Human exposure to hydrocarbons (HCs) is a common problem. In 2010, U.S. poison centers reported 43,000 exposures to HCs, with the majority of cases managed in an outpatient setting. Exposure to HCs of patients who present to the emergency department (ED) can generally be classified into four types. The first is the accidental ingestion involving children younger than 5 years. This is the most common scenario causing fatality, and it usually involves significant pulmonary injury. Second is the intentional inhalational abuse of volatile HCs. Recreational abuse has been a medical problem since solvent inhalation became popular during the late 1800s. Fatalities in this group will typically occur within distinct demographic groups (Native Americans, homosexual men, and teenagers). Third is the accidental inhalational or dermal exposure to HCs in the household or workplace setting. The fourth type is massive oral ingestion of HCs in a suicide attempt.

HCs are a diverse group of organic compounds that contain hydrogen and carbon. Most HCs (e.g., gasoline) are byproducts of crude oil and are therefore called petroleum distillates. Some products, such as turpentine, are derived from pine oil, not petroleum. HCs can also be classified by their structure. The two main categories are straight chain HCs (aliphatic, such as propane) and those containing a benzene ring structure (aromatic, such as toluene). HCs can also have multiple nonorganic side chains. For example, halogenated HCs usually will have one or more bromide, chloride, fluoride, or iodide moieties (e.g., carbon tetrachloride). Finally, HCs are used as the solvent base for many toxic chemicals, such as insecticides and metals, that in turn can cause a separate distinct syndrome of poisoning. Although the range of toxicity of HCs can vary widely, the majority of human exposures are confined to petroleum distillates.

Acute HC toxicity usually affects three main target organs: the lungs, the heart, and the central nervous system (CNS). Although certain HCs can enter the body through the skin or gastrointestinal tract, HCs cause the most damage through the lungs. Despite the fact that there are thousands of different types of HCs, their potential for acute toxicity depends on four characteristics:

1. Viscosity: The capacity to resist flow or change. Low viscosity allows a substance to spread rapidly, and low-viscosity HCs spread easily into the airway and lungs.
2. Volatility: The ease for a liquid to turn into a gas. High volatility often has a detectable odor. HCs with high volatility can displace alveolar oxygen and cause hypoxia. Butane and propane are types of HCs with high volatility.
3. Surface tension: The capacity for a substance to collect on a liquid surface. Low surface tension enables a substance (e.g., turpentine) to disperse easily.
4. Chemical side chains: Often increase potential toxicity. These toxic side chains include metals (e.g., arsenic), halogens (e.g., carbon tetrachloride), and aromatic structures (e.g., toluene).

The primary target organ for toxicity is the lung. Fatalities after ingestion usually occur with an accompanying aspiration. A small amount of HC in the trachea can be devastating, whereas a much larger amount of the same compound in the stomach remains benign.

HCs affect the lungs through several mechanisms. HCs are usually poorly water soluble and penetrate into the lower airways, producing bronchospasm and an inflammatory response. Second, volatized HCs can displace oxygen in the alveolar space, causing hypoxia. Third, HCs cause direct injury to pulmonary alveoli and capillaries, producing distinct uniform lesions. Autopsy findings of these lesions include hyperemia, diffuse hemorrhagic exudative alveolitis with granulocytic infiltration, and microabscesses. Finally, HCs can inhibit surfactant function, leading to alveolar instability and collapse. These mechanisms lead to alveolar dysfunction, ventilation-perfusion mismatch, hypoxemia, and respiratory failure.

Certain HCs cause CNS depression (i.e., toluene, benzene, gasoline, butane, and chlorinated HCs). After respiratory exposure, most HCs passively diffuse through the pulmonary alveolus and are highly absorbed in blood and tissues. These HCs can cause euphoria, disinhibition, confusion, and obtundation. With an isolated single exposure, these effects usually have a rapid onset of intoxication and rapid recovery. For these reasons, substance abusers seek these HCs for recreational use. Inhalation of these substances avoids hepatic first-pass metabolism and generates high concentrations in the CNS. Chronic use of inhaled HCs can remain benign.
cause severe abnormalities in nervous system function, which include peripheral neuropathy, cerebellar degeneration, neuropsychiatric disorders, chronic encephalopathy, and dementia. More than 50% of patients who abuse toluene for more than 10 years will have cerebral cortical atrophy with histologic changes that include loss of neurons, diffuse gliosis, and axonal degeneration.3,7

Cardiac Pathophysiology

HCs can precipitate sudden death, especially after intentional inhalation. These compounds are thought to produce myocardial sensitization of endogenous and exogenous catecholamines, which then precipitates ventricular dysrhythmias and myocardial dysfunction. This is particularly true for halogenated and aromatic HCs (e.g., difluoroethane found in computer keyboard cleaning canisters).

Other Pathophysiology

Various HCs have been reported to be toxic to other organ systems. Certain recognized syndromes include toluene-induced renal tubular acidosis, benzene-induced bone marrow toxicity and leukemia, methylene chloride–induced carbon monoxide poisoning, and chlorinated HC–induced centrilobular hepatic necrosis and renal failure. Direct skin exposure of certain HCs can cause extensive chemical burns.

CLINICAL FEATURES

After oral ingestion, severe poisoning is related to aspiration, which is manifested with early respiratory symptoms, including cyanosis, coughing, grunting (small children), noisy respirations, or repeated bouts of vomiting. A patient may initially have mild symptoms and then develop tachypnea, dyspnea, bronchospasm, wheezing, rales, and fever during several hours.9,12 A change in mental status can be a manifestation of hypoxia or hypercapnia, but it is also a direct effect of HCs. In extreme cases, patients may have frank respiratory failure. Various additives or solutes can produce symptoms independently (e.g., seizures from camphorated HCs or cyanosis from nitrite-induced methemoglobinemia). Pesticides are often dissolved in an HC base. With pesticide exposures, it can be difficult to distinguish acute respiratory distress syndrome induced by HC aspiration from pulmonary edema induced by organophosphate exposures (see Chapter 163). Ingestion or injection of HC as a mechanism for deliberate self-harm is exceedingly rare.

The second scenario for HC toxicity is with solvent abuse. Solvent abuse is associated with various paraphernalia, such as plastic bags used for “bagging” (a method of pouring HCs in a bag or container and then inhaling deeply) and HC-soaked cloth used for “huffing” (a method in which abusers inhale through a saturated cloth). Patients often have the distinctive odor associated with organic HCs. A characteristic rash may be present over the mouth and nose (“glue-sniffer’s rash”) (Fig. 158-1). These patients can also present to the ED with CNS intoxication with euphoria, agitation, hallucinations, confusion, or bizarre behavior. This may progress to CNS depression and seizures. In the extreme case, these patients will be in cardiac arrest. This typically involves an individual who has inhaled solvents, performed some type of physical activity, such as running, and then suddenly collapsed. This is thought to be due to cardiac sensitization by endogenous catecholamine and the ensuing development of dysrhythmias.

Drug abusers who chronically inhale HCs may be brought to medical attention not specifically for treatment of their drug abuse
but rather for behavioral problems or nonspecific medical symptoms caused by their abuse. The long-term chronic abuser may clinically appear similar to the long-term “skid row” alcoholic, with peripheral neuropathy, cerebellar degeneration, and encephalopathy.7,13,14

A third common scenario is the accidental dermal or inhaled (nonaspiration) respiratory exposure to HCs in the workplace or home. Such exposures are rarely life-threatening. Most cases do not present for medical care or are handled primarily through lay discussion with consultants at local poison control centers.1 The few patients who present to the ED typically will be asymptomatic or have transient nonspecific symptoms, such as headache, dizziness, or nausea. Those with significant respiratory exposure may have persistent pulmonary complaints and physical findings such as coughing, wheezing, and cyanosis. Patients with significant acute dermal exposures may have pain and evidence of chemical burns consisting of erythema, swelling, blistering, and dermal destruction.

The patient who intentionally ingests or intravenously injects HCs in a suicide attempt is rare. However, these patients often ingest other substances. In the absence of aspiration or congestion of another toxic substance, the massive oral ingestion of most commonly available HCs is not associated with significant morbidity or mortality. These patients risk aspiration if they vomit.

**DIAGNOSTIC STRATEGIES**

The diagnosis is usually self-evident, supported by history and the patient’s characteristic odor. Ideally, a prehospital provider, police officer, school official, or family member brings the offending agent to the ED. The local poison control center may help identify and verify substances containing HCs. When verification of an unknown substance is required, a reference laboratory is usually needed. Laboratory identification of HCs is usually time-consuming and does not help ED management.

History and examination should focus on possible aspiration. These symptoms include cough, difficulty breathing, and shortness of breath. Signs of a significant exposure include tachypnea, tachycardia, wheezing, and hypoxemia. Patients with a significant HC exposure should have a chest radiograph taken. Radiographic changes can occur within 30 minutes of ingestion and may identify pathologic processes not recognized by auscultation in more than 50% of cases.2 Hypoxemia noted on pulse oximetry or arterial blood gas measurement provides supportive evidence for aspiration.

Patients who chronically abuse HCs may present to the ED for behavioral problems or nonspecific symptoms caused by their drug addiction. Similar to patients with alcoholism, these patients require evaluation for underlying medical disease. For example, serum electrolyte analysis will identify distal renal tubular acidosis associated with chronic toluene abuse.

**DIFFERENTIAL CONSIDERATIONS**

Organophosphate, salicylate, and paraquat poisonings can mimic the signs and symptoms of HC aspiration. In the scenario of the recreational abuser, multiple drugs of abuse are often present. Behavioral disorders and confusion can be caused by hypoxia and respiratory compromise as well as by the drugs themselves. In the chronic abuser, it can be difficult to differentiate between a functional and an organic confusional state.

**MANAGEMENT**

Dermal exposures to HCs can cause extensive burns, and exposed patients require early decontamination. Contaminated clothing should be removed, and the skin should be washed with soap and copious lukewarm water. Gastrointestinal decontamination should be avoided. The aphorism “the safest place in the body for hydrocarbons is the duodenum” holds true for most HCs regardless of the volume ingested. HCs are much more toxic to lungs than to the gastrointestinal tract, where they have a low potential for systemic absorption. In rare situations, gastrointestinal decontamination may be considered, in consultation with a regional poison center, but there is no sound evidence supporting gastric emptying, even in such cases. CHAMP is a long-standing mnemonic used to help identify the most serious ingestions for which toxicity of the HCs or additives is potentially life-threatening: C camphor, which can cause seizures and status epilepticus H halogenated HCs, which can cause dysrhythmias and hepatotoxicity A aromatic HCs, which can cause bone marrow suppression and cancer M metals (e.g., arsenic, mercury, and lead) P pesticides, which can cause cholinergic crises, seizures, and respiratory depression

If gastric emptying is desired, the use of a small-caliber nasogastric tube to evacuate stomach contents is recommended.

Because HC toxicity can cause rapid decompensation of a patient’s pulmonary, cardiac, and CNS functions, all patients should be in a well-observed area with cardiac monitoring and pulse oximetry. In severe cases, early intubation for airway control and positive end-expiratory pressure has been advocated to minimize aspiration risks and to counteract HC-induced alveoli collapse. However, no studies have proved this to be more beneficial than standard respiratory care. High-frequency jet ventilation, surfactant therapy, and extracorporeal membrane oxygenation have been attempted as treatments of children with respiratory failure secondary to aspiration,13,14 but no studies have been done to demonstrate benefit. Corticosteroids and antibiotics have not been shown to be beneficial in HC aspiration, but the differentiation between bacterial and chemical pneumonia may be difficult. More than 50% of children with significant HC poisoning will have a fever and leukocytosis.2 Theoretically, endogenous catecholamine might cause dysrhythmias in HC-sensitized myocardium, so most toxicologists recommend avoidance of cardiac-active catecholamines, such as epinephrine.

In most cases of HC ingestion or inhalation, supportive care and close observation and monitoring are the cornerstone of management. Currently, there are no specific antidotes for HCs.
Patients with exposures to known, relatively benign HCs should have a 4- to 6-hour period of observation. If pulmonary symptoms develop, a chest radiograph will show developing pneumonitis (Fig. 158-2), requiring hospital admission for prolonged observation.

Patients who present after an episode of recreational inhalation of HCs should be observed until clinical symptoms are improving or resolved, usually 1 to 3 hours.

**Key Concepts**

- Aspiration is the major toxic risk of HC poisoning.
- Gastrointestinal decontamination is potentially harmful in cases of HC ingestion and should be avoided.
- Symptoms of toxicity, especially aspiration, can be delayed, so asymptomatic patients should be observed for 4 to 6 hours and given clear instructions to return if symptoms develop after discharge.

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

**Figure 158-2.** Chest radiograph of a patient with hydrocarbon ingestion 6 hours after exposure.
References