# Chapter 13

# SOLVENTS, FLUOROCARBONS, AND PAINTS

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## INTRODUCTION

A large segment of the Department of Defense (DoD) industrial workforce, including both civilian and military personnel, is employed in occupations that involve potential exposures to toxic environments. Organic solvents are a major source of exposure. A *solvent* is a material, usually a liquid, that is capable of dissolving another substance. Solvents include highly polar substances like water, as well as nonpolar organic compounds. Because the vapor pressure of water-based solvents is low, and therefore the potential for inhaling their vapors is also low, water-based solvents are not discussed in this chapter. Many of the nonpolar organic solvents, however, are relatively volatile and pose significant inhalation hazards.

The organic solvents include aliphatic and aromatic hydrocarbons, chlorinated hydrocarbons, alcohols, ether esters, and ketones. This textbook treats fluorocarbons separately from the other halogenated hydrocarbons; they are used as solvents, but their nonsolvent uses as refrigerants, propellants, and in fire-suppression systems have greater military and medical significance. Oil-based paints not only contain solvents that are used as diluents, they also contain resins, vehicles, and additives that are associated with a variety of illnesses to which occupational health professionals must respond.

## SOLVENTS

The DoD is a major user of solvents. More than 25 installations—including shipyards, air logistics centers, army depots, and aviation repair and maintenance facilities—each use over 27,500 gallons of solvent per year, and at least 120 installations use lesser amounts.[1]

In general, occupational exposures to solvents in the uniformed and civilian defense workforce duplicate those found in civilian industry. Solvents are used as dry-cleaning agents, chemical intermediates in manufacturing, drying compounds, general-purpose cleaners, paint thinners and removers, and in the manufacture of materiel. This chapter considers solvents as they are used in four military applications: vapor degreasers, cold-dipping cleaners, precision cleaners, and solvents that are associated with paints.

In vapor degreasing, solvent contained in degreasing tanks is heated to the boiling point—which is low, relative to water—and the vapors form a cloud over the surface. The items to be cleaned are lowered into the vapors, which then condense on the cold item and dissolve its surface oils and greases. The degreasing tanks are usually fitted with a cooling coil near the top that condenses the vapor, minimizing the solvent loss. The solvents that are commonly used in these operations include perchlorethylene, methyl chloroform, trichloroethylene, methylene chloride, and trichloro-fluoroethane.[2] Vapor degreasing is used by the army for a number of industrial operations including cleaning engines and other vehicle parts in vehicle-maintenance facilities and degreasing large-bore gun tubes after milling (Figures 13-1 and 13-2).

In cold-dipping degreasing, the item to be cleaned is simply dipped into a tank of solvent. These solvents—from petroleum distillates to mixtures that include aliphatic and aromatic hydrocarbons, ketones, cellosolves, and creosote—tend to have lower volatility than those used in vapor degreasers and therefore the risks of inhalation are less for workers. These procedures are also typical at vehicle maintenance facilities, particularly for smaller parts.

Precision cleaners—generally fluorocarbons—are also used in their liquid state, usually in cold dipping or as sprayed aerosols. They are generally used to clean sensitive electronic components. Large quantities of solvents are also used in paints and related products such as varnishes and lacquers, and paint thinners, primers, and strippers. Exposures occur during mixing, applying, or drying.

### General Toxicity

The potential health effects from exposure to solvents depend on a number of factors including the physical and chemical properties of the solvents, how the solvents are used, the way in which workers are exposed, and the duration (acute or chronic) of the exposure. In the industrial setting, there are two primary routes of exposure: inhalation of vapors or aerosolized mists and dermal absorption. The concentration of solvent vapors in air is expressed in *volume per volume* units (eg, parts per million). Concentrations can also be expressed as milligrams per cubic meter, which is a *weight per volume* measure.

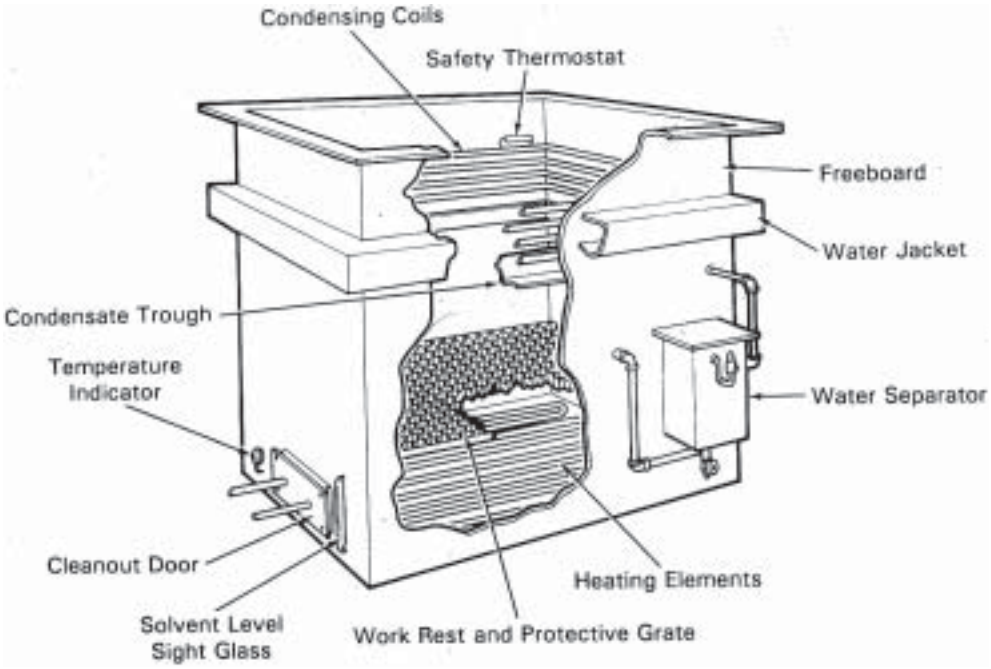



**Fig. 13-1.** A typical degreasing tank. Vapor degreasing is accomplished by lowering the object to be cleaned into the vapor zone above the liquid solvent. Solvent vapors condense on the cold item and loosen grease and oil, which then collect in the bottom of the tank. Condensing coils located near the top of the tank condense the solvent vapors, which collect in the condensate trough and are returned to the reservoir. The item is held in the vapor zone until the parts reach the temperature of the solvent vapor. At that time, condensation stops and the item is dry.


**Fig. 13-2**. Degreasing tanks located in a pit must be surrounded by a railing. Only authorized personnel can operate a vapor degreaser, and they must be wearing appropriate personal protective equipment. During routine operations, employees should wear chemical safety goggles and solvent-resistant aprons and gloves. During tank cleaning operations, a rescue harness, lifeline, and self-contained breathing apparatus should be worn. A second person, fully equipped to enter the tank, should be stationed outside the tank ready to assist if required.

Particulates suspended in air, such as dusts, are only expressed as weight per volume units.

*Acute* exposures are isolated and of short duration (minutes to several hours). In the occupational setting, acute exposures usually occur when pipes or supply lines are accidentally broken, spills occur, or workers enter chemical storage tanks to clean or paint. Acute exposures to most organic solvents cause central nervous system (CNS) depression and narcosis. Signs of toxicity include disorientation, euphoria, giddiness, and confusion, which are reversible in most cases when the victim is removed from the toxic environment. With sufficiently high exposures, the signs may progress to paralysis, convulsions, unconsciousness, and death due to respiratory or cardiovascular arrest.[3] Airborne concentrations of solvents sufficient to cause acute toxic effects are typically in the range of 1,000 to 10,000 ppm, although these are compound specific.

*Chronic* exposures to solvents are repeated, daily exposures to low concentrations. Again, concentrations sufficient to cause chronic toxicity are compound specific, but generally range from hundreds of parts per million to less than 1. Dermatitis is a common result of prolonged or repeated dermal contact. This is a result of the defatting of skin by the solvents, which causes dryness and fissuring of the skin.

The organic solvents are toxic to a number of organ systems. Well-defined CNS lesions have been described for n-hexane and n-butyl ketone. There are also reports of nonspecific behavioral, intellectual, and psychological effects among workers exposed to mixed solvents: house and car painters and jet-fuel handlers have reported impairment of visual perception, hand–eye coordination, and memory, as well as abnormal results on psychological tests.[4]

Many solvents are also toxic to the blood, liver, and kidneys. In many instances, the specific toxicity results from *biotransformation products* of the parent solvent with the formation of *reactive metabolites*.[3] The hepatotoxic effects of carbon tetrachloride and alcohol are thought to result from biotransformation products; however, they have yet to be thoroughly described. Similarly, some blood dyscrasias produced by benzene are believed to occur as a result of reactive metabolites.

## Respiratory Uptake of Solvents

Among other physiological factors, the rate and depth of pulmonary ventilation and the cardiac output affect the respiratory uptake of solvents, and these ultimately influence the toxic effects. Solvent uptake in the lungs is through simple diffusion: the difference in concentration between inspired air and the blood is the driving force that causes a solvent to enter the blood and be distributed throughout the body. Gas in the alveoli equilibrates rapidly with blood in the pulmonary capillaries. Blood solvent levels depend on the solubility of the solvent vapors in blood. For very soluble solvents such as chloroform, very little remains in the alveolar gas for expiration. Soluble compounds, however, require longer to equilibrate with the blood than low-soluble compounds because a greater amount of compound is required to reach equilibrium. An increasing ventilation rate will increase the delivery of solvent vapors to the lungs and decrease the time required for equilibration. Uptake of these solvent vapors is said to be *ventilation limited* (ie, equilibration is dependent on the rate and depth of respiration). In contrast, the vapors of solvents with low solubility take less time to equilibrate with the blood, and the rate is dependent on blood flow through the lungs. Uptake of these substances is said to be *perfusion limited*.[3,5] The solubility of a solvent, the time necessary for it to reach equilibrium in the blood, and the concentration of the solvent in the blood at equilibrium are (*a*) related through the solubility coefficient (S) and (*b*) limited by ventilation, perfusion, and cardiac output (Table 13-1). The solubility coefficient represents the ratio of the concentration of a vapor or gas in an aqueous medium to the concentration in the gas phase.

## Specific Toxicity

Specific toxicological effects can be described for individual solvents (Table 13-2). However, in most industrial settings exposures will be to complex mixtures; the chemical composition of these solvent mixtures will vary with the particular lot of solvent furnished by various suppliers, in many cases. The only way to be certain of the specific solvent composition is to consult the Material Safety Data Sheet supplied by the manufacturer for the specific lot of solvent in use.

### *Aliphatic Hydrocarbons*

The aliphatic hydrocarbons include the saturated, straight-chain paraffins (alkanes) and the unsaturated olefins (alkenes). Compounds with chain lengths of 5 to 16 carbon atoms are usually liquids, and those exceeding 16 carbons are usually solids. Most compounds containing carbon chains exceeding eight units have low volatility and pose little inhalation threat. Typical aliphatic hydrocarbons include hexane, which

**TABLE 13-1**

**RESPIRATORY UPTAKE**

| Chemical | S* | Equilibration Time | [Blood] at Equilibrium | Physiological Parameter Limiting Uptake |
|---|---|---|---|---|
| Ethanol | 1,100.0 | Slow | High | Ventilation |
| Acetone | 245.0 | ↓ | ↓ | |
| Methyl ethyl ketone | 202.0 | | | |
| Benzene | 7.8 | | | |
| Carbon tetrachloride | 2.4 | | | |
| Ethylene | 0.15 | Fast | Low | Perfusion |

*Solubility coefficient

Source: Alarie Y. *Inhalation and Toxic Responses of Lung*. Kansas City, Kan: Mid America Toxicology Course; 1981: 285–400.

is used frequently as a solvent in adhesives and is found in many paints and varnishes[6]; kerosene, which is used as a fuel, a carrier for pesticides, and a cleaning solvent; and stoddard solvent, which can be a mixture of more than 100 different aliphatics and is used extensively as a degreasing agent.

***Hexane.*** Commercial hexane, sometimes called hexane isomers, can consist of up to 100% n-hexane. Until the mid-1960s, n-hexane was considered to be relatively nontoxic because workers could be exposed to high concentrations of vapor with no discomfort. However, in l964, workers using products containing n-hexane began to exhibit sensory and motor deficits. Exposures were estimated to be 500 to 2,500 ppm. Methyl-n-butyl ketone (MNBK) was shown to produce a similar neuropathy.[3]

Hexane is readily absorbed and has an affinity for fatty tissues. An extensive research program in both animals and humans identified 2,5-hexanedione as a common metabolite of both n-hexane and MNBK, and this metabolite is believed to be responsible for the neurotoxicity.[5] 2,5-Hexanedione was also shown to produce a neuropathy in animals that was identical to that produced in humans by n-hexane and MNBK.[3]

The American Conference of Government Industrial Hygienists (ACGIH) has recommended a Threshold Limit Value (TLV) of 50 ppm (176 mg/m$^3$) for n-hexane, although recommended levels for other hexane isomers are 10-fold higher.[7] The current Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) follows the ACGIH guidance: 50 ppm for n-hexane and 500 ppm for mixed hexane isomers.[8]

***Stoddard Solvent.*** Several commercial solvent mixtures are derived from the distillation of petroleum. The nomenclatures for stoddardlike solvents include varsol, mineral spirits, and white spirits. Although these mixtures differ in their distillation ranges and performance characteristics, their toxicological properties are similar. Stoddard solvents cause the typical hydrocarbon-solvent effects of CNS depression from high inhalation exposures and skin irritation from dermal exposures. In addition, scattered reports in the literature demonstrate that chronic exposures to stoddard solvents cause myelotoxic effects and liver toxicity.[6] Both the ACGIH TLV and OSHA PEL for stoddard solvent is 100 ppm (525 mg/m$^3$).[7,8]

***Kerosene.*** Kerosene is a mixture of petroleum hydrocarbons with carbon chain lengths of 9 to 16 units, although its composition varies with the source of crude oil and the refining methods. Other common names for kerosene include astral oil, coal oil, and No. 1 fuel oil. Typical constituents of kerosene include mixtures of aliphatic, naphthenic, and aromatic hydrocarbons. Regardless of its name and exact composition, kerosene has a relatively low acute systemic toxicity, although it is a significant health hazard when the liquid directly enters the trachea and lungs or is aspirated from the stomach. Milliliter quantities of kerosene can cause pneumonitis, pulmonary edema, hemorrhage, and necrosis. Other petroleum-derived solvents pose a similar threat.[6] Dermal exposure to kerosene has also been shown to cause skin irritation, and inhalation exposures may cause bone marrow depression, although the latter response may be due to contamination of the kerosene with benzene (discussed later in this chapter).[9] TLV and PEL Values have not been established for kerosene.

**TABLE 13-2**

**TOXICITY OF REPRESENTATIVE SOLVENTS**

| Solvent | Toxicity Rating* | Target Organ or Effect of Chronic Toxicity | WOE† | TLV (ppm) | PEL |
|---|---|---|---|---|---|
| *Aliphatic Hydrocarbons* | | | | | |
| n-Hexane | 2–1 | neurotoxic, testicular atrophy | UE | 50 | 50 |
| Kerosene | 3 | pneumonitis, edema | NE | — | — |
| VM&P naphtha | 2 | CNS depression, skin irritation | NE | 100 | — |
| Stoddard solvent | 1 | — | — | — | — |
| *Aromatic Hydrocarbons* | | | | | |
| Benzene | 3 | CNS, aplastic anemia, leukemia | A | 10 | — |
| Toluene | 3 | CNS depressant, dermal irritant | D | 100 | 100 |
| Cresol | 2 | cancer, dermal irritant | C | 5 | — |
| Phenol | 3 | developmental toxicity | D | 5 | — |
| Xylene | 2 | liver | D | 100 | 100 |
| *Chlorohydrocarbons* | | | | | |
| 1,2-Dichloroethane | 2 | circulatory system | B2 | 200 | 100 |
| Trichloroethylene | 3 | lung, liver | B2 | 50 | 50 |
| Carbon tetrachloride | 3 | liver | B2 | 5 | 2 |
| Perchlorethylene | 3 | liver, blood | B2 | 50 | 25 |
| Methylene chloride | — | lung, liver | B2 | 50 | — |
| *Fluorocarbons* | | | | | |
| Dichlorodifluoromethane | 1 | lung, liver | NE | — | — |
| Trichlorofluoroethane | 2–1 | — | — | — | — |
| Trichlorotrifluoroethane | 1 | — | — | — | — |
| Chloropentafluoroethane | 2–1 | — | — | — | — |
| Bromochloro-difluoromethane | 1 | — | — | — | — |
| Dibromotetrafluoroethane | 2 | — | — | — | — |
| *Aldehydes and Ketones* | | | | | |
| Formaldehyde | 3 | irritant, sensitizer | B1 | 0.30 | 0.75 |
| Acetone | 2–1 | liver, kidney carcinogen | D | 200 | 200 |
| Methyl ethyl ketone | 3 | CNS neurotoxin, fetotoxin | D | — | — |
| Methyl n-butyl ketone | 2 | CNS | — | — | — |
| *Glycols* | | | | | |
| Ethylene glycol | 2–1 | liver, kidney | NE | 50 | 50 |
| Propylene glycol | 2–1 | kidney, blood | NE | — | — |
| *Glycol Ethers* | | | | | |
| Ethylene glycol monomethyl ether | 2 | toxic encephalopathy, bone-marrow depression | NE | 5 | 5 |
| Ethylene glycol monoethyl ether | 2 | testicular atrophy | NE | 5 | 5 |

*Toxicity Rating: a qualitative ranking of compound toxicity; categories range from 1 to 3. 1: low or slight; 2: moderate; 3: severe. Source of toxicity rating: Sax NJ. *Dangerous Properties of Industrial Materials*. 6th ed. New York: Van Nostrand Reinhold; 1984.

†Weight of Evidence: a qualitative classification of the potential for a compound to produce cancer in humans (developed by the EPA)

A: Human carcinogen (based on sufficient information from epidemiology studies to support a causal relationship)

B1: Probable human carcinogen (based on sufficient information from animal studies and limited information from human epidemiology studies)

B2: Probable human carcinogen (based on sufficient information from animal studies, but evidence of carcinogenicity in humans is inadequate)

C: Possible human carcinogen (based on limited information from animal studies)

D: Cannot be classified

E: No evidence of carcinogenicity in humans

UE: Compound is undergoing evaluation for carcinogenicity

NE: Compound has not been evaluated for its potential human carcinogenicity

### *Aromatic Hydrocarbons*

Aromatic hydrocarbons containing an unsaturated six-carbon ring structure are used extensively and are toxic to multiple organ systems. These solvents are primary skin irritants, due to their defatting properties, and their vapors are irritating to the mucous membranes and airways. Systemically, aromatic hydrocarbons are CNS depressants, and several of these compounds have toxic actions on other target organs. Aromatic hydrocarbons are important in the production of plastics and rubber products, and are used in some paints.[9]

***Benzene.*** Benzene is the most toxic member of this group. It is a common ingredient in paint and varnish removers and paint thinners as well as a contaminant of many of the petroleum-based solvents. In addition to its CNS-depressing actions, chronic exposure to benzene suppresses the hematopoietic system. Acute and chronic lymphocytic leukemias and aplastic anemia (which has a mortality rate of 70% over a 5-y period) have been associated with worker exposures.[3,9] Epidemiological studies have also implicated benzene as a cause of acute myelogenous leukemia. The mechanisms and etiology of chronic lymphocytic leukemia and aplastic anemia have not been described; however, they appear to be initiated by an unidentified metabolite of benzene.[3] Clinically, chronic benzene exposures have been associated with decreased numbers of circulating erythrocytes and leukocytes, conditions which may be an early indication of benzene toxicity.[3] The ACGIH TLV for benzene is currently 10 ppm; however, the ACGIH has proposed lowering this value to 0.1 ppm.[7] The OSHA PEL for benzene is 1 ppm.[8]

***Alkyl Benzene.*** The alkyl benzenes include *toluene* (methylbenzene), the *xylenes* (ortho, meta, and para isomers of dimethyl benzene), and the *cresols* (monomethyl phenols). The evidence from studies done with animals suggests that this group of solvents does not have the hematopoietic toxicity that is characteristic of benzene. The acute and chronic effects of toluene are CNS depression and dermal irritation.[10] The xylenes are also CNS depressants; the most common symptoms reported from occupational exposures are headache, fatigue, lassitude, irritability, and gastrointestinal disturbances.[8] Cresol is a strong dermal irritant with systemic effects on the kidneys and liver; its toxicity is similar to that of phenol. For work-place exposures to toluene and xylene, the ACGIH TLVs and OSHA PELs are 100 ppm; for cresol, both values are 5 ppm.[7,8]

### *Halogenated Hydrocarbons*

These solvents are composed of carbon, hydrogen, and a halogen (usually chlorine or fluorine), and can be divided into the simple chlorohydrocarbons and the chlorofluorocarbons. The simple chlorohydrocarbons contain chlorine as the only halogen moiety and are excellent solvents for oils, fats, and other organic compounds. Simple chlorohydrocarbons are often used in both vapor and cold-dip degreasing. They are also used extensively in paint removers, solvents, and thinners.

The most common acute toxic effect of the simple chlorohydrocarbons is CNS depression; high exposures may cause respiratory depression or circulatory failure resulting in death. The vapors of the simple chlorohydrocarbons are not especially irritating to the upper airways, but repeated skin exposures may cause dermatitis due to their defatting actions.[2]

The solvent methylene chloride (dichloromethane) has a unique toxic action of which occupational health professionals should be aware. This very volatile chlorohydrocarbon is widely used in paints, paint strippers, degreasing operations, and as an aerosol propellant. In addition to the typical CNS depression, methylene chloride has also been found to be rapidly metabolized to carbon monoxide. Short exposures to high levels of this solvent have been shown to produce carboxyhemoglobin levels over 10%. This could have a significant adverse health effect in workers with existing cardiovascular disease.[9]

With chronic exposures, many of the chlorinated hydrocarbons are hepatotoxic, causing both fatty infiltration and necrosis. Carbon tetrachloride, chloroform, and 1,1,2-trichloroethane are also toxic to the kidneys; however, the other chlorohydrocarbons do not share this effect.[3] A number of chlorinated solvents have been demonstrated to cause cancer in laboratory animals (see Table 13-2). Despite extensive data from studies on animals that indicate potential carcinogenicity, however, evidence from epidemiological studies on humans has been inconclusive. Data on worker exposures to trichlorethylene, fluorocarbons, and methylene chloride have all been negative.[9]

One further concern is the potential toxicity of the thermal decomposition products of halogenated hydrocarbons. At high temperatures, the chlorohydrocarbons decompose to hydrogen chloride and phosgene. Toxic levels of these hazardous gases can be produced during welding operations in atmospheres of chlorinated hydrocarbon solvents.[10]

The chlorofluorocarbons contain both chlorine and fluorine as the halogen moiety and are used extensively in the military as precision cleaners for electronic components and in vapor degreasing. Their nonsolvent uses are discussed separately in this chapter.

### *Aldehydes and Ketones*

Although aldehydes and ketones are grouped together based on their chemical structures, they are used for distinctly different purposes. The most common aldehyde, formaldehyde, is used in adhesives, industrial coatings, and in the production of certain dyes. The ketones are widely used as solvents.

Aldehydes and ketones also have somewhat different toxicological effects. Formaldehyde is a potent skin and eye irritant and a well-known sensitizer. Allergic responses are commonly seen in persons who come in contact with formaldehyde. Studies with animals also show that inhalation of 15 ppm of formaldehyde produces carcinomas in the nasal cavities of rats.[8] The common ketones including acetone, methyl ethyl ketone (MEK), and methyl isopropyl ketone (MIPK) are also irritants to the eyes and mucous membranes. At high concentrations, ketones are CNS depressants. MNBK is uniquely neurotoxic, as was discussed previously.

The potential health effects that distinguish the aldehydes from the ketones are also the rationale for the differences in their exposure limits. The ACGIH TLV for formaldehyde is 1 ppm, but this value may be reduced to 0.3 ppm.[7] OSHA has established a PEL of 1 ppm, with a 2 ppm *ceiling value*, and is also considering a reduction (a ceiling value is an exposure level that cannot be exceeded at any time during an 8-hour workday). Both the ACGIH TLV and OSHA PEL are 5 ppm for MNBK and 200 ppm for MEK and MIPK.[7,8]

### *Glycols and Glycol Ethers*

Glycols and glycol ethers differ in both their degree and their mechanisms of toxicity, although they are usually grouped together by virtue of their chemical structures. The two are also used differently. Glycols are used commonly as industrial solvents for nitrocellulose and cellulose acetate, and in the production of pharmaceuticals. The glycol ethers are used extensively as solvents for lacquers, varnishes, resins, dyes, and inks.

Glycols have low vapor pressure and do not present a significant inhalation hazard unless they are heated or aerosolized. However, ethylene glycol has greater oral toxicity in humans than has been demonstrated in other mammalian species. The primary toxic action associated with ethylene glycol is metabolic acidosis, which is caused by its metabolite glycolic acid. Calcium oxalate, another metabolic byproduct of ethylene glycol, tends to accumulate in the proximal renal tubules and causes necrosis and functional changes in the kidneys.[3]

The glycol ethers include ethylene glycol monomethyl ether (EM), ethylene glycol monoethyl ether (EE), and the propylene series of glycol ethers. While they are not acutely toxic when ingested or inhaled, they have a unique reproductive toxicity: testicular atrophy and hematological effects were found when animals were administered EM and EE by various routes. The propylene series of glycol ethers does not appear to cause these reproductive effects.[3]

Due to these toxic effects, the ACGIH recently lowered the TLV for EM and EE; current recommendations are 5 ppm.[7] The OSHA PELs for these solvents are also 5 ppm.[8]

### Exposure Controls

Two of the many controls used in vapor degreasing operations are condensers and local exhaust ventilators, although other controls may be designed for other operations that require solvents. Properly designed vapor degreasing units are equipped with condensers that surround the tank, which minimize the escape of solvent vapors into the ambient environment of the workplace. All of these units should be equipped with thermostatic controls mounted above the normal vapor level, which will shut off the heat source if the vapors rise above the condensing surface. Local exhaust ventilation controls may be necessary depending on the type of installation. Even with these controls, open flames, electric heating elements, and welding operations must not be located near a degreaser.[2]

Vapor degreasers also require periodic cleaning to remove the sludge and metal chips that have accumulated at the bottom of tanks. This requires that the solvent be distilled off until the heating surface is nearly exposed. The unit is then allowed to cool, and the sludge and remaining solvent are drained off. Personnel who enter a degreasing tank to perform this operation should wear respiratory protective equipment and a lifeline that is held by an attendant.[2]

The exposure controls for cold-dip degreasing and precision cleaning operations are much less extensive. Lids should be provided for the dip tanks to minimize evaporation of the solvent. Personal protective equipment (PPE) such as a faceshield and gloves must be worn to prevent the solvent from coming in contact with the skin and eyes.

## FLUOROCARBONS

The first fluorocarbon, carbon tetrafluoride, was isolated in 1926.[11] Although no such compound occurs in nature, numerous additional fluorocarbons have been synthesized in substantial quantities since the 1940s. Commercial interest in fluorocarbons centered around their chemical and thermal stability and led to their extensive use as aerosol propellants, refrigerants, plastic foaming agents, heat-exchange agents, and solvents; as propellants for therapeutic agents and antiasthmatic drugs; and as general anesthetics. By the mid-1970s, production of these chemicals—originally considered to be inert refrigerants—had exceeded 2 billion pounds, most of which was released eventually into the environment.[12]

Fluorocarbons can be divided into the fully halogenated (nonhydrogenated) and the hydrogenated species. A further subdivision of these two groups—into the chlorinated and nonchlorinated species—is necessary to understand the significance that these chemical structures have in the current depletion of the earth's ozone layer (Table 13-3). This process involves the nonhydrogenated fluorocarbons, which are the more stable and therefore have the greater probability of reaching the ozone layer. Of the nonhydrogenated species, the chlorinated fluorocarbons are postulated to be the most detrimental.

**TABLE 13-3**

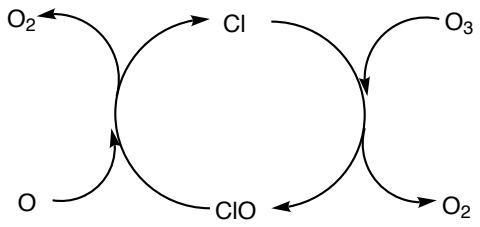
**REPRESENTATIVE FLUOROCARBONS**

| Type | Formula |
|---|---|
| **Nonhydrogenated** | |
| *Chlorinated* | |
| Dichlorodifluoromethane | $CCl_2F_2$ |
| Trichlorofluoromethane | $CCl_3F$ |
| *Nonchlorinated* | |
| Octafluorocyclobutone | $C_4F_8$ |
| Dibromotetrafluoroethane | $C_2F_4Br_2$ |
| **Hydrogenated** | |
| *Chlorinated* | |
| Chlorodifluoromethane | $CHClF_2$ |
| Chlorodifluoroethane | $CH_3–CClF_2$ |
| *Nonchlorinated* | |
| Difluoromethane | $CH_2F_2$ |
| Trifluoroethane | $CH_2CF_3$ |

The fluorocarbons are similar in composition to the chlorinated hydrocarbons, with the addition of a fluorine moiety. Numerous possible moiety combinations of carbon with hydrogen, fluorine, bromine, and chlorine have been prepared with the aliphatic hydrocarbon series. The fluorocarbons are usually clear, colorless, highly volatile liquids with a mild, somewhat ethereal odor. They are nonflammable, have high density, low viscosity, low surface tension, low toxicity, and are very stable.

Fluorocarbons do not react with most metals at temperatures below 200°C, or with most acids or oxidizing agents. However, under unusual circumstances, they can be made to react with highly reactive metals: for example, trichlorofluoromethane (CFC-11) reacts with concentrated sulfuric acid and sulfur trioxide at room temperature.

With few exceptions, the fluorocarbons are relatively inert to chemical reactions on earth and in the lower atmosphere. When compared to other halogenated compounds, the hydrolysis rates for the fluorocarbons are quite low, although there is considerable variation within the group. At atmospheric pressure, the rate of hydrolysis is too low to be measured by most analytical methods. This low rate of hydrolysis prevents the fluorocarbons from degrading in the troposphere.[13] However, it is their inherent stability—which allows them to migrate, intact, as high as the stratosphere—that is the source of concern surrounding the depletion of the ozone layer. Through photodegradation and free-radical reactions, chlorofluorocarbons provide a large reservoir of free chlorine atoms, which then catalyze the destruction of ozone:




It is postulated that this is a chain reaction; it allows one chlorine atom to continue to react, thus destroying thousands of molecules of ozone. Much of the research done on this problem is, of necessity, theoretical; furthermore, the contribution that free chlorine from natural sources might make to this process is neither well documented nor well understood. However, sufficient data exist for the international scientific community to call for a ban on the release of

chlorofluorocarbons into the environment. The nonchlorinated fluorocarbons are more stable, are not sources of free chlorine, and are therefore not of such environmental concern.

### Current Status of Ozone-Depleting Substances

The Montreal Protocol, an international agreement to limit the release and production of ozone-depleting substances, was signed in September 1987.[14] This agreement, ratified by the U.S. Senate, became effective in January 1989; it stringently restricts the international production and use of chlorofluorocarbons and *halons* (a trademarked name for several tetrafluoroethylene polymers) (Exhibit 13-1). A DoD Directive (DoDI) issued on 13 February 1989 clarified the army's position concerning specific chlorofluorocarbons and halons (Table 13-4).[15] This directive affects all DoD components, establishes policy, and assigns the responsibility for

- managing the production of chlorofluorocarbons and halons,
- identifying chlorofluorocarbon and halon applications and prioritizing their uses,
- identifying a long-term process to decrease the DoD's dependence on chlorofluorocarbons and halons,
- developing research and development programs to produce or evaluate suitable substitutes for chlorofluorocarbons and halons, and
- designing a tracking system to document the DoD's annual requirements for chlorofluorocarbons and halons.

**EXHIBIT 13-1**

**CFC AND HALON RESTRICTIONS OF THE MONTREAL PROTOCOL**

| Restriction | Year of Accomplishment |
|---|---|
| Freezing of CFC consumption at 1986 levels* | 1989 |
| Freezing of halon consumption at 1986 levels | 1992 |
| Further reduce CFC consumption by 20%† | 1993 |
| Further reduce CFC consumption by 30%‡ | 1998 |

*Represents an initial reduction of approximately 15%
†Reduced from 1986 level
‡Reduced from 1993 level
Source: Treaty Document 100-10. December 21, 1987. *Montreal Protocol on Substances That Deplete the Ozone Layer.* Done at Montreal on September 16, 1987, to the Vienna Convention for the Protection of the Ozone Layer. Ratified by the US Senate March 14, 1988. A copy of the treaty document may be obtained from the Library of Congress, Washington, DC.

**TABLE 13-4**

**CFCs AND HALONS AFFECTED BY THE DoD DIRECTIVE AS OF AUGUST 1988**

| Ozone Depleter | Formula | Chemical Name |
|---|---|---|
| CFC-11 | $CCl_3F$ | Trichlorofluoromethane |
| CFC-12 | $CCl_2F_2$ | Dichlorodifluoromethane |
| CFC-113 | $C_2Cl_3F_3$ | Trichlorotrifluoroethane |
| CFC-114 | $C_2Cl_2F_4$ | Dichlorotetrafluoroethane |
| CFC-115 | $C_2ClF_5$ | Chloropentafluoroethane |
| Halon 1211* | $CBrClF_2$ | Bromochlorodifluoromethane |
| Halon 1301* | $CBrF_3$ | Bromotrifluoromethane |
| Halon 2402* | $C_2Br_2F_4$ | Dibromotetrafluoroethane |

*Registered trademark of Allied Chemical Corporation.
Source: US Department of Defense. *Chlorofluorocarbons (CFCs) and Halons*. Washington, DC: DoD; 1989. DoD Directive 6050.9.

Both the Montreal Protocol and the DoDI will substantially affect the use of chlorofluorocarbons and halons in the near future. The phased-in restrictions should result in a reduction of CFCs by over 60%, as measured from 1989 consumption levels.

The effects of compliance will be at least these two: (1) the use of present compounds will be curtailed, and (2) replacement chemicals will enter the DoD's supply channels. Unfortunately, replacement chemicals have not yet been designated and therefore cannot be discussed in this chapter, but the search for replacement chemicals will obviously center around the hydrogenated nonchlorinated compounds.

The mandated phaseout of the chlorofluorocarbons and halons has generated tremendous interest in developing replacement chemicals. However, current research appears to be driven more by the need to find and produce chemicals that are environmentally safe rather than physiologically nontoxic. Occupational health professionals may soon find themselves confronting new replacement chemicals that are accompanied by little or no medical or toxicological information.

### Civilian and Industrial Exposures

The fluorocarbons and halons are used as refrigerants, polymer intermediates, propellants, anesthetics, fire extinguishers, foam-blowing agents, dry-cleaning agents, and as degreasing solvents in the electronics industry. Workers who produce and package fluorocarbons are at highest risk of exposure to high concentrations of these chemicals, but significant exposures also occur when fluorocarbons are used as solvents or cleaners. Because fluorocarbons disperse rapidly when released, only minimal exposure appears to occur from their use as refrigerants and propellants. However, recent uses of fire extinguishers to protect computer and electronic areas have created the potential for short-term, high-concentration exposures when large volumes of the material are released in confined spaces. Chronic occupational exposure to hospital operating-room personnel from the use of anesthetic gases is also significant.

### Militarily Unique Exposures

In addition to the civilian and industrial uses already discussed, fluorocarbon use in the military includes specialized applications such as the use of CFC-114 for submarine refrigeration to eliminate vibration that might lead to detection by the enemy. Halon 1211 and Halon 1301 are used to suppress fires in the crew compartments of tactical vehicles, aircraft, shipboard systems, and in command, control, and communication centers (see Chapter 6, Health Hazard Assessments). Significant amounts of these materials are used by DoD workers. Based on 1986 production figures, the DoD procured just under 35% of the United States's total production of Halon 1211, approximately 6% of Halon 1301, and just under 5% of the total production of the regulated chlorofluorocarbons.[16]

Fluorocarbons, as they are used in submarines and other tactical vehicles, expose military personnel to hazardous situations not usually encountered by civilians: specialized weapons systems are used in essentially closed environments. Military personnel are not always able to simply leave a contaminated environment; their duties may oblige them to stay and man their equipment, thereby exposing them to higher concentrations for longer times than may be typical in the civilian community.

For example, the crew of an M60A3 tank can be exposed to Halon 1301 if the tank's automatic fire extinguishing system is activated during a system malfunction or an actual fire. In either situation, the crew could be exposed to oxygen deficiency, the toxic effects of Halon 1301 itself, or the toxic effects of the decomposition products of Halon 1301:

> The discharge of four 7-pound (net weight) bottles of Halon 1301 into the closed, unventilated M60A3 crew compartment (estimated volume 12.7 m) would be expected to initially depress the oxygen concentration in the crew compartment to an average value of 17.5 percent. This figure is based on the assumption that the discharged Halon displaces crew-compartment air with nearly 100 percent efficiency ….[M]inimum oxygen concentrations at various locations…range from about 13 percent to 19 percent, with the lowest concentrations occurring near floor level. Exposure to oxygen concentrations in the 13 to 16 percent range can cause increased breathing and pulse rate, and slight impairment of concentration and muscular coordination.
>
> . . . .
>
> [The toxic] effects of exposure to Halon 1301 concentrations in the 7 to 20 percent range are varying degrees of central nervous system depression. Exposures in the 7 to 10 percent range cause mild anesthetic effects including dizziness and tingling of the extremities. Exposures to concentrations above 10 percent are usually accompanied by pronounced dizziness, and reduced physical dexterity and mental acuity.
>
> . . . .
>
> Halon 1301 decomposes upon exposure to flame. The decomposition products (hydrogen fluoride, hydrogen bromide, free bromine, and phosgene analogues) are severely irritating (even at low concentrations) to the eyes and the respiratory tract….Therefore, the occurrence of any crew-compartment fire during training necessitates immediate evacuation of the M60A3.[17(pp2–4)]

It is the risk of exposure to the decomposition products of halon fire-extinguishing agents within enclosed spaces that is the immediate hazard to the crew (Table 13-5). These decomposition products are all significantly more toxic than their parent compounds; even at low concentrations, they are severely irritating to the eyes and respiratory tract. As the intensity of the fire increases, the concentration of the toxic byprod-ucts also increases; in enclosed spaces, halon decomposition products can reach extremely toxic concen-trations.

### Pharmacological Effects

Although they are relatively nontoxic, numerous fluorocarbon injuries have occurred. The ban on fluorocarbon propellant use has already substantially reduced human exposure, but the Consumer Product

**TABLE 13-5**

**EXPOSURE LIMITS FOR COMBUSTION AND DECOMPOSITION PRODUCTS OF COMMON HALONS**

| Product | Exposure Limit* (ppm) | Type of Limit |
|---|---|---|
| HBr | 3.0 | Ceiling† |
| HCl | 5.0 | Ceiling |
| HF | 3.0 | Ceiling |
| $Br_2$ | 0.3 | STEL‡ |
| $Cl_2$ | 1.0 | STEL |
| $F_2$ | 2.0 | STEL |
| $COBr_2$ (Carbonyl bromide) | 0.1§ | — |
| $COCl_2$ (Phosgene) | 0.1 | 8-Hour |
| $COF_2$ (Carbonyl fluoride) | 5.0 | STEL |

*All exposure limits, except carbonyl bromide, established by ACGIH

†Ceiling: the value above which concentrations must never rise

‡STEL: short term exposure limit

§No value established (phosgene value assumed)

Source: US Department of the Army. *Health Hazard Evaluation and Test Support of the Special Study of Halon Fire Extinguishing Agents.* Washington, DC: DA; 1985. TECOM Project 1-VC-080-060-153.

Safety Commission records for 1975 (prior to any propellant restrictions) show that 5,700 aerosol-related injuries were treated in hospital emergency rooms. These injuries resulted from spraying (66%), inhalation and ingestion (12%), fragmenting of the container (10%), and cold (3%); the causes of 8% were unspecified (the published data were not rounded to 100%).[18]

### *Acute Exposures*

Fluorocarbons as a class have very low toxicity and the predominant hazard they pose is from simple asphyxiation. Early in the history of fluorocarbon use, deaths associated with exposure were usually attributed to asphyxia, but sufficient data were accumulated during the late 1960s and early 1970s to associate mortality with abuse of these products. In particular, deaths due to propellant "sniffing" warranted closer scrutiny. The possibility of fluorocarbon toxicity and abuse was raised in 1970 when the deaths of more than 100 youths who had died while sniffing various aerosol products were investigated.[19] Other research led to similar findings; these clearly documented that fluorocarbons sensitize the myocardium to sympathomimetic drugs, which can lead to severe cardiac arrhythmia and death on subsequent exposure. Further experimental and epidemiological data tend to corroborate the unusual finding that, unlike most other sensitization reactions, the myocardial sensitization is a transient occurrence that quickly and completely disappears if the affected individual is removed from contact with the chemical.[12]

Similar and equally serious consequences resulted from the use of pressurized bronchodilator aerosols. The original bronchodilators were mixtures of the sympathomimetic drugs epinephrine and isoproterenol, which were aerosolized by both trichlorofluoromethane (CFC-11) and dichlorodifluoromethane (CFC-12). During the few years that these broncho-dilators were used, physicians began to document a high incidence of bronchospasm and death. By 1968, sufficient evidence had accumulated to ban the over-the-counter sale of these devices.[20]

As a group, the fluorocarbons have a low lipid solubility and are poorly absorbed in the lung. Once in the bloodstream they are apparently not metabolized and are slowly excreted through the lung, unchanged, via expired air.

### *Chronic Exposure*

Because fluorocarbons are rapidly disseminated in the environment, chronic exposures are limited to relatively few occupations. Workers who manufacture and package these chemicals, electronics workers who use them as cleaning solvents on printed circuit boards, refrigeration mechanics, and hospital operating-room personnel appear to have the highest exposure potential. Federal workplace exposure standards have been promulgated for most of the commercially important fluorocarbons and range from 100 to 1,000 ppm for the entire series.[21]

Current epidemiological data indicate that no significant hazards are involved with chronic exposure to most fluorocarbons at low concentrations. Their rapid dissemination limits the possibility of high exposure concentrations in most applications. Some fluoro- and chlorofluorocarbons are used as anesthetic gases; this appears to be the most prevalent route of chronic exposure to chlorofluorocarbons (see also Chapter 5, Health Hazards to Healthcare Workers). The handling and processing of waste anesthetic gases (as well as their intended use) cause the potential for daily exposure to operating room personnel. Epidemiological studies of nurse anesthetists and other female hospital personnel have indicated a correlation between exposure to anesthetic gases and the occurrence of cancers and spontaneous abortions in the study population, and congenital anomalies in their infants. Based on these findings, the National Institute for Safety and Health (NIOSH) recommended severely lowering the occupational standard for anesthetic gases to 5 ppm.[22]

A researcher who performed an extensive mutagenicity study on a series of 21 different chlorofluorocarbons concluded that they are not biologically inert: the series contains bacterial mutagens, cell-transforming agents, and rodent carcinogens. For this series of compounds at least, prokaryotic mutation does not accurately predict carcinogenic potential. [23]

Long-term carcinogenicity bioassays have been completed in rats and mice for the three major chlorofluorocarbons: trichlorofluoromethane (CFC-11), dichlorodifluoromethane (CFC-12), and chlorodifluoromethane (CFC-22). These inhalation studies ran for 104 weeks in rats and 78 weeks in mice. All three of these chemicals failed to demonstrate any carcinogenicity.[24]

**Physical Effects**

Exposure to fluorocarbons such as Halon 1301 can cause both traumatic auditory damage and cold injury. Military vehicles are frequently equipped with automatic fire-suppression systems that contain fluorocarbons stored under very high pressures (750 psi). These systems rapidly trigger when they sense a fire and quickly flood the vehicle. The extremely high storage pressures and short response times required of the system cause noise levels to be exceptionally high: impulse noise levels greater than 160 dBP have been measured with these systems, a level sufficient to cause permanent auditory damage. Furthermore, transitory subzero temperatures can be produced near the discharge nozzles and cause medically significant cold injury to crew members who are close to the discharge. The Health Hazard Assessment of the M60A3 tank found that

> During gunnery training and M60A3 motion, crew members are likely to be at their normal stations and are required by U.S. Army hearing conservation policy…to wear [hearing protective devices]. At these positions, the impulse noise levels caused by [the automatic fire extinguishing system] activation range up to 161 dBP at over 200 millisecond B-duration. Such levels exceed the 140 dBP hazard criterion but are within the allowable exposure range for personnel wearing [hearing protective devices]. Activation of the [automatic fire extinguishing system] at such time would not expose protected personnel to hazardous impulse noise. If personnel are not wearing [hearing protective devices] and the [automatic fire extinguishing system] is activated, permanent hearing loss may occur with repeated exposure, but is less likely for a once in a lifetime exposure.
>
> . . . .
>
> The rapid discharge of the 7-pound Halon 1301 bottles has the potential for freezing tissues in the discharge stream.…[S]ubzero temperatures would be expected on skin surfaces for very short durations.…Cold injuries…have been documented with [the automatic fire extinguishing system] in…vehicles [other than the M60A3] during accidental Halon discharge….[17(pp4–6)]

## PAINTS

Paints are widely used in industry and the military for aesthetic purposes and also to produce a surface coating for protection against weathering.[2] Workers can be exposed during application of the paint, during drying and curing as a result of solvent evaporation, and during paint-grinding or -stripping operations. For this discussion, the term *paint* refers to a range of solvent-based products including conventional and epoxy paints, varnishes, enamels, and lacquers. Conventional paints consist of a pigment dispersed in a vehicle. Varnishes are usually a nonpigmented resin that is dissolved in a solvent. They dry by solvent evaporation and the oxidation and polymerization of the binder. Most enamel paints are like varnishes, but with a pigment added. Lacquers are clear or pigmented finishes, usually based on a cellulose ester in a solvent, and cure through solvent evaporation.[25] A number of water-based paints and enamels are also available, but because these coatings do not pose a significant inhalation hazard, they will not be discussed further in this chapter. Epoxy paints consist of epoxy resins in reactive diluents. These are mixed with curing agents to create tough, inert surface coatings. Generally the components are not volatile and are not an inhalation hazard.[26]

Solvent-based paints generally consist of three components: a vehicle, fillers, and additives (Table 13-6). The *vehicle*, which includes a binder dissolved in the solvent, makes up the liquid portion of the paint and allows it to be thinned to a consistency suitable for the chosen method of application. The *binder*, either a naturally occurring oil or resin or a synthetic material, cements the paint film to the substrate.[25] *Fillers* include pigments to color the coating and extenders to control gloss, texture, and viscosity. *Pigments* are usually finely powdered, insoluble solids that are dispersed in the liquid medium. In addition to providing color and opacity to the finish, some pigments also act to inhibit corrosion. *Additives* include agents that promote drying, inhibit mildew growth, and prevent the pigment from settling and the paint film from sagging. Although a variety of additives are

**TABLE 13-6**

**COMPOSITION OF PAINT**

| Vehicles | Function | Fillers | Function | Additives | Function |
|---|---|---|---|---|---|
| *Solvents* | Adjust viscosity | Epoxy | | Calcium carbonate | |
| (thinners) | | *Pigments* color, opacity | Provide | Silica | |
| Xylene | | Titanium dioxide | | Bentonite | |
| Toluene | | Zinc oxide | | *Driers* | Speed drying |
| Benzene | | Iron oxides | | *Biocides* | Prevent growth of molds and fungus |
| Naphthas | | Lead | | | |
| Perchlorethylene | | Chromium | | *Flattening Agents* | Reduce luster |
| Mineral spirits | | Cadmium | | | |
| Methyl ethyl ketone | | Aluminum | | | |
| Methyl isobutyl ketone | | Bronze | | | |
| Ethyl acetate | | Carbon black | | | |
| Trichlorethylene | | Lamp black | | | |
| Butyl alcohol | | *Extenders* | Build body | | |
| Ethyl alcohol | | Talc | | | |
| Cyclohexanol | | | | | |
| *Resins* | Form film | | | | |
| Alkyd | | | | | |
| Phenolic | | | | | |
| Acrylic | | | | | |
| Vinyl | | | | | |
| Amino | | | | | |
| Cellulose | | | | | |
| Polystyrene | | | | | |
| Polyurethane | | | | | |

Source: Burgess WA. *Recognition of Health Hazards in Industry: A Review of Materials and Processes.* New York: John Wiley & Sons; 1981.

used in formulating paints, they generally compose only a small percentage of the paint.

### Toxic Constituents

The toxic constituents of paints are contained in the solvents, pigments, extenders, and resins. Dermal and ocular exposures and inhalation can occur while paints are being mixed or applied; the route and degree of exposure depend to some extent on the method of application. During roller or brush painting, solvent vapors can be inhaled as these constituents volatilize from the freshly painted surface. Spray painting, however, poses a much greater respiratory hazard because both the liquid and solid constituents are aerosolized.

Chronic exposure to the mixed solvents that are found in paints can also cause a neurasthenia that is sometimes referred to as *painter's syndrome*. Toxic symptoms include headache, fatigue, difficulty in concentrating, deficits in short-term memory, irritability, depression, and alcohol intolerance. (These symptoms have also been reported by workers in the plastic boat industry and among jet-fuel handlers).[4] In general, as the severity of the solvent-related effects increase, reversibility becomes less likely.[27] The solvent-related neurasthenia was first described in the Scandinavian literature during the late 1970s, but is still not universally recognized. A recent study evaluated workers in two paint-manufacturing plants in the United States, but failed to document these specific symptoms of toxicity.[28] This study evaluated 187 workers using three standardized psychological/neurological assessment batteries. Exposure durations ranged from 6 to 36 years and concentrations were below the TLVs or PELs. No significant associations were found between solvent exposure and test scores.[29]

### Solvents

Paints can contain solvents from virtually every chemical class and toxicity level (described previously in this chapter). Because many are highly toxic, such as benzene, and potentially carcinogenic, such as the halogenated hydrocarbons, attempts have been made in recent years to reduce the use of these toxic solvents in paints by substituting less-toxic solvents.[30] However, the workplace hazard remains significant because workers continue to encounter these agents in older formulations, or as constituents of current paints.

### Inorganic Pigments

Pigments provide paint with opacity, color, durability, and film hardness. They typically constitute 20% to 60% of a paint by weight, and are composed of finely divided inorganic solids.[31]

***Titanium Dioxide.*** Titanium dioxide is a white pigment that has virtually replaced the older lead-based white pigments. (It is also used in food products and cosmetics as a whitening agent.) Titanium dioxide is generally thought to be physiologically inert.[3] Other white pigments include calcium carbonate, barium sulfate, and aluminum silicate.[31] Historically, these pigments have been considered physiologically inert, and they have a low order of toxicity. Recently, however, concerns have been raised over *pulmonary alveolar proteinosis*, a physical condition thought to result from exposure to particulates. The condition has been reported among workers exposed to several particulates including the inert dusts. Other studies suggest that these may, in fact, be biologically active, and NIOSH has reported data suggesting that titanium dioxide is a potential occupational carcinogen.[8]

The ACGIH TLV for these white pigments is 10 $mg/m^3$. OSHA PELs are 10 $mg/m^3$ for barium sulfate and titanium dioxide, and 15 $mg/m^3$ for calcium carbonate. OSHA also set a PEL of 5 $mg/m^3$ as a respirable dust for all three pigments.[7,8]

***Carbon Black and Lamp Black.*** Carbon black, produced during the incomplete combustion of petroleum gas, and lamp black, produced during the incomplete combustion of oil, are the most common black pigments used in paints. Incomplete combustion may produce a variety of polynuclear aromatic hydrocarbons (PAHs), which are likely to contaminate these black pigments. A number of the PAHs have been found to be both mutagenic, when tested in in vitro test systems, and carcinogenic in animal bioassays. No excess tumor incidence has been found in epidemiological studies of carbon black workers, however.[31] The ACGIH TLV for carbon black is 3.5 $mg/m^3$; OSHA has not set a PEL for this substance. No exposure limits have been set for lamp black.[7]

***Iron Oxides.*** Iron oxides, found in red and brown inorganic pigments, have low dermal and oral toxicity. Workers in the metal or pigment industries who inhale iron oxide dust or fumes may develop a mild form of pneumoconiosis. Because the causal agent is iron, this condition is more specifically termed a siderosis, and apparently does not become fibrotic.[7,8] The ACGIH TLV for iron oxide is 5 $mg/m^3$; the OSHA PEL is 10 $mg/m^3$.[7,8]

***Chromium.*** Chromates, used extensively in yellow and orange pigments, can act as sensitizers. Chromate dermatitis has been reported in workers in a number of industries. Exposures to chromium in the chrome-production and -pigment industries have been associated with an increased incidence of respiratory cancers.[32]

Chromium exists in oxidation states ranging from divalent to hexavalent; the trivalent state is the most common form found in nature. The carcinogenic activity associated with exposure to chromium has been attributed to the hexavalent form. Epidemiological studies suggest that the acid-soluble, water-insoluble hexavalent chromium is produced in refining operations. This form is also corrosive and reportedly can cause chronic ulceration and perforation of the nasal septum. In contrast, trivalent chromium is neither irritating nor corrosive.[8]

The ACGIH recommends a TLV of 0.5 $mg/m^3$ for chromium metal, di- and trivalent chromium and water-soluble hexavalent chromium compounds. Water-insoluble hexavalent chromium is a known human carcinogen and has a TLV of 0.05 $mg/m^3$. OSHA has a PEL of 0.1 $mg/m^3$ as a ceiling value for $CrO_3$.

### Organic Pigments

In addition to the inorganic pigments, there are hundreds of organic pigments used in the paint industry. However, data on the toxicity of most organic pigments are limited: in most cases, the only data consist of acute oral $LD_{50}$ values for rodents. The human toxicology of chronic exposures to most organic pigments remains largely unknown.[25]

### Extenders

A number of minerals such as silicas, silicate clays, mica, and talc are used as extenders, which act to build body in paint formulations. While the toxicological effects of pigments depend on the specific type of pigment, *all* extenders have the potential to produce

fibrosis of the lung. Limited epidemiological data suggest that a mixed-dust pneumoconiosis is prevalent among painters, which might be due, in part, to exposure to extender materials.[25]

### *Resins*

Resins are polymers. As a group they include the alkyd, acrylate and methacrylate, vinyl, cellulose, epoxy, and polyurethane resins. They have low volatility and are generally soluble in organic solvents and insoluble in water. In paints and varnishes, resins provide film hardness, gloss, surface adhesion, and resistance to weathering.[31]

***Alkyd Resins.*** Alkyd resins form as a condensation product of a polybasic acid and a polyhydric alcohol. Their toxicity is relatively low and they have a long history of use without indication of a chronic hazard.[31]

***Acrylate and Methacrylate Resins.*** Acrylate and methacrylate resins are polymers formed from acrylic and methacrylic acids. These substances are used extensively in latex paints. Unreacted monomers are primary irritants and skin sensitizers. Once they are polymerized, however, they are no longer health hazards. Reacted polymers have a low order of toxicity, and the Food and Drug Administration has approved polymethacrylate as an indirect food additive.[31]

***Vinyl Resins.*** The vinyl resins are polymers of a number of monomers including vinyl chloride, vinylidene chloride, vinyl acetate, and derivatives of styrene. The principal health hazard associated with the vinyl resins is the potential for exposure to unreacted vinyl chloride, which the EPA considers to be a known human carcinogen. Epidemiological studies have associated workers' exposures to the vinyl resins with increased incidence of liver angiosarcoma and, possibly, brain tumors.[33] The ACGIH recommends a TLV of 5 ppm for exposure to the vinyl resins.[7]

***Cellulose Resins.*** The cellulose resins are natural products and include nitrocellulose, cellulose acetate, and cellulose acetate butyrate. These compounds do not present any known toxic hazard.[25,31]

***Epoxy Resins.*** The epoxy resins used most frequently in paints and coatings are usually made by reacting epichlorhydrin and *bis*-phenol-A. Exposures to epoxy resins that have not cured completely have been associated with skin and eye irritation and allergic skin reactions. Liquid epoxy resins are used primarily in two-component epoxy paints. These liquid resins are modified when reactive diluents—which are also skin, eye, and respiratory irritants—are added. Aliphatic and aromatic polyamines and polyamides are also typically used as curing agents in two-component epoxy coatings. These substances are also potential sensitizers and irritants.[25,31]

Because they are not volatile compounds, there are no TLVs or PELs. Operations such as grinding and sanding can produce nuisance dusts, however, and the TLVs for the individual components are then relevant.

***Polyurethane Resins.*** Polyurethane resins are formed by polymerization of an isocyanate such as toluene diisocyanate (TDI). Uncured polyurethane resins contain small quantities of unreacted monomers and these monomers can cause health problems in exposed workers. Isocyanates can cause severe irritation to the conjunctiva, may cause respiratory distress, and are associated with sensitization-type reactions. Inhalation of isocyanate vapors or aerosols can produce asthmalike symptoms including constricted airways, difficulty in breathing, and a dry irritant cough. In sensitized individuals, even a very low exposure to an isocyanate can produce anaphylactic shock or other such dramatic response.[25] The ACGIH TLV and OSHA PEL are both .005 for the unreacted monomer.[7,8]

### *Additives*

Until recently, mercury biocides were added to latex paints as a preservative to prevent bacterial and fungal growth and to control mildew on exterior surfaces. These biocides were derived from mercurial compounds including phenylmercuric acetate (PMA), 3-(chloromethoxy)propylmercuric acetate (CMPA), and phenylmercuric oleate (PMO). At least one report has linked these mercury compounds to acrodynia, a rare form of mercury poisoning found in children. This disease is characterized by pink-colored fingers and toes, peeling of the soles and palms, pain in the extremities, impaired motor control, photophobia, and mental apathy. The EPA, working with paint manufacturers, decided to remove mercury from interior paints. Only PMA will remain registered for use in exterior paints and for miscellaneous interior uses (spackling and patching compounds).[34]

## Chemical Agent Resistant Coatings

Until the early- to mid-1980s, military vehicles were painted with standard alkyl and acrylic paints, but these absorbed chemical warfare (CW) agents. Up to 25% of liquid CW agent applied to surfaces painted with standard paints is absorbed within 30 minutes. The CW agent can then desorb slowly over several weeks, creating a residual toxic hazard. Standard alkyl paints are also soluble in decontamination solu-

tions. To avoid these hazards, the military converted to polyurethane paints (PUPs), which are chemical agent resistant coatings (CARCs), on all combat, combat-support, and tactical-wheeled vehicles, and aircraft and essential ground-support equipment.

There are two primary paints in the CARC system. The first, for exterior use, consists of aliphatic PUP applied over epoxy paint, which is used as a primer coat. The second, for interior use, consists of an epoxy-polyamide paint used over the epoxy primer.[35] Epoxies were initially considered for all CARC applications. However, because they break down with prolonged exposure to ultraviolet light, they can only be used indoors.

PUPs provide a measure of chemical resistance. However, the camouflage pigments and flattening agents required for military vehicles are more porous than are high-gloss paints. CW agents can enter the pores of PUPs, but are not adsorbed to CARC paints—unlike alkyd and acrylic paints—and they can be removed with standard decontamination procedures. CARC paints are also resistant to the components of the decontamination systems.

Two polyurethane topcoats—a two-component paint and a single-component paint—are available. The two-component system incorporates component A (containing the polyester resin, solvent, and pigment), which reacts with component B (an aliphatic polyisocyanate combined with volatile solvents) to form a tough, resilient coating. In the single-component system, the paint cures by reacting with moisture from the air.[35]

Both of the PUP CARC systems contain unreacted isocyanate groups in the uncured resin, which irritate the skin and sensitize the respiratory system. The newer PUP used currently in CARC paints includes hexamethylene diisocyanate. The monomeric hexamethylene diisocyanate is usually reacted to form a higher-molecular-weight prepolymer that is less volatile than the hexamethylene diisocyanate monomer, which tends to reduce the potential hazard from inhalation.[35]

**Fig. 13-3.** The operator in this walk-in spray booth is wearing coveralls, gloves, a head cover, and an air-purifying respirator. He has also positioned the items to be painted between himself and the booth's exhaust ventilation. This serves two purposes: it minimizes the potential for the painter's exposure and it prevents vapors and aerosols from leaving the spray booth.

### Exposure Controls

Industrial painting operations use several methods to control worker exposures and reduce potential health effects. Most flow- and spray-painting operations with solvent-based paints require exhaust ventilation to reduce the airborne paint mists and solvent vapors to acceptable levels. Ventilation controls include spray booths and rooms or tunnels, which are designed to contain the aerosol mists (Figure 13-3). These enclosures are equipped with a filtration system (or mist arrestor) to remove paint mists from the exhaust air. Spray-paint operators must wear effective PPE: goggles and a face mask with a charcoal filter. Effective spray-booth controls also require that the operator not position him- or herself between the object being painted and the point of exhaust. Personnel should also ensure that booths equipped with dry filters receive regular maintenance and cleaning to prevent the filter from clogging.[25]

The method of paint application influences worker exposures. Conventional air spraying is the most common method used in spray-finishing operations. Exposures to paint mists as a result of overspray and rebound are principal hazards of these operations. Airless spray techniques reduce overspray by approximately 50% when spraying flat surfaces. Another application method, electrostatic spraying, involves charging the paint mist so it is attracted to the item to be painted. This technique eliminates almost 90% of the overspray associated with conventional air atomization.[2,25]

Spray-finishing applications in which engineering controls are impractical, or operations in which highly toxic materials are present require that PPE, including respiratory protection, be used. Airline respirators may be required during painting operations in confined spaces such as storage tanks, boilers, ventilation ducts, or other areas where airflow is restricted. In other situations, conventional half-facepiece respirators with mist-removing prefilters and organic-vapor filters may provide adequate protection. Additional PPE should include cloth coveralls, eye protection, gloves, and head coverings. Workers should be prohibited from wearing contact lenses while painting.[35]

## MEDICAL SURVEILLANCE

Workers who could be exposed to the constituents of paints should be enrolled in a comprehensive medical surveillance program designed to prevent or control occupational diseases. Exposure to solvents can occur in a variety of occupations and often involves complex mixtures; therefore, the installation's medical authority should determine the specific details concerning the scope and frequency of medical surveillance examinations. The physician can find detailed recommendations on medical surveillance from the Medical Information Module of the Occupational Health Management Information System (OHMIS), which is discussed in Chapter 4, Industrial Hygiene, or from other medical guidance.

Medical evaluations should include a preemployment examination and a regular periodic examination, both of which should include detailed medical and occupational histories. In addition to the standard evaluation for smoking and alcohol use, the physician should pay particular attention to any history of previous exposures to toxic substances, especially organic solvents or other agents associated with neurotoxic effects (see Table 13-6). Due to the neurotoxic effects of solvents, the physician should also consider obtaining a neuropsychiatric evaluation.

Because painters can be exposed not only to solvents but also to the other constituents of paint, they require medical surveillance. The most serious occupational health concerns for painters are their potential for exposures to mixed solvents and isocyanates. During the preplacement or baseline evaluation, the physician should screen for previous exposures to isocyanates. The physician should note the patient's allergies, respiratory diseases, and smoking habits. Because sensitized individuals who are subsequently exposed to isocyanates may have serious allergic reactions, *hypersensitive individuals and workers with a history of chronic respiratory illness should not work with these substances.* The physical examination and clinical tests should also thoroughly evaluate the respiratory system and include, in addition to the routine chest X ray, pulmonary function tests with forced expiratory volume in 1 second ($FEV_1$), forced vital capacity (FVC), and $FEV_1$/FVC. A periodic examination should be performed at least annually after the preplacement examination.[35]

For a medical surveillance program to be effective, health education must accompany the evaluations. Health education includes training for both employees and employers, and should provide information on the potential hazards of the chemicals in use, the measures to control exposures, and the proper use of personal protective equipment. Training should also be updated and repeated when new chemicals are added to the workplace.[36,37]

## MEDICAL TREATMENT

There are no militarily unique medical treatments for exposure to solvents and paints or to chlorofluorocarbons. In most cases, simply removing the victim from the hazardous environment is the only treatment required. Contaminated clothing should be removed to prevent additional exposure. The physician should ensure that an adequate airway and respiration are maintained. Urinary output should be monitored and fluids administered either intravenously or orally. Diazepam can be used to treat convulsions.

### Solvents and Paints

Usually, the only treatment necessary for acute exposures to solvents is removal from the toxic atmosphere. However, acute exposures in high-enough concentrations can cause paralysis, convulsions, and unconsciousness that can progress to death. In instances of ingested solvents (including halogenated hydrocarbons, kerosene, and stoddard solvent), medical personnel must take care to prevent the victim from aspirating the toxic agent into the lungs. Even small quantities of a solvent like kerosene in the respiratory tract can cause pneumonitis, pulmonary edema, hemorrhage, and necrosis. If aspiration occurs, the emergency treatment is the same as for any oily liquid.

### Fluorocarbons

As a group, fluorocarbons and chlorofluorocarbons have very low acute toxicity; the greatest hazard of exposure to these compounds is simple asphyxiation. Exposure to very high concentrations (> 50,000 ppm) has been shown to produce CNS depression and eventual respiratory failure. Simply removing the victim from the hazardous environment is usually the only treatment required; all toxic signs rapidly disappear when the patient is removed to fresh air. The most severe threat from fluorocarbon exposure is the ability of these chemicals to sensitize the lungs and myocardium. In some individuals this can cause bronchospasm and cardiac arrhythmias. *Should overexposures to halogenated hydrocarbons—including the chlorofluorocarbons—occur, epinephrine or other sympathomimetic amines and adrenergic activators are contraindicated.* These solvents are cardiac sensitizers and will further sensitize the heart to the development of arrhythmias. They must not be administered.

#### *Toxic Combustion and Decomposition Products*

Exposure to the combustion and decomposition products of halons when they are used as fire extinguishers is probably the most significant medical problem posed by chlorofluorocarbons. These products are extreme irritants; consequently, exposed individuals will attempt to escape from the contaminated environment. Personnel inside vehicles or confined spaces, where escape is difficult, may be exposed to long-term, high concentrations of these severe irritants and can suffer chemical burns of the eyes, skin, mucous membranes, and lungs. Medical treatment should be the same as that for any other severe irritant. Pulmonary edema will undoubtedly be a complication if exposure to high concentrations has occurred.

#### *Physical Trauma*

Halons used in fire-extinguishing or other pressurized, closed systems can cause hearing damage from excessive noise. Hearing protection should be recommended for areas where the probability of sudden release of these chemicals is high. The subzero temperatures that occur when pressurized fluorocarbons such as the halons are suddenly released can also freeze unprotected skin, eyes, and mucous membranes. Treatment for freezing injury requires no special procedures; the fluorocarbons will rapidly volatilize and the resultant injuries can be treated like any routine cold injury.

## SUMMARY

Although large quantities of solvents, fluorocarbons, and paints are used annually in DoD industrial operations, exposure potentials in the military are similar to those in comparable civilian occupations. Despite the increased use of engineering controls and PPE, severe injury or debilitation can result from overexposure to these compounds.

Solvents are used extensively in the military to degrease metal parts, in paints and paint removers, and as intermediates in the manufacture of other items. Most of the organic solvents discussed in this chapter have similar acute, toxic, anesthetic-like ef-

fects to the CNS. Chronic exposures also may affect the CNS; other target organs include the liver, kidneys, and blood. Some solvents, including benzene and the chlorinated hydrocarbons, are animal carcinogens, and some are human carcinogens. Target organs for carcinogenicity are blood, liver, and skin.

Fluorocarbons as a class are relatively nontoxic after acute exposure. Chronic exposure also does not appear to pose a high level of risk. They are potentially toxic to both the cardiovascular and bronchopulmonary systems; this fact is of major significance when medical personnel are required to treat exposed individuals. The greatest medical hazard posed by fluorocarbons is the extreme toxicity of their highly irritant combustion and decomposition products. Current use in the military is significant and will probably rise. As replacement chemicals for banned fluorocarbons are selected, the toxicological and medical databases for them will be fragmentary at best.

Painters can be exposed to a variety of toxic components. The most prevalent exposures are to the solvents. Other constituents of paint can also have toxic effects. However, most constituents of paint are relatively safe in the concentrations used in paint formulations. Allergic sensitization reactions can still occur in sensitized individuals who are exposed to the isocyanates used PUP and CARC paints.

Military occupational health professionals should continue to work with industry to minimize the potential risks through engineering controls and, where possible, through substitution with less-toxic materials.

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