

EMERGENCY MEDICINE PRACTICE
AN EVIDENCE-BASED APPROACH TO EMERGENCY MEDICINE

TOXICOLOGY DIAGNOSIS AND MANAGEMENT:
A RATIONAL APPROACH TO THE POISONED PATIENT

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*“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)

Introduction

Toxic overdose can present with a great variety of clinical symptoms, including nausea and vomiting, altered mental status, seizures, cardiac dysrhythmias, and respiratory depression. These may be the only clues to diagnosis when the cause of toxicity is unknown at the time of initial assessment and management. The diagnosis may be complicated by the possibility that the patient has taken multiple drugs.

The prognosis and clinical course of recovery of a patient poisoned by a specific agent depends largely on the quality of care delivered within the first few hours in the emergency setting. 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

Although several published position statements (3-7) and practice guidelines or consensus statements (8) exist regarding clinical toxicology diagnosis and management, the majority of the literature is based on retrospective case series analysis or isolated case reports (class IIb evidence) with isolated animal/bench research. Well-controlled, randomized, human trials with adequate sample sizes are infrequent.

Regional Poison Centers data exists and is updated on an annual basis to document changing trends in poisoning epidemiology. This national database is predominantly presented in a retrospective fashion.(9) However, it is important to note that the majority of severe cases resulting in death never arrive to hospitals (ie- medical examiner cases). Therefore, published studies based on poison center data is skewed towards mild-moderate poisonings and may misrepresent a small, but important segment of toxic agents. (10) Unfortunately, well designed forensic, toxicology data is limited in the literature.

Epidemiology and Etiology

In the year 1999, over 2.2 million human exposures to toxins were reported to the American Association of Poison Control Centers. (9) Over 75% were reported from the home and 13% from a health care facility. Two thirds of the reported exposures involved pediatric patients under 20 years of age. The leading agents were cleaning substances (10%), followed by analgesics, and cosmetics/personal care products. There were 873 poisoning fatalities reported with children under 6 yrs of age representing only 3% of these deaths. The leading fatal agents were analgesics, antidepressants, cardiovascular drugs, stimulants and street drugs.

Pathophysiology

As described by the Renaissance toxicologist Paracelsus, any substance should be considered as a potential poison dependent on the dose and duration of exposure. Once exposed, the pharmacokinetic movement of a poison through the human body can be described in terms of its absorption, distribution, and elimination. Toxicokinetics is used to describe the absorption, distribution, and elimination of poisonous substances at doses that produce clinical toxicity. In the overdosed patient, toxicokinetic concepts are most often used in the interpretation of drug concentrations in plasma or urine. Toxicokinetics may also be used to predict the onset of symptoms and duration of toxicity. (11)

Differential Diagnosis

Any symptomatic patient can be a potential drug overdose. Altered mental status, GI complaints, cardiovascular compromise, seizures, temperature-related disorders can all be toxin-related. Some are subtle such as the “flu-like” symptoms seen with CO poisoning, while cardiotoxins such as digitalis, may mimic intrinsic heart disease. In the differential diagnosis, the clinician should also consider similar agents—with acetaminophen think aspirin, with methanol also consider ethylene glycol, with digitalis also add B-blockers and calcium channel antagonists to the list of possible responsible culprits.

Prehospital Care

Apart from basic stabilization measures, (oxygen administration, cardiac monitoring and venous access establishment) paramedics should do very little in the field with the overdosed patient; particularly if the transport time to the nearest hospital is short. Regarding gastric decontamination, paramedics should avoid giving ipecac, due to delayed effects and potential aspiration in the comatose or combative patient. Some clinical trials have studied the efficacy of pre-hospital charcoal administration (12,13) documenting some clinical efficacy. In a patient with a depressed mental status paramedics should check a rapid serum glucose, and administer intravenous dextrose when necessary. Small doses of naloxone may be required if opiates are highly suspected and the patient

is hypoxic or suffering airway compromise. Benzodiazepines can be given for toxic-induced seizures. Pre-hospital intravenous sodium bicarbonate administration for known TCA overdoses demonstrating a widened QRS complex on the cardiac monitor may also be indicated.

History

Historical facts should include the type of toxin or toxins, time of exposure (acute versus chronic), amount taken, and route of administration (eg- ingestion, intravenous, inhalation). Also inquire as to why the exposure occurred (accidental, suicide attempt, euphoric reasons, therapeutic misadventure). Also ask of prior suicide attempts or psychiatric history. Question the patient about all drugs taken, including prescription, over the counter medications, vitamins, and herbal preparations. Toxic patients can be unreliable historians, particularly if suicidal, psychotic, presenting with altered mental status, or under the influence of recreational drugs (14-16). If unavailable from the patient, information solicited from family and friends may also prove helpful. Although issues of confidentiality may arise, it is advisable to err on the side of acting in the patient's best interest. Paramedics or EMTs are also 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.

(See Table A for common drugs of abuse and their respective street names)

In some cases, it may be worthwhile to send someone back to the scene to look for clues or a suicide note. Ask about the nature and progression of signs and symptoms. Further history can be obtained by consulting the patient's other physicians or by obtaining old medical records. In case of occupational exposure, obtain a description of the work environment and contact persons at the site for relevant information.

Information regarding specific toxins may also prove useful. For example with hydrocarbons, did the child have a coughing spell upon ingestion? With a caustic ingestion, did the child drool or vomit? Following an iron ingestion, did the patient experience bloody emesis, with tricyclic antidepressants, did the patient have any seizure activity? With carbon monoxide exposure, did the patient lose consciousness?

Physical Examination

In the emergency setting, performing an overly detailed physical examination is a low priority compared with patient stabilization. However, even a directed examination can yield important diagnostic clues. Once the patient is stable, a more comprehensive physical examination can reveal additional signs suggesting a specific poison. Additionally, a dynamic change in clinical appearance over time may be a more important clue than a single exam.

Vital Signs.

In many cases, the clinician may be able to deduce the class of drug or toxin taken simply by addressing the patient's vital signs. Mnemonics and phrases may help narrow the differential diagnosis when the patient has signs such as tachycardia, hyperthermia or hypotension.

(See Table 1).

Neurologic exam.

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 "patchy" neurologic impairment. Toxicologic causes of coma rarely cause focal neurologic deficits. These findings, along with a prolonged comatose state, loss of midbrain papillary function, and decerebrate or decorticate posturing should prompt the clinician to rule-out an intracranial process. (17) The often quoted Glasgow Coma Scale, while useful in head trauma victims, has little role in predicting the prognosis of the poisoned patient (18,19).

Seizures are a common presentation of unknown overdose, and the lists of toxins that can induce a convulsion is lengthy (see table 2). Classic papillary findings include miosis (opioids) and mydriasis (sympathomimetic agents). (see table 3). Nystagmus suggests phenytoin or PCP, along with carbamazepine, lithium, ethanol, barbiturates and sedative hypnotics. Rotary nystagmus suggests PCP toxicity whereas vertical nystagmus represents a brainstem lesion until proven otherwise. Thiamine depletion, found in Wernicke's disease, produces ophthalmoplegia. Optic neuritis and vision loss, while seen in multiple sclerosis, may indicate advanced methanol poisoning. Other general neurologic signs include fasciculations, (organophosphate poisoning), rigidity, (tetanus and strychnine), tremors, (lithium and methyl-xanthines), speech-mumbling (anticholinergics) and dystonic posturing, (neuroleptic agents).

Skin.

A careful examination of the skin should not be overlooked. 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 and sympathomimetic 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 axilla as a result of prolonged immobility. They may be associated with any sedative hypnotic drug-induced coma, but are classically described with barbiturate poisoning. (20) One of the most common skin findings is needle tracks, suggesting intravenous or subcutaneous opiate or cocaine abuse. Blue skin indicates methemoglobinemia or hypoxia; (determine central cyanosis versus acrocyanosis). Red skin may suggest niacin, or boric acid exposure. (table 4).

Odors.

Some poisons produce odors characteristic enough to suggest the diagnosis, such as oil of wintergreen (methyl-salicylates), or garlic (organophosphate insecticides). Some smells may be more subtle, as with the bitter-almond scent associated with cyanide (missed by approximately 50% of the population). (21) Certain odors may be overpowering and easily noted by anyone managing the patient. For example, sulfur dioxide and hydrogen sulfide produce a strong rotten egg smell. (see table 5).

Laboratory Tests

Routine tests.

Several simple, readily available laboratory tests may provide important diagnostic clues in the symptomatic overdosed 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 child-bearing age, a pregnancy test is essential since these patients often overdose for suicidal or abortifacient reasons. (22)

To check for anion gap metabolic acidosis, calculate the anion gap using serum mEq/L measurements:

$$\text{Na} - (\text{Cl} + \text{HCO}_3)$$

Although 8 to 12 mEq/L is traditionally accepted as the normal range for an anion gap, the measured and calculated anion gap can vary considerably. (23) When a patient presents with an elevated anion gap, the mnemonic METALACID GAP will assist in identifying most of the common toxic causes (see table 6). In addition, knowledge of the dynamic relationship between the rise in anion gap and the fall in bicarbonate is also important. (ie- Δ AG – Δ HCO₃). (24) If positive and greater than 6, a metabolic alkalosis is usually manifested. A difference is less than 6, suggests that a hyperchloremic acidosis is present.

When a patient presents with an unexplained metabolic acidosis, a serum osmolality should be measured and the osmolar gap calculated. 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 commonly determined by the following formula:

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

An osmolar gap of greater than 10 mOsm/kgH₂O has been arbitrarily defined as increased, and indicates the presence of an unknown low-molecular-weight, osmotically active substance in the serum. (25) The mnemonic ME DIE will help recall the major toxins that produce an increased osmolar gap (see table 7). While an increased gap may be helpful, a “normal gap” (ie-less than 10 mOsm) does not absolutely exclude substances known to be osmotically active. For example, ethylene glycol has such a large molecular weight that even toxic amounts may contribute minimally to a patient’s overall osmolality. Furthermore, depending on the amount of metabolism, which correlates with the time of ingestion, little parent compound may be present when a patient presents with toxicity (26). As a result, the range of a normal gap may vary from –5 to 15 mOsm, depending on the equation used to determine the gap and the time after the ingestion the calculation was made. Thus, although the osmolar gap can lead an important diagnostic clue while awaiting more definitive serum levels, the sensitivity and specificity of this gap is variable. (27) As with the calculation of ETOH/4.6, if quantitative serum levels of the other toxic alcohols are not readily available, these levels can also be estimated by using the following denominators in the above equation: methanol: 3.2; ethylene glycol: 6.2; and isopropanol: 6.0 (25)

Toxicology screens.

The majority of toxicologic diagnoses and therapeutic decisions are still made on a clinical or historical basis, even though technology has provided the ability to measure many toxins. The application of these laboratory measurements are limited by practical considerations. Laboratory turnaround time is often longer than the critical intervention time course of an overdose, and hospitals cannot support the cost of maintaining the procedures, instruments, training, and specialized labor that would be needed to analyze every toxin. (28)

While commonly ordered in a “shotgun” fashion, toxicology screens have their limitations. Most limited immunoassay screens are capable of detecting commonly abused drugs such as marijuana and cocaine. However, many other common dangerous drugs and poisons, such as isoniazid, digitalis glycosides, calcium antagonists, beta blockers, heavy metals, and pesticides, are not routinely included. Therefore, a negative screen does not rule out the possibility of poisoning. On the other hand, some drugs that present in therapeutic amounts, such as opioids and benzodiazepines, may be detected by the screen even though they are causing no contributing clinical symptoms. Additionally, 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 (28-31). Most laboratories do not report a drug unless it has been confirmed by two procedures or by a single method that is known to be highly specific (eg-GC-MS). Immunoassays are most widely used for discrete analysis, and gas chromatography techniques are used for broad screens.

The toxicology screen may have little clinical correlation if specimens are collected too early or late for detection. In general, urine specimens are more useful than blood because drug metabolites in the urine can be detected as long as two to three days after exposure compared with 6-12 hours in the blood. A comprehensive urine toxicology screen is labor intensive and intended to detect as many drugs as possible using common techniques. Usually detected 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 physicians in evaluating the patient if the diagnosis remains unclear. (32)

Quantitative blood tests should be ordered only for those drugs for which blood levels will predict subsequent toxicity or guide specific therapy, (eg- acetaminophen, salicylate, theophylline, lithium, lead, iron, carbon monoxide, methemoglobin, toxic alcohols, anticonvulsants and digoxin). For unknown ingestions, a routine quantitative serum acetaminophen level is recommended since this is agent is contained in many OTC preparations and in overdose may not exhibit early diagnostic clues. (15) Although some sources advocate the analysis of gastric contents, this is usually reserved for forensic cases.

Urine analysis.

Detailed laboratory urine analysis may reveal important diagnostic clues concerning the overdosed patient. Calcium oxalate crystals are considered pathognomonic for ethylene glycol poisoning. However, these are usually discovered late in the clinical course, and may be absent in the urine if early after ethylene glycol ingestion or not detected at all if timely therapy has been instituted.(33). Additionally, past sources suggest the use a Wood’s lamp to detect urine fluorescence following an ethylene glycol ingestion (34). However, a more recent trial refutes the utility of this technique citing numerous false positive results. (35). Urinalysis showing occult blood, with no evidence of red blood cells, suggests myoglobinuria or hemolysis consistent with sympathomimetic drugs. A positive ferric chloride test usually indicates the presence of phenothiazines, salicylates, or ketones.

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 monitoring bicarbonate therapy for salicylate overdose.

Radiologic Studies

Abdominal films.

A KUB radiograph can reveal radiopaque pills, drugfilled packets or condoms, or other toxic material. Drugs or toxins that are likely to be visible on films can be recalled by the mnemonic COINS: They include Chloral hydrate and cocaine packets; Opiate packets; Iron and other heavy metals such as lead, arsenic, and mercury; Neuroleptics; and Sustained-release or enteric-coated preparations, which may be only faintly visible (see table 8). (36)

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 this reason, in cases of body packers (aka- drug smugglers), a KUB may be clinically useful. Whereas, body stuffers, who quickly “swallow the evidence,” the majority of KUB films are negative for foreign body detection (37) For many slightly radio dense 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.

In practice, the only prescription medication worth looking for on KUB is iron. In these cases, (as with lead paint chips), serial KUBs may be used to monitor response to gastric decontamination, such as whole bowel irrigation.

Chest Films.

Patients with tachypnea, coma or obtundation should have radiograph to search for potential causes of hypoxemia, including chemical or aspiration pneumonia, cardiogenic or noncardiogenic pulmonary edema, and atelectasis. Drugs that can cause noncardiogenic pulmonary edema can be remembered by the mnemonic MOPS: Meprobarnate and methadone; Opiates; Phenobarbital; Propoxyphene; and Salicylates (see table 9). Chest films are also useful for detecting pneumothorax or pneumomediastinum in patients abusing cocaine or other sympathomimetic agents.

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 is also useful for anticipating other symptoms that may occur. Cholinergics, anticholinergics, sympathomimetics, and narcotics all have characteristic toxidromes, and withdrawal from many addictive agents will produce its own distinctive constellation of symptoms (see table 10). 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 clinical effects may negate each other and cloud the clinical picture. In addition, the onset of specific toxic complications can be delayed (24). Toxidrome recognition can also improve the efficiency of drug screening when these findings are communicated to laboratory personnel. (38)

Supportive Measures

Management of any clinically significant poisoning should begin with basic supportive measures.

ABCs.

While the majority of patients with poisoning are awake and have stable vital signs, some may present unconscious, hemodynamically unstable, 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. In many cases, intubation, with supplemental oxygen or assisted ventilation, may be required. The patient’s oxygenation status can be monitored with bedside pulse oximetry. However, in cases of specific toxins, the pulse oximetry may be read as normal despite the fact the patient is severely poisoned. This is particularly true in carbon monoxide poisoning where the pulse ox is unreliable in estimating O₂Hb saturation and should therefore be interpreted cautiously (39). Also, the pulse oximetry does not assess the patient’s acid-base status, and therefore, cannot be substituted for an arterial blood gas or serum bicarbonate level.

In symptomatic overdose 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, as some toxins will produce delayed symptoms (eg- hypotension, or seizures) that may make later intravenous access difficult.

Whether or not the patient is unconscious or hemodynamically compromised on arrival in the emergency department, 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. Frequent or continuous cardiac monitoring along a 12 lead electrocardiogram is indicated following exposure to any potential cardiotoxin, for example, sympathomimetic agents, cyclic antidepressants, digitalis, B-blockers, calcium channel antagonists, antihypertensive agents, arsenic, cyanide and carbon monoxide. Unlike patients with more classic cardiopulmonary disease states (CHF, COPD, Asthma) where the progression of respiratory compromise is more predictable and familiar, 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.

“Coma cocktail”.

Hypoglycemia, may be caused by a toxic agent or malnutrition. Give 50% dextrose to all comatose patients, unless a rapid finger stick glucose assessment demonstrates euglycemia or hyperglycemia. While some sources caution against giving a hypertonic glucose bolus to any patient with stroke or cerebral ischemia, this concern is probably unwarranted. (40) (41).

Naloxone, a specific opioid antagonist, may have both therapeutic and diagnostic value, as patients with opioid overdoses usually become fully awake soon after its administration. Give 0.4 to 2 mg IV if you suspect narcotic abuse, and clinical signs are consistent with the opioid toxidrome (42). Tolerant or chronic abusers typically require smaller amounts of the antidote for effect. One may need to go as high as 10 mg in patients who have taken a resistant opioid such as diphenoxylate/atropine, propoxyphene, pentazocine, or codeine, although the literature on this dosing is largely anecdotal (43), (44), (45). If possible, restrain the patient before giving naloxone, particularly with a chronic or “hard core” abuser, as the antidote can precipitate acute opiate withdrawal, causing the patient to become belligerent and even violent.(46) Fortunately, the drug has a half-life of only 60 to 90 minutes, and withdrawal symptoms wear off in one of two hours. The dosage should be titrated until the desired response is achieved. With longer-acting opioids, an intravenous naloxone drip may be required. The drip is classically mixed with 2/3rds the dose required to awaken the patient, given per hour, IVPB. However, this dose is widely variable, depending on the patient’s time of exposure and tolerance. If necessary, naloxone can also be given intralingually or endotracheally. There is also a study recommending twice the dose subcutaneously when IV access is delayed, with equal clinical efficacy (47)

Longer-acting opioid antagonists, such as nalmefene, are now available. (48, 49) These agents have a half-life of approximately ten hours. Nalmefene has been shown to reverse opioid intoxication for as long as 8 hours theoretically reducing the need for continuous monitoring of intoxicated patients and repeated doses of naloxone. However, the long duration of action may cause unnecessary extended withdrawal reactions in chronically opioid-dependent patients. (50) Therefore, cautious and limited use in the ED is recommended 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. (40) In addition, intravenous thiamine carries a small, but real risk of anaphylaxis. (51)

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.” (52-56)

Physostigmine, while formerly considered part of the “coma cocktail”, is contraindicated in comatose patients with an unclear cause. This antidote is only indicated in cases of severe anticholinergic poisoning. It is contraindicated with tricyclic antidepressant overdoses since it may actually exacerbate cardiotoxicity. (57)

Skin And Eye Decontamination

Data regarding proper decontamination methods are limited, but fundamental principles can be found in military chemical battlefield and radiation accident protocols (58). If possible, Haz-mat decontamination is best performed in the pre-hospital 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 (58). 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 alkali 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)

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. (59)(60) Numerous experimental and clinical trials have examined gastric emptying techniques, but their overall effectiveness remains limited. Regardless of the method of gastric decontamination, a significant amount of toxin is not removed, available for absorption. (61)

Ipecac-induced emesis.

Once the preferred technique for gastric emptying, syrup of ipecac is no longer recommended in the emergency department. 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, with limited documented support, ipecac still may have a role in the domestic setting. A 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 health care facility is delayed. (3,62-64)

Gastric lavage.

In the early 1800's, Edward Jukes, a British surgeon performed gastric lavage on himself following an ingestion of landanum (a tincture of opium). Other than mild GI complaints followed by a brief 3 hour nap, he survived with no adverse side effects. The experiment was documented as a success. (103) Present-day, 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 250mL of warm fluid such as tap water or normal saline (in a child, use 10mL/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)(64-69) However, even in this scenario, there is no solid evidence that its use improves clinical outcome. While some clinicians anecdotally claim that delayed lavage (beyond one hour) may be efficacious when the ingested drug can cause either slowing of peristalsis, (eg- anticholinergics or opioids), or formation of large concentrations, (eg- salicylates), there are no well- designed trials which support this opinion. Whether gastric lavage is of clinical benefit in decontaminating hemodynamically unstable patients with an unknown type and time of ingestion, remains unproven.

Gastric lavage should not be performed 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. (5,59-62, 64-70)

The 19th century French pharmacist PF Tourey, rather ahead of his time, demonstrated 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 was under appreciated by his peers. (72) Much 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) As a result, giving oral activated charcoal has become first-line treatment for patients who have ingested a potentially toxic amount of drug. However, its routine administration in nontoxic ingestions is not indicated. As with gastric lavage, the effectiveness of activated charcoal decreases with time, and 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 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 gram (or 1gm/kg) loading dose in adults, a more accurate dose of charcoal is to provide at least a 10:1 ratio of AC to toxin (61). If this ratio cannot be achieved in one single dose, then serial dosing may be required. 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 25grams, every 2-4 hours, without a cathartic, is recommended in the adult. Some of the drugs removed by repeat-dose activated charcoal can be recalled by the mnemonic ABCD: Antimalarials (quinine), Aminophylline (theophylline), Barbiturates (phenobarbital) Carbamazepine; and Dapsone (see table 11). Although controversial, there are currently no good clinical trials supporting the use of multiple dosing of activated charcoal to increase elimination of drugs such as amitriptyline, digitalis, digoxin, phenytoin or salicylate. A trial using a dog model investigated lavage versus charcoal versus 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. (73)

Those substances not well adsorbed by charcoal can be recalled by the mnemonic PHAILS: Pesticides; Hydrocarbons; Acids, and alkali; Iron; Lithium; and Solvents. Activated charcoal also appears to be most efficacious and safe decontamination method when the ingested substance is unidentified.

Adverse side effects of activated charcoal administration, while rare, include aspiration pneumonitis in the unprotected airway, bowel obstruction and perforation. (74,75)

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. Furthermore, the administration of a cathartic alone has no role in the management of the poisoned patient. The most popular cathartics are magnesium sulfate (10% solution @4ml/kg) and sorbitol (35% solution @ 4ml/kg; diluted 1:1 with water). The latter offers the added benefit of making charcoal more sweet and 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 (61). 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 use of sodium cathartics in patients with renal or cardiac failure. (6)

Whole bowel irrigation. (7,76)

Originally used to cleanse the gut before surgical or endoscopic procedures, whole bowel irrigation has recently been adopted for gut decontamination after certain ingestions. Although volunteer studies have demonstrated decreased bioavailability of certain toxins using WBI, there is currently no conclusive evidence that this intervention improves clinical outcome of poisoned patients. It may be effective for large ingestions of iron, heavy metals, lithium, and other drugs poorly adsorbed to activated charcoal. It may also prove useful for sustained-release or enteric-coated products not well adsorbed to charcoal. Whole bowel irrigation can also be used to 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 liter/hr in adults, 25cc/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 four to six 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 are not for indiscriminate use (see table 12). Paracelsus, truly 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 overuse often complicates the clinical situation. In weighing the benefits and risks of giving a particular antidote, consider the patient's clinical status, laboratory values, the expected pharmacologic action of the toxin, and possible adverse reactions associated with the antidote. The clinician should be familiar with the availability and indication of specific antidotes (77)

Enhance Elimination

Methods of enhanced elimination include urine alkalinization and extracorporeal measures such as hemodialysis and hemoperfusion. Urinary alkalinization with sodium bicarbonate may be useful in salicylate overdoses. 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. Alkalinization promotes excretion of weakly acidic agents through ion trapping at the renal tubules. Urinary acidification has been recommended in the past for phencyclidine and amphetamine toxicity, but it is dangerous, and contraindicated as it may precipitate myoglobinuria and rhabdomyolysis.(78) Therefore, urine acidification is never indicated.

Hemodialysis.

In the unstable overdosed patient, consultation with a 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 13). 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. (see table 14). (79,80)

Special circumstances (High risk poisoned patients):

Pediatric Poisonings

There has been a 95% decline in the number of poisoning deaths in children <6 years of age over the past few decades, with 450 reported deaths in 1961 and 24 in 1999. (9,81). Child-resistant product packaging, heightened parental awareness of potential household toxins, and more sophisticated medical intervention at the poison control and emergency and intensive care levels have all contributed to reduce morbidity and mortality. Two thirds of poisonings reported to the American Association of Poison Control Centers occur in individuals less than 20 years. Most exposures in this age group are accidental ingestions and result in minimal toxicity. (9)

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 worse case scenario. Although considered minimally toxic in adults, some medications in small doses may be potentially fatal in a child. (82-84) These agents include: cyclic antidepressants, calcium channel antagonists, camphor, benzocaine, lomotil, chloroquine, and methylsalicylate.

As mentioned above, several clinical trials have been conducted to determine which gastric decontamination modalities are most efficacious. However, the investigations either involve adult volunteers taking sub-toxic amounts who receive decontamination at a set post-ingestion time, or involve mildly to moderately poisoned patients, excluding patients with significant overdoses. Very few children have been included in these trials.

In the pediatric patient, there is currently no role for syrup of ipecac in the EM setting. However, it may have limited use in the home when the ingestion is very recent, the child is alert, the poison is not a caustic or hydrocarbon, the toxin is not well absorbed by activated charcoal, and the hospital transport time is delayed. As with adults, gastric lavage may be indicated in the poisoned child presenting within one hour of a potentially life-threatening agent. In this setting, if the child has a depressed level of consciousness, airway protection by endotracheal intubation prior to lavage may be necessary. 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 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 button batteries.

If a child has ingested or been exposed to a potentially dangerous dose 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.(81) Furthermore, children with seemingly small overdoses of potentially life-threatening toxins (82-83) 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 may be taking several medications resulting in acute or chronic toxicity along with potential adverse drug interactions. Poisoning should always be considered in any geriatric patient presenting with an altered mental status, cardiac symptoms or GI complaints. Often times, the elderly patient has poorer prognosis due to preexisting cardiopulmonary disease states, hepato-renal compromise and considerable delays in diagnosis.

Immunocompromised patients such as oncology and AIDS victims, may be on several organotoxic 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. (85) (86)

Pregnant patients may be suffering depression secondary to an untimely pregnancy or often overdose for abortifacient reasons. (22) In these scenarios, if you treat the mother, you will be treating the fetus- (eg- HBO for CO poisonings, NAC for APAP overdoses, deferoxamine for iron toxicity) Both maternal and fetal deaths have been reported in cases where antidotes or appropriate aggressive interventions have been withheld over concerns of potential teratogenicity or fetal toxicity (87)

Medical legal issues:

In the suicidal or intoxicated patient who refuses care, be vigilant about documenting the level of competency. A competent, nonsuicidal 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 and medical treatment. (88), (89), (90)

Controversies/ Cutting Edge

The newest toxicology antidotes include 4-methylpyrazole (4-MP, Antizole) for ethylene glycol and methanol poisoning (91)(92) specific immune therapy with purified Fab fragments for rattlesnake antivenin (93), and high dose insulin and dextrose rescue for refractory calcium channel antagonist toxicity.(84) Current controversies include challenging the widely accepted 72 hour oral NAC treatment course for acetaminophen toxicity, suggesting a more abbreviated regimen. Also, a recent large scale Australian study questions whether hyperbaric oxygen therapy is indicated (at all) in carbon monoxide poisoning (95)

Disposition (96-98)

Persons with a potentially serious overdose should be observed for several hours before discharge. If signs or symptoms of intoxication develop during that time, admit the patient for further observation and treatment. Exceptions to this rule include agents with delayed or sustained-release properties (calcium channel antagonists, theophylline, lithium, methadone, Lomotil, MAOIs, and oral hypoglycemics). These overdoses often require up to 24 hours of continuous observation.(99) 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. Suicidal patients need to be admitted and closely observed. Those with a substance abuse problem should be considered for drug counseling.

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 emergency department 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 health care professionals in poison management. (100-101)

Summary

The toxic patient can be challenging both diagnostically and therapeutically. Oftentimes medical legal issues arise regarding competency of the uncooperative overdosed patient. Clinical toxicology requires hyper-vigilant detective work and can be very rewarding to the clinician and lifesaving to the patient. It is wide-open field in regards to potential evidence-based clinical research.

Table A: Drugs of Abuse and Street Names

<u>Marijuana</u>	<u>Heroin</u>	<u>Cocaine</u>
Acapulco Gold	Boy	All American drug
Bhang	China White	Coke
Doobie	Dust	Crack
Ganja	Harry	Girl
Grass	Horse	Mother of Pearl
Joint	Junk	Nose Candy
Mary Jane	Monkey	Peruvian Powder
Pot	Smack	Snow
Rope	Speed ball (with cocaine)	Toot
Reefer	Atom bomb (with MJ)	White Lady
<u>Amphetamines</u>	<u>PCP</u>	<u>LSD</u>
Black Beauties	Angel Dust	Acid
Crank	Goon	Blotters
Crystals	Horse Tranquilizer	Microdots
Cat (Methcathinone)	Hog	Paper Acid
Ice	Sherman	Pyramids
Ecstasy	Tank	Window Pane
Love Drug	Wickie Stick (with MJ)	Zen
Meth		
Pep Pills		
Smart Drug (Ritalin)	<u>GHB</u>	
Speed	Bioski	
Uppers	Georgia Home Boy	
XTC	Grievous Bodily Harm	
	Liquid G	
	Liquid Ecstasy	
	Somatomax	
	Cow Growth Hormone	

Table 1: DIAGNOSING TOXICITY FROM VITAL SIGNS

<p>Bradycardia (PACED) Propranolol or other beta blockers, poppies (opiates) Anticholinesterase drugs Clonidine, calcium channel blockers Ethanol or other alcohols Digoxin</p>	<p>Hypotension (CRASH) Clonidine, calcium channel blockers Reserpine or other antihypertensive agents Antidepressants, aminophylline Sedative-hypnotics Heroin or other opiates</p>
<p>Tachycardia (FAST) Free base or other forms of cocaine Anticholinergics, antihistamines, amphetamines Sympathomimetics (cocaine, amphetamines), solvent abuse Theophylline</p>	<p>Hypertension (CT SCAN) Cocaine Thyroid supplements Sympathomimetics Caffeine Anticholinergics, amphetamines Nicotine</p>
<p>Hypothermia (COOLS) Carbon monoxide Opiates Oral hypoglycemics, insulin Liquor Sedative-hypnotics</p>	<p>Rapid respiration (PANT) PCP, paraquat, pneumonitis (chemical) ASA and other salicylates Noncardiogenic pulmonary edema Toxin-induced metabolic acidosis</p>
<p>Hyperthermia (NASA) Neuroleptic malignant syndrome, Nicotine Antihistamines Salicylates, sympathomimetics Anticholinergics, antidepressants</p>	<p>Slow respiration (SLOW) Sedative-hypnotics Liquor Opiates, sedative-hypnotics Weed (marijuana)</p>

Table 2: Agents That Cause Seizures (OTIS CAMPBELL*)

<p> Organophosphates Tricyclic antidepressants Isoniazid, insulin Sympathomimetics Camphor, cocaine Amphetamines Methylxanthines (theophylline, caffeine) Phencyclidine (PCP) Benzodiazepine withdrawal, botanicals (water hemlock), G H B Ethanol withdrawal Lithium, lidocaine Lead, lindane </p>
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**Famous T.V. "town drunk" on the Andy Griffith Show*

Table 3: AGENTS THAT AFFECT PUPIL SIZE

<p> Miosis (COPS) Cholinergics, clonidine Opiates, organophosphates Phenothiazines, pilocarpine, pontine hemorrhage Sedative-hypnotics </p> <p> Mydriasis (AAAS) Antihistamines Antidepressants Atropine and other anticholinergics Sympathomimetics </p>

Table 4: AGENTS THAT CAUSE SKIN SIGNS

<p> Diaphoretic skin (SOAP) Sympathomimetics Organophosphates Acetylsalicylic acid or other salicylates Phencyclidine </p>	<p> Flushed or red appearance Anticholinergics Boric acid Carbon monoxide (rare) Cyanide (rare) </p>
<p> Dry Skin Antihistamines, anticholinergics </p>	<p> Cyanosis Ergotamine Nitrates Nitrites Aniline dyes Phenazopyridine Dapsone </p> <p> Any agent causing hypoxemia, hypotension, or methemoglobinemia. </p>
<p> Bullae Barbiturates and other sedative-hypnotics </p>	
<p> Acneiform rash Bromides Chlorinated aromatic hydrocarbons </p>	

Table 5: ODORS THAT SUGGEST THE 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

**Table 6: 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
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Table 7: AGENTS INCREASING THE OSMOLAR GAP (ME DIE)

Methanol Ethylene glycol Diuretics (mannitol) Isopropyl alcohol Ethanol

Table 8: AGENTS VISIBLE ON ABDOMINAL FILMS (COINS)

Chloral hydrate, cocaine packets, calcium Opium packets Iron, other heavy metals such as lead, arsenic, mercury Neuroleptic agents Sustained-released or enteric coated agents
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Table 9: DRUGS CAUSING PNEUMONITIS OR PULMONARY EDEMA (MOPS)

Meprobamate, methadone Opiates Phenobarbital, propoxyphene Salicylates

Table 10: COMMON TOXIDROMES

<p>Cholinergic (organophosphates) (DUMBELS)</p> <p>Diarrhea, diaphoresis Urination Miosis Bradycardia, bronchosecretions Emesis Lacrimation Salivation</p>	<p>Narcotic (heroin, methadone)</p> <p>Miosis Bradycardia Hypotension Hypoventilation Coma</p>
<p>Anticholinergic (antihistamines, tricyclic antidepressants)</p> <p>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 and retention</p>	<p>Withdrawal (from alcohol, opioids, benzodiazepines, barbiturates, antihypertensives)</p> <p>Diarrhea Mydriasis Goose Flesh Tachycardia Lacrimation Hypertension Yawning Cramps Hallucinations Seizures (with ETOH and benzodiazepine withdrawal)</p>
<p>Sympathomimetic (cocaine, amphetamines)</p> <p>Mydriasis Tachycardia Hypertension Hyperthermia Seizures</p>	

Table 11: AGENTS RESPONSIVE TO MULTIPLE DOSES OF ACTIVATED CHARCOAL

<p>Adsorbable (ABCD) Antimalarials (quinine), Aminophylline (theophylline) Barbiturates (phenobarbital) Carbamazepine Dapsone</p> <p>Not adsorbable (PHAILS) Pesticides Hydrocarbons Acids, alkali Iron Lithium Solvents</p>

Table 12: ANTIDOTES AND THEIR INDICATIONS

Antidote	Indication (agent)
N-acetylcysteine	Acetaminophen
Ethanol / 4-MP Oxygen / HBO	Methanol/Ethylene glycol
Naloxone / Nalmefene	Carbon monoxide
Physostigmine	Opioids
Atropine/2-PAM	Anticholinergics
Methylene blue	Organophosphates
Nitrites	Methemoglobinemia
Deferoxamine	Cyanide
BAL (chelating agent)	Iron
Succimer (chelating agent)	Arsenic
Fab fragments	Lead, Mercury
Glucagon	Digoxin, Colchicine, Crotalids
Sodium bicarbonate Calcium / Insulin / Dextrose	Beta Blockers
	Tricyclic antidepressants Calcium channel antagonists

Table 13: TOXINS ACCESSIBLE TO HEMODIALYSIS (ISTUMBLE)

Isopropanol Salicylates Theophylline Uremia Methanol Barbiturates Lithium Ethylene glycol
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Table 14 ENHANCED ELIMINATION BY CHARCOAL HEMOPERFUSION

Theophylline Barbiturates Carbamazepine

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Educational Objectives

- Form a differential diagnosis after presentation of clinical scenarios involving toxicology
- Discuss the approach to management of a variety of specific poisonings including antidote use.
- Identify the utility of laboratory data and other aids for diagnosis of poisoned patients
- Determine by critical evaluation the validity of previously rigid standards of care in toxicology.
- Evaluate the role of various poisoning treatment modalities using evidence-based medicine.

CME Questions

- 1) Activated charcoal will absorb all of the following medications except:
 - a) Ferrous sulfate (*)
 - b) Phenoarbutal
 - c) Theophylline
 - d) Verapamil
 - e) Salicylates
- 2) Bradycardia is commonly associated with all the following overdoses except:
 - a) Clonidine
 - b) Digoxin
 - c) Propranolol
 - d) Methadone
 - e) Amphetamines (*)
- 3) Hyperthermia is often seen with all of the following overdose situations except:
 - a) Ethanol withdrawal
 - b) Oral hypoglycemics (*)
 - c) Salicylates
 - d) Phencyclidine
 - e) Cocaine
- 4) A high anion gap metabolic acidosis would be anticipated in each of the following toxic ingestions except:
 - a) Ethylene glycol
 - b) Salicylates
 - c) INH
 - d) Lithium (*)
 - e) Iron
- 5) 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
- 6) 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
- 7) All of the following ingested toxins have been found to be radiopaque except:
 - a) Ferrous sulfate
 - b) Acetaminophen (*)
 - c) Chloral hydrate

- d) Mercury
e) Cocaine packets
- 8) The finding on chest x-ray of noncardiogenic pulmonary edema have been associated with all of the following overdoses except:
- a) Isopropanol (*)
b) Heroin
c) Barbiturates
d) Salicylates
e) Meprobamate
- 9) 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
- 10) All of the following toxins are correctly matched with their respective antidote except:
- | | |
|--------------------|------------------------|
| a) Cyanide(*) | Methylene blue (*) |
| b) INH | Pyridoxime |
| c) Ethylene glycol | 4-methylprazole (4-MP) |
| d) Carbon monoxide | Oxygen |
| e) Acetaminophen | N-acetylcysteine |
- 11) 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
- 12) All of the following drugs can cause miotic pupils except:
- a) MDMA (*)
b) Clonidine
c) Organophosphates
d) Heroin
e) Codeine
- 13) All of the following toxins may result in an elevated osmolar gap except:
- a) Isopropanol
b) Mannitol
c) Ethylene glycol
d) Ethanol
e) Isoniazid (*)
f)
- 14) All of the following are acceptable routes of naloxone administration except:
- a) Intramuscular (IM)
b) Subcutaneous (SQ)
c) Intravenous (IV)
d) Intranasally (IN) (*)
e) Via an endotracheal (ET) tube
- 15) 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 |
| f) | |
- 16) Who is considered the Renaissance Father of Toxicology?

- a) Nostradamus
- b) Hippocrates
- c) Paracelsus (*)
- d) Leonardo de Vinci
- e) Aristotle
- f)

Clinical Pathway:

Gastric Decontamination

For all procedures

Use in a patients with an unprotected airway (Class III)

Gastric Lavage

Patients ingesting a potentially life-threatening amount of a poison, presenting within one hour after an ingestion (Class II b)

Patients ingesting a potentially life-threatening amount of a poison, presenting after one hour after ingestion (Indeterminate)

Patients ingesting a substance that is not absored by activated charcoal (e.g. iron, lithium, lead) (Indeterminate, theoretical benefit)

Syrup of Ipecac (Class III)

Cathartics (Indeterminate)

Activated charcoal

Patients presenting within one hour after ingestion (Class II b)

Patients presenting after one hour after ingestion (Indeterminate)

Multiple Dose Activated Charcoal

Patients suffering from overdoses of the following substances:

II b

- Theophylline
- Phenobarbital
- Carbamazepine
- Quinine
- Dapsone

Indeterminate

- Amitriptyline
- Astemizole
- Chlorpropamine
- Dextropropoxyphene
- Digitoxin
- Digoxin
- Disopyramine
- Doxepin
- Imipramine

- Methotrexate
- Nadolol
- Phenylbutazone
- Phenytoin
- Piroxicam
- Salicylate
- Tobramycin
- Valproate
- Vancomycin

Whole Bowel Irrigation

Indeterminate or Theoretically based:

- Enteric -coated or sustained release drugs
- Substances where activated charcoal is not effective (lead, zinc, iron, lithium (II b))
- Ingested packets of illicit drugs (body stuffers and body packers)

Class I: Definitely recommended

Class II a: Acceptable and useful. Very good evidence provides support

Class II b: Acceptable and useful. Fair-to-good evidence provides support.

Class III: Not acceptable, not useful, may be harmful

Indeterminate: Continuing area of research

13 EXCUSES THAT DON'T WORK IN COURT

- 1) "The patient said she only took one pill- How was I supposed to assume she ingested the whole bottle of meds?"

Patients who overdose can be unreliable historians- particularly if they are suicidal, psychotic or using recreational drugs. Always assume the worst case scenario when estimating the potential amount of drug ingested or abused.

- 2) The 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 (eg-methanol/ethylene glycol ingestion) or with an intracranial catastrophe (eg- subdural hematoma).

- 3) "The patient looked great, so I thought 1 to 2 hours 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 fully examine."

While the overdosed 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 main central area. If overly combative, when necessary, the patient may require physical or chemical restraints.

- 5) "The patient refused any form of gastric decontamination"

Most poisoned patients want nothing to do with any type of gastric decontamination. (Have you ever undergone a round of whole bowel irrigation?) If the patient has ingested a potentially life-threatening amount of toxin and presents within one hour, some form of decontamination is indicated. Usually, these patients can at least be convinced to swallow an adequate dose of activated charcoal. Forcing gastric lavage on a combative patient may result in significant morbidity and is therefore highly discouraged.

- 6) "The guy overdosed on heroin and woke right after given a dose of naloxone- He was alert, but angry and demanded to be discharge- So I signed him out AMA."

Narcotic abusers are never pleased with your bedside manner when their euphoric "high" has been abruptly interrupted by naloxone administration. However, because the clinical effects of heroin may outlast the counteractive properties of naloxone, be careful to observe these patients until the CNS and respiratory depressive actions of the abused opioid have effectively worn-off. If the clinician does decide to discharge the patient under these circumstances, be vigilant about documenting the patient's adequate level of competency.

- 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 therapeutic 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 (eg- 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, intermediate/transition treatment options such as ethanol drips for the toxic alcohols and alkalization for salicylate poisoning should be considered.

- 9) "We have no access to poison/overdose information."

Even if your medical center does not have a clinical toxicologist on staff, every region of the U.S. has access to a poison center "hotline." In addition, several EDs are equipped with computerized poison information systems or, updated toxicology textbooks.

- 10) "The toxicology screen was negative, so it couldn't have been a drug overdose."

While qualitative toxicology screens are very sensitive and specific for detecting the more commonly abused drugs (eg- 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 (ie- obtained too early or late in the clinical course).

- 11) "The patient was just lying to get away from the police and get out of jail- I really didn't think he 'swallowed the evidence'."

Yet another easy trap to fall into. Many patients do misrepresent the truth in order to avoid police custody- However, when you minimize these types of patients, they can come back to haunt you if their ingested drug packets rupture resulting in severe toxicity.

12) "I knew the patient was unstable, but we withheld treatment because we didn't have the blood level back"

While blood levels for many medications may help guide antidote administration and predict clinical outcome, if the patient is unstable, the clinician may have institute life-saving measures without having the opportunity to interpret a complete set of laboratory results. If treatment is withheld because the "level wasn't back" the patient may not survive.

13) "I thought the child was OK 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 (ie- TCAs, methylsalicylate, calcium channel antagonists). When in doubt, assume the worst case scenario, and admit these patients for close observation.

Cost Effective Strategies for Managing the Poisoned Patient

1. Initiating public education measures stressing poison prevention

The most cost effective means of dealing with the rising costs of managing poisoned victims is to prevent the exposures from ever occurring. This would obviously have an impact on diminishing poison morbidity and mortality.

2. Use of pediatric safety measures ("child proofing" medications containers)

One the most effective means of preventing pediatric poisonings from occurring was the novel introduction of "safety caps" on medications bottles in the early 1970s.

3. Providing easily accessible poison control information.

Free public access to poison information using a toll free telephone number is not only cost effective in the long run, but is a public health necessity. Accessible poison information also assists health care professionals in the early intervention of poisoned patients potentially avoiding costly intensive care unit admissions when definitive management is delayed.

4. Instituting regional poison treatment centers.

This concept is similar to that established for level I trauma centers, where overdosed patients would be bypassed to a tertiary care facility properly staffed to manage more complicated or unstable patients. (102)

5. Avoid "shot gunning" laboratory data with every overdose.

Ordering excessive laboratory tests is commonplace in overdose settings since the offending agent is either unknown or the clinician is unfamiliar with the toxin. Consult the regional poison center or a comprehensive toxicologic data-base in order to narrow the scope of lab acquisitions. In other words, every toxic ingestion does not require liver function tests, thyroid function tests and an amylase.

6. Limiting toxicology qualitative screening panels, particularly when the results will not alter clinical management or patient disposition.

It is a knee-jerk response to order a "tox screen" on every overdose patient. This may not be necessary depending on the ingestion or exposure. This is particularly true when the patient presents with an accurate history of ingestion and has no clinical evidence of exposure to drugs of abuse (ie- the pediatric patient with reliable parents).

7. Using specific serum levels that will actually guide management and help predict outcome.

Rather than ordering nonspecific qualitative drugs screens, quantitative serum levels such as digoxin, theophylline, salicylate, acetaminophen, and carbon monoxide will help direct therapeutic interventions, such as antidote administration, and better document the progress and prognosis of the patient.

8. Tightening antidote administration criteria to avoid unnecessary use of expensive antidotes.

Although the glamorous part of toxicology is to administer life-saving antidotes, it is probably only necessary in 10-15% of all cases as the majority of patients will recover uneventfully with supportive care alone. Specific administration criteria already exist for antidotes such as Digibind for digoxin toxicity, and NAC therapy for acetaminophen overdose. In addition, cavalier administration of antidotes when not indicated can also result in dangerous clinical side-effects, particularly if the offending agent is misdiagnosed.

9. Starting psychosocial interventions earlier in the medical management

This is monumental. Poisoned patients tend to be "repeat offenders" if drugs of abuse are involved or if the patient has a psychiatric history. As with primary care in general, early intervention may not only be life-saving, but extremely cost effective.

10. Use of observation units in the Emergency Department to avoid unnecessary hospital admissions.
The majority of poisoned patients may not require a full admission and may require less than 24 hours of observation. ED observation units already developed for asthma and chest pain patients are a perfect model for overdosed victims.
11. Every overdosed patient does not require an intensive care (ICU) admission.
Admitting all poisoned patients to an ICU setting is an over-used practice. While clearly indicated in an unstable poisoned patient, it more often the result of needing one-on-one nursing for psychiatric patients or a general lack of familiarity with the specific poison. 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.
12. Providing more comprehensive detoxification programs with improved federal and state funding.
The patients who are in the greatest need for psychosocial intervention (such as addiction programs) tend to be the least insured. Federal, state and locally funded programs are key to this intervention.