CASE REPORT

Michael B. McGee,[1] M.D.; Robert F. Meyer,[2] Ph.D.; and Subhash G. Jejurikar,[3] Ph.D.

A Death Resulting from Trichlorotrifluoroethane Poisoning

REFERENCE: McGee, M. B., Meyer, R. F., and Jejurikar, S. G., "**A Death Resulting from Trichlorotrifluoroethane Poisoning,**" *Journal of Forensic Sciences*, JFSCA, Vol. 35, No. 6, Nov. 1990, pp. 1453–1460.

ABSTRACT: Fatalities due to accidental exposure to chlorinated hydrocarbon in an industrial setting have been infrequently reported. The deaths in these cases have occurred within poorly ventilated, enclosed compartments or areas. A case is presented of a 16-year-old male who died as a result of exposure to trichlorotrifluoroethane while working in an open pit. Chromatographic results and tissue concentrations are presented.

KEYWORDS: toxicology, trichlorotrifluoroethane, death

Trichlorotrifluoroethane (fluorocarbon 113, Freon 113) is a fluorocarbon that was first introduced as a refrigerant and is used today primarily in an industrial setting as a refrigerant, degreaser, and dry-cleaning solvent. Reports of accidental deaths due to exposure to this compound are rare. A case is presented of a 16-year-old male who expired as a result of such exposure during the course of his employement.

Case Report

A 16-year-old male was assigned the duty of cleaning out a floor collection pit within a glass lens manufacturing facility. The collection pit consisted of a subfloor pit with an open top, constructed of cement and measuring 0.91 by 1.22 m (3 by 4 ft) with a depth of 1.42 m (4.8 ft) (Fig. 1). The waste fluid and sludge produced during the manufacturing process sometimes included methylene chloride, acetone, isopropanol, ethylene glycol, cerium oxide, chromic acid, lead, glass, and a fluorocarbon-containing compound. Waste material was conducted to this pit by a series of covered troughs and drainpipes located throughout the plant. The fluorocarbon-containing compound, Genesol DS (1,1,2-trichlorotrifluoroethane, Allied Chemical Co., Morristown, New Jersey), is used in the

Received for publication 18 Sept. 1989; revised manuscript received 24 Nov. 1989; accepted for publication 14 Dec. 1989.
[1]Chief medical examiner, Ramsey County Medical Examiner's Office, St. Paul, MN.
[2]Forensic toxicologist, Forensic Science Laboratory, State of Minnesota, St. Paul, MN.
[3]Chief of forensic toxicology, Forensic Science Laboratory, State of Minnesota, St. Paul, MN.

Copyright © 1990 by ASTM International

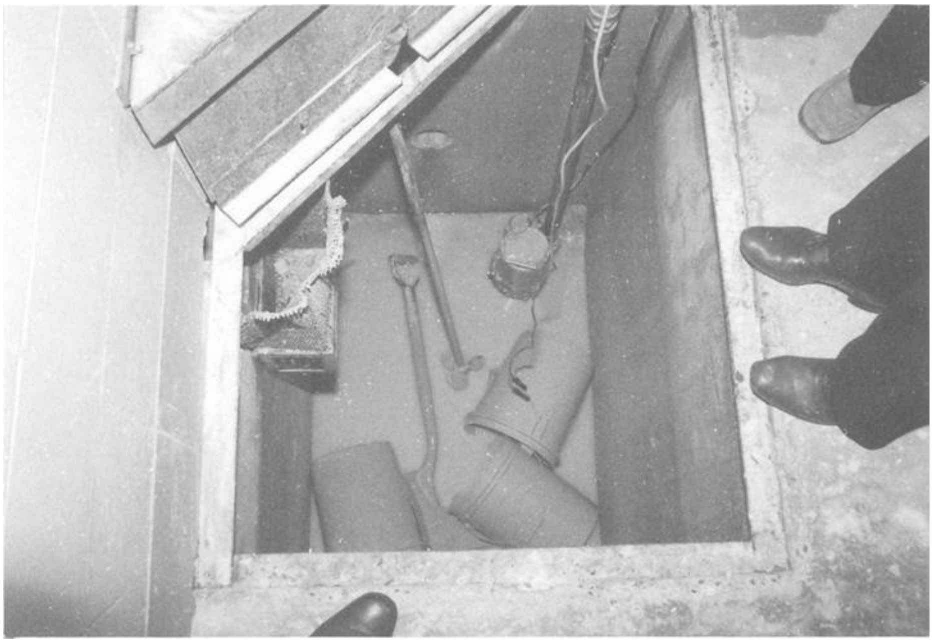

FIG. 1—*Floor collection pit within the manufacturing facility. The shovel and buckets at the bottom were used for cleaning when the subject collapsed.*

final cleaning step of the lens manufacturing process. Investigators believe that the fluorocarbon may have been accidentally spilled during routine filling of machinery and may thereby have entered the drain system, which carried it to the floor collection pit. The normal procedure for cleaning the pit included pumping off of any standing liquid before anyone enters the pit and then manual removing of any sludge deposited at the base of the pit. After the subject entered the pit to complete this cleaning, he suddenly collapsed. A fellow worker, who retrieved him from the pit, noted a sweet gas-like smell coming from within the pit moments before he began to feel dizzy and light-headed. Upon arrival at a local hospital emergency room, the 16-year-old was unconscious and in ventricular tachycardia/fibrillation, which would not respond to cardioversion or medical intervention. He expired 1 h after collapsing. The fellow worker was examined and released.

At the time of the postmortem examination, the body was noted as that of a normally developed 16-year-old Caucasian male. The traumatic injuries consisted of small isolated abrasions on the dorsum of the left hand and the right lower extremity, while the anterior facies were congested and cyanotic. The internal examination findings were unremarkable with the exception of the lungs, which were congested and edematous. The microscopic tissue results were unremarkable with the exception of the lung tissue, which exhibited a chronic peribronchitis.

Specimens collected at the time of the examination included heart blood, vitreous fluid, and urine from the urinary bladder, as well as gastric contents, and bile. Tissue samples were collected from the brain, kidney, lung, liver, adipose tissue, and skeletal muscle. Whole blood specimens were placed in sterile 10-cm glass test tubes with rubber stoppers and containing potassium oxalate and sodium fluoride additives. Samples of vitreous fluid and centrifuged serum were retained in sterile 10-cm glass test tubes without additives. These specimens were stored at 3°C (37°F). Bile, urine, and gastric contents specimens were placed in sterile plastic containers with screw tops, while tissue samples from the brain, kidney, lung, liver, adipose tissue, and skeletal muscle were separately sealed in polyethylene heat-sealable pouches. These specimens were immediately frozen and stored at −20°C (−4°F).

Analysis of these specimens in the medical examiner's toxicology laboratory failed to reveal the presence of ethyl alcohol, carbon monoxide, sedative/hypnotics, antidepressants, cocaine, narcotics, phencyclidine, or marijuana metabolites. Diphenylhydantoin was detected within the subject's serum and was within normal therapeutic levels. No odor was noted on examination of the tissue samples. Toxicologic specimens obtained at the autopsy were submitted to the Minnesota Forensic Science Laboratory, along with samples of fluid and sludge obtained from the floor collection pit during the scene investigation. A sample of Genesol DS was also submitted.

Toxicology

Toxicological analyses of the tissue specimens and the sludge pit samples revealed the presence of trichlorotrifluoroethane.

Trichlorotrifluoroethane or fluorocarbon 113 (FC 113) was analyzed by a modified method described by Christopoulos and Kirch [*1*]. Measured volumes of blood or urine and measured weights of finely diced samples of a number of different frozen tissues were extracted with 3.0 mL of benzene. One-microlitre aliquots of the benzene extract were injected into the gas chromatograph. Samples from the sludge pit in which the victim had been working just prior to death were also analyzed.

Reference standards were made using whole blood spiked with FC 113 to give concentrations ranging from 0.28 to 1.58 μg/mL.

FC 113 was analyzed with a Hewlett-Packard 5890 gas chromatograph fitted with an electron capture detector. The 15-m DB wax capillary column was 0.53 mm in inside diameter (ID) and 1.0 μm in film thickness (J & W Scientific Co.). The helium carrier flow was 1.5 mL/min. The oven temperature was 50°C.

The presence of FC 113 was confirmed using a Hewlett-Packard 5988A gas chromatograph/mass spectrometer (GC/MS). A 12-m cross-linked methyl silicone capillary column, 0.2 mm in ID and 0.33 μm in film thickness, was used. The oven temperature was 50°C.

The FC 113 analytical results are reported in Table 1. Figure 2 includes the total ion chromatogram and the mass spectrum of FC 113 present in the brain sample.

The reference spectrum of FC 113 is shown in Fig. 3.

TABLE 1—*1,1,2-Trichlorotrifluoroethane concentrations.*

Blood, μg/mL	2.3
Tissue, μg/g	
Lung	2.5
Brain	1370.1
Liver	80.6
Kidney	475.8
Muscle	8.8
Fat	5.4
Urine	not detected
1st liquid sample (sludge pit)	present
2nd liquid sample (sludge pit)	present
Pit sludge	present
Genisol DS	present

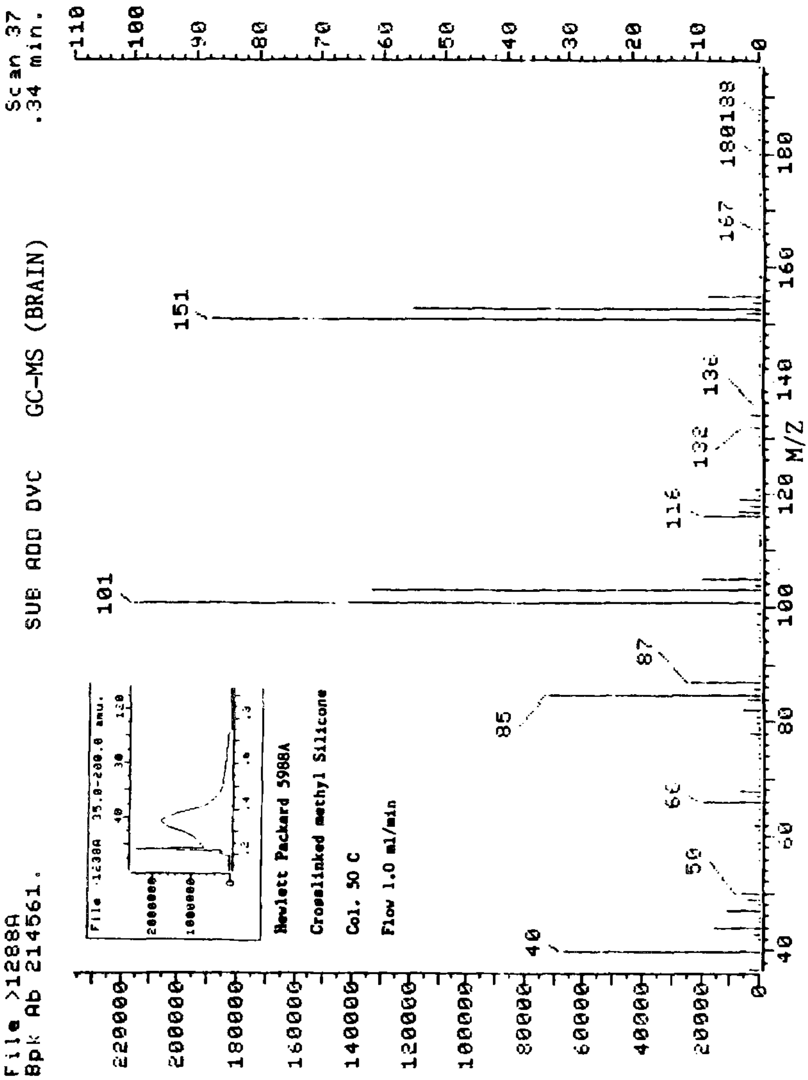

FIG. 2—Mass spectrum of the FC 113 present in the brain sample.

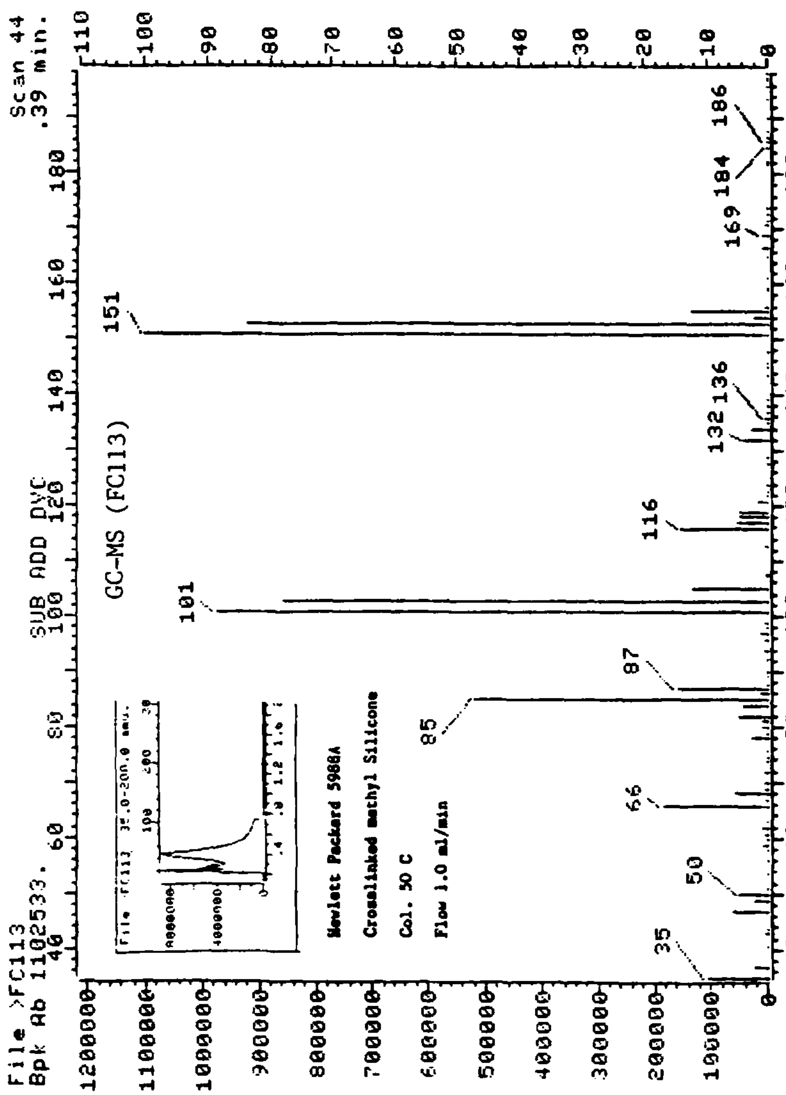

FIG. 3—The reference mass spectrum of FC 113.

Discussion

The fluorocarbons comprise a group of halogen-substituted methane and ethane derivatives, which have found extensive commercial use as aerosol propellants and refrigerants [*2*]. The first fluorinated hydrocarbon utilized as an aerosol propellant was originally used by the armed forces in World War II before it reached the civilian market in 1947. Production of various aerosolized products steadily increased to over 1 billion units by 1963 [*3*]. The propellants most widely used included fluorocarbons 11, 12, and 114, as well as isobutane, propane, and vinyl chloride [*4*]. Fluorocarbons 11 and 12 are currently being removed from the market in the United States because of their effect on the ozone concentration in the atmosphere [*5*].

A number of deaths associated with inhalation abuse of hydrocarbon-containing solvents were first observed in the late 1950s, which continued into the following decade. Bass [*6*] reported on 110 of these deaths and found that the most frequently abused hydrocarbons were trichloroethane and the fluorinated refrigerants. Subsequent reports of deaths due to fluorocarbon exposure have continued to appear in the literature [*7–15*]. Baselt [*16*] has summarized the fluorocarbon concentrations in fatalities due to exposure to fluorocarbons 11, 12, and 22 (Table 2). Clark et al. [*17*] reported three fatalities due to exposure to trichlorotrifluoroethane (TCTFE) in an enclosed, poorly ventilated compartment of a ship. Despite the victims' ability to climb a ladder out of the compartment, all three victims were in cardiopulmonary arrest within 3 to 5 min after collapsing on the deck of the ship. In these cases, the lung tissue concentration of TCTFE was between 0.05 and 1.0 μg/mL. In the current case, TCTFE was found in all specimens tested with the exception of urine. The highest concentrations were present in brain tissue, reflecting the high solubility of hydrocarbons within this particular tissue. The lung tissue concentration levels were comparatively low and may reflect the efforts at resuscitation.

Reports from the scene of the accident and from the clinical presentation of the subject in the emergency room, and the nonspecific results of the postmortem examination suggest a rapidly developing cardiac arrhythmia as the mechanism of death. Bass [*6*] suggested that fluorocarbons in high concentration act synergistically with endogenous catecholamines to induce cardiac arrhythmias. This response within the heart to catecholamines is known as cardiac sensitization and was first demonstrated by Levy and Lewis [*18*] and by Levy [*19*] in animal models. It has since been reported in the literature by a number of investigators [*20–24*]. Reinhardt et al. [*4*] demonstrated that cardiac sensitization could occur even after brief exposure to high concentrations of fluorocarbons.

In the present case, the subject's death was due to exposure to TCTFE gas, which vaporized out of sludge that was being removed from the base of a floor collection pit. Because of its specific gravity of 1.55, the fluorocarbon initially settled through the standing liquid into the sludge at the bottom of the pit. This settling was confirmed by investigators when a sample of Genesol DS was added to a 30-mL flask containing water.

TABLE 2—*Fluorocarbon concentrations.*

	Fluorocarbon		
Tissue	FC 11	FC 12	FC 22
Blood, μg/mL	12.0	3.0	371.0
Brain, μg/g	30.0	4.3	715.0
Lung, μg/g	43.0	33.0	248.0
Liver, μg/g	21.0	4.7	358.0
Kidney, μg/g	18.0	1.3	83.0

The water formed a layer on top of the fluorocarbon that acted as a barrier to evaporation. When the standing liquid was removed from the pit, this barrier was removed and evaporation of the TCTFE was able to occur. Mechanical disturbance of the sludge, occurring when the subject began to remove the material with a shovel, hastened this process, filling the pit with trichlorotrifluoroethane gas and displacing air upwards. Air samples were collected from the pit using a Gastec Model 400 gas detector tube pump and a Gillian Model HFS 113 Hi-Flow personal sampler. Analysis of these air samples by gas chromatograph and mass spectrometer confirmed elevated levels of TCTFE.

Summary

While deaths due to abuse of volatile substances have been widely reported in the literature, reports of fatalities due to hydrocarbon exposure in the workpalce are much more infrequent. Those deaths due to hydrocarbon exposure have been reported as occurring within enclosed, poorly ventilated areas or sites. We report here a death due to TCTFE exposure that occurred in an open pit located within a manufacturing facility.

Acknowledgment

The authors wish to thank Patty McGee for her kind assistance in the preparation of this manuscript.

References

[*1*] Christopoulos, G. N. and Kirch, E. R., "Estimation of Fluoroalkane Propellants," *Journal of Forensic Sciences,* Vol. 19, No. 1, Jan. 1974, pp. 168–171.

[*2*] Houts, M., Baselt, R. C., and Cravey, R. H., *Courtroom Toxicology,* Matthew Bender, New York, 1986, pp. FLUO 31–45.

[*3*] Martin, E. A., *Pharmaceutical Sciences,* Mack Publishing Co., Easton, PA, 1965, pp. 641–662.

[*4*] Reinhardt, C. F., Azar, A., Maxfield, M., Smith, P. E., Mullin, L. S., et al., "Cardiac Arrhythmias and Aerosol Sniffing," *Archives of Environmental Health,* Vol. 22, Feb. 1971, pp. 265–279.

[*5*] Wofsy, S. C., McElroy, M. B., and Sze, N. D., "Freon Consumption: Implications for Atmosphere Ozone," *Science,* Vol. 187, 1975, pp. 535–537.

[*6*] Bass, M., "Sudden Sniffing Death," *Journal of the American Medical Association,* Vol. 212, No. 12, June 1970, pp. 2075–2079.

[*7*] Baselt, R. C. and Cravey, R. H., "A Fatal Case Involving Trichloromonofluoromethane and Dichlorodifluoromethane," *Journal of Forensic Sciences,* Vol. 13, No. 3, July 1968, pp. 407–410.

[*8*] Stahl, D. J., Fatteh, A. V., and Dominguez, A. M., "Trichloroethane Poisoning: Observations on the Pathology and Toxicology in Six Fatal Cases," *Journal of Forensic Sciences,* Vol. 14, 1969, p. 393.

[*9*] Standefer, J. C., "Deaths Associated with Fluorocarbon Inhalation: Report of a Case," *Journal of Forensic Sciences,* Vol. 20, No. 3, July 1975, pp. 548–551.

[*10*] Kamm, R. C., "Fatal Arrhythmias Following Deodorant Inhalation," *Forensic Science,* Vol. 5, 1975, pp. 91–93.

[*11*] Muita, M., Miki, A., Kozama, H., and Sakata, M., "Case Report of Deaths Caused by Freon Gas," *Forensic Science,* Vol. 10, 1977, pp. 253–260.

[*12*] Garriott, J. and Petty, C. S., "Death from Inhalant Abuse: Toxicological and Pathological Evaluation of 34 Cases," *Clinical Toxicology,* Vol. 16, No. 3, 1980, pp. 305–315.

[*13*] Greer, W. E. R. and Giovaschini, R. P., Letter to the Editor, *Journal of the American Medical Association,* Vol. 254, No. 13, Oct. 1985, pp. 1721-1722.

[*14*] Fogel, S., Letter to the Editor, *Canadian Medical Association Journal,* Vol. 114, April 1976, pp. 671–672.

[*15*] Poklis, A., "Aerosol Propellant Abuse and Toxicity," *Canadian Society of Forensic Science Journal,* Vol. 8, No. 3, 1975, p. 87.

[16] Baselt, R. C., "Disposition of Toxic Drugs and Chemicals in Man," *Biomedical Publications*, Davis, Philadelphia, PA, 1982.
[17] Clark, M. A., Jones, J. W., Robinson, J. J., and Lord, J. T., "Multiple Deaths Resulting from Shipboard Exposure to Trichlorotrifluoroethane," *Journal of Forensic Sciences*, Vol. 30, No. 4, Oct. 1985, pp. 1256–1259.
[18] Levy, A. G. and Lewis, T., "Heart Irregularities, Resulting from the Inhalation of Low Percentages of Chloroform Vapor, and Their Relationship to Ventricular Fibrillation," *Heart*, Vol. 3, 1911/1912, pp. 79–111.
[19] Levy, A. G., "The Exciting Causes of Ventricular Fibrillation in Animals Under Chloroform Anesthesia," *Heart*, Vol. 4, 1913, pp. 319–378.
[20] Taylor, G. J. and Harris, W. S., "Cardiac Toxicity of Aerosol Propellants," *Journal of the American Medical Association*, Vol. 214, No. 1, Oct. 1970, pp. 81–85.
[21] Flowers, N. C. and Horan, L. G., "The Electrical Sequelae of Aerosol Inhalation," *American Heart Journal*, Vol. 83, No. 5, May 1972, pp. 644–651.
[22] Flowers, N. C. and Horan, L. G., "Nonanoxic Aerosol Arrhythmias," *Journal of the American Medical Association*, Vol. 219, No. 1, Jan. 1971, pp. 33–37.
[23] Harris, W. S., "Toxic Effects of Aerosol Propellants on the Heart," *Archives of Internal Medicine*, Vol. 131, Jan. 1973, pp. 162–166.
[24] Wyse, D. G., "Deliberate Inhalation of Volatile Hydrocarbons: A Review," *Canadian Medical Association Journal*, Vol. 108, Jan. 1973, pp. 71–74.

Address requests for reprints or additional information to
M. B. McGee, M.D.
Ramsey County Medical Examiner's Office
155 Hill St.
St. Paul, MN 55102