rhythm, which may appear to be ventricular tachycardia, can result from poisoning of the cardiac sodium channels. In the case of hemodynamically stable ventricular tachycardia associated with cocaine, the drugs of choice include sodium bicarbonate[87,88] and perhaps lidocaine.[89]

Sodium bicarbonate is also the drug of choice for the treatment of ventricular dysrhythmias and / or hypotension secondary to TCA poisoning.[90,91] Hyperventilation[92] and hypertonic saline[93] may also be useful, but clinical and experimental experience with these modalities is less extensive than with sodium bicarbonate. In patients with severe toxicity, give enough bicarbonate to achieve a serum pH of 7.50-7.55. Intermittent boluses of sodium bicarbonate are preferred to a constant infusion.[22] Procainamide is often discouraged in the case of arrhythmias secondary to tricyclics. This mostly theoretical concern is due to the fact that most tricyclics have Class Ia antiarrhythmic properties similar to procainamide.

"Coma Cocktail"

A toxic agent or malnutrition may cause hypoglycemia. Give 50% dextrose to all patients with altered mental status, unless a rapid fingerstick glucose assessment demonstrates euglycemia or hyperglycemia. While some sources caution against giving a hypertonic glucose bolus to any patient with a potential stroke or cerebral ischemia, this concern is probably unwarranted.[94,95]

Naloxone, a specific opioid antagonist, may have both therapeutic and diagnostic value, as patients with an opioid overdose usually become fully awake soon after its administration. Give 0.4-2.0 mg IV if the clinical signs are consistent with the opioid toxidrome.[23] If necessary, naloxone can also be given intramuscularly, subcutaneously, intralingually, or endotracheally. One study recommends that when using naloxone subcutaneously, begin with at least 0.8 mg SC.[21]

Chronic abusers typically require smaller amounts of the antidote. In these patients, begin with 0.1-0.2 mg intravenously, repeated to a total dose of 0.4 mg, instead of the more traditional 2 mg.[22] Giving a full dose of naloxone to an opioid-dependent patient could transform a peaceful snoozer into an extremely belligerent doctor-basher.[96,97] Fortunately, the drug has a half-life of only 60-90 minutes, and withdrawal symptoms wear off in one or two hours. If possible, restrain the patient before giving naloxone, particularly with a "hard-core" abuser.

Some opioids, such as diphenoxylate / atropine, propoxyphene, pentazocine, or codeine, may be resistant to standard doses (2 mg) of naloxone, and the emergency physician may need to administer as much as 10 mg to achieve arousal. Admittedly, the literature on this dosing is largely anecdotal.[98-100] Naloxone may also be ineffective in patients who have co-ingested sedatives such as benzodiazepines or alcohol, or in those who have injected heroin containing adulterants.

The dosage should be titrated until the patient has stable respirations (respiratory rate > 10 or greater and pulse oximetry ≥ 92%). With longer-acting opioids, an intravenous naloxone drip may be required. The drip is classically mixed with two-thirds of the dose required to awaken the patient given per hour using a medication pump. However, this dose is widely variable, depending on the patient's time of exposure and tolerance.

Longer-acting opioid antagonists, such as nalmefene, are now available.[101,102] These agents have a half-life of approximately 10 hours. Nalmefene can reverse opioid intoxication for as long as eight hours, theoretically reducing the need for continuous monitoring of intoxicated patients and repeated doses of naloxone. However, the long duration of action may cause extended withdrawal reactions in chronically opioid-dependent patients.[103] Some authorities

believe its use in the ED should be limited unless the patient is going to be admitted for observation.

Reserve thiamine for alcoholic, malnourished patients. Although it is an inexpensive water-soluble vitamin, giving thiamine to every comatose patient in order to prevent Wernicke's encephalopathy is probably unwarranted.[94]

Flumazenil, a specific benzodiazepine antagonist, can rapidly reverse coma in benzodiazepine overdose. The drug, however, may also induce seizures in patients with mixed drug overdoses, such as with a cyclic antidepressant or sympathomimetic, and it may provoke acute withdrawal in those addicted to benzodiazepines. Flumazenil should, therefore, be used judiciously rather than administered routinely as part of the "coma cocktail."[104-108] "Judicious" in this case means limited to the patient with a known isolated benzodiazepine overdose who has persistent hypoxia, or progressive respiratory depression. Flumazenil may be used in this hypothetical patient to avoid intubation.

A few longtime physicians may recall that physostigmine was once part of the "coma cocktail." However, this drug is contraindicated in comatose patients with an unclear cause and should only be used in cases of severe, isolated anticholinergic poisoning. It is *absolutely contraindicated* with TCA overdoses since it may exacerbate cardiotoxicity.[109]

Skin And Eye Decontamination

While scientific data regarding proper dermal and ocular decontamination methods are limited, fundamental principles can be found in military chemical battlefield and radiation accident protocols.[110] If possible, HAZMAT decontamination is best performed in the prehospital setting. In patients with dermal exposures, all clothing should be removed and the skin copiously irrigated and washed with a mild soap and water. Avoid using hot water, strong detergents, or harsh abrasives.[110] Never delay decontamination in order to search for the offending agent. Emergency care providers should wear gloves, water-resistant gowns, splash-resistant goggles, and masks to protect themselves from dermal exposure (particularly with insecticides). Ocular exposures to acids and alkalis can be devastating; copiously irrigate with several liters of normal saline solution and monitor the pH of the conjunctival sac before starting other therapeutic or diagnostic interventions.[8] The ocular pH should be 7.4 before terminating irrigation.

Gastric Decontamination

Controversy still exists concerning the roles of emesis, gastric lavage, activated charcoal, and cathartics in decontaminating the gastrointestinal tract. Specific circumstances may dictate which technique is the most appropriate.[111,112] Numerous experimental and clinical trials show that the effectiveness of gastric emptying techniques is limited. Regardless of the method of gastric decontamination, a significant amount of toxin is not removed and remains available for absorption.[113]

Ipecac-Induced Emesis

Once the preferred technique for gastric emptying, syrup of ipecac is no longer recommended in the ED. There is no evidence from clinical trials that ipecac improves the outcome of poisoned patients. Furthermore, persistent vomiting after ipecac administration may cause aspiration and frequently delays the administration of activated charcoal. Although controversial, ipecac still may have a role in the domestic setting. This specific scenario involves alert children who have very recently ingested known substances that are not well adsorbed by activated charcoal and for whom transport time to a healthcare facility is delayed.[3,114-116] Ipecac should never be given to anyone who has ingested a toxin that has the potential to decrease consciousness or cause seizures, as aspiration may occur. Furthermore, it is contraindicated if the poison is a hydrocarbon or corrosive. For these reasons, parents should contact a Poison Center before administering ipecac.

The American Academy of Clinical Toxicology and the European Association of Poison Centres and Clinical Toxicologists issued a position statement on ipecac. Their strong language states: "Syrup of ipecac should not be administered routinely in the management of poisoned patients….There is no evidence from clinical studies that ipecac improves the outcome of poisoned patients, and its routine administration in the emergency department should be abandoned."[117]

"The lethal dose of cannabis is a two-kilo block dropped on your head from the 25th floor of a high-rise building."
—from Everything You Always Wanted to Know about Drugs, but Were Afraid to Ask Your Children

Gastric Lavage

In the early 1800s, Edward Jukes, a British surgeon, performed gastric lavage on himself following an ingestion of laudanum (a tincture of opium). (We rarely see such dedication to research nowadays.) Other than mild GI complaints followed by a three-hour nap, he survived with no adverse side effects.[118]

Currently, gastric lavage involves using a large-bore (36-40 French) tube. During the procedure, the patient should be placed in the left lateral / head down position. The technique is carried out using small aliquots of liquid. In adults, use 250 mL of warm fluid such as tap water or normal saline (in a child, use 10 mL / kg body weight of warm normal saline). The volume of the lavage fluid returned should approximate the amount of fluid administered. This process should be continued until the recovered solution is clear of particulate matter or pill fragments.

Gastric lavage is no longer indicated for small-to-moderate ingestions of most substances, particularly if activated charcoal can be given promptly. In experimental models, the amount of toxin removed by gastric lavage is highly variable and significantly diminishes over time. Gastric lavage may be considered if a patient has ingested a potentially life-threatening amount of toxin and presents within one hour of ingestion.[4,116,119-123] However, even in this

scenario, there is no solid evidence that its use improves clinical outcomes. While some clinicians anecdotally claim that delayed lavage (beyond one hour) may be efficacious when the ingested drug can cause either slowing of peristalsis (e.g., anticholinergics or opioids) or formation of large concentrations (e.g., salicylates), there are no well-designed trials that support this opinion. Whether gastric lavage is of clinical benefit in decontaminating hemodynamically unstable patients with an unknown type and time of ingestion remains unknown.

Again, gastric lavage is contraindicated when a patient has ingested a corrosive substance or a hydrocarbon. It should never be used as a punitive measure in cases of nontoxic overdoses or forced on patients who are combative and uncooperative. Additionally, oral intubation of a patient solely to perform gastric lavage is highly discouraged. Lavage is not a benign procedure; it has been associated with complications including aspiration, esophageal perforation, epistaxis, hypothermia, and death.[4]

Activated Charcoal

The use of activated charcoal is well-described in the literature.[5,111-114,116,119-124] The 19th-century French pharmacist P.F. Tourey established the beneficial effects of charcoal when he ingested a potentially life-threatening amount of strychnine mixed with a primitive charcoal preparation in front of the French Academy of Medicine. He survived to prove his point, but his demonstration was not met with thunderous applause.[125] More recent studies suggest that, even when given alone without previous gastric emptying, activated charcoal is more effective than emesis or gastric lavage for most toxins.[5] A dog study investigated lavage vs. charcoal vs. a charcoal-lavage-charcoal approach in salicylate overdose. In this study, charcoal was found to be superior to lavage alone. Although the combined approach tended toward more efficacy, it was not found to be statistically significant.[126]

Activated charcoal has become the first-line treatment for patients who have ingested a potentially toxic amount of drug. Activated charcoal also appears to be the most efficacious and safe decontamination method when the ingested substance is unidentified. However, routine administration in nontoxic ingestions is not indicated.

As with gastric lavage, the effectiveness of activated charcoal decreases with time, and it is most beneficial if administered within one hour post-ingestion. Several different activated charcoal products are commercially available. Regardless of the product, it is important to ensure that the activated charcoal is re-suspended and thoroughly mixed in water or sorbitol to achieve a 25% concentration prior to use. Although commonly administered in an arbitrary 50 g (or 1 g/kg) loading dose in adults, a more accurate dose of charcoal is to provide at least a 10:1 ratio of activated charcoal to toxin.[113] If this ratio cannot be achieved in one single dose, then serial dosing may be required.

Those substances *not* well adsorbed by charcoal can be recalled by the mnemonic "PHAILS": **P**esticides; **H**ydrocarbons; **A**cids and **a**lkali; **I**ron; **L**ithium; and **S**olvents. Adverse side effects of activated charcoal administration, while rare, include aspiration pneumonitis in the unprotected airway as well as bowel obstruction and perforation.[127,128]

Multiple-Dose Charcoal

Repeated doses of activated charcoal can also reduce the elimination half-life of some drugs by interrupting enterohepatic or enteroenteric recirculation. For repetitive dosing, administration of 25 g every 2-4 hours, *without a cathartic*, is recommended in the adult (although some centers use a cathartic with the first dose only). Some of the drugs removed by repeat-dose activated charcoal can be recalled by the mnemonic "ABCD": **A**ntimalarials (quinine) and **a**minophylline (theophylline); **B**arbiturates (phenobarbital) and **b**eta-blockers (nadolol);[129] **C**arbamazepine; and **D**apsone. (See **Table 12**.) The data are mixed regarding the efficacy of multiple dosing of activated charcoal to increase elimination of amitriptyline, digitalis, digoxin, phenytoin,[130] and salicylate.

Cathartics

The efficacy of cathartics in reducing the absorption or increasing the elimination of toxins has never been established in the literature.[6] Although cathartics are generally used with activated charcoal to hasten the elimination of the toxin bound to charcoal, studies have not shown that administration improves decontamination efficacy. Because some believe the risks associated with cathartics outweigh the proven benefits, they recommend against cathartics in the acute management of the poisoned patient. However, this recommendation remains controversial. Others argue that the administration of charcoal without any cathartics (especially multiple doses or in the case of impaired peristalsis) can result in gastrointestinal obstruction.[131] The administration of a cathartic without charcoal has no role in the management of the poisoned patient. The most popular cathartics are magnesium sulfate (10% solution at 4 mL/kg) and sorbitol (35% solution at 4 mL/kg, diluted 1:1 with

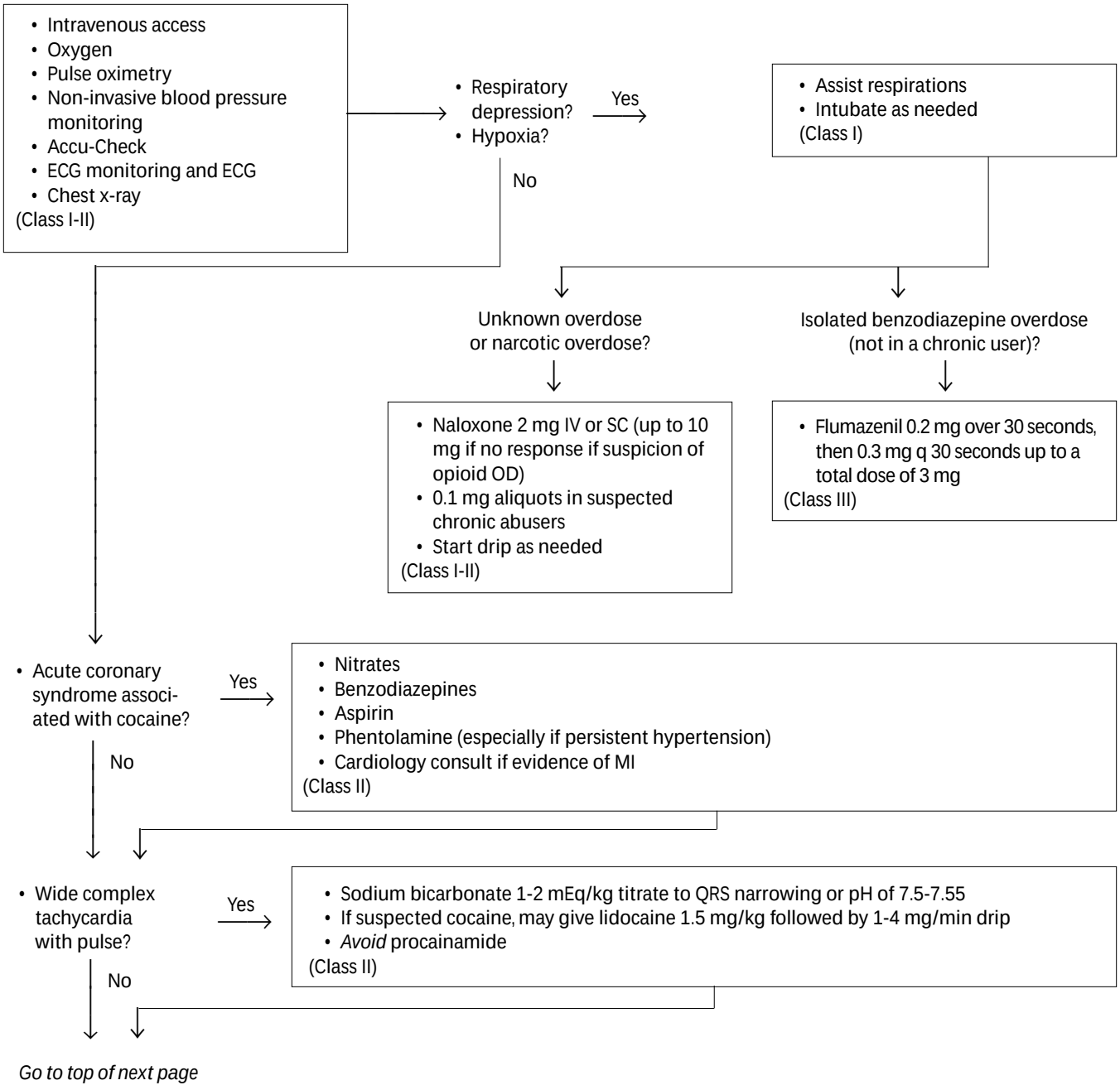
Continued on page 19

Table 12. Agents Responsive To Multiple Doses Of Activated Charcoal.

Adsorbable (ABCD)
- **A**ntimalarials (quinine), aminophylline (theophylline), possibly aspirin
- **B**arbiturates (phenobarbital), possibly beta-blockers (Nadolol)
- **C**arbamazepine
- **D**apsone, possibly dilantin

Not adsorbable (PHAILS)
- **P**esticides
- **H**ydrocarbons
- **A**cids, alkali
- **I**ron
- **L**ithium
- **S**olvents

Clinical Pathway: Management Of The Life-Threatening Overdose

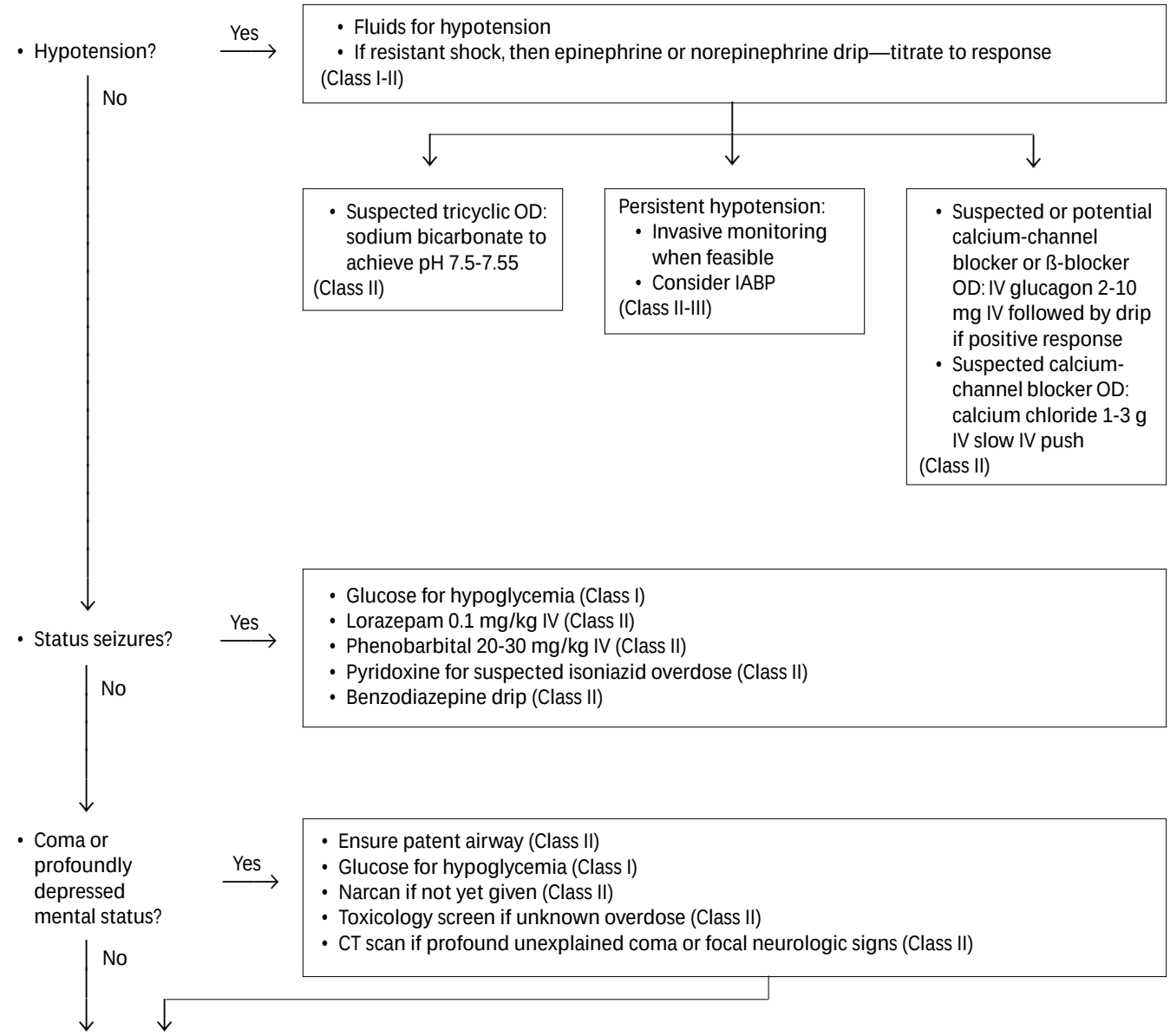


Go to top of next page

The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Clinical Pathway: Management Of The Life-Threatening Overdose (continued)



Go to "Clinical Pathway: Basic Toxicologic Interventions"

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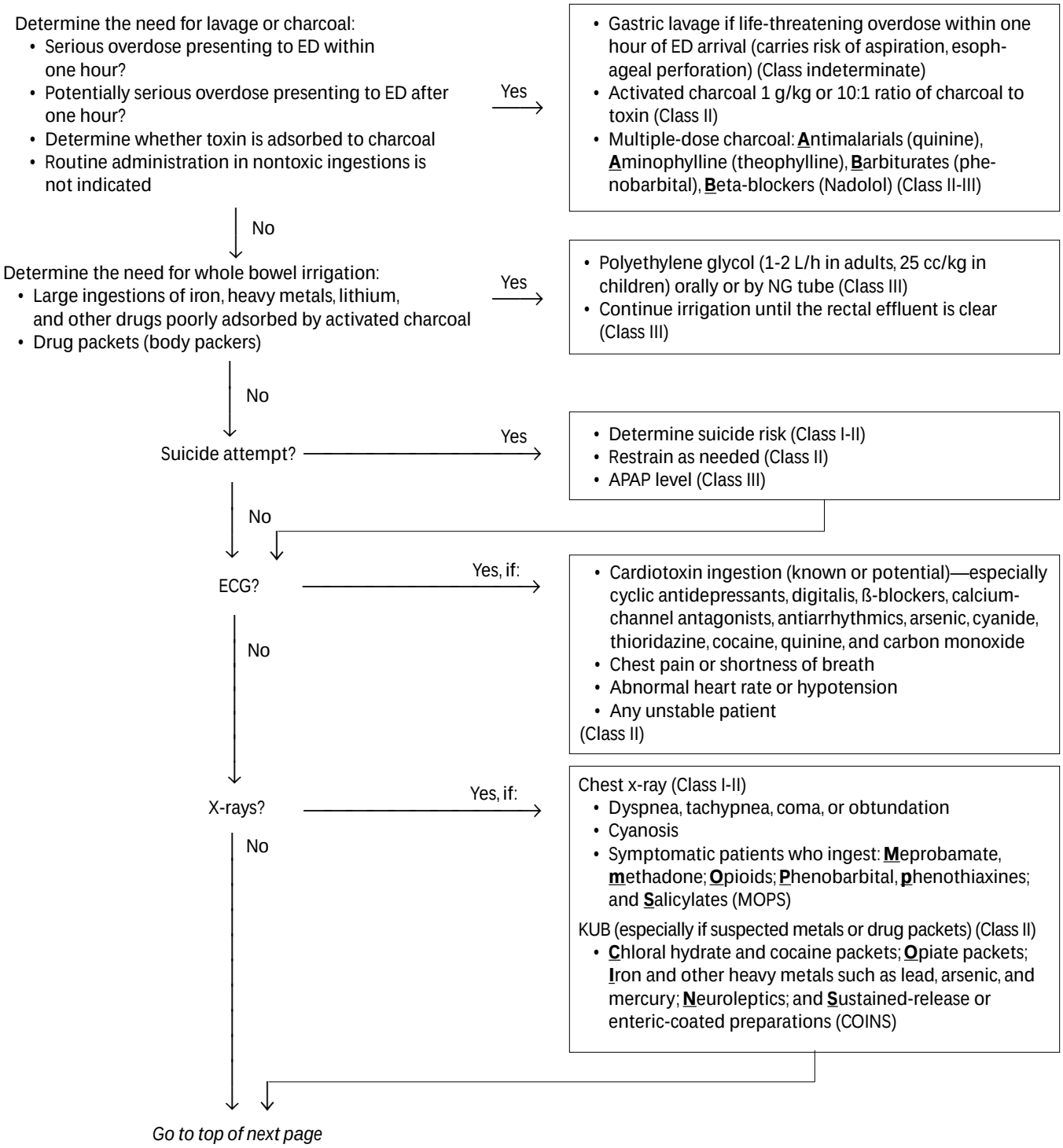
Clinical Pathway: Basic Toxicologic Interventions



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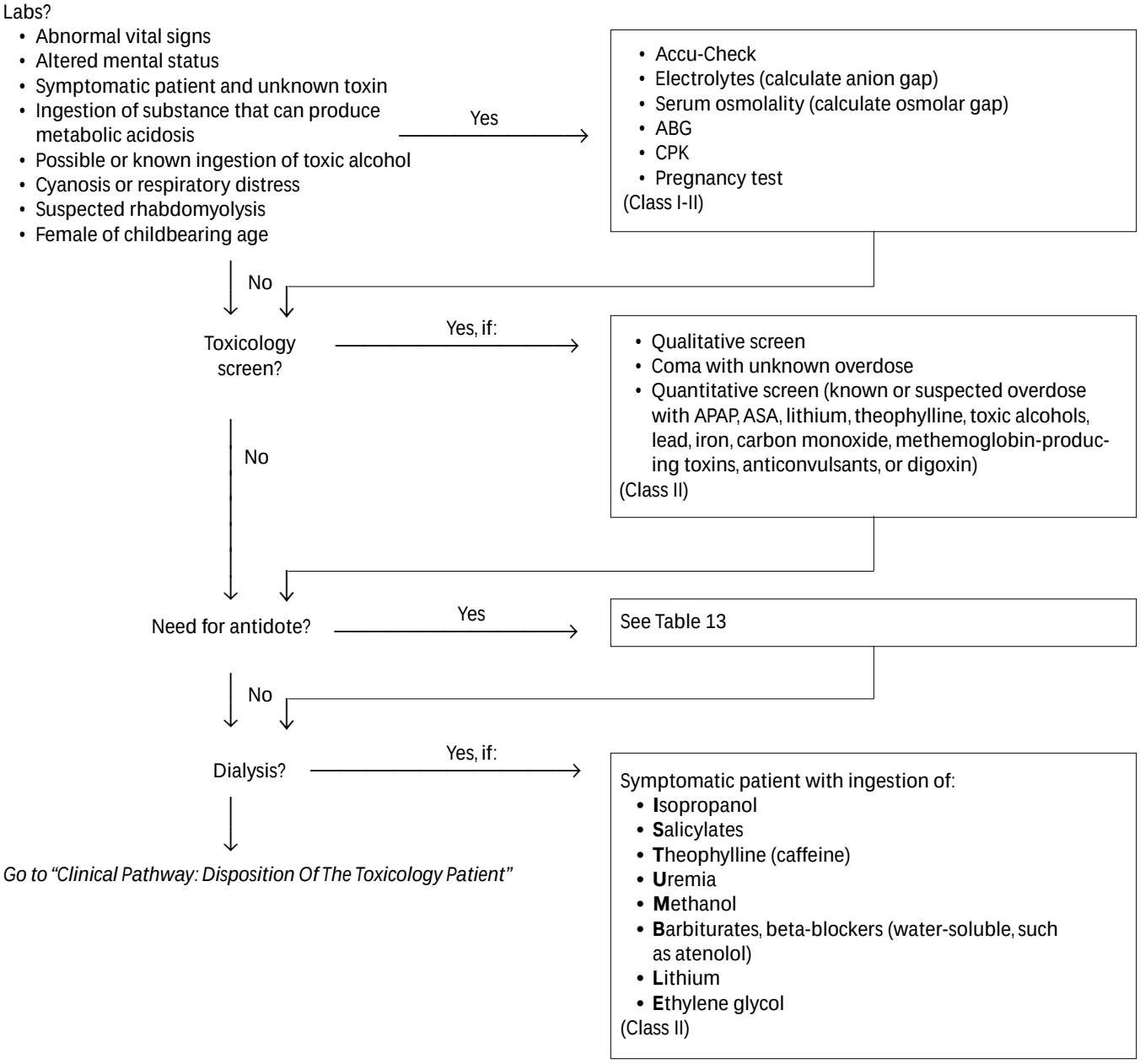
Clinical Pathway: Basic Toxicologic Interventions (continued)



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Clinical Pathway: Disposition Of The Toxicology Patient

- Unstable vital signs?
- Potentially lethal overdose?
- Cardiotoxic overdose?

Yes → ICU admission (Class I-II)

No ↓

- Moderately symptomatic patient with low potential for fatal outcome?

Yes → Hospital admission (Class II)

No ↓

- Overdose with delayed or sustained-release properties (calcium-channel antagonists, theophylline, lithium, methadone, Lomotil, MAOIs, and oral hypoglycemics)?

Yes → Prolonged observation or admission (12-24 hours in the asymptomatic patient) (Class III)

No ↓

- Mildly symptomatic patient, low-lethality ingestion?
- Asymptomatic patient, unknown ingestion?

Yes → ED observation for 4-6 hours (Class II-III)

No ↓

- Suicidal patient?

Yes → Psychiatric consult on inpatient or outpatient basis depending upon suicidal risk and home situation (Class I-II)

No ↓

- Reliable patient and non-toxic overdose?

Yes → Short or no observation and discharge home (return for worsening)

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water). The latter offers the added benefit of making the charcoal more palatable.

While a single dose of a cathartic is usually well-tolerated, repetitive dosing can lead to serious complications. Large doses of sorbitol, especially in the very young or old, have been associated with electrolyte imbalance and dehydration.[113] Magnesium-containing cathartics may cause hypermagnesemia, particularly in patients with renal insufficiency. Cathartics should be avoided in patients with severe diarrhea, ileus, recent bowel surgery, and electrolyte imbalance. Also avoid the use of sodium cathartics in patients with renal or cardiac failure.[6]

Whole Bowel Irrigation

Originally used to cleanse the gut before surgical or endoscopic procedures, whole bowel irrigation has recently been adopted for gut decontamination after certain ingestions.[7,132] Although volunteer studies have demonstrated decreased bioavailability of certain toxins using whole bowel irrigation, there is no conclusive evidence that this intervention improves the clinical outcome of poisoned patients. It may be effective for large ingestions of iron, heavy metals, lithium, and other drugs poorly adsorbed by activated charcoal. It may also be useful for sustained-release or enteric-coated products not well adsorbed to charcoal. Whole bowel irrigation can remove drug-filled packets or other potentially toxic foreign bodies.

The technique employs a large volume of polyethylene glycol solution to clean the gut by mechanical action without fluid or electrolyte shifts. The solution can be given orally (1-2 L/h in adults, 25 cc/kg in children), but because most patients refuse to drink adequate volumes, administration by nasogastric tube is often required. Continue irrigation until the rectal effluent is clear. If the procedure is performed correctly, the gut should empty almost completely in 4-6 hours. Whole bowel irrigation is usually well-tolerated by most patients and has been used safely in children.

Antidotes

Effective antidotes are limited in number, and they are not for indiscriminate use. (See **Table 13**.) Paracelsus accurately observed that all substances are potentially toxic (including antidotes), and that only the dose differentiates a poison from a cure. Employ antidotes carefully, particularly in the patient with an unknown ingestion or overdose, as misuse may complicate the clinical situation. In weighing the benefits and risks of giving a particular antidote, consider the patient's clinical status, laboratory values, the expected pharmaceutical action of the toxin, and possible adverse reactions associated with the antidote. The clinician should be familiar with the indication, availability, dose, and potential adverse effects of specific antidotes.[133]

Be sure that your hospital has the necessary antidotes to care for common poisonings. In one survey of 82 hospitals, fewer than 10% stocked all 14 common antidotes, and even fewer had adequate supplies of Crotalid antivenin, digoxin-specific Fab antibodies, or pyridoxine.[133]

Enhanced Elimination

Methods of enhanced elimination include urine alkalinization and extracorporeal measures such as hemodialysis and hemoperfusion. Alkalinization promotes excretion of weakly acidic agents through ion trapping at the renal tubules. Urinary alkalinization with sodium bicarbonate may be useful in salicylate overdoses. In salicylate overdoses, alkaline urine will increase drug elimination by fourfold for each unit increase in urine pH.[134] To alkalinize the urine, give the patient 1-2 mEq/kg of sodium bicarbonate over five minutes and then begin a bicarbonate drip. The drip is prepared by adding three ampules of sodium bicarbonate to 850 cc D_5W plus 20-40 mEq of potassium. The drip can be started at 150-250 cc per hour and adjusted based on the urinary pH. Target the urine pH to 7.5-8.0.[135] In this process, it is important to monitor the potassium, as the hypokalemic patient will not develop alkaline urine without adequate potassium stores. Although alkalinization significantly lowers the serum half-life of phenobarbital, whether it actually improves clinical outcome and decreases the length of hospital stay is unclear. A comparison study of healthy volunteers who ingested phenobarbital showed that multiple-dose charcoal was superior to urinary alkalinization.[136]

Urinary acidification has been recommended in the past for phencyclidine or amphetamine toxicity, but it is dangerous and may precipitate myoglobinuria and rhabdomyolysis.[137] Therefore, urine acidification is never indicated.

Hemodialysis

In the unstable overdose patient, consultation with a

Table 13. Antidotes And Their Indications.

Antidote	Toxin
N-acetylcysteine	Acetaminophen, possibly carbon tetrachloride
Ethanol/4-MP	Methanol/ethylene glycol
Oxygen/HBO	Carbon monoxide
Naloxone/nalmefene	Opioids
Physostigmine	Anticholinergics
Atropine/pralidoxime	Organophosphates
Methylene blue	Methemoglobinemia
Nitrites and thiosulfate, hydroxycobalamin	Cyanide
Deferoxamine	Iron
BAL (chelating agent)	Arsenic
Succimer (chelating agent)	Lead, mercury, arsenic
Fab fragments	Digoxin, colchicine, crotalid
Glucagon	ß-blockers
Sodium bicarbonate	Tricyclic antidepressants and other sodium-channel blockers
Calcium/insulin/ dextrose/glucagon	Calcium-channel antagonists

nephrologist may be indicated before definitive diagnostic studies or drug levels become available. This is particularly important when the suspected agent is a salicylate, lithium, theophylline, or toxic alcohol. (See **Table 14**.) Hemodialysis will enhance removal of substances with low protein binding, small volumes of distribution, high water solubility, and low molecular weight. Charcoal hemoperfusion is useful for theophylline, barbiturate, and carbamazepine overdose.[138,139] (See **Table 15**.)

Special Circumstances: High-Risk Poisoned Patients

Pediatric Poisonings

There has been a 95% decline in the number of poisoning deaths in children younger than 6 years over the past few decades, with 450 reported deaths in 1961 and 24 in 1999.[10,140] Child-resistant product packaging, heightened parental awareness of potential household toxins, and more sophisticated medical interventions have all helped reduce morbidity and mortality. Still, two-thirds of poisonings reported to the American Association of Poison Control Centers occur in individuals younger than 20 years. Most exposures in this age group are accidental ingestions and result in minimal toxicity.[10]

As with the adult patient, history includes the toxin or medication to which the child was exposed, the time of the exposure or ingestion, what other medications were available to the child, and how much was taken. It is prudent to assume the worst-case scenario. Although considered minimally toxic in adults, some medications in small doses (even one pill) may be potentially fatal in a child.[141-143] These agents include cyclic antidepressants, calcium-channel antagonists, camphor, benzocaine, Lomotil, chloroquine, methylsalicylate, and oral hypoglycemics.

In the pediatric patient, there is currently no role for syrup of ipecac in the emergency setting. As with adults, gastric lavage *may* be indicated in the poisoned child presenting within one hour of exposure to a potentially life-threatening agent. Airway protection by endotracheal intubation prior to lavage may be necessary if the child has a depressed level of consciousness. The majority of poisoned children who are not critically ill can be managed safely and effectively in the ED setting with charcoal alone. Cathartic agents should be used with extreme caution (if at

Table 14. Toxins Accessible To Hemodialysis.

I STUMBLE
- **I**sopropanol
- **S**alicylates
- **T**heophylline (caffeine)
- **U**remia
- **M**ethanol
- **B**arbiturates, beta-blockers (water soluble, such as atenolol)
- **L**ithium
- **E**thylene glycol

Table 15. Enhanced Elimination By Charcoal Hemoperfusion.

- Theophylline
- Barbiturates
- Carbamazepine

Ten Excuses That Don't Work In Court

1. "The patient said she only took one pill—how was I supposed to know she ingested the whole bottle?"

Patients who overdose can be unreliable historians—particularly if they are suicidal, psychotic, or using recreational drugs. Always assume the worst when estimating the potential amount of drug ingested or abused. Four to six hours of observation is prudent in the case of an unknown overdose.

2. "That patient is a frequent flyer to our ED. He's always intoxicated, and we never work him up with a bunch of labs."

This attitude is a set-up for a morbidity/mortality conference or medical/legal case. Just when you think it's another ethanol intoxication, the patient presents acidotic (e.g., methanol/ethylene glycol ingestion) or with an intracranial catastrophe (e.g., subdural hematoma). Re-examine such patients frequently enough to determine that their mental status is improving and not deteriorating.

3. "The patient looked great, so I thought one hour of observation was enough."

With the majority of accidental nontoxic ingestions, 2-6 hours' observation time may be adequate. However, with ingestions of potentially life-threatening or sustained-release medications, up to 24 hours of observation may be indicated.

4. "The patient was too belligerent to examine fully."

While the overdose patient may be "difficult to deal with," a complete physical examination is essential. All too often, these patients are placed in rooms away from the central area. The combative patient may require physical or chemical restraints.

5. "The patient refused any form of gastric decontamination."

Most poisoned patients want nothing to do with gastric decontamination. However, if the patient ingested a potentially life-threatening amount of toxin and presents within one hour, some form of decontamination is indicated. Usually, these patients can be convinced to swallow activated charcoal (a nasogastric tube being their less-appealing alternative). Forcing gastric lavage on a combative patient may result in significant morbidity and is highly discouraged.

6. "The guy overdosed on heroin and woke right after naloxone. He cursed at me, so I kicked him out."

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all), as excessive use can result in dehydration and electrolyte imbalances. Whole bowel irrigation is safe in children and may be indicated in ingestions of iron, lead paint chips, and, rarely, button batteries.

If a child has ingested or been exposed to a potentially dangerous amount of toxin, is manifesting mild-to-moderate toxicity, requires antidote therapy, or the child's home environment is not considered safe, a general pediatric or ICU admission is indicated.[140] Furthermore, children with seemingly small overdoses of potentially life-threatening toxins[141,142] may require more prolonged observation. In the case of accidental ingestions, it is important that parents be taught prevention strategies. When child abuse is suspected, order a social service referral and file a report with local child protective services.

Geriatric Patients

Geriatric patients may be taking multiple medications that result in acute or chronic toxicity or that interact in adverse ways. Poisoning should always be considered in any geriatric patient presenting with altered mental status, cardiac symptoms, GI complaints, or acid-base disorder. Often, elderly patients have a worse prognosis due to preexisting cardiopulmonary disease states, hepato-renal compromise, and considerable delays in diagnosis.

Immunocompromised Patients

Patients with cancer or HIV may be on several toxic drugs. Often, these agents are under investigational protocols awaiting FDA approval with underreported side effects and drug interactions. In addition, these types of patients are potentially depressed and suicidal secondary to their chronic disease states.[144,145] HIV patients may also be taking isoniazid to treat or prevent tuberculosis. This is highly toxic when taken in overdose and can result in status seizures, rhabdomyolysis, and acidosis.[146]

Pregnancy

Pregnant patients may be suffering depression secondary to an untimely pregnancy or may take an overdose to induce an abortion.[59] In these scenarios, if you treat the mother, you will be treating the fetus (e.g., HBO for CO poisonings, NAC for APAP overdoses, deferoxamine for iron toxicity). Do not withhold treatment in a symptomatic pregnant woman for fear of fetal toxicity. Both maternal and fetal deaths have been reported in cases where antidotes or appropriate aggressive interventions were withheld over concerns of potential teratogenicity or fetal toxicity.[147] The need for antidotal treatment may be especially important to protect the fetus from toxicity associated with acetaminophen and carbon monoxide.[148,149]

Medicolegal Issues

In the suicidal or intoxicated patient who refuses care, be vigilant about documenting the level of competency and evaluate the potential for suicide. A competent, non-suicidal adult who is fully informed of the risk of his or her decision may refuse treatment—even potentially lifesaving measures. Suicidal patients (adult or child) are deemed incompetent by law and lose their right to refuse medical treatment.[150-152]

Ten Excuses That Don't Work In Court (continued)

Narcotic abusers are never pleased when their "high" has been abruptly terminated. However, because the clinical effects of heroin may outlast the counteractive properties of naloxone, convince them to stay for several hours of observation. If they refuse, determine whether they are competent to leave, warn them of the risks, and above all, *document everything*.

7. "We didn't have the proper antidote in our hospital, so we couldn't give it to the patient."

Many hospitals are not fully stocked with every state-of-the-art antidote. However, if the patient needs a specific antagonist, the clinician must either locate a hospital or poison center that can deliver the antidote, or the patient should be transferred to a more comprehensive treatment center.

8. "We don't have a nephrologist at our institution, so we could not dialyze the patient."

Several potentially life-threatening toxins (e.g., toxic alcohols, salicylates, lithium) may ultimately require hemodialysis if the patient is in critical condition. If the hospital does not have an adequate renal service, the patient may require transfer to another medical center with dialysis capabilities. While awaiting transfer, consider intermediate/transition treatment options such as fomepizole or an ethanol drip for the toxic alcohols and alkalinization for salicylate poisoning.

9. "The toxicology screen was negative, so it couldn't have been an overdose."

While qualitative toxicology screens are very sensitive and specific for detecting the more commonly abused drugs (e.g., cocaine, amphetamines, PCP), a negative toxicology screen does not rule out a toxic exposure. Most screens will not detect many of the newer designer drugs. Furthermore, the timing of the screen may not correlate with the timing of the ingestion or use (i.e., the screen is obtained too early or late in the clinical course).

10. "I thought the child was okay since she only took one pill."

Be careful. There are several toxins that can kill a small child if only one teaspoon or pill is ingested. These include TCAs, methylsalicylate, calcium-channel antagonists, and hypoglycemic agents. When in doubt, assume the worst-case scenario, and admit these patients for close observation. ▲

The emergency physician must also prevent the patient from further self-harm. This may mean restraining the patient who took an overdose and providing a sitter to watch him or her in the ED. It is ironic that whenever pill bottles are brought to the ED after an overdose, they are almost always placed on the table within reach of the suicidal patient.

One common problem regarding competency involves the narcotic abuser who is revived with naloxone. Frequently, such patients are not interested in remaining in the ED for further monitoring and state that "they have places to go and things to do." Since the half-life of most opioids is longer than the half-life of naloxone, there is a potential for recurrent respiratory depression. Some physicians argue that the patient who wakes from naloxone is no longer under the influence of the narcotic and must be allowed to make a competent (although possibly stupid) decision to leave against medical advice. Others believe that patient safety is paramount and hold the patient against his or her will for several hours of observation—a possibly risky strategy from a legal standpoint.

One recent study suggests that it may be safe to allow patients to leave against medical advise after awakening with naloxone. In this one-year observational study, over 300 prehospital patients received naloxone and refused further treatment. None went on to die from their overdose.[153]

Controversies/Cutting Edge

The newest toxicology antidotes include 4-methylpyrazole (4-MP, Antizol) for ethylene glycol and methanol poisoning,[154,155] specific immune therapy with purified Fab fragments for rattlesnake envenomation,[156] and high-dose insulin and dextrose rescue for refractory calcium-channel antagonist toxicity.[143] Current controversies include challenging the widely accepted 72-hour oral NAC treatment course for acetaminophen toxicity, suggesting a more abbreviated regimen.[157] Also, a recent large Australian study questions the dogma of whether hyperbaric oxygen therapy is beneficial in CO poisoning.[158]

Disposition

Asymptomatic patients with a potentially serious overdose should be observed for 4-6 hours before discharge.[159-161] The six-hour limit may have been based on reports of delayed complications in patients who ingested TCAs.[162] Recent data suggest that the period of observation may be safely shortened in some asymptomatic patients. In one multicenter study of 260 overdose patients, no patient who was believed to be safe for medical clearance at either two or four hours had a complication within the six-hour time period.[161] Possible exceptions to this rule may include agents with delayed or sustained-release properties such as calcium-channel antagonists, theophylline, lithium, methadone, Lomotil, MAOIs, and oral hypoglycemics. Since these overdoses may require prolonged observation, consulting a Poison Control Center may be valuable.[163]

If signs or symptoms of intoxication develop during observation, admit the patient for further observation and treatment. Although many patients admitted will require observation in the ICU, some can be managed on the general medical floor or in an observation unit. Consulting with a medical toxicologist at a Poison Control Center can help to determine appropriate disposition.

All patients with intentional poisoning should have a psychiatric evaluation (although, depending on the circumstances, this can be arranged on an outpatient basis). Actively suicidal patients should be admitted and closely observed. Those with a substance abuse problem should be considered for drug counseling. Among motivated individuals, substance abuse counseling can reduce future drug use by 44%.[164]

Poison Control Centers

Poison Control Centers have had a positive impact on the management and prevention of poisonings in the general population. Specific health and economic benefits of regional poison centers have included: 1) reduction of unnecessary ED visits and inappropriate use of medical resources; 2) reduction in the time required to diagnose and establish definitive care for the poisoned victim; 3) minimization of public health effects of community exposure to toxic materials; 4) reduction in unintentional poisoning in the home and workplace; and 5) education of other healthcare professionals in poison management.[165,166]

Summary

Because the presentation and clinical course of poisoned patients can vary tremendously, it is important for emergency physicians to maintain a high index of suspicion for overdose. Likewise, it is necessary to remain vigilant throughout the patient encounter, as the clincial scenario may change rapidly.

Despite the diagnostic and therapeutic challenges, with appropriate diagnosis and treatment, most patients have a good prognosis. A thorough evaluation, early intervention, and appropriate management are key. ▲

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in the paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

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Physician CME Questions

17. Activated charcoal will adsorb all of the following medications *except:*
a. ferrous sulfate.
b. phenobarbital.
c. theophylline.
d. verapamil.
e. salicylates.

18. Bradycardia is commonly associated with all the following overdoses *except:*
a. clonidine.
b. digoxin.
c. propanolol.
d. methadone.
e. amphetamines.

19. Hyperthermia is often seen with all of the following overdose situations *except:*
a. ethanol withdrawal.
b. oral hypoglycemics.
c. salicylates.
d. phencyclidine.
e. cocaine.

20. A high anion gap metabolic acidosis would be anticipated in each of the following toxic ingestions *except:*
a. ethylene glycol.
b. salicylates.
c. isoniazid.
d. lithium.
e. iron.

21. A comatose patient with an acute exposure to an unknown toxin should receive all of the following therapeutic interventions *except:*
a. flumazenil.
b. naloxone.
c. oxygen.
d. thiamine.
e. dextrose (or Accu-Check).

22. Mydriasis, tachycardia, urinary retention, diminished bowel sounds, and dry mucous membranes would be expected for all of the following ingestions *except:*
a. Jimson weed.
b. tricyclic antidepressants.
c. diphenhydramine.
d. amphetamines.
e. cogentin.

23. All of the following ingested toxins have been found to be radiopaque *except:*
a. ferrous sulfate.
b. acetaminophen.
c. lead.
d. mercury.
e. cocaine packets.

24. Whole bowel irrigation is recommended in all of the following ingestions *except:*
a. lead paint chips.
b. cocaine packets.
c. button batteries.
d. hydrocarbons.
e. sustained-release lithium tablets.

25. All of the following toxins are correctly matched with their respective antidote *except:*
a. cyanide / methylene blue.
b. isoniazid / pyridoxine.
c. ethylene glycol / 4-methylprazole.
d. carbon monoxide / oxygen.
e. acetaminophen / N-acetylcysteine.

26. All of the following drugs can cause miotic pupils *except:*
a. MDMA.
b. clonidine.
c. organophosphates.
d. heroin.
e. codeine.

27. Who is considered the Renaissance "Father of Toxicology"?
a. Nostradamus
b. Hippocrates
c. Paracelsus
d. Leonardo da Vinci

28. All of the following toxins may result in an elevated osmolar gap *except:*
a. isopropanol.
b. mannitol.
c. ethylene glycol.
d. ethanol.
e. isoniazid.

29. All of the following are acceptable routes of naloxone administration *except:*
a. intramuscular.
b. subcutaneous.
c. intravenous.
d. intranasal.
e. via an endotracheal tube.

30. All of the following toxins are properly matched with their associated odor *except:*
a. cyanide / bitter almonds.
b. methylsalicylate / oil of wintergreen.
c. naphthalene / mothballs.
d. mercury / garlic.
e. sulfur dioxide / rotten eggs.

31. The finding on chest x-ray of noncardiogenic pulmonary edema has been associated with all of the following overdoses *except:*

a. isopropanol.
b. heroin.
c. barbiturates.
d. salicylates.
e. meprobamate.

32. Dialysis is recommended with all of the following toxins in the setting of severe overdose *except:*

a. theophylline.
b. methanol.
c. iron.
d. salicylates.
e. lithium.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives an alpha-numerical score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case-control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA* 1992;268(16):2289-2295.

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