ric acid (xylene) are excreted in the urine and can be used to document exposure, but urine levels do not correlate with systemic effects.

B. Other useful laboratory studies include CBC, electrolytes, glucose, BUN, creatinine, liver transaminases, CPK, and urinalysis. Chest radiography and pulse oximetry (or arterial blood gas measurements) are recommended for severe inhalation or if pulmonary aspiration is suspected.

V. Treatment

A. Emergency and supportive measures

1. **Inhalational exposure.** Maintain an open airway and assist ventilation if necessary (see pp 1–7). Administer supplemental oxygen and monitor arterial blood gases and chest x-rays.
   - **a.** If the patient is coughing or dyspneic, aspiration pneumonia is likely. Treat as for hydrocarbon pneumonia (see p 219).
   - **b.** If the patient remains asymptomatic after a 6-hour observation period, chemical pneumonia is unlikely and further observation or chest x-ray is not needed.
2. Treat coma (see p 18), arrhythmias (pp 13–15), and bronchospasm (p 7) if they occur. ***Caution:*** Epinephrine and other sympathomimetic amines can provoke or aggravate cardiac arrhythmias. Tachyarrhythmias may be treated with **propranolol** (p 504), 1–2 mg IV, or **esmolol** (p 449), 0.025–0.1 mg/kg/min IV.

B. Specific drugs and antidotes. There is no specific antidote.

C. Decontamination. Patients exposed only to solvent vapor who have no skin or eye irritation do not need decontamination. However, victims whose clothing or skin is contaminated with liquid solvent can secondarily contaminate response personnel by direct contact or through off-gassing vapor.

1. **Inhalation.** Remove the victim from exposure and give supplemental oxygen if available.
2. **Skin and eyes.** Remove contaminated clothing and wash exposed skin with soap and water. Flush exposed or irritated eyes with plain water or saline.
3. **Ingestion.** Administer activated charcoal orally if conditions are appropriate (see Table I–38, p 51). Consider gastric lavage for large ingestions (> 1–2 oz) if it can be performed within 30 minutes of ingestion.

D. Enhanced elimination. There is no role for enhanced elimination.

► TRICHLOROETHANE, TRICHLOROETHYLENE, AND TETRACHLOROETHYLENE

Josef G. Thundiyil, MD, MPH

Trichloroethane and trichloroethylene are widely used solvents that are ingredients in many products, including typewriter correction fluid ("white-out"), color film cleaners, insecticides, spot removers, fabric-cleaning solutions, adhesives, and paint removers. They are used extensively in industry as degreasers. Trichloroethane is available in two isomeric forms, 1,1,2- and 1,1,1-, with the latter (also known as methyl chloroform) being the more common. Tetrachloroethylene (perchloroethylene) is another related solvent that is used widely in the dry cleaning industry.

I. Mechanism of toxicity

A. These solvents act as respiratory and CNS depressants and skin and mucous membrane irritants. As a result of their high lipid solubility and CNS penetration, they have rapid anesthetic action, and both tricholorethylene and trichloroethane were used for this purpose medically until the advent of safer agents. Peak blood levels occur within minutes of inhalation exposure or 1–2 hours after ingestion. Their proposed mechanism of action includes neuronal calcium channel blockade and GABA stimulation.

B. Trichloroethane, trichloroethylene, their metabolite trichloroethanol, and tetrachloroethylene may sensitize the myocardium to the arrhythmogenic effects of catecholamines.

C. Trichloroethylene or a metabolite may act to inhibit acetaldehyde dehydrogenase, blocking the metabolism of ethanol and causing "degreaser's flush."

D. **Carcinogenicity.** The National Institute of Occupational Safety and Health (NIOSH) and the International Agency for Research on Cancer (IARC) consider tetrachloroethylene and trichloroethylene probable carcinogens (Class 2A). Although 1,1,2-trichloroethane is a NIOSH-suspected carcinogen, there is insufficient evidence to label 1,1,1-trichloroethane a carcinogen.

II. **Toxic dose**

A. **Trichloroethane.** The acute lethal oral dose to humans is reportedly between 0.5–5 mL/kg. The recommended workplace limits (ACGIH TLV-TWA) in air for the 1,1,1- and 1,1,2-isomers are 350 and 10 ppm, respectively, and the air levels considered immediately dangerous to life or health (IDLH) are 700 and 100 ppm, respectively. Anesthetic levels are in the range of 10,000–26,000 ppm. The odor is detectable by a majority of people at 500 ppm, but olfactory fatigue commonly occurs.

B. **Trichloroethylene.** The acute lethal oral dose is reported to be approximately 3–5 mL/kg. The recommended workplace limit (ACGIH TLV-TWA) is 50 ppm (269 mg/m^3), and the air level considered immediately dangerous to life or health (IDLH) is 1000 ppm.

C. **Tetrachloroethylene.** The recommended workplace limit (ACGIH TLV-TWA) is 25 ppm (170 mg/m^3), and the air level considered immediately dangerous to life or health (IDLH) is 150 ppm.

III. **Clinical presentation.** Toxicity may be a result of inhalation, skin contact, or ingestion.

A. **Inhalation or ingestion** may cause nausea, euphoria, headache, ataxia, dizziness, agitation, confusion, and lethargy and, if intoxication is significant, respiratory arrest, seizures, and coma. Hypotension and cardiac arrhythmias may occur. Inhalational exposure may result in cough, dyspnea, and bronchospasm. With severe overdose, renal and hepatic injury may be apparent 1–2 days after exposure.

B. **Local effects** of exposure to liquid or vapors include irritation of the eyes, nose, and throat. Prolonged skin contact can cause defatting, dermatitis and, in the case of trichloroethane and tetrachloroethylene, scleroderma-like skin changes.

C. **Ingestion** can produce GI irritation associated with nausea, vomiting, and abdominal pain. Aspiration into the tracheobronchial tree may result in hydrocarbon pneumonia (see p 219).

D. **Degreaser's flush.** Workers exposed to these vapors may have a transient flushing and orthostatic hypotension if they ingest alcohol, owing to a disulfiram-like effect (see Disulfiram, p 184).

E. **Other.** Severe exposures have caused development of cranial nerve neuropathies, optic neuritis, and skeletal muscle toxicity. Trichloroethylene has been shown to cross the placenta and is associated with preeclampsia and spontaneous abortion. Tetrachloroethylene is present in breast milk.

IV. **Diagnosis** is based on a history of exposure and typical symptoms. Addictive inhalational abuse of typewriter correction fluid suggests trichloroethylene poisoning.

A. **Specific levels**

1. Although all three solvents can be measured in expired air, blood, and urine, levels are not routinely rapidly available and are not needed for emergency evaluation or treatment. Confirmation of exposure to trichloroethane may be possible by detecting the metabolite trichloroethanol in the blood or urine. Hospital laboratory methods are not usually sensitive to these amounts.

2. Breath analysis is becoming more widely used for workplace exposure control, and serial measurements may allow for estimation of the amount absorbed.

B. Other useful laboratory studies include electrolytes, glucose, BUN, creatinine, liver transaminases, arterial blood gases, chest radiography, and ECG monitoring.

V. Treatment

A. Emergency and supportive measures

1. Maintain an open airway and assist ventilation if necessary (see pp 1–7). Administer supplemental oxygen and treat hydrocarbon aspiration pneumonitis (p 219) if it occurs.
2. Treat seizures (see p 22), coma (p 18), and arrhythmias (pp 12–15) if they occur. ***Caution:*** Avoid the use of epinephrine or other sympathomimetic amines because of the risk of inducing or aggravating cardiac arrhythmias. Tachyarrhythmias caused by myocardial sensitization may be treated with **propranolol** (p 504), 1–2 mg IV, or **esmolol** (p 449), 0.025–0.1 mg/kg/min IV.
3. Monitor for a minimum of 4–6 hours after significant exposure.

B. Specific drugs and antidotes. There is no specific antidote.

C. Decontamination (see p 45)

1. **Inhalation.** Remove the victim from exposure and administer supplemental oxygen, if available.
2. **Skin and eyes.** Remove contaminated clothing and wash exposed skin with soap and water. Irrigate exposed eyes with copious tepid water or saline.
3. **Ingestion.** Do ***not*** give activated charcoal or induce vomiting because of the danger of rapid absorption and abrupt onset of seizures or coma. Perform gastric lavage only if the ingestion was very large and recent (less than 30 minutes). The efficacy of activated charcoal is unknown.

D. Enhanced elimination. These procedures are not effective or necessary.

► VACOR (PNU)

Neal L. Benowitz, MD

Vacor rat killer (2% *N*-3-pyridylmethyl-*N'*-*p*-nitrophenylurea; PNU) is a unique rodenticide that causes irreversible insulin-dependent diabetes and autonomic nervous system injury. It was removed from general sale in the United States in 1979 but is still available in some homes and for use by licensed exterminators. The product was sold in 39-g packets of cornmeal-like material containing 2% PNU.

I. Mechanism of toxicity. PNU is believed to antagonize the actions of nicotinamide and, in a manner similar to that of alloxan and streptozocin, injure pancreatic beta cells. The mechanisms of autonomic neuropathy and CNS effects are unknown. Adrenergic neurons acting on blood vessels but not the heart are affected. As a result, orthostatic hypotension associated with an intact reflex tachycardia is the usual picture.

II. Toxic dose. Acute toxicity usually has occurred after ingestion of one 39-g packet of Vacor (approximately 8 g of PNU). The smallest dose reported to cause toxicity was 390 mg.

III. Clinical presentation. Initial symptoms include nausea and vomiting. Occasionally, confusion, stupor, and coma occur after several hours. After a delay of several hours to days, irreversible autonomic neuropathy, peripheral neuropathy, and diabetes may occur.

A. Autonomic dysfunction. Dizziness or syncope or both caused by severe orthostatic hypotension occur with an onset from 6 hours to 2 days after ingestion. Orthostatic hypotension usually is accompanied by intact reflex ta-