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Polyvinyl Chloride Toxicity in Fires

Hydrogen Chloride Toxicity in Fire Fighters

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• **Polyvinyl chloride, of all the plastic polymers, has been implicated primarily in causing the most serious problem in fire fighting today because it releases hydrogen chloride gas when burning. One hundred seventy fire fighters who experienced symptoms from its toxicity have been studied from 1970 to 1975. One died.**

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A RECENTLY identified hazard that is of paramount importance to practicing physicians is that of chemical injury incurred by persons exposed to fumes from fires. Specifically, such injuries are caused by the inhalation of vapors created by the thermal degradation of polyvinyl chloride, a plastic polymer. Such substances have now become widely used in the construction of homes, furnishings, office equipment, electric wire, telephone and cable covering, and vehicles. A particular hazard exists in the case of the fire fighter, although it could be equally serious in any circumstance in which an individual might be exposed to such plastic combustion products.

See also p 390.

THE FIRE

At 10:30 AM on Jan 6, 1970, an alarm sounded for a fire reported to be on the sixth floor of Building 213 at the Washington, DC, Navy Yard. Units of the District of Columbia Fire Department arriving on the scene found the floor filled with a heavy concentration of toxic fumes. Although all of the fire fighters carried masks, none were used, since neither the fire nor the smoke was so excessive as to prevent entry without masks. The fire, which was confined to an office copying machine constructed of plastic and Teflon parts, was quickly extinguished.

The fire companies entered the building via the elevator, placed several portable fans for ventilation, and left the building about 20 minutes later. On returning to their quarters, many of the fire fighters experienced unusual and unexplainable symptoms, including a constricting tightness localized to the anterior part of the chest. This chest discomfort was described as a searing, burning sensation accompanied by dyspnea as well as a "burning sensation appearing to close off the throat." These symptoms were in addition to the usual headache, dizziness or vertigo, and nausea, which, in our experience, is frequently seen in cases of nonchemical smoke inhalation. Some symptoms persisted over a course of several days following exposure to the fumes. The dyspnea was noted on mild exertion and was associated with moderate apprehension by all of the affected fire fighters. A number of the fire fighters exposed at this fire experienced severe conjunctivitis, lacrimation, and dyspnea persisting for 24 hours after the fire. An intense headache, localized anteriorly and persisting about 24 hours, was also common.

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REPORT OF A CASE

Approximately 24 hours after the fire, a 33-year-old fire fighter fainted in the sitting room of the truck company. It was also reported that he had a muscle spasm similar to an epileptic seizure, but recovered, was fully conscious, and got up immediately. His superior officer asked how he felt, and he replied that he was "fine" and walked into the kitchen. Following lunch, the acting sergeant on duty looked for the man to check his condition for driving and found him lying on the floor next to his bed. He was cyanotic and, despite all resuscitative efforts by an ambulance crew and the rescue squad, was pronounced dead on arrival by a fire surgeon at the George Washington University Hospital at 1:10 PM. Postmortem examination by the District of Columbia Department of Public Health Medical Examiner showed severe pulmonary hemorrhage and edema due to chemical pneumonitis secondary to exposure to chemical smoke and fire. The pathologist also reported coronary atherosclerosis.

EFFECT OF FUMES

It has been suspected for several years that the smoke and fumes from electrical fires produced greater injury now than similar fires in the past.[1,2] It was strongly suspected that hydrogen chloride released by thermal degradation of PVC, a common electrical wiring insulation material, was the lethal product causing death in this instance. The medical examiner's report and the microscopic findings subsequently confirmed our original clinical impression of the cause of death.

Hydrogen chloride, inhaled either in the gaseous state or combined with water vapor, acts as an irritant to the mucous membranes of the eyes and

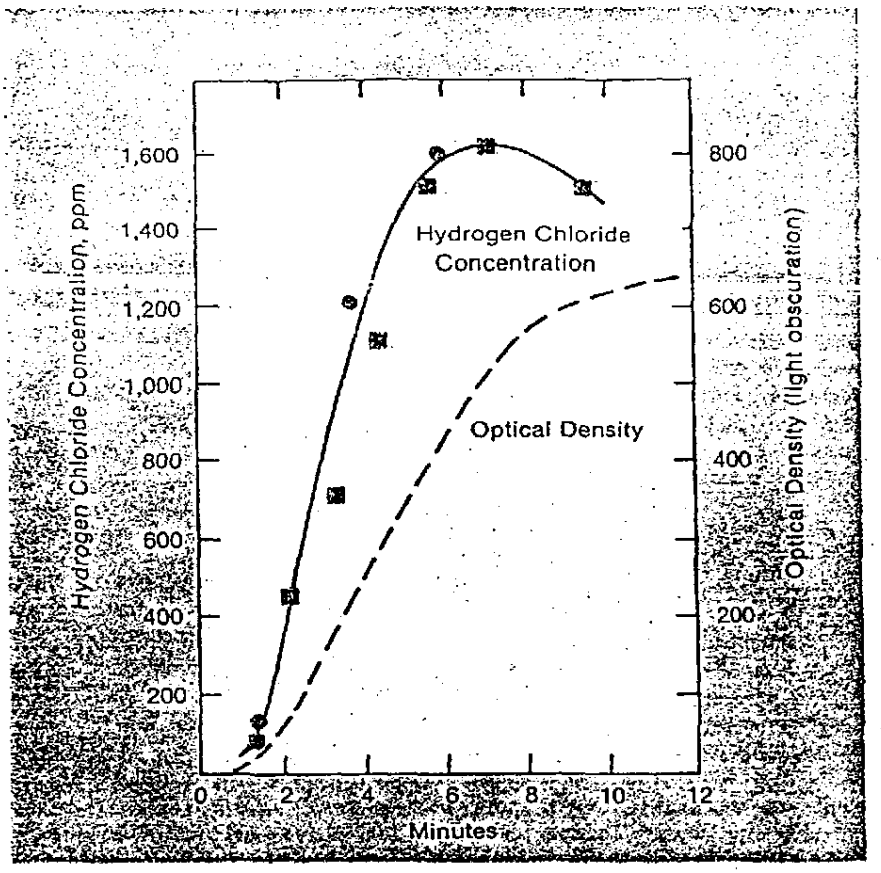

Fig 1.—Hydrogen chloride concentration and optical density of polyvinyl chloride electrical insulation decomposition products.

the respiratory tract. An HCl concentration of 15 ppm causes localized irritation to the throat after short exposure. Hydrogen chloride concentrations of 50 to 100 ppm are tolerable for one hour by humans. More severe exposures result in pulmonary edema and often in laryngeal spasm. Concentrations of 1,000 to 2,000 ppm of HCl are dangerous even with brief exposure[3] (Fig 1).

Quantitative release of HCl on thermal degradation of PVC has been well documented in the scientific and medical literature. Polyvinyl chloride, of all the synthetic plastic polymers, has been implicated as causing one of the most insidious, serious problems in fire fighting today, due to its release of HCl gas while burning. Mists of hydrochloric acid are considered less harmful than anhydrous HCl because the droplets have no hydrating action.

Animal Experiments

The toxicity of PVC has been substantiated by Kishitani[4] in his comprehensive animal experiments performed at the University of Tokyo. With respect to the toxicity of carbon monoxide, Kishitani states that the injurious nature of PVC is unique. All mice exposed to harmful gas products died, but the carboxyhemoglobin concentration in the dead animals was low (average, 21.2%). This clearly indicated that the fatalities were not due directly to CO poisoning. At autopsy, the eyes of the mice were closed, their mucous membranes were injured by chemicals, and the sclerae were discolored. The latter had become white due to the action of chlorine gas. All of the mice died in a period of 15 minutes after exposure to PVC fumes. Electrocardiographic abnormalities appeared prior to generation of large quantities of smoke. This indicated that a harmful gas produced at an early stage was causing the effect. Similar results were found on exposure to polyurethane gas. Kishitani concluded that the injurious properties of burning materials such as wood, fire retardant plywood, melamine finishing board, and acrylic resin are due to CO.

In the fatalities due to CO, the average concentration of CO was 45%. The burning of cellulose material resulted in nothing but the release of CO. In the mouse study with exposure to burning building materials, the ECG abnormalities were recorded before the smoke from the burning materials was evident.

In regard to the harmful properties of PVC to mice, as well as those from polyurethane, polysterene, and phenol, the pathologic effects of the gases, rather than those of the smoke, appeared first, as shown by atypical ECG changes. It requires less CO in combination with HCl to cause death. In conclusion, HCl and CO have a reciprocal, potentiating adverse effect.

EFFECT OF SMOKE

Smoke is a suspension of small particles in hot air and gases. It has a particulate fraction and a gaseous fraction. The particles consist of carbon and are coated with combustible products such as organic acids and aldehydes. The gaseous fraction has an extremely variable composition. Carbon monoxide and carbon dioxide are always present and constitute the bulk of this fraction.

However, a wide variety of other toxic gases are present at the scene of a fire. These may be formed by the combustion process or leak from commercial processing or storage equipment. The particulate and gaseous fractions combine to exert a large space-occupying effect and can fill an enclosed space at the expense of air. The gaseous fraction of smoke is much more dangerous to life than the particulate fraction. The nose, mouth, and throat, which filter out and trap the particulate fraction, cannot filter out the toxic gases. These gases enter the upper respiratory tract and lungs freely when inhaled. Damage to the lungs may result from exposure to a group of gases and vapors called pulmonary irritants (chlorine, phosgene, nitrogen dioxide, sulfur dioxide, and ammonia). On entering the lungs, these agents react chemically with water to produce strong acids or alkalis. A violent inflammatory response occurs, causing destruction of lung tissue.

Fire fighters and victims of fires in homes, offices, and industrial complexes are exposed to smoldering and

burning plastics and synthetics from furniture, floor and wallcoverings, textiles, and building materials. Many plastics produce large volumes of pulmonary-irritant gases when burned. Polyvinyl chloride is used extensively in furniture, both in the foamed plastic stuffing as well as the covering. Burning 450 gm of this material produces 320 gm of chlorine, phosgene, and HCl, all of which are strong pulmonary irritants. In all, there are 75 known products of PVC degradation. Practically every structure today contains plastics capable of producing lung-damaging gases when burned. Therefore, the fire fighter faces a great, new risk in almost any fire.

It became obvious that a particular pattern was emerging in which certain fire situations were productive of insidious and unpredictable fire casualties. The lethal chemical smoke was determined to be HCl resulting from the decomposition of PVC plastic used in telephone cable and electrical equipment installations.

We have found electrical and telephone cable fires to be especially hazardous to the fire fighters, due to the toxic gases produced. We have found that when a short circuit developed in a main switch, usually located in a sub-basement or penthouse of an office building or apartment house, the area was always poorly ventilated, and the plastic-covered switches quickly heat to temperatures in excess of 93.3 C. The heated conductors cause the plastic insulation to decompose and emit white, mist-like HCl fumes that resemble steam. When the temperature range of 232 to 321 C is attained, the gas may be invisible. The fire fighter may be fatally deceived if he does not know plastic-covered cable is involved in the fire he is fighting.

OVERHAUL PHASE

The period of time just after the fire is extinguished is termed the "overhaul phase." This is when the fire fighters clean up the fireground. Toxic smoke and fumes are still being emitted. This is a serious time for toxic gas inhalation to occur since many fire fighters remove their self-contained breathing apparatus during this phase. Another special hazard during this phase is the "heat sink," ie, concrete that retains a great amount of heat and releases fumes throughout the overhaul phase. Our testing has shown highly toxic concentrations of HCl in this concrete for as long as one hour after the fire has been extinguished.

FUEL LOADING

In the late 1960s, the type of "fuel loading" in office and high-rise buildings (ie, contents of the buildings) changed significantly and initiated a new awareness. The noncombustible buildings were not burning, but furniture within them, building insulation, ceiling tile, wall paneling, and decorations made partly or wholly of plastic were fueling disasters.

While a building may be constructed of fire-resistant elements, there is no control over the combustible material that an individual may elect to bring into the structure. This is the "fuel load" and may be expressed as kilograms of combustible material per square meter of floor area. Large differences exist in combustibility, smoke, and toxic gas release among different combustible materials. More than 50 sq m of red oak, all burning at its maximum rate, would be required to produce the same rate of smoke as 2.5 meters of acrylonitrile-butadiene-styrene plastic pipe. A fire of plastic materials develops extremely high temperatures because plastics possess a heat of combustion 2½ times that of other combustibles. Therefore, the fire would magnify the normal problems of radiant heat, structural damage, and exposure. The fire would exhibit a very high burning rate because the flammability of plastics is much higher than that of other combustibles.

The four most common plastics produced in the United States in the past few years are polystyrene, polyethylene, polypropylene, and PVC. A chlorethylene polymer is a plastic solid marketed under a variety of trade names, and currently being manufactured at a rate over 8.3 billion kilograms annually.

Polyvinyl chloride (Fig 2) is a plastic solid widely used as a rubber substitute and as electric and telephone wire and cable covering. It is also utilized frequently in electrical fixtures, plastic drains, as well as waste pipe

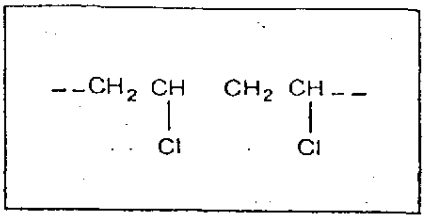

Fig 2.—Polyvinyl chloride.

for interior plumbing, pliable thin sheeting, film finishes for textiles, "noninflammable" upholstery, raincoats, tubing, belting, gaskets, shoe soles, disposable unbreakable plastic bottles, packaging materials, automobile and aircraft seatcovers, corrugated fence toppings, tarpaulins, shower curtains, phonograph records, plastic trays, baby pants, toys, curtains, and office copying-machines. It is used as an exterior finish in many vehicles, including buses and subway cars, because of its high resistance to wear and soiling.

When PVC is heated to 225 C, nothing happens. When heated above that, up to 475 C, PVC does not burn. The combustion range or flame point is 475 C. Between 225 C and 475 C, PVC loses over 60% of its weight. During this process, it thermally degrades and HCl is released. Four hundred fifty-three grams of PVC at degradation releases 320 gm of chlorine, phosgene, and HCl.

The individual toxicity factors of phosgene and the other major decomposition products of PVC, ie, benzene, toluene, total xylenes, naphthalene, and vinyl chloride, are very small, compared with the toxicity factor of HCl. Further, the total or additive toxicity factors of these minor components are also very small in comparison with HCl toxicity. Unfortunately, very little is known about the synergistic effects of toxic gases (ie, the ability of one chemical to increase or decrease the toxicity of another). The main toxic risks from the decomposition products of PVC are HCl and CO.[5]

Stone et al[6] elucidated the nature of smoke aerosol and its role in the transport of toxic gases in fire situations. They confirmed the fact that the toxic gas, HCl, is adsorbed on soot aerosol. Possible inhalation hazards of such soot are positively related to the size of the soot particles and particle agglomerates. It has been shown that

the amount and location of particle deposition in the respiratory tract depends on the particle size.[6] On inhalation, soot aerosols would enter the lower lung region and, to an extent, be retained there. Soot particle clusters, with sizes ranging from 0.1μ to 2.5μ in diameter, would be retained in the alveolar sacs to an extent of from 20% to 40% of those particles inhaled.[6] At a breathing rate of 18 liters/min for an exposure of one hour, a maximum of 0.7 gm of soot bearing 13 mg of loosely bound HCl would be retained in the lower lungs.

The effect of HCl in the gas phase is largely limited to irritability, mainly affecting the upper respiratory tract, whereas loosely bound HCl condensed on soot aerosol gains access to the lower lungs. Experiments have shown that water droplets of respirable size rapidly adsorb HCl from the gas phase and approach equilibrium concentrations in fractions of a second.[7]

SMOKE INHALATION

Respiratory distress may develop in fire fighters or fire victims immediately or within one to two days of exposure to combustion products. The best protection against these agents is the mandatory use of the self-contained breathing apparatus by every fire fighter. When anyone without a breathing apparatus is exposed to more than a few breaths of "choking smoke" characteristic of burning or smoldering plastics, he should receive prompt medical attention.

Prior to our investigation, we considered most fireground pulmonary casualties to be due to "smoke inhalation" or CO poisoning. Carbon monoxide had long been considered the leading cause of death and pulmonary morbidity in fires. Though CO is not noxious and does not actually cause serious pulmonary injury, the amount of CO in a patient is a good indicator of whether more harmful gases are present.

Before the introduction of massive fire loads of combustible and thermally decomposable plastic products, fire surgeons usually considered the CO victim to be "home safe" in four hours after exposure due to the fast half-life of CO in the blood. After exposure to plastic degradation products, a period of one to six hours elapses between exposure and onset of severe respiratory and chest symptoms. Treatment is most effective if given during this quiescent phase. Once the symptoms of difficult breathing, cyanosis, or other symptoms, as noted in the reported case, have appeared, it may be too late to prevent severe morbidity or even mortality. The "overhaul phase" after the fire is extinguished is dangerous with respect to fumes also.

A Harvard research team headed by Donald P. Dressler, MD, reported in 1973 that when burned, plastics and modern fibers produced colorless, odorless fumes that quickly displace oxygen and cause a dramatic rise in the CO_2 level of the atmosphere.[8] Therefore, a person either dies or is rendered unconscious in the early stages of a fire involving these materials. If he is unconscious, he soon dies of other lethal fume inhalations.[8] Tests performed in Cambridge, Mass, showed that fire fighters were probably affected adversely by the high level of CO_2 rather than by the initial release of toxic gases.[8]

In establishing the importance of detecting the presence of certain toxic gases, the difficulty in detecting CO has been emphasized.[9] Some human response mechanisms are more sensitive than others to CO inhalation.[9] Fairly high levels of CO affect a human's vision, but even higher levels do not affect his sense of timing. A research team headed by R. A. McFarland, MD, of Harvard University, found that when CO replaces 11% to 17% of the blood's normal oxygen, the peripheral vision is affected and the subject has difficulty viewing stimuli at a 20° angle from the line of sight.[9] The Harvard team also found that at a 17% concentration of COHb, some subjects had a momentary lapse of attention and failed to respond to all the stimuli presented. This is important in disproving the once-held concept that once an individual has detected the sharp odor of HCl he can walk away. It has been our observation that the fire fighter cannot escape the HCl fumes.

Cornish and Abar reported that CO and hydrochloric acid were the major toxic products from the combustion of PVC.[10] They pyrolized PVC polymers in a stream of air by gradually increasing the temperature from ambient to 600 C. Exposure to this air stream supplemented with oxygen produced pulmonary and interstitial edema in rats. Some of these animals also showed focal bronchial and intra-alveolar hemorrhage.

In a sealed environment (such as a centrally air-conditioned house, office, high-rise unit, or hospital), fumes from plastic degradation could kill, even if the fire is brought under control. It has been observed that a sleeping person needs an estimated 12 to 15 minutes to awaken, react to danger, and take appropriate action to save himself. With inhalation of fumes from burning plastic, he will not have that much time.

COHb LEVEL AND CIRCULATION

As noted by E.P. Radford (written communication), 43 deaths due to fire demonstrated that a blood COHb level of over 65% is nearly always lethal due to the depression of respiration and circulation. Ventricular fibrillation from acute myocardial hypoxia induced by a rapid rise of the COHb level in individuals with a restricted coronary circulation was found to be the likely explanation of a lower terminal blood COHb level. These individuals were found to have severe, preexisting coronary vascular disease.

We believe that any compromise of the coronary circulation, eg, atherosclerosis such as found in the fatal case, would be potentiated by highly irritating chemical fumes. We also believe that other pyrolysis products, especially HCl, have contributed to ventricular fibrillation since its concentration is comparable to that of CO in real fire situations involving plastics.

Kishitani has demonstrated that chlorine affects the myocardium more rapidly and more severely than CO. Carbon monoxide may cause changes in enzyme activities suggestive of myocardial damage. Hydrogen chloride has been found to be a more potent myocardial irritant whose effects may be difficult to determine by clinical observation. This is in conjunction with the development of pulmonary edema over a 24-hour period following exposure to HCl. This further complicates and compromises coronary artery oxygen saturation. This could lead to sudden death syn-

drome, similar to that in the reported case. Although in the cases of CO poisoning noted by G. Gordon, (written communication) the ECG changes did appear late if at all and the COHb varied in individuals at the same fire as well as in different fire situations, it has been our experience that all fire fighters exposed to PVC do manifest ECG signs of its toxicity. There are variable levels of its toxic effect on the lungs, mucous membranes (tracheal peeling effect), eyes, and other parts of the body.

CLINICAL STUDY

In Washington, DC, a clinical study of fire fighters suspected and later known to have been exposed to toxic products from thermal degradation of PVC was conducted by the DC Board of Police and Fire Surgeons between January 1970 and September 1975. One hundred seventy individuals were exposed from one to four different times to these products and reported two or all of the symptoms of acute toxicity, ie, pain in the anterior aspect of the chest, neck and throat pain, dyspnea, severe headache, dizziness, and irregular pulse within a few hours after exposure.

Initial physical examinations were performed by the fire surgeon on the fireground, in the hospital emergency room, or at the Police and Firemens Clinic. Laboratory studies including complete blood cell count, determinations of serum glutamic oxaloacetic transaminase, blood urea nitrogen, or creatinine, and a complete urinalysis were done. Electrocardiograms were performed initially when there was chest pain, extrasystoles, or cardiac arrhythmia. However, ECG studies were not incorporated as part of the physical evaluation until the latter part of the study when their importance was appreciated.

About one fifth of the fire fighters had extrasystoles during the first few hours after exposure. Fourteen of the last 26 firemen exposed had premature ventricular contractions. Some have had recurrence of extrasystoles within the subsequent year. Chest x-ray films were done at the initial examination and have remained normal. Serum electrolyte and arterial blood gas determinations were performed on 12 of the fire fighters who were more acutely ill, or required hospitalization. Because of the excellent clinic facilities provided by the District of Columbia government, all fire fighters were observed in a holding area after exposure and examined at the clinic or the hospital emergency room after transportation from the fireground. Twelve men were admitted to the hospital for treatment. If symptoms did not worsen over the first 24 hours in the hospital, they were released and followed up on an outpatient basis at the clinic.

Specific treatment, including oxygen given intranasally at 5 liters/min, bronchodilators, antihistamines, and decongestants in oral suspension, has been utilized in controlling most of the acute respiratory symptoms. Steroids were given intravenously to three of the fire fighters during hospitalization due to the severity of their signs and symptoms of respiratory distress.

Initial and follow-up liver function tests have remained normal in all of these patients. There has been no direct correlation between severity of symptoms and history of cigarette smoking.

None of the fire fighters has had to retire for permanent airway disorders. Serial studies and follow-up examinations will continue. Since the onset of this study among professional fire fighters, the use of the self-contained breathing apparatus has been required at all fires where toxic fumes could be present. This is necessary during the "overhaul phase" also. We urge all physicians to advocate restriction of unsupervised use of PVC products. Fire research activity should be maintained at a national level to continue study as well as preventive measures for HCl toxicity in PVC fires.

CONCLUSIONS

Hazards of Plastic Fires.—Respiratory distress may develop in fire fighters and fire victims after exposure to combustion products. The complexity of the problem is emphasized when it is recognized that the thermal degradation of PVC results in the formation of at least 75 identifiable potentially toxic compounds. Three characteristics of plastic fires are the extremely high temperatures, very high burning rates, and thick toxic smoke. The main danger to exposed fire fighting personnel at the fireground results from the massive formation of HCl gas, and in many instances, its adsorption on spherical soot particles that are carried into the victims' respiratory tracts.

Prevention.—The mandatory use of the self-contained breathing apparatus by all who will be vulnerable to toxic smoke will aid in the prevention of plastic fires. Control of hazardous building designs, including equipment and ventilation is also necessary. Protection by masks during the "overhaul phase" should be required. Sophisticated air-sampling techniques are required for definitive study, including gas chromatography. Electrocardiographic studies utilizing base line tracings directly after HCl exposure are stressed.

Areas of Future Study.—All atmospheres should be suspected by the fire fighter. There should be a search for an effective gas analyzer that can immediately assess gas concentrations released by burning or smoldering plastics. Further definitive cardiologic studies are indicated. The term "smoke inhalation" should be abandoned and replaced by "inhalation of toxic combustion products."

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