CHAPTER 147

General Approach to the Poisoned Patient

Ken Kulig

PERSPECTIVE

Most poisoned patients seen in the emergency department are adults with acute oral drug overdoses. Other common clinical scenarios include accidental poisoning in children; drug abuse through smoking, snorting, or injection; chronic poisoning from drug abuse or from environmental, industrial, and agricultural chemical exposure; medication reactions or interactions; and envenomation. Management requires both a general supportive approach and specific actions directed at the particular toxin or toxins involved, as outlined in the various chapters in this section. Clinical studies have modified the management of poisoned patients, such as the use of gastric decontamination, but much of the toxicology literature, especially with unusual poisonings, remains case based. Regional poison centers and medical toxicologists have a concentrated experience in management of poisoned patients and can be called on for advice and assistance.

INITIAL APPROACH TO THE POISONED PATIENT

Patients who are contaminated with an agent that might injure health care personnel require decontamination before treatment to avoid disabling of the hospital staff or the entire health care facility. Except for specific lifesaving antidotes against certain toxins, most poisoned patients require only supportive therapy for recovery. The initial workup should determine whether a specific patient has been exposed to an agent for which an antidote (or other specific treatment) exists.[1] A thorough poisoning history and toxicologic physical examination may be followed by the selective use of laboratory tests.

After initial stabilization of a critically ill patient, specific antidote therapy is administered while a more detailed history and physical examination are performed. Hypoglycemia should be considered in a patient with altered mental status or seizures and should be evaluated by bedside glucose testing. *Naloxone* can be given to patients with suspected opioid overdose and respiratory depression while preparations are made to secure the airway because a positive response may obviate the need for intubation. *Flumazenil* is not indicated in an undifferentiated overdose patient, and its use should be limited to confirmed acute benzodiazepine overdose in a patient who is not a chronic benzodiazepine user (e.g., an adolescent who impulsively ingests a parent's benzodiazepine). Indiscriminate use may cause a chronic benzodiazepine user to have severe benzodiazepine withdrawal. The patient may have ingested tricyclic antidepressants or other drugs likely to cause seizures. In either case, the use of flumazenil can carry a substantial risk of seizures. *Thiamine* should be administered when dextrose is given to nutritionally compromised, alcoholic patients with altered mental status (100 mg in the maintenance intravenous line is sufficient and safe).[2] It is common to give alcoholic patients intravenous multivitamins ("yellow bag") containing thiamine, folate, and B_{12}. However, serum levels of these vitamins are usually normal in alcoholic patients, and the practice is not justified.[3]

A complete *overdose history* may be time-consuming but is important (Box 147-1). Valuable clues often come from unexpected sources, such as the patient's previous medical records, the pharmacy where prescriptions were filled, online regulatory databases, or the prescribing physician as listed on the patient's prescription bottles. Whenever possible, prehospital personnel should bring the patient's medications and other available medications from the patient's residence to the hospital with them. If the ingested agent is a hazardous chemical (e.g., pesticide) that might endanger hospital personnel, it should be brought to the hospital in an airtight container or secured at the scene. Precise product identification information should be obtained so that a hazardous materials reference system can be consulted. When it is suspected that the contents of the container are not the original product, the substance should be checked against the product label. It is easy to confuse the different types of chemical agents with agents with similar names found in many homes, and some may have specific properties that affect treatment. In rare cases, overdose patients may deliberately attempt to deceive caregivers by hiding the ingested agents.

Accurate vital signs, including pulse oximetry, are important in the diagnosis of poisoning and should be repeated as indicated. At least one measurement of temperature should be included. Respirations should be counted, not estimated. A cardiac monitor or 12-lead electrocardiogram should be evaluated for QRS and QT intervals, morphology, and rhythm. The physical examination in a comatose patient should ensure that concomitant treatable conditions (e.g., trauma, intracranial hemorrhage, central nervous system [CNS] infection) are not missed. Focal neurologic findings could be possible indicators of intracranial disease or severe head trauma.

The pupillary examination may give misleading information. Some opioid agonists, especially propoxyphene and pentazocine, may not produce the characteristic miosis of opioid intoxication. When multiple drugs are ingested, the expected pupillary findings related to any particular agent may be modified or absent.

Physical stigmata of injection drug use (track marks) should be sought in both usual (e.g., antecubital fossa) and unusual (e.g., under the tongue and top of the feet) locations.

Signs of infection (sepsis, abscess, endocarditis) should be investigated in patients suspected of injection drug abuse.[4] A patient may be critically ill as a result of "body packing" or "body

BOX 147-1 Obtaining an Overdose History

- Obtain all prescription bottles and other containers when possible. Perform a pill count. Be sure that the bottles contain the medications listed. Identify any unknown tablets.
- Contact the prescribing physician or the pharmacy as listed on the bottles to determine previous overdoses or other medications that the patient may have available. Identify underlying medical and psychiatric disorders and medication allergies. Review past medical records.
- Talk to the patient's family and friends in the emergency department. If necessary, call the patient's home to ask questions of others. The persons providing the important elements of the history should be identified in the chart.
- Search the patient's belongings for drugs or drug paraphernalia. A single pill hidden in a pocket, for example, may provide *the* most important clue to the diagnosis.
- Have family members (or the police) search the patient's home, including the medicine cabinet, clothes drawers, closets, and garage; such searches may also provide clues that make the diagnosis. This has the added benefit of involving the family in the patient's care.
- Look for track marks on the patient. Consider body packing or body stuffing.

Table 147-1 Odors in Overdose History

ODOR	POSSIBLE INTOXICANT
Bitter almonds	Cyanide
Carrots	Water hemlock (cicutoxin)
Fishy	Zinc or aluminum phosphide
Fruity	Ethanol, acetone, isopropyl alcohol, chlorinated hydrocarbons (e.g., chloroform)
Garlic	Arsenic, dimethyl sulfoxide (DMSO), organophosphates, yellow phosphorus, selenium, tellurium
Glue	Toluene, other solvents
Pears	Chloral hydrate, paraldehyde
Rotten eggs	Disulfiram, hydrogen sulfide, *N*-acetylcysteine, dimercaptosuccinic acid (DMSA)
Shoe polish	Nitrobenzene
Wintergreen	Methyl salicylate

stuffing," complicated by rupture of packets of cocaine, heroin, or amphetamines (see Chapters 154 and 162).[5] Rectal, vaginal, and radiographic examination of the abdomen should be performed in these circumstances.

Another important physical finding is evidence of aspiration or noncardiogenic pulmonary edema on chest auscultation. Bowel sounds may be increased or decreased if agents affecting the cholinergic nervous system have been ingested. A rectal examination to detect melena or hematochezia may also provide evidence of a coagulopathy (e.g., ingestion of anticoagulant medication). Skin examination may reveal evidence of skin popping, cyanosis (hypoxia or methemoglobinemia), or pressure marks if the patient has been unconscious.

Unusual odors of the patient's breath, skin, clothing, vomitus, or nasogastric aspirate may also provide useful diagnostic clues (Table 147-1).[6] The absence of such odors, however, should not be taken as evidence that the agents listed are not present.

Table 147-2 Common Toxic Syndromes (Toxidromes)

Anticholinergic	
Common signs	Delirium with mumbling speech, tachycardia, dry flushed skin, dilated pupils, myoclonus, slightly elevated temperature, urinary retention, decreased bowel sounds. Seizures and dysrhythmias may occur in severe cases.
Common causes	Antihistamines, antiparkinsonians, atropine, scopolamine, amantadine, antipsychotics, antidepressants, antispasmodics, mydriatics, muscle relaxants, many plants (e.g., jimson weed, *Amanita muscaria*)
Sympathomimetic	
Common signs	Delusions, paranoia, tachycardia (or bradycardia with pure alpha-agonists), hypertension, hyperpyrexia, diaphoresis, piloerection, mydriasis, hyper-reflexia. Seizures, hypotension, and dysrhythmias may occur in severe cases.
Common causes	Cocaine, amphetamine, methamphetamine and its derivatives, over-the-counter decongestants (phenylpropanolamine, ephedrine, pseudoephedrine). In caffeine and theophylline overdoses, similar findings, except for the organic psychiatric signs, result from catecholamine release.
Opioid/Sedative/Ethanol	
Common signs	Coma, respiratory depression, miosis, hypotension, bradycardia, hypothermia, pulmonary edema, decreased bowel sounds, hyporeflexia, needle marks. Seizures may occur after overdoses of some narcotics (e.g., propoxyphene).
Common causes	Narcotics, barbiturates, benzodiazepines, ethchlorvynol, glutethimide, methyprylon, methaqualone, meprobamate, ethanol, clonidine, guanabenz

TOXIC SYNDROMES AND ANTIDOTES

The term *toxidrome* refers to a syndrome or constellation of physical findings attributed to a specific class of toxins that can provide important clues to narrow the differential diagnosis.[1] The general rules outlined here have many exceptions, and polydrug overdoses may result in overlapping and confusing mixed syndromes. Nevertheless, this approach may confirm the history, provide the clinician with a starting point for management, and suggest useful laboratory tests. The most common toxidromes are the anticholinergic syndrome, sympathomimetic syndrome, opioid/sedative/ethanol syndrome, cholinergic syndrome, and serotonin syndrome (Table 147-2).

The *anticholinergic syndrome* occurs frequently because many common medications and plants have anticholinergic properties. Anticholinergic CNS poisoning causes mild temperature elevation and acute delirium with mumbling speech and typical "picking movements" of the fingers. Suppression of cholinergic inhibition of the heart rate leads to tachycardia. Inhibition of the secretory functions of the integument causes dry mouth and skin, and the face is typically flushed. Unopposed sympathetic drive of the ciliary apparatus causes wide pupillary dilation. Most patients

Box 147-2 Toxins Causing Delirium

- Alcohol and alcohol withdrawal
- Anticholinergics
- Anticonvulsants
- Antidepressants
- Antihypertensives
- Antiparkinson medications
- Antipsychotics
- Cardiac medications
- Cocaine
- Lithium
- Monoamine oxidase inhibitors
- Muscle relaxants
- Mushrooms with muscimol or ibotenic acid
- Opiates
- Phencyclidine
- Salicylates
- Sedative withdrawal
- Solvents
- Steroids
- Sympathomimetics (cocaine, amphetamines)

recover with supportive therapy, but the delirium may last a day or more. *Physostigmine* may be a useful antidote in carefully selected patients; this is discussed in detail in Chapter 150.

The *sympathomimetic syndrome* is usually seen after acute or chronic abuse of cocaine, amphetamines, or decongestants (e.g., phenylephrine). Patients may be delusional; methamphetamine, in particular, may cause complicated, intricate, and paranoid delusions. Seizures may occur, and the postictal state can contribute to the altered mental status. Blood pressure is usually elevated, the pulse is rapid (except with pure alpha-adrenergic agonists, which can cause reflex bradycardia), the pupils are dilated, and piloerection may be seen. In massive overdoses of sympathomimetic agents, cardiovascular collapse can occur with the development of shock and wide-complex dysrhythmias. This clinical picture can mimic that of overdose of cardioactive drugs or cyclic antidepressants. In contrast to the dry skin seen with anticholinergic syndrome, diaphoresis would be expected with the sympathomimetic syndrome.

An extreme presentation of sympathomimetic excess can be excited delirium (Box 147-2). In this state, patients are agitated, hyperthermic, and violent and possess "superhuman strength." Frequently, many security personnel are required to control these individuals. These individuals may have a severe metabolic acidosis and hyperkalemia, which can cause sudden cardiovascular collapse. It is critical to sedate these patients and to control hyperthermia aggressively while treating their acidosis and hyperkalemia simultaneously. Dantrolene has been reported to be effective in treatment of MDMA-related hyperpyrexia.[7]

All sedative-hypnotic agents, when they are taken in sufficient dosage, cause general anesthesia with a complete loss of awareness and reflex activity. The CNS depressant (opioid/sedative/ethanol) syndrome is the most common toxic syndrome seen in the emergency department, and a depressed sensorium is its hallmark. Mixing agents in this class (e.g., ethanol and benzodiazepines) is common. As the drugs are absorbed at higher doses, the patient becomes increasingly obtunded, the deep tendon reflexes diminish, and finally the vital signs deteriorate as medullary drive of respiration and cardiovascular function is attenuated.

Respiratory depression is particularly pronounced with opioid overdose, and the respiratory rate is often diminished before decreases in blood pressure or pulse occur. The diagnosis of opioid overdose is confirmed by the use of naloxone (Narcan) or nalmefene (Revex) in adequate doses. Management of opioid overdose is discussed in Chapter 162. Comatose patients often present without a history and need to be managed aggressively, with securing of the airway when it is needed. These patients may need basic laboratory tests and a head computed tomography scan if their presentation or course is suggestive of stroke, infection, or head trauma while consideration is given to a drug overdose alone or in combination with one of these medical conditions. A serum or breath ethanol level that is not commensurate with the level of CNS depression raises suspicion of intracranial injury, hemorrhage, or infection.

The *cholinergic syndrome* is uncommon, but it is important to recognize it because lifesaving treatment is available. Cholinergic syndrome causes the patient to be "wet," as opposed to the anticholinergic syndrome, which causes the patient to be "dry." The wetness is manifested by profuse sweating and excessive activity of virtually the entire exocrine system, often accompanied by vomiting, diarrhea, diaphoresis, and urinary incontinence. The mnemonic SLUDGE is used to recall the specific elements of the syndrome: *s*alivation, *l*acrimation, *u*rination, *d*efecation, *g*astrointestinal cramping, and *e*mesis. The CNS (e.g., confusion, coma, and seizures) and the skeletal muscles (e.g., weakness and fasciculations) can also be involved. The pupils are often miotic. Cholinergic syndrome is most frequently caused by organophosphate or carbamate pesticide exposure, which may be through unsuspected dermal contamination. Anticholinergic agents are also the foundation of "nerve agents" such as sarin, which was used in the Tokyo subway attack. Recognition of the syndrome led to the use of atropine and cholinesterase regenerators, with a subsequent good outcome in many patients.[8]

Serotonin syndrome ensues when there is a drug interaction involving the selective serotonin reuptake inhibitors (SSRIs) or an overdose of an SSRI.[9] Fluoxetine (Prozac), sertraline (Zoloft), paroxetine (Paxil), fluvoxamine (Luvox), and citalopram (Celexa) are commonly used SSRIs. Other drugs inhibit the reuptake of other neurotransmitters and are also serotonin reuptake inhibitors (SRIs) that can cause the syndrome. These drugs include venlafaxine (Effexor), nefazodone (Serzone), and mirtazapine (Remeron). Drug interactions between many drugs can cause the serotonin syndrome described in Chapter 151. These drugs include the SSRIs, SRIs, monoamine oxidase inhibitors (MAOIs), tryptophan, sympathomimetics, tricyclic and other antidepressants, meperidine, dextromethorphan, and lithium. Because of the long-lasting effects of the SSRIs, the syndrome can occur when one of the active agents is ingested even weeks after use of an SSRI has been discontinued.

Serotonin syndrome is characterized by altered mental status, fever, agitation, tremor, myoclonus, hyper-reflexia, ataxia, incoordination, diaphoresis, shivering, and sometimes diarrhea.[7] The diagnosis relies on the drug history, and it is difficult to distinguish serotonin syndrome from an overdose of cocaine, lithium, or MAOIs and from the neuroleptic malignant syndrome or thyroid storm. Patients may deteriorate slowly and become critically ill after an apparently benign manifestation.

As possible toxidromes are investigated, the use of specific antidotes should be considered (Table 147-3).

A relatively new antidote, Intralipid, for the treatment of cardiotoxicity from local anesthetics has been reported to be lifesaving in some cases. It has been successfully used for beta-blocker, calcium channel blocker, and tricyclic antidepressant poisoning and many other mostly lipophilic drugs.[10-12] The American College of Medical Toxicology has recently published guidelines for its use.[13]

TOXICOLOGY LABORATORY

A toxicology screen (usually urine, sometimes blood and urine) may identify the class of drugs ingested but may also be misleading.[14-17] The typical urine toxicology screen (U-tox) does not screen for many substances, even commonly ingested agents that are capable of causing critical illness (Box 147-3).[18] Some drugs, such as γ-hydroxybutyrate (GHB), are present relatively briefly in blood and urine, and therefore findings may be negative in samples collected even on the same day. Conversely, drugs found on screening may not be those currently responsible for the toxidrome, and drugs ingested therapeutically and not in overdose

Table 147-3 Antidotes Used in the Emergency Department

TOXIN USED FOR	ANTIDOTE	DOSE AND COMMENTS
Acetaminophen	*N*-Acetylcysteine	140 mg/kg PO, then 70 mg/kg q4h for up to 17 doses OR 150 mg/kg IV load during 1 hour with 50 mg/kg during 4 hours followed by 100 mg/kg during 16 hours
Anticholinergics	Physostigmine	1-2 mg IV in adults, 0.5 mg in children during 2 minutes for anticholinergic delirium, seizures, or dysrhythmias
Arsenic, lead, and mercury	BAL D-Penicillamine	3-5 mg/kg IM only 20-40 mg/kg/day; 500 mg tid in adults; may cross-react with penicillin in allergic patients
Benzodiazepines	Flumazenil	0.2 mg, then 0.3 mg, then 0.5 mg, up to 5 mg; not to be used if patient has signs of TCA toxicity; not approved for use in children but probably safe
Black widow spider bite	*Latrodectus* antivenin	One vial by slow IV infusion is usually curative; may cause anaphylaxis
Beta-blockers	Glucagon Insulin and glucose Intralipid 20%	5-10 mg in adults, then infusion of same dose per hour 1 unit/kg regular insulin IV push followed by 1-10 unit/kg/hr; blood glucose should be checked frequently; D10 at 100 mL/hr with boluses of D50 if hypoglycemia occurs 1.5 mL/kg during 2-3 minutes followed by infusion of 0.25 mL/kg/min Reported to be effective for highly protein bound drugs with large volumes of distribution, such as local anesthetics, beta-blockers, calcium channel blockers, and tricyclic antidepressants
Calcium channel blockers	Calcium Glucagon Insulin and glucose	1 g calcium chloride IV in adults, 20-30 mg/kg/dose in children, during a few minutes with continuous monitoring; repeat as needed 5-10 mg in adults, then infusion of same dose per hour Same dose as for beta-blockers
Cyanide	Hydroxycobalamin	5 mg in 100 mL of NS during 15 minutes; repeat if necessary
Cyanide, hydrogen sulfide	Sodium thiosulfate Sodium nitrite Hydroxycobalamin	50 mL of 25% (12.5 g; 1 ampule) in adults; 1.65 mL/kg IV in children 10 mL of 3% (300 mg; 1 ampule) in adults; 0.33 mL/kg slowly IV in children 5 g in 100 mL of NS given during 15 minutes
Digitalis glycosides	Digoxin-specific Fab	10-20 vials if patient is in ventricular fibrillation; otherwise dose fragments on the basis of serum digoxin concentration or amount ingested
Ethylene glycol	Fomepizole Pyridoxine Thiamine	15 mg/kg × 1, then 10 mg/kg q12h × 4, until ethylene glycol < 20 mg/dL; adjust dose during dialysis 100 mg IV daily 100 mg IV
Hydrofluoric acid	Calcium gluconate	3.5 g in 5 oz of KY jelly topical; apply liberally to affected skin
Iron	Deferoxamine	15 mg/kg/hr IV; higher doses reported to be safe
Isoniazid, hydrazine, and monomethylhydrazine	Pyridoxine	5 g in adults, 1 g in children, if ingested dose unknown; antidote may cause neuropathy in very large doses
Lead	DMSA (succimer) EDTA	Reported useful for arsenic and lead as well; one 100-mg capsule per 10-kg body weight tid for 1 week, then bid, with chelation breaks 75 mg/kg/day by continuous infusion; watch for nephrotoxicity, best done in hospital
Local anesthetics and others	Intralipid 20%	1.5 mL/kg during 2-3 minutes followed by infusion of 0.25 mL/kg/min Reported to be effective for highly protein bound drugs with large volumes of distribution, such as local anesthetics, beta-blockers, calcium channel blockers, tricyclic antidepressants, and others
MDMA-related hyperpyrexia	Dantrolene	2.5 mg/kg IV Lower doses have also been reported to be effective
Neuroleptics	Dantrolene	2.5 mg/kg IV for neuroleptic malignant syndrome
Methanol	Folate or leucovorin Ethanol Fomepizole	50 mg IV q4h in adults while patients have serious toxicity Loading dose, 10 mL/kg of 10%; maintenance dose, 0.15 mL/kg/hr of 10%; double rate during dialysis 15 mg/kg × 1, then 10 mg/kg q12h × 4, until methanol < 20 mg/dL; adjust dose during dialysis
Methemoglobin-forming agents	Methylene blue	1-2 mg/kg IV, one 10-mL dose of 10% solution (100 mg) is typical for an adult without anemia
Opioids	Nalmefene Naloxone	0.5-1 mg; much longer half-life than naloxone 2 mg; less to avoid narcotic withdrawal, more if inadequate response; same dose in children

Continued

Table 147-3 Antidotes Used in the Emergency Department—cont'd

TOXIN USED FOR	ANTIDOTE	DOSE AND COMMENTS
Organophosphates and carbamates	Atropine	Test dose, 1-2 mg IV in adults, 0.03 mg/kg in children; titrate to drying of pulmonary secretions
	Protopam	Loading dose, 1-2 g IV in adults, 25-50 mg/kg in children; adult maintenance, 500 mg/hr or 1-2 g q4-6h
Rattlesnake bite	CroFab antivenin	Five vials minimum dose by infusion in normal saline; increase is rate dependent on patient's tolerance; may cause anaphylaxis
Serotonin syndrome	Cyproheptadine	4 mg PO or by nasogastric tube as needed; no parenteral form available; antidote may cause anticholinergic effects
Sulfonylureas	Octreotide	50 µg SC q12h, 5-10 µg/kg/24 hr IV
Tricyclic antidepressants	Bicarbonate	44-88 mEq in adults, 1-2 mEq/kg in children; best used by IV push and not by slow infusion
	Intralipid 20%	1.5 mL/kg during 2-3 minutes followed by infusion of 0.25 mL/kg/min Reported to be effective for highly protein bound drugs with large volumes of distribution, such as local anesthetics, beta-blockers, calcium channel blockers, and tricyclic antidepressants
Valproic acid	Carnitine	100 mg/kg IV or PO loading dose with 25 mg/kg q6h

BAL, British anti-Lewisite; *DMSA,* dimercaptosuccinic acid; *EDTA,* ethylenediaminetetraacetic acid; *TCA,* tricyclic antidepressant.

Box 147-3 Drugs, Chemicals, and Groups *Not Detected* by a Comprehensive Toxicology Screen

Ammonia
Anesthetic gases
Antibiotics
Anticoagulants
Beta-blockers
Borates
Bromides
Caustics and corrosives
Colchicine
Cyanide
Digitalis glycosides
Disulfiram
Ergot alkaloids
Ethylene glycol
Fentanyl and its derivatives
Fluorides
H_2 antagonists
Hallucinogens (e.g., LSD)
Herbicides
Household products
Hypoglycemics
Insect repellents
Isoniazid
Laxatives
Lithium
Metals
Monoamine oxidase inhibitors
Most antihypertensives
Most cardiac medications
Muscle relaxants
Mushrooms
Newer antidepressants (e.g., fluoxetine, sertraline, paroxetine, bupropion, buspirone)
Nitrates and nitrites
Nonsteroidal anti-inflammatory drugs
Paraquat
Pesticides
Phenol
Plants
Solvents
Thyroid hormone
Vitamins

(e.g., Adderall containing amphetamine) and foods (e.g., poppy seeds containing morphine and codeine) can cause a misleading positive U-tox result.[19]

Drugs with a large volume of distribution or high fat solubility may be detected in urine long after the last dose. Most street drugs are detected in urine for days and marijuana for weeks (in rare cases even for months) after the last exposure.[19] Screening results rarely change the immediate clinical management of patients but may help document substance abuse or class of drugs responsible for the current clinical condition, as long as clinicians remember that false-positive or false-negative U-tox results occur. Quantitative serum measurements of certain drugs (e.g., acetaminophen, aspirin) may be helpful. In the agitated or seizing patient, elevated salicylate or lithium levels would significantly alter the management.

Electrolyte levels help identify metabolic acidosis by the carbon dioxide content ("bicarbonate level"); the determination should be repeated if it is low to ensure that the acidosis is resolving. A persistent, unexplained metabolic acidosis should prompt urine examination for oxalate crystals (suggestive of ethylene glycol poisoning), serum osmolality, serum salicylate level, and methanol and ethylene glycol levels. A normal arterial blood gas or electrolyte measurement does not rule out such ingestion because metabolic acidosis is delayed and does not appear until after metabolism of the acids from ethylene glycol and methanol or until after erratic and slow absorption of salicylate. Co-oximetry will confirm carboxyhemoglobin and methemoglobinemia, which will not be detected by pulse oximetry.

Rhabdomyolysis should be suspected when there is severe agitation or hyperthermia or if the patient is thought to have been unresponsive for a prolonged time (i.e., by history of presence or pressure sores). A urine dipstick test for blood (myoglobin) and determination of a serum creatine kinase concentration should be performed. Rhabdomyolysis and its treatment are discussed in Chapter 127. Noncardiogenic pulmonary edema on a chest radiograph suggests opioid or salicylate overdose. Some drugs are radiopaque (e.g., heavy metals, phenothiazines, potassium, calcium, and chlorinated hydrocarbons such as chloral hydrate), but radiography is rarely helpful in the evaluation of a poisoned patient except to monitor the decontamination of iron, lead, or body packets.

DECONTAMINATION

Gastric decontamination should not be undertaken routinely as it may result in morbidity, and patients often present too late for it to be effective.[20-24] Gastric lavage is rarely if ever indicated. In the rare circumstances in which gastric lavage is performed, a large (30-F or larger) orogastric tube is used, and specially designed lavage systems with large-bore tubes are available for this purpose.

Although activated charcoal may decrease drug absorption even if it is given hours after ingestion, it has not been proved to improve outcome. It might be considered in selected high-risk cases (such as with calcium channel blockers or salicylates) to prevent absorption when the patient is still likely to have a toxic amount of a drug or chemical in the gastrointestinal tract that is known to be absorbed by charcoal.

Whole-bowel irrigation with a polyethylene glycol solution is sometimes recommended by poison centers for overdose of metals such as iron and lead, in patients with ingestion of

sustained-release formulations, or for the evacuation of drug packets from body packers or body stuffers.[25] It usually but not always requires placement of a small nasogastric tube. The scientific evidence that this procedure improves outcomes is lacking.

Exposure of the eye to caustic chemicals and irritants requires immediate irrigation with large amounts of water or readily available fluid, as outlined in Chapter 64. Irrigation is begun immediately after exposure, before transfer to the emergency department. Exposure to a gas does not require decontamination because the patient and rescuers are not at risk once the patient is removed from the toxic environment. The exception is when the patient's skin or clothing is contaminated with a liquid that is evaporating. The most important intervention to limit dermal exposure is to remove all clothing as soon as possible, ideally at the scene. Skin should be irrigated with warm water, with special attention to skin folds and other areas that might be missed. This includes the axilla, beneath nails, behind the knees, the genitalia, and in the scalp. For hydrocarbons or solvents, soap can be added. The skin should not be abraded with overly aggressive scrubbing, which could increase skin absorption. Ideally, skin decontamination occurs as soon as possible after exposure, at an out-of-hospital site before transport.

DISPOSITION AND CONSULTATION

The decision to admit a patient is not difficult when the patient manifests serious toxicity. When the patient is minimally symptomatic but has ingested a potentially dangerous substance, the decision is more difficult. Identification of an agent that causes a particular risk for the patient, especially cardiovascular instability, seizures, or respiratory depression, generally suggests admission to the hospital or to an observation unit in the emergency department. For benign medications, such as nonsteroidal anti-inflammatory drugs, observation is not necessary. For others, a 6-hour period of observation usually is sufficient to determine whether there is any significant toxicity. For extended-release preparations of toxic medications (e.g., beta-blockers), a longer observation period is warranted if drug effect is not subsiding by 6 hours. Patients with cardiac dysrhythmia, conduction disturbance, altered mental status requiring intubation, or the need for frequently titrated agents (e.g., pressors) should be admitted to a monitored bed or the intensive care unit. If the patient is acutely suicidal, a sitter or secure environment may be required.

Recognition of drug-induced prolongation of the QTc presents a disposition challenge. Many drugs have been reported to cause QTc prolongation, a known risk factor for torsades de pointes.[26-28] If the QTc is longer than 500 msec, even in a relatively asymptomatic drug overdose patient, monitoring is indicated until the QTc is decreasing.

Regional poison centers use a single nationwide toll-free number (1-800-222-1222) and can provide specific, current advice, especially for more esoteric or unfamiliar poisons. Consultation with a medical toxicologist is particularly helpful when an uncommon agent has been ingested, the patient is not following the anticipated clinical course, or specific interventions (such as administration of antidote therapy or dialysis) are contemplated.

KEY CONCEPTS

- A thorough history from multiple sources is often the key to toxicologic diagnosis.
- Common toxidromes should guide selective use of antidotes.
- Extensive laboratory investigation is usually unnecessary.
- Activated charcoal and other forms of gastric decontamination are not indicated in most poisoned patients. Gastric lavage is unlikely to be beneficial even if it is done soon after an oral drug overdose.
- Poison center and medical toxicologist consultations can be extremely helpful in diagnosis and management of the poisoned patient.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

References

1. Kulig K: Initial management of ingestion of toxic substances. *N Engl J Med* 1992; 326:1677.
2. Wrenn K, Murphy F, Slovis C: A toxicity study of parenteral thiamine hydrochloride. *Ann Emerg Med* 1989; 18:867.
3. Li SF, Jacob J, Feng J, Kulkarni M: Vitamin deficiencies in acutely intoxicated patients in the ED. *Am J Emerg Med* 2008; 26:792-795.
4. Kaushik KS, Kapila K, Praharaj AK: Shooting up: The interface of microbial infections and drug abuse. *J Med Microbiol* 2011; 60:408-422.
5. Mandava N, et al: Establishment of a definitive protocol for the diagnosis and management of body packers (drug mules). *Emerg Med J* 2011; 28:98-101.
6. O'Malley GF, Deitch KR, Dominici P, Young M: A modified Goldfrank sniffing bar improves accuracy in identifying simulated toxins in a case-based teaching model. *J Med Toxicol* 2008; 4:11-15.
7. Grunau BE, Wiens MO, Brubacher JR: Dantrolene in the treatment of MDMA-related hyperpyrexia: A systematic review. *CJEM* 2010; 12:4335-4342.
8. Suzuki T, et al: Sarin poisoning in a Tokyo subway. *Lancet* 1995; 345:980.
9. Boyer EW, Shannon M: The serotonin syndrome. *N Engl J Med* 2005; 352:1112.
10. Felice KL, Schumann HM: Intravenous lipid emulsion for local anesthetic toxicity: A review of the literature. *J Med Toxicol* 2008; 4:184-191.
11. Jamaty C, et al: Lipid emulsions in the treatment of acute poisoning: A systematic review of human and animal studies. *Clin Toxicol (Phila)* 2010; 48:1-27.
12. Di Gregorio G, Neal JM, Rosenquist RQ, Weinberg GL: Clinical presentation of local anesthetic systemic toxicity. A review of published cases, 1979-2009. *Reg Anesth Pain Med* 2010; 35:181-187.
13. American College of Medical Toxicology: ACMT Position Statement: Interim guidance for the use of lipid resuscitation therapy. *J Med Toxicol* 2011; 7:81-82.
14. Rentsch KM: Laboratory diagnostics in acute poisoning: Critical overview. *Clin Chem Lab Med* 2010; 48:1381-1387.
15. Wu AH: Limitations of point-of-care testing in the ED or ICU: A role for regional centralized toxicology laboratories. *Clin Pharmacol Ther* 2010; 88:295-298.
16. Moeller KE, Lee KC, Kissack JC: Urine drug screening: Practical guide for clinicians. *Mayo Clin Proc* 2008; 83:66-76.
17. Tenore PL: Advanced urine toxicology testing. *J Addict Dis* 2010; 21:436-448.
18. Kulig K: *The appropriate utilization of toxicology screens*. In: Cantrill SV, Karas SJ, eds. *Cost-Effective Diagnostic Testing in Emergency Medicine*. Dallas, Tex: American College of Emergency Physicians; 2000.
19. Medical Review Officer Certification Council, Swotinsky R, Smith D: *The Medical Review Officer's Manual. MROCC's Guide to Drug Testing*, 4th ed. Beverly Farms, Mass: Occupational and Environmental Medicine (OEM) Press; 2010.
20. Chyka PA, Seger D: Position statement: Single-dose activated charcoal. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. *J Toxicol Clin Toxicol* 2005; 2:51-87.
21. Vale JA: Position statement: Gastric lavage. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. *J Toxicol Clin Toxicol* 2004; 42:933-943.
22. Jurgens G, Hoegberg LCG, Graudal NA: The effect of activated charcoal on drug exposure in healthy volunteers: A meta-analysis. *Clin Pharmacol Ther* 2009; 85:501-505.
23. Bond GR: The role of activated charcoal and gastric emptying in gastrointestinal decontamination: A state-of-the-art review. *Ann Emerg Med* 2002; 39:273.
24. Heard K: The changing indications of gastrointestinal decontamination in poisonings. *Clin Lab Med* 2006; 26:1.
25. Tennenbein M: Position statement: Whole bowel irrigation. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. *J Toxicol Clin Toxicol* 2004; 42:843-854.
26. Woosley RL: *Drugs that prolong the QT interval and/or induce torsades de pointes*. Available at www.torsades.org. Accessed July 16, 2002.
27. Pickham D, et al: How many patients need QT interval monitoring in critical care units? Preliminary report of the QT in Practice study. *J Electrocardiol* 2010; 43:572-576.
28. Waring WS, et al: Evaluation of a QT nomogram for risk assessment after antidepressant overdose. *Br J Clin Pharmacol* 2010; 70:881-885.