

The Approach to the Patient with an Unknown Overdose

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Toxic overdose can present with various clinical symptoms, including abdominal pain, vomiting, tremor, 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 of a multiple-drug ingestion.

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 never delay life-saving supportive care, however. 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 if other measures to enhance elimination are necessary [1].

Clinical guidelines

Although several published position statements [2–6] and practice guidelines or consensus statements [7] exist regarding clinical toxicology diagnosis and management, most of the literature is based on retrospective case series

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analysis or isolated case reports with isolated animal or laboratory research. Well-controlled, randomized, human trials with adequate sample sizes are infrequent and difficult to perform.

Regional Poison Control Center data exist and are updated on an annual basis to document changing trends in poisoning epidemiology. This national database is predominantly presented in a retrospective fashion [8]. It is important to note, however, that most severe cases resulting in death never arrive to hospitals (ie, medical examiner cases). Published studies based on poison center data therefore are skewed toward mild to moderate poisonings and may underrepresent a small but important segment of toxic agents [9]. Unfortunately, well-designed forensic toxicology data are limited in the literature. Recently, the poison exposure data of the National Health Interview Survey and the Toxic Exposure Surveillance System have been compared to identify age-adjusted poisoning episode rates to provide a broader perspective [10].

Epidemiology

In the year 2004, more than 2.4 million human exposures to toxins were reported to the American Association of Poison Control Centers [8]. More than 75% were reported from the home and 15% from a health care facility. Two thirds of the reported exposures involved pediatric patients less than 20 years of age. The leading agents were cleaning substances, followed by analgesics and cosmetics/personal care products [8]. There were 1183 reported poisoning fatalities with children less than 6 years of age representing only 2% of these deaths. The leading fatal agents were analgesics, antidepressants, cardiovascular drugs, stimulants, and street drugs [8].

Toxicokinetics

What is it that is not a poison? All things are poison and nothing is without poison. Solely, the dose determines that a thing is not a poison.

—Paracelsus (1493–1541), the Renaissance Father of Toxicology, in his *Third Defense* [11]

As described by the Renaissance toxicologist Paracelsus, any substance should be considered a potential poison depending on the dose and duration of exposure. The pharmacokinetic movement of a xenobiotic through the human body can be described in terms of pharmacokinetics—absorption, distribution, and elimination. Toxicokinetics describes the absorption, distribution, and elimination of xenobiotics at doses capable of producing 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 [12].

Differential diagnosis of the poisoned patient

Any symptomatic patient can be a potential drug overdose. Altered mental status, gastrointestinal complaints, cardiovascular compromise, seizures, and temperature-related disorders can all be toxin-related. Some are subtle, such as the flulike symptoms seen with carbon monoxide poisoning, whereas cardiotoxins, such as digitalis, may mimic intrinsic heart disease. In the differential diagnosis, the clinician should also consider similar agents. For example, the following should be considered together as possible culprits: acetaminophen and salicylates, methanol and ethylene glycol, digitalis and beta-blockers and calcium channel antagonists.

Prehospital care

Apart from basic stabilization measures (such as oxygen administration, cardiac monitoring, and venous access establishment), emergency medical system (EMS) personnel need to do little in the field with the overdosed patient, especially when the transport time to the nearest hospital is short. EMS personnel should avoid giving ipecac because of the possibility of delaying definitive therapy and the potential for aspiration in the comatose or combative patient. Some clinical trials have studied the efficacy of prehospital charcoal administration documenting some clinical efficacy [13–15]. There is still a lack of definitive evidence to advocate the administration of charcoal in the prehospital setting, however. In the patient who has a depressed mental status EMS personnel should check a rapid serum glucose and administer intravenous dextrose when necessary. Small doses of naloxone may be required if opioids are highly suspected and the patient is hypoxic or suffering airway compromise. Benzodiazepines can be given for toxin-induced seizures. Prehospital intravenous sodium bicarbonate administration for known overdoses of cardiac sodium channel blocking agents (ie, cyclic antidepressants, propoxyphene, and cocaine) demonstrating a widened QRS complex on the cardiac monitor may also be considered.

The general approach

The general approach to the diagnosis and management of the poisoned patient can be described using a two-pronged model as seen in Fig. 1. In practice, the two prongs occur simultaneously. The left-sided prong begins with basic emergency medical care—the ABCs (airway, breathing, circulation). The mnemonic *DONT* stands for dextrose, oxygen, naloxone, thiamine. In most potentially poisoned patients, a rapid blood glucose measurement should be obtained and any derangements corrected. Supplemental oxygen, naloxone, and thiamine should be considered in the appropriate cases and situations. The various methods of decontamination should

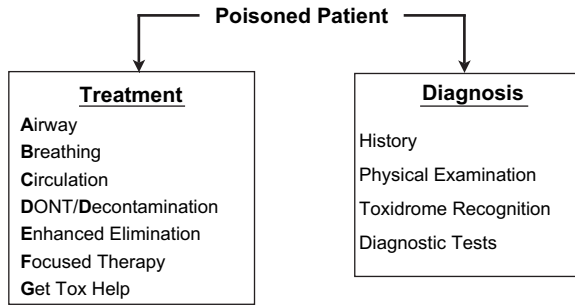


Fig. 1. The two-pronged approach to the poisoned patient.

be considered in any poisoned patient. The exact method used should be based on each individual clinical situation. Once a poisoning has been identified, methods of enhanced elimination should be considered. Focused therapy involves antidote administration when appropriate or aggressive supportive care tailored to the poison in question. Finally, when treating any poisoned patient it is prudent to consider early consultation with a toxicology service or regional poison center for further guidance.

The right-sided prong on the diagram focuses on obtaining the poisoning and other patient history, performing a focused physical examination with attention to toxidrome recognition, and deciding on the appropriate diagnostic tests to be performed. The two prongs often occur simultaneously and are integral to the diagnosis and management of the poisoned patient.

History

Historical facts should include the type of toxin or toxins, time of exposure (acute versus chronic), amount taken, and route of administration (ie, ingestion, intravenous, inhalation). It is also important to understand why the exposure occurred (accidental, suicide attempt, euphoria, therapeutic misadventure) and whether there is history of psychiatric illness or previous suicide attempts. Furthermore, it is important to inquire about all drugs taken, including prescription, over-the-counter medications, vitamins, and herbal preparations. Patients may incorrectly name the drugs they have ingested; for example, they may refer to ibuprofen as acetaminophen or vice versa. Poisoned patients can be unreliable historians, particularly if suicidal, psychotic, presenting with altered mental status, or under the influence of recreational drugs [16–18]. 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 emergency medical technicians are also good sources of information because they may be able to furnish details, such as the presence of empty pill bottles or drug paraphernalia that were at the scene (see [Box 1](#) for common drugs of abuse and their respective

Box 1. Drugs of abuse and street names*Marijuana*

Acapulco gold

Bhang

Doobie

Ganja

Grass

Joint

Mary Jane

Pot

Rope

Reefer

Amphetamines

Black beauties

Crank

Crystals

Cat (Methcathinone)

Ice

Ecstasy

Meth

Pep pills

Smart drug (Ritalin)

Speed

Uppers

Ecstasy

Adam

E

Lollies

Love Drug

Smarties

Vitamin E

XTC

Heroin

Boy

China white

Dust

Harry

Horse

Junk

Monkey
Smack
Speed ball (with cocaine)
Atom bomb (with marijuana)

PCP

Angel dust
Goon
Horse tranquilizer
Hog
Sherman
Tank
Wickie stick (with marijuana)

γ -hydroxybutyrate (GHB)

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

Cocaine

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

LSD

Acid
Blotters
Microdots
Paper acid
Pyramids
Window pane
Zen

street names). In some cases it may be worthwhile to send someone to the scene to look for clues or a suicide note. Emergency personnel should inquire 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 the case of an occupational exposure, personnel should obtain a description of the work environment and contact people at the site for relevant information.

Information regarding specific toxins may also prove useful. For example, the following may be noted following certain ingestions:

- Protracted coughing with hydrocarbon ingestions
- Inability to swallow or drooling with caustic ingestions
- Hematemesis with iron ingestions
- Intractable seizures with isoniazid overdose
- Loss of consciousness with carbon monoxide

Physical examination

In the emergency setting, performing an overly detailed physical examination is a low priority compared with patient stabilization. Even a directed examination can yield important diagnostic clues, however. 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 findings on a single examination. There are classic presentations that occur with specific drug classes. Even these classic presentations may not always be seen, however, and their appearance depends on the dose that the patient has ingested, the patient's premorbid condition, other substances that may have been taken, and complications that may occur in the course of the poisoning (ie, aspiration pneumonia, rhabdomyolysis, and anoxic brain injury). Emergency physicians should be aware of the classic drug syndromes that may occur, but also realize the limitations depending on other confounding factors.

Toxic 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 (**Box 2**) [19,20].

Neurologic examination

A systematic neurologic evaluation is important, particularly with patients exhibiting altered mental status. In contrast to the patient who has structural brain injury, the patient who has a toxic-metabolic cause of coma may exhibit "patchy" neurologic impairment. Toxicologic causes of

Box 2. Diagnosing toxicity from vital signs

Bradycardia (PACED)

Propranolol (beta-blockers), poppies (opiates), propoxyphene, physostigmine
Anticholinesterase drugs, antiarrhythmics
Clonidine, calcium channel blockers
Ethanol or other alcohols
Digoxin, digitalis

Tachycardia (FAST)

Free base or other forms of cocaine, freon
Anticholinergics, antihistamines, antipsychotics, amphetamines, alcohol withdrawal
Sympathomimetics (cocaine, caffeine, amphetamines, PCP), solvent abuse, strychnine
Theophylline, TCAs, thyroid hormones

Hypothermia (COOLS)

Carbon monoxide
Opioids
Oral hypoglycemics, insulin
Liquor (alcohols)
Sedative-hypnotics

Hyperthermia (NASA)

Neuroleptic malignant syndrome, nicotine
Antihistamines, alcohol withdrawal
Salicylates, sympathomimetics, serotonin syndrome
Anticholinergics, antidepressants, antipsychotics

Hypotension (CRASH)

Clonidine, calcium channel blockers
Rodenticides (containing arsenic, cyanide)
Antidepressants, aminophylline, antihypertensives
Sedative-hypnotics
Heroin or other opiates

Hypertension (CT SCAN)

Cocaine
Thyroid supplements
Sympathomimetics
Caffeine

Anticholinergics, amphetamines

Nicotine

Rapid respiration (PANT)

PCP, paraquat, pneumonitis (chemical), phosgene

ASA and other salicylates

Noncardiogenic pulmonary edema, nerve agents

Toxin-induced metabolic acidosis

Slow respiration (SLOW)

Sedative-hypnotics (barbiturates, benzodiazepines)

Liquor (alcohols)

Opioids

Weed (marijuana)

COMA

L: Lead, lithium

E: Ethanol, ethylene glycol, ethchlorvynol

T: Tricyclic antidepressants, thallium, toluene

**H: Heroin, hemlock, hepatic encephalopathy, heavy metals,
hydrogen sulfide, hypoglycemics**

**A: Arsenic, antidepressants, anticonvulsants, antipsychotics,
antihistamines**

R: Rohypnol (sedative hypnotics), risperidone

G: GHB

I: Isoniazid, insulin

C: Carbon monoxide, cyanide, clonidine

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 evaluate for an intracranial process [21]. The **Glasgow Coma Scale (GCS)**, although useful in head trauma victims, has a limited role in predicting the prognosis of the poisoned patient [22,23]. One recent study investigated the use of the **Alert/Verbal/Painful/Unresponsive scale (AVPU)** as a simple rapid method of assessing consciousness in most poisoned patients [24]. Both the GCS and AVPU scales can be used as tools to communicate level of consciousness and for evaluating the need for intubation; however, neither is suitable as a predictor of outcome in the poisoned patient.

Seizures are a common presentation of an unknown overdose, and the list of toxins that can induce a convulsion is lengthy (**Box 3**). Classic pupillary findings include miosis (opioids) and mydriasis (sympathomimetic agents) (**Box 4**). **Nystagmus suggests phenytoin or phencyclidine (PCP)**, along with

Box 3. Agents that cause seizures (OTIS CAMPBELL^a)

Organophosphates, oral hypoglycemics

Tricyclic antidepressants

Isoniazid, insulin

Sympathomimetics, strychnine, salicylates

Camphor, cocaine, carbon monoxide, cyanide, chlorinated hydrocarbons

Amphetamines, anticholinergics

Methylxanthines (theophylline, caffeine), methanol

Phencyclidine (PCP), propranolol

Benzodiazepine withdrawal, botanicals (water hemlock, nicotine), bupropion, GHB

Ethanol withdrawal, ethylene glycol

Lithium, lidocaine

Lead, lindane

^a Famous television "town drunk" on the Andy Griffith Show.

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, although seen in multiple sclerosis, may indicate advanced methanol poisoning. Other general neurologic signs include fasciculations (ie, organophosphate poisoning), rigidity (ie, tetanus and strychnine), tremors (ie, lithium and methylxanthines), speech-mumbling (ie, anticholinergics), and dystonic posturing (ie, neuroleptic agents).

Box 4. Agents that affect pupil size

Miosis (COPS)

Cholinergics, clonidine, carbamates

Opiates, organophosphates

Phenothiazines (antipsychotics), pilocarpine, pontine hemorrhage

Sedative-hypnotics

Mydriasis (SAW)

Sympathomimetics

Anticholinergics

Withdrawal

Skin

A careful examination of the skin should be performed. The patient's clothing should be removed and the skin assessed for color, temperature, and the presence of dryness or diaphoresis. The absence of diaphoresis is an important clinical distinction between anticholinergic and sympathomimetic poisoning. The presence of bites or similar marks may suggest spider or snake envenomations. The presence of a rash or bullae may also help provide a diagnosis. Although uncommon, bullous lesions are typically located on dependent portions of the body, such as between the fingers, knees, axilla, and back, as a result of prolonged immobility. They may be associated with any sedative hypnotic drug-induced coma, but are classically described with barbiturate poisoning [25]. Such lesions could also be indications of rhabdomyolysis or the development of compartment syndrome. A common skin finding is the presence of track marks, suggesting intravenous or subcutaneous opiate or cocaine abuse. Blue skin indicates methemoglobinemia or hypoxia; red skin may suggest niacin or boric acid exposure (Box 5). The skin examination should also include a search for pharmaceutical patches, such as opioids like fentanyl. In cases of drug abuse, these patches may be present in unusual locations, such as the vagina and scrotum.

Odors

Some poisons produce odors characteristic enough to suggest the diagnosis, such as oil of wintergreen (methylsalicylates), or garlic (organophosphate insecticides, arsenic). Some odors may be more subtle, such as the freshly mowed hay smell of phosgene or the bitter-almond scent associated with cyanide, which 30% of the population cannot detect [26]. (It is important to note that bitter almonds, which are not readily available in the United States, have a unique musty odor like dirty tennis shoes when compared with the common, or sweet, almond.) Certain odors may be overpowering and easily noted by anyone managing the patient. For example, sulfur dioxide and hydrogen sulfide produce a noxious rotten-egg smell (Table 1).

Laboratory tests

It is commonplace for many health care providers to order excessive laboratory tests when treating a poisoned patient. This testing often occurs because the offending agent is unknown or the clinician is unfamiliar with the toxin. Consultation with the regional poison center or medical toxicologist may help to narrow the scope of testing.

Routine tests

Several simple, readily available laboratory tests may provide important diagnostic clues in the symptomatic overdosed patient. These include

Box 5. Agents that cause skin signs

Diaphoretic skin (SOAP)

Sympathomimetics

Organophosphates

Acetylsalicylic acid or other salicylates

Phencyclidine

Dry Skin

Antihistamines, anticholinergics

Bullae

Barbiturates and other sedative-hypnotics,

Bites: Snakes and spiders

Acneiform rash

Bromides

Chlorinated aromatic hydrocarbons (dioxin)

Flushed or red appearance

Anticholinergics, niacin

Boric acid

Carbon monoxide (rare)

Cyanide (rare)

Cyanosis

Ergotamine

Nitrates

Nitrites

Aniline dyes

Phenazopyridine

Dapsone

Any agent causing hypoxemia, hypotension, or methemoglobinemia.

measurements of electrolytes, blood urea nitrogen, 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 because these patients often overdose for suicidal or abortifacient reasons [27].

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

Table 1
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
Freshly mowed hay	Phosgene

[28]. When a patient presents with an elevated anion gap, the mnemonic METAL ACID GAP assists in identifying most of the common toxic causes (Box 6). In addition, knowledge of the dynamic relationship between the increase in anion gap and the decrease in bicarbonate is also important ($\Delta AG = \Delta HCO_3^-$) [29]. If positive and greater than 6, a metabolic alkalosis is usually present. A difference of 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 osmolal gap calculated. When an elevated osmolal gap is accompanied by anion gap acidosis immediate consideration should be given to poisoning by methanol, ethylene glycol, and other less common toxic alcohols. The osmolal gap is the difference between

Box 6. Agents causing an elevated anion gap (METAL ACID GAP)

Methanol, metformin, massive overdoses
Ethylene glycol
Toluene
Alcoholic ketoacidosis
Lactic acidosis
Acetaminophen (large overdoses)
Cyanide, carbon monoxide, colchicine
Isoniazid, iron, ibuprofen
Diabetic ketoacidosis
Generalized seizure-producing toxins
Acetylsalicylic acid or other salicylates
Paraldehyde, phenformin

measured serum osmolality (most accurately determined by the freezing-point depression method) 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 osmolal gap of 10 has been arbitrarily defined as normal. Further investigation, however, has revealed that the range of normal is approximately -15 to $+10$ [30]. An increase in the osmolal gap indicates the presence of a low molecular weight, osmotically active substance in the serum. The mnemonic ME DIE describes the major toxins that produce an increased osmolal gap (Box 7). It is important to understand that with toxic alcohols, the parent compound is the osmotically active component; the metabolites are not osmotically active [31]. It is generally accepted that a markedly increased osmolal gap is suggestive of toxic alcohol intoxication. Furthermore, a normal osmolal gap does not rule out toxic alcohol intoxication for several reasons: (1) the patient's baseline osmolal gap is not known; (2) as metabolism of the parent toxic alcohol compound ensues, the osmolal gap narrows (with a concomitant increase in the anion gap); (3) the contribution of any osmotically active compound to the osmolal gap is related to the compound's molecular weight. Compounds with larger molecular weights contribute less to the osmolal gap (eg, ethylene glycol has a relatively large molecular weight and a small increase in the osmolal gap results from toxic ethylene glycol levels) [30–32].

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 [33].

Toxicology screens

Although technology has provided the ability to measure many toxins, most toxicologic diagnoses and therapeutic decisions are made based on historical or clinical information. The application of laboratory measurements is limited by several practical considerations: (1) laboratory turnaround time

Box 7. Agents increasing the osmolar gap (ME DIE)

Methanol

Ethylene glycol

Diuretics (mannitol), Diabetic ketoacidosis (acetone)

Isopropyl alcohol

Ethanol

can often be longer than the critical intervention time course of an overdose; (2) the cost and support of maintaining the instruments, staff training, and specialized labor involved in some analyses are prohibitive; (3) for many toxins there are no established cutoff levels of toxicity, making interpretation of the results difficult [34].

Although commonly ordered in a “shotgun” fashion, toxicology screens have several limitations. Most limited immunoassay screens are capable of detecting commonly abused drugs, such as marijuana and cocaine. Many other common dangerous drugs and poisons, such as **isoniazid, digitalis glycosides, calcium antagonists, beta-blockers, heavy metals, and pesticides, are not routinely included**. A negative screen therefore does not rule out the possibility of poisoning. Conversely, 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 [34–37]. Immunoassays are most widely used for discrete analyses; gas chromatography and mass spectrometry techniques are used for broad screens.

The toxicology screen may have little clinical correlation if specimens are collected too early or too late for detection. Urine drug tests often detect drug metabolites and may remain positive for several days after the exposure. Blood or serum drug tests are generally positive for much shorter time periods. A comprehensive urine toxicology screen is labor intensive and intended to detect as many drugs as possible. The compounds 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 [38]. Care should be given to indiscriminate ordering of such comprehensive tests; the history, physical examination, and common laboratory tests can usually narrow the differential to a few potential culprits. Additionally, potent opioids (eg, fentanyl) or sedative hypnotic agents (eg, rohypnol) may be present, but the detection limits are set too high to produce a positive result [39].

Many clinicians reflexively order a “**tox screen**” on all poisoned patients. **This practice should be avoided**. Qualitative screening panels should be used when the results will alter patient management or disposition.

Quantitative blood tests should be ordered only for those drugs or toxins for which blood levels predict toxicity or guide specific therapy. Such drugs include **acetaminophen, salicylates, theophylline, lithium, lead, iron, carbon monoxide, methemoglobin, toxic alcohols, anticonvulsants, and digoxin**. For unknown ingestions, a routine quantitative serum acetaminophen level is recommended because this agent is contained in many over-the-counter

preparations and in overdose may not exhibit early diagnostic clues [17]. Although some sources advocate the analysis of gastric contents, this is usually reserved for forensic cases.

Urine testing

Detailed laboratory urine analysis may reveal important diagnostic clues concerning the overdosed patient. Calcium oxalate crystals are generally present in ethylene glycol poisoning. These are usually discovered late in the clinical course, however, and may be absent early in the clinical course or not detected at all if timely therapy has been instituted [40]. The Wood's lamp has been used to detect urine fluorescence following ethylene glycol ingestion [41]. More recent studies demonstrate the lack of specificity with this, however [42–44]. Urinalysis showing occult blood, with no evidence of red blood cells, suggests myoglobinuria or hemolysis. Additionally, the urinary pH is an important measurement, especially when monitoring bicarbonate therapy for salicylate overdose.

Urine color may also provide a diagnostic clue. The following are examples:

- An orange to red-orange color with phenazopyridine, rifampin, deferoxamine, mercury, or chronic lead poisoning
- A pink color with ampicillin or cephalosporins
- A brown color with chloroquine or carbon tetrachloride
- A greenish-blue color with copper sulfate or methylene blue

Radiologic studies

Abdominal films

A plain abdominal radiograph (KUB) can reveal radiopaque pills, drug-filled packets, or other toxic material. Drugs or toxins that are likely to be visible on films can be recalled by the mnemonic COINS as demonstrated in Box 8 [45].

In some cases, the vehicle in which the drug is contained, such as an enteric coating or latex, is more radiopaque than the drug itself. For this reason, a KUB may be useful in cases of body packers (drug smugglers). On the other hand, body stuffers who quickly swallow the evidence to evade the authorities typically have abdominal films that are negative for foreign body detection [46]. For many slightly radiodense drugs, such as neuroleptics and salicylates, visibility depends on the time of ingestion. A patient presenting several hours after the ingestion rarely has a positive radiograph. In practice, the KUB is probably most useful to determine the presence of a heavy metal foreign body in the gastrointestinal tract and to monitor the progress of gastrointestinal decontamination (such as whole bowel irrigation).

Box 8. Agents potentially visible on abdominal radiographs (COINS)

Chloral hydrate, cocaine packets, calcium

Opium packets

Iron; other heavy metals, such as lead, arsenic, mercury

Neuroleptic agents

Sustained-release or enteric-coated agents

Chest films

Patients who have tachypnea, hypoxia, obtundation, or coma should have a chest radiograph performed to search for potential causes of hypoxemia: chemical or aspiration pneumonitis, cardiogenic or noncardiogenic pulmonary edema (acute lung injury), and atelectasis. **Drugs that can cause noncardiogenic pulmonary edema can be remembered by the mnemonic MOPS (Box 9).** Chest films are also useful for detecting occasional pneumothorax or pneumomediastinum seen 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 who have unknown overdoses, a toxidrome can assist in making a diagnosis and is also useful for anticipating other symptoms that may occur. Cholinergic, anticholinergic, sympathomimetic, and narcotic agents all have characteristic toxidromes; withdrawal from many addictive agents may also produce distinctive constellations of symptoms [19]. **Box 10** lists the common toxidromes. 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 be present or may negate

Box 9. Drugs causing pneumonitis or pulmonary edema (MOPS)

Meprobamate, methadone

Opioids

Phenobarbital, propoxyphene, paraquat, phosgene

Salicylates

Box 10. Common toxidromes

Cholinergic (Examples: organophosphates, carbamates, pilocarpine)

(DUMBELLS)

Diarrhea, diaphoresis

Urination

Miosis

Bradycardia, bronchosecretions

Emesis

Lacrimation

Lethargic

Salivation



Nicotinic (recalled by the days of the week)

Monday: Miosis

Tuesday: Tachycardia

Wednesday: Weakness

Thursday: Tremors

Friday: Fasciculations

Saturday: Seizures

Sunday: Somnolent

Anticholinergic (Examples: antihistamines, cyclic antidepressants, atropine, benztrapine, phenothiazines, scopolamine)

Hyperthermia (HOT as a hare)

Flushed (RED as a beet)

Dry skin (DRY as a bone)

Dilated pupils (BLIND as a bat)

Delirium, hallucinations (MAD as a hatter)

Tachycardia

Urinary urgency and retention

Sympathomimetic (Examples: cocaine, amphetamines, ephedrine, phencyclidine, pseudoephedrine)

Mydriasis

Tachycardia

Hypertension

Hyperthermia

Seizures

Opioid (Examples: heroin, morphine, codeine, methadone, fentanyl, oxycodone, hydrocodone)

Miosis

Bradycardia

Hypotension

Hypoventilation

Coma

Withdrawal

Diarrhea

Mydriasis

Goose flesh

Tachycardia

Lacrimation

Hypertension

Yawning

Cramps

Hallucinations

Seizures (with alcohol and benzodiazepine withdrawal)

each other and cloud the clinical picture. In addition, the clinical onset of a specific toxic agent may be delayed when multiple substances have been ingested concomitantly. Toxidrome recognition can improve the efficiency of drug screening when these findings are communicated to laboratory personnel [47].

Treatment

The management of any clinically significant poisoning should begin with basic supportive measures. Most poisoned patients do well with supportive care alone.

ABCs

Most poisoned patients are awake and have stable vital signs. Some patients may present with an altered state of consciousness, hemodynamic instability, or active convulsions. The first priority is to stabilize the ABCs and manage life-threatening complications. When intubation is necessary, rapid sequence induction is indicated with a preference toward using short-acting paralytic agents. The emphasis should be placed on short-acting paralytics for fear of masking toxin-induced seizures. For cases in which hyperkalemia or poisoning with cholinesterase inhibitors is suspected, non-depolarizing agents are preferred.

A patient's oxygenation status can be monitored with a bedside pulse oximeter. Certain toxins, however, may demonstrate a normal pulse oximetry reading despite severe poisoning. This observation is particularly true in

carbon monoxide poisoning, in which the pulse oximeter is unreliable in detecting carboxyhemoglobin [48]. The pulse oximeter only provides information regarding oxygen saturation and does not assess acid–base status. It should not be used as a substitute for obtaining an arterial blood gas or serum bicarbonate level when such information is clinically warranted.

Intravenous access should be considered in all potentially poisoned patients. This practice should be maintained even when the patient seems stable and asymptomatic. Many toxins produce delayed effects, such as hypotension or seizures, that could make obtaining intravenous access difficult.

The poisoned patient should be kept under close observation with frequent evaluations of the level of consciousness, oxygenation status, and vital signs. This observation is important, particularly with the patient who presents in a stable condition, because continued absorption of an ingested substance may lead to delayed clinical evidence of poisoning. An electrocardiogram and continuous cardiac monitoring are indicated for any agent with potential cardiac toxicity. Examples include sympathomimetic agents, cyclic antidepressants, cardiac glycosides, beta-blockers, calcium channel antagonists, antihypertensive agents, arsenic, cyanide, carbon monoxide, antidysrhythmics, citalopram, diphenhydramine, venlafaxine, chloral hydrate, methylxanthines, propoxyphene, phenothiazines, and quinine. **An electrocardiogram should also be routinely obtained in patients who have polysubstance ingestions and in cases of unknown suicidal ingestions.** Unlike patients who have chronic cardiopulmonary diseases whose condition worsens progressively, the poisoned patient may unexpectedly become unstable.

The patient who has an unknown poisoning may present convulsing or with a history of seizure activity. Toxin-induced seizures tend to be global central nervous system (CNS) processes as opposed to focal processes like those seen in patients who have epilepsy or CNS structural lesions [49]. Benzodiazepines, barbiturates, and valproic acid, therefore, are considered the first- and second-line therapies for toxin-induced seizures [49]. Phenytoin is generally not effective in treating toxin-induced seizures or seizures from alcohol or sedative-hypnotic withdrawal [49].

The “coma cocktail”

The “coma cocktail” refers to the empiric administration of certain medications or delivery of interventions to patients who present with unconsciousness or coma. The most common components of the coma cocktail are **dextrose, naloxone, and thiamine**. Flumazenil and **physostigmine** are sometimes included also. The approach to the unconscious poisoned patient, however, should be deliberate; empiric therapy should be used with caution.

Many toxins can potentially cause hypoglycemia. A rapid capillary glucose measurement should be obtained in all comatose patients.

Hypoglycemia should be rapidly corrected with a dextrose bolus. In situations in which a capillary glucose measurement cannot be performed, it is reasonable to administer dextrose empirically. Although some sources caution against giving a hypertonic glucose bolus to patients who have acute cerebral ischemia, this concern is probably unwarranted [50,51].

Naloxone, an opiate antagonist, may have therapeutic and diagnostic value. Patients who have opioid overdose usually become fully awake soon after its administration. If the clinical picture is consistent with the opioid toxidrome, 0.4 to 2 mg of naloxone may be given by intravenous infusion [52]. Tolerant or chronic opioid abusers typically require smaller amounts of the antidote for effect. Patients who overdose on certain potent or resistant opioids may need larger doses of naloxone. These opioids include diphenoxylate, propoxyphene, pentazocine, codeine, and fentanyl [53–55]. Although the literature is largely anecdotal, up to 10 mg of naloxone may be required in such cases [53–55]. Naloxone can precipitate acute opiate withdrawal in the opioid-dependent patient. Caution should be exercised because acute withdrawal can be accompanied by belligerence and violence [56]. Fortunately, naloxone has a short half-life, and withdrawal symptoms wear off in 1 or 2 hours. The naloxone dose should be titrated until the desired response is achieved. With long-acting opioids, an intravenous naloxone infusion drip may be required. The hourly infusion rate is two thirds of the dose required to awaken the patient. This dose is variable depending on the time of exposure and the patient's level of tolerance. If necessary, naloxone can be given intralingually, endotracheally, or subcutaneously [57]. Recently, a fatal North American fentanyl epidemic has been described, resulting in medical centers depleting their stock of naloxone [39].

Nalmefene is a long-acting opioid antagonist with a half life of approximately 10 hours. The long-lasting effect could reduce the need for continuous monitoring and repeated dosing of naloxone in opioid-intoxicated patients. The long duration of action, however, may lead to unnecessarily extended withdrawal signs and symptoms in opioid-dependent patients [58]. For this reason, cautious and limited use of nalmefene in the emergency department is warranted.

The administration of thiamine should be reserved for alcoholic patients who are malnourished. Giving thiamine to every comatose patient in an attempt to prevent Wernicke's encephalopathy is probably unwarranted [50]. Additionally, intravenous thiamine carries a small but significant risk for anaphylactoid reactions [59].

Flumazenil, a benzodiazepine antagonist, can rapidly reverse coma in benzodiazepine overdose. The drug may also induce seizures in patients who have mixed drug overdoses, however, such as cyclic antidepressant or sympathomimetic agents; it may provoke acute withdrawal in those addicted to benzodiazepines. Flumazenil should therefore be used judiciously and not administered routinely as part of the coma cocktail [60–64].

Physostigmine should not be empirically administered as part of a coma cocktail in a comatose patient who has an unknown cause. It is indicated in the case of isolated, **severe anticholinergic poisoning**. It is contraindicated with **tricyclic antidepressant** overdoses because it may exacerbate cardiotoxicity [65]. **Recently, physostigmine has been recommended in potential GHB poisoning; however, efficacy in this setting is controversial [66].**

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 [67]. If possible, hazardous materials–type decontamination is best performed in the prehospital setting. In patients who have dermal exposures, all clothing should be removed and the skin copiously irrigated and washed with a mild soap and water. The use of **hot water, strong detergents, or harsh abrasives should be avoided [67].** Decontamination should not be delayed while awaiting identification of the offending agent. Emergency care providers should wear gloves, water-resistant gowns, splash-resistant goggles, and masks to protect themselves from dermal exposure. Ocular exposures to acids and alkali can be devastating. The eye should be copiously irrigated with several liters of normal saline solution and the pH of the conjunctiva closely monitored before starting other therapeutic or diagnostic interventions [7].

Gastrointestinal decontamination

Gastrointestinal decontamination is the process of preventing or reducing absorption of a substance after it has been ingested. Controversy exists concerning the **roles of induced emesis, gastric lavage, activated charcoal, and cathartics in decontaminating the gastrointestinal tract.** Individual circumstances determine which technique is the most appropriate in a given clinical situation [68,69]. Several experimental and clinical trials have examined gastric emptying techniques; their overall effectiveness remains limited. Regardless of the method of gastric decontamination, a significant amount of toxin remains and is available for absorption [70].

Ipecac-induced emesis

Once a preferred technique for gastric emptying, syrup of ipecac is no longer recommended for use in the emergency department [2]. Research has demonstrated no improvement in clinical outcome with its use [2]. Furthermore, the persistent vomiting that often occurs after ingestion of syrup of ipecac may delay other modes of therapy, such as administration of activated charcoal. Currently there is no role for syrup of ipecac in the prehospital management of poisonings. In 2003, the American Academy of Pediatrics issued a statement that ipecac should no longer be used

routinely as a home treatment strategy and that existing ipecac in the home should be disposed of safely [71]. On June 12, 2003, the FDA Nonprescription Drugs Advisory Committee met to discuss whether there is sufficient evidence of the benefits of ipecac syrup to outweigh the potential for misuse, abuse, and adverse effects associated with it as an over-the-counter drug. At the conclusion of the meeting, the committee recommended by a six to four vote that the FDA rescind ipecac's over-the-counter status. There is no reduction in resource use or improvement in patient outcome from the use of syrup of ipecac at home. [72] The American Heart Association First Aid Task Force has made the administration of syrup of ipecac a Class III Action (more harm than benefit).

Gastric lavage

In the early 1800's Edward Jukes, a British surgeon, performed gastric lavage on himself following an ingestion of laudanum (a tincture of opium). Aside from mild gastrointestinal upset followed by a 3-hour nap, he survived with no adverse side effects. The experiment was considered a success [73]. Gastric lavage currently involves inserting a large-bore, 36- to 40-French orogastric tube. The patient should be placed in the left lateral decubitus position with the head of the bed in the Trendelenburg position. The procedure is performed by instilling approximately 250 mL (10 mL/kg in pediatric patients) of water or saline with immediate lavage of that same quantity of fluid. The technique is repeated until the recovered solution is clear of particulate matter or pill fragments [3].

Gastric lavage is no longer indicated for most ingestions [3]. Gastric lavage may be considered if a patient has ingested a potentially life-threatening amount of a toxin and presents within 1 hour of ingestion [3,74–79]. Even in this scenario, however, there is no clear evidence that its use improves clinical outcome. Gastric lavage should not be performed when a patient has ingested a corrosive substance or a volatile hydrocarbon. It should never be used as a punitive measure in cases of nontoxic overdoses or forced on patients who are combative or otherwise uncooperative. Additionally, endotracheal intubation solely to perform gastric lavage is discouraged; the decision to intubate should be independent of the decision to perform gastric lavage. Orogastric lavage is not a benign procedure and has been associated with aspiration, esophageal perforation, epistaxis, hypothermia, and death [3].

Activated charcoal

The nineteenth-century French pharmacist P.F. Tourey 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 but remained

underappreciated by his peers [80]. Recent evidence suggests that activated charcoal is more effective than induced emesis or gastric lavage for gastric decontamination [4]. As a result, activated charcoal administration has become the preferred method of decontamination for most poisons and is most effective when administered early after the ingestion [4]. Its routine administration in nontoxic ingestions is not indicated.

Several activated charcoal products are commercially available. Regardless of the brand, it is important to ensure the activated charcoal is thoroughly resuspended in water to achieve a 25% concentration before use. Although commonly dosed at 1 g/kg body weight, the more appropriate dose is a 10:1 ratio of activated charcoal to toxin [70]. If this dose cannot be achieved in a single dose, then serial dosing may be required.

Although studies have demonstrated reduced drug absorption with activated charcoal use, it is important to note that there is no evidence that administration of activated charcoal improves patient outcome [4]. The use of activated charcoal is contraindicated when airway reflexes are not intact or protected [4].

Multiple dose activated charcoal (MDAC) is a potential method of enhanced elimination. Some drugs undergo enterohepatic and enteroenteric recirculation. MDAC can interrupt enterohepatic and enteroenteric recirculation when such drugs have been absorbed, acting as “gut dialysis.” Box 11 provides a memory aid for the drugs that may be removed using MDAC. There is no standard dose for MDAC administration. Twenty-five grams every 2 to 4 hours is a reasonable regimen. It is important that a cathartic (such as sorbitol) is not administered with MDAC because serious electrolyte abnormalities may result.

Box 11. Agents responsive to multiple doses of activated charcoal

Substances adsorbable by activated charcoal (ABCD)

Antimalarials (quinine), **aminophylline** (theophylline)

Barbiturates (phenobarbital)

Carbamazepine

Dapsone

Substances not adsorbable by activated charcoal (PHAILS)

Pesticides, potassium

Hydrocarbons

Acids, alkali, alcohols

Iron, insecticides

Lithium

Solvents

The substances not well adsorbed by charcoal can be recalled by the mnemonic PHAILS as listed in Box 11. The most common side effect of activated charcoal administration is constipation [4]. When given with sorbitol, gastrointestinal upset with diarrhea may result [4]. Complications of activated charcoal administration, although uncommon, include pneumonitis if aspirated, bowel obstruction, and perforation [81,82]. The reported adverse events following single-dose activated charcoal administration are few, but as with any medical intervention, a risk-to-benefit comparison should be carefully assessed [83].

Cathartics

The efficacy of cathartic use in reducing the absorption or increasing the elimination of toxins has not been established [5]. There are no published data demonstrating an improved outcome with cathartic use alone or combined with activated charcoal [5]. Cathartics, typically sorbitol, are often used with activated charcoal to reduce the gastrointestinal transit time of the toxin-charcoal mixture; however, this has not been shown to improve decontamination efficacy. Cathartic use alone has no role in the management of the poisoned patient [5]. Sorbitol may have a role in increasing charcoal therapy compliance because the sweet taste may make the activated charcoal more palatable.

A single dose of a cathartic, such as sorbitol, is typically well-tolerated. Repetitive dosing, however, can lead to serious complications. Large doses of sorbitol, especially in the extremes of age, have been associated with electrolyte imbalance and dehydration [70]. This finding is particularly important when considering multiple-dose activated charcoal because many commercially-available charcoal products contain sorbitol, and repeated doses of sorbitol may be inadvertently administered. Magnesium-containing cathartics may cause hypermagnesemia, particularly in patients who have renal insufficiency. Cathartic use should be avoided in patients who have diarrhea, ileus, recent bowel surgery, and electrolyte imbalance. Sodium-containing cathartics should be avoided in patients who have renal disease or cardiac failure [5].

Whole bowel irrigation

Whole bowel irrigation (WBI) is the process of using a large volume of a polyethylene glycol solution to clean the gastrointestinal tract by mechanical action without affecting the fluid or electrolyte balance [6]. A similar, although less rigorous, procedure has been well established to prepare patients for surgical or endoscopic procedures. Although volunteer studies have demonstrated decreased bioavailability of certain drugs using WBI, there is currently no conclusive evidence that WBI improves clinical outcome of poisoned patients [6]. In patients who are hemodynamically

stable and have normal bowel function and anatomy, it is reasonable to consider using WBI with ingestions of the following substances: heavy metals, lithium, sustained-release and enteric-coated products, and substances not adsorbed by charcoal. Whole bowel irrigation may also be considered when the patient has ingested drug-filled packets or other potentially toxic foreign bodies.

Whole bowel irrigation is performed by placing a nasogastric tube and administering 1 to 2 L/h (25–50 mL/kg/h in pediatric patients) of the polyethylene glycol solution. WBI should be continued until the rectal effluent is clear. In most patients, this usually occurs within 4 to 6 hours. This procedure is generally well tolerated by most patients and has been used safely in children [6].

Antidotal therapy

The number of effective antidotes is limited and they are not for indiscriminate use. Table 2 lists selected antidotes and the substances for which they are indicated. As Paracelsus observed, all xenobiotics are potentially toxic. This observation is true for purported antidotes also. Antidotal therapy should be used carefully and in clinical circumstances when specifically indicated. With the exception of naloxone, antidotal therapy use is limited in the patient who has an unknown poisoning [84]. The clinician should be familiar with the indications for use and the availability of antidotal therapy [84]. Although administering so-called “life-saving” antidotes is often considered to be the glamorous aspect of clinical toxicology, antidotal therapy is used in a minority of poisonings. Most poisoned patients have an uneventful recovery when routine supportive care is appropriately provided.

Table 2
Antidotes and their indications

Antidote	Indication (agent)
<i>n</i> -acetylcysteine	Acetaminophen
Ethanol/fomepizole (4-MP)	Methanol/ethylene glycol
Oxygen/hyperbarics	Carbon monoxide
Naloxone/nalmefene	Opioids
Physostigmine	Anticholinergics
Atropine/pralidoxime (2-PAM)	Organophosphates
Methylene blue	Methemoglobinemia
Nitrites	Cyanide
Deferoxamine	Iron
Dimercaprol (BAL)	Arsenic
Succimer (DMSA)	Lead, mercury
Fab fragments	Digoxin, colchicine, crotalids
Glucagon	Beta-blockers
Sodium bicarbonate	Tricyclic antidepressants
Calcium/insulin/dextrose	Calcium channel antagonists
Dextrose, glucagon, octreotide	Oral hypoglycemics

Enhanced elimination

Enhancing elimination is the process of removing a toxin from the body once absorption has already occurred. Methods of enhanced elimination include multiple-dose activated charcoal, urinary alkalinization, and extracorporeal elimination. The role of multiple-dose activated charcoal was discussed previously. Urinary alkalinization involved the use of an intravenous sodium bicarbonate infusion and promotes urinary elimination of substances that are weak acids. It is important to maintain a normal potassium level when performing alkalinization because appropriate alkalinization cannot be achieved when hypokalemia is present. A common use of urinary alkalinization is in the salicylate-poisoned patient. Another use may be in patients who overdose on phenobarbital, although no outcome data has demonstrated this to be beneficial in terms of survivability. As an aside, urinary acidification has been recommended in the past as a method of enhanced elimination with poisoning by weak bases, such as phencyclidine and amphetamine. This procedure is no longer recommended because of the high risk for myoglobinuria and rhabdomyolysis [85].

Extracorporeal elimination

In the unstable poisoned patient, consultation with a nephrologist may be indicated before definitive diagnostic studies or drug levels become available. This consultation is particularly important when the suspected agent is a salicylate, lithium, theophylline, or a toxic alcohol (Box 12). Hemodialysis enhances removal of substances with low protein binding, small volumes of distribution, high water solubility, and low molecular weight. Charcoal hemoperfusion, if available, may be useful for theophylline, barbiturates, and carbamazepine overdose (Box 13) [86,87].

Box 12. Toxins accessible to hemodialysis (UNSTABLE)

Uremia

No response to conventional therapy

Salicylates

Theophylline

Alcohols (isopropanol, methanol)

Boric acid, barbiturates

Lithium

Ethylene glycol

Box 13. Enhanced elimination by charcoal hemoperfusion

Theophylline

Barbiturates

Carbamazepine

Paraquat

Glutethimide

Cutting-edge toxicology

The latest toxicology antidotes include fomepizole for ethylene glycol and methanol poisoning [88–90], specific immune therapy with purified Fab fragments for rattlesnake antivenin [91–94], and high-dose insulin for calcium channel antagonist poisoning [95,96]

Current controversies include challenging the widely accepted 72-hour oral *n*-acetylcysteine (NAC) treatment course for acetaminophen toxicity. Many are now suggesting a more abbreviated regimen [97]. An intravenous form of NAC has recently been approved by the US Food and Drug Administration for use in the United States [98,99]. This is a 21-hour infusion protocol with the total administered NAC dose of 300 mg/kg.

There continues to be controversy about the use of hyperbaric oxygen therapy for carbon monoxide-poisoned patients. Two recent studies show conflicting results [100,101]. Each study used distinctly different treatment protocols, which makes comparison difficult. The current Cochrane Database concludes that existing randomized trials do not establish whether the administration of HBO to patients who have carbon monoxide poisoning reduces the incidence of adverse neurologic outcomes [102].

Emergent orthotropic liver transplantation is currently the only standard treatment of fulminant hepatic failure. There is often a prolonged wait time for transplantation. The concept of a liver support device (“liver dialysis”) may in the future provide a bridge to transplantation or potential recovery without transplantation [103]. Currently there are few studies in this area, especially with application in the poisoned patient, but liver support devices may play a future role in poisoned patients. Finally, the use of intravenous fat or lipid emulsion is showing promise with lipid soluble drug toxicity to treat cardiotoxicity and hemodynamic instability. Recent efficacy has been demonstrated in an animal model and in isolated human cases [104,105].

Disposition

People who have a potentially serious overdose should be observed for several hours before discharge. If signs or symptoms of intoxication develop during that time, the patient should be admitted for further observation and

treatment. Some agents may require a more prolonged observation period. Such agents include sustained-release products and agents with known delayed or prolonged onset of action: calcium channel antagonists, theophylline, lithium, methadone, Lomotil, monoamine oxidase inhibitors, and oral hypoglycemic agents. Overdose with these substances may require up to 24 hours of continuous observation [106]. Although some patients admitted require observation in an intensive care unit, others can be appropriately managed on the general medical floor or in an observation unit. Consultation with a medical toxicologist or regional poison control center can help to determine the appropriate disposition.

Because many poisoned patients require less than 24 hours of observation, unnecessary hospitalization may be avoided by the use of observation units. Such units have already been developed for asthma and chest pain patients. This model may also work well for poisoned patients.

All patients presenting with an intentional poisoning should have a psychiatric evaluation and will likely require psychiatric admission. Substance abusers should be considered for drug abuse counseling.

Summary

The management of the poisoned patient who has an unknown exposure can be diagnostically and therapeutically challenging. The history and physical examination, along with a small dose of detective work, can often provide the clues to the appropriate diagnosis. The two-pronged approach as outlined in Fig. 1 provides a framework for evaluating poisoned patients. Consultation with a regional poison center or clinical toxicologist early in the care of poisoned patient can have a profound impact on the management and disposition of such patients.

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