symptomatic. Symptoms may range from confusion or fatigue to nausea, headache, tachycardia, and profound coma. The diagnosis is based on clinical grounds and supported by laboratory data. Carbon monoxide intoxication can occur from vehicle exhaust in enclosed automobiles, methylene chloride–based paint strippers, and by exposure to kerosene heaters or charcoal fires in closed spaces.

In suggested cases, arterial blood gas levels should be obtained with measured (not calculated) hemoglobin-oxygen saturation. A carbon monoxide level should be measured in patients with a measured systemic arterial oxygen saturation ($Sa_{O_2}$) lower than the calculated $Sa_{O_2}$ obtained from the arterial oxygen tension. Carbon monoxide does not alter $Pa_{O_2}$, which is a measure of the partial pressure of oxygen dissolved in plasma. Treatment of carbon monoxide poisoning is breathing 100% oxygen. Hyperbaric oxygen may be useful, but this therapy may not be readily available.

Inhalation of caustic substances such as ammonia, chlorine, and hydrogen fluoride causes acute symptoms of eye and upper airway inflammation. Pain, lacrimation, rhinorrhea, and upper airway symptoms usually prompt the individual to flee the environment. Inhalation of nitrogen dioxide (i.e., silo filler's disease) occurs in farmers who work in silos where fermentation of grain produces large quantities of the gas. Most patients recover without sequelae, but a few may develop bronchiolitis obliterans, an irreversible obstruction of small airways.

Metal fume fever causes influenza-like symptoms as a result of the inhalation of metal oxides generated by welding. Inhalation of platinum, formalin, and isocyanates may precipitate asthma. Pneumonitis can be induced by high-intensity inhalation of cadmium and mercury vapors.

Smoke inhalation may cause direct thermal injury that is usually confined to the upper airways, but it may also produce injury to the lower airways if exposure to sufficient steam occurs as a result of the high thermal content of water. Laryngeal edema, airway inflammation, and mucus can lead to airway obstruction, which requires intubation. Anoxia occurs from consumption of oxygen by fire and from cytotoxic injury from gases such as carbon monoxide, cyanide, and oxidants liberated during combustion. The combustion of natural and synthetic polymers often produces aldehydes, acetaldehyde, and acrolein, which also have a high irritant potential.

Cyanide poisoning uncouples oxygen from energy production by binding to cytochrome $a,a_3$ (i.e., cytochrome *c* oxidase), preventing electron transfer to oxygen, and it requires prompt treatment with 100% oxygen and sodium thiosulfate. Sodium thiosulfate facilitates the conversion of cyanide to thiocyanate. Recently, the U.S. Food and Drug Administration (FDA) has also approved the use of hydroxocobalamin, which combines with cyanide to form cyanocobalamin (vitamin $B_{12}$).

The treatment of inhalation injuries is supportive, with close attention to the airway. Oxygen should be provided, and continuous monitoring of cardiac and hemodynamic status is necessary. Sometimes, intubation and mechanical ventilation are needed to overcome airway obstruction and the development of respiratory failure.

*For a deeper discussion on this topic, please see Chapter 110, "Acute Poisoning," in* Goldman-Cecil Medicine, *25th Edition.*

## DRUG OVERDOSES

Drug overdoses are common causes of admission to the ICU. The presenting complaints and management of the more common overdoses that result in medical emergencies are summarized in Table 22-3.

**TABLE 22-3** COMMON DRUG OVERDOSES

| DRUG OVERDOSE | CLINICAL SYNDROME | TREATMENT |
|---|---|---|
| Acetaminophen (paracetamol) | 0.5-24 hr: nausea, vomiting<br>24-72 hr: nausea, vomiting, right upper quadrant pain; abnormal liver function tests and prothrombin time<br>72-96 hr: liver necrosis, coagulation, defects, jaundice, renal failure, hepatic encephalopathy<br>4 days-2 wk: resolution of liver dysfunction | Elimination: gastric lavage (if <1 hr after ingestion); activated charcoal (if <4 hr after ingestion); both longer if sustained-release product<br>Treatment: *N*-acetylcysteine for toxic ingestion |
| Amphetamines | Hypertension, tachycardia, arrhythmias, myocardial infarction, vasospasm, seizures, paranoid psychosis, diaphoresis, tachypnea | Elimination: activated charcoal for oral ingestion<br>Agitation or seizures: benzodiazepines<br>Hypertension: control agitation, α-antagonists (phentolamine), vasodilators (nitroglycerin, nitroprusside, nifedipine)<br>Hyperthermia: control agitation, external cooling |
| Iron | 0.5-6 hr: nausea, vomiting, gastrointestinal discomfort, gastrointestinal bleed, drowsiness, hypoglycemia, hypotension<br>6-24 hr: latency, quiescence (may not occur in severe ingestions)<br>6-48 hr: shock, coma, seizures, coagulopathy, acidosis, cardiac failure<br>2-7 days: hepatotoxicity, coagulopathy, metabolic acidosis, renal insufficiency<br>1-8 wk: gastrointestinal disorders, achlorhydria | Elimination: gastric lavage and/or whole bowel irrigation with polyethylene glycol electrolyte solution, especially after ingestion of tablets containing radiopaque iodinated dye for kidney-ureter-bladder radiography<br>Shock: intravenous fluids and blood (for hemorrhage); vasopressors if needed<br>Antidote: deferoxamine to chelate iron, when iron levels >500 µg/dL or severe ingestion suspected |
| Tricyclic antidepressants | Wide-complex tachyarrhythmias, hypotension, seizures | Tachyarrhythmias: alkalinize blood (pH 7.5-7.55) with intravenous bicarbonate<br>Seizures: benzodiazepines<br>Hypotension: fluid resuscitation, vasopressors |
| Salicylate | Respiratory alkalosis (initially), metabolic acidosis (after substantial absorption), pulmonary edema, platelet dysfunction, nausea, vomiting, hearing loss, agitation, delirium | Elimination: activated charcoal, hemodialysis (for severe poisoning), alkalinization of urine<br>Agitation or delirium: alkalinize blood with intravenous bicarbonate |