CASE REPORT

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Multiple Deaths Resulting from Shipboard Exposure to Trichlorotrifluoroethane

REFERENCE: 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*, JFSCA, Vol. 30, No. 4, Oct. 1985, pp. 1256-1259.

ABSTRACT: During the course of dockside ship maintenance, a compartment was partially flooded with tricholotrifluoroethane gas. One sailor entered the compartment, collapsed, and was then rescued by two other men. All three victims then climbed a 11-m (36-ft) ladder and collapsed. They all experienced a rapid development of cardiac arrest. We report on the pathologic, toxicologic, and pathophysiologic aspects of the incident.

KEYWORDS: pathology and biology, toxicology, trichlorotrifluoroethane

Halogenated hydrocarbons were first widely used in the 1930s as refrigerants and industrial solvents. They were regarded as relatively safe and nontoxic and by the early 1960s were ubiquitous as propellants in a variety of spray can products. Bass in 1970 [*1*] reviewed a number of cases of sudden death associated with the inhalation abuse of spray can propellants including fluoroalkanes. A number of case reports and reviews have subsequently been published [*2-5*].

Besides being used as spray can propellants, fluoroalkanes are widely used as refrigerants—particularly in industrial applications. Because of their known cardiotoxicity in high concentrations [*6*], there is a potential for fatal accidental occupational exposure to fluoroalkanes.

Report of Incident

During the course of routine dockside maintenance on the air conditioning system of a ship, a manifold fitting ruptured without the defect being detected. A hose was attached to this manifold and the hose was run to the deck of the ship where tricholotrifluoroethane (TCTFE)

The opinions or assertions expressed herein are those of the authors and are not to be construed as official or as necessarily reflecting the views of the Department of the Navy or the naval service at large.

Received for publication 2 Oct. 1984; revised manuscript received 8 Dec. 1984; accepted for publication 10 Dec. 1984.

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was pumped through the hose into the refrigeration system. Because of the undetected leak, the compartment containing the refrigeration manifold became filled with TCTFE. One of two members of the ship's crew, making routine security rounds, entered the compartment and collapsed. The other crew member, seeing that his shipmate was in trouble, entered the compartment and rescued him. Together, they began climbing a ladder, whereupon they were aided by a third sailor who happened upon the scene. The three victims managed to climb 11 m (36 ft) then collapsed on the deck. Medical help was summoned, and within 3 to 5 min of collapsing on deck all three men were in full cardiopulmonary arrest. Basic cardiopulmonary resuscitation (CPR) was begun and the victims were subsequently taken off the ship and transferred to a local hospital where advanced CPR, including open cardiac massage, and multiple attempts at direct electrical defibrillation were performed. Initially, an electrical myocardial response was obtained in all three victims, but within minutes the rhythm degenerated to refractory asytole. All three victims were pronounced dead approximately 1 h after the initiation of CPR.

The six paramedical personnel, who administered CPR at the scene and while in route to the hospital, and one emergency room staff member complained of weakness, chest pain, and palpitations. They were admitted to the hospital for observation. Their symptoms disappeared within 2 to 3 h and all are alive, well, and without complaints two-and-a-half years following the incident.

Autopsy Findings

Postmortem examinations were performed on all three victims the following morning approximately 14 h after death. Aside from evidence of advanced CPR procedures, generalized visceral congestion was the only constant anatomic finding in the three autopsies. Agonal aspiration of gastric contents was noted in one case, and a small area of remote posterior left ventricular scarring was seen in another case.

At the time of autopsy, the left mainstem bronchus was ligated and the left lung of each victim removed intact. The lungs were sealed in glass jars and immediately frozen. Samples of blood were also obtained for toxicologic analysis.

Toxicologic Studies

Analyses for TCTFE were performed on samples of blood and lung obtained at autopsy, and the samples of blood drawn in the emergency room (and immediately frozen) which were taken from the three victims and the seven paramedical personnel who became symptomatic while administering CPR. A sample of TCTFE, taken from the ship's air conditioning system on the day following the deaths, was used as a standard. This standard showed a linear relationship between concentration and peak height or peak area, and it was used as a reference for analysis of lung tissue and blood samples.

Weighed samples of frozen lung tissue and blood were sealed in vials with septum caps and crimped aluminum rings. These vials were inserted into a Perkin-Elmer headspace analyzer for injection. A Perkin-Elmer Sigma IB gas chromatograph with flame ionization and electron capture detectors was used for qualitative and quantitative analyses. The carrier gas was helium at a flow gate of 40 mL/min with column, injector, and detector temperatures of 250, 180, and 285°C, respectively. A 1.8-m (6-ft), 1.8- by 4-mm glass column was used containing Porapak Q 80-100 mesh.

TCTFE was detected only in the lung samples of the three victims (Table 1), and it was not detected in their blood or in the blood of the paramedical personnel who became symptomatic while administrating CPR.

TABLE 1—*Toxicologic and autopsy findings in victims.*

Victim	Age, Years	TCTFE Concentration in Lung Tissue, mg/kg	Anatomic Autopsy Findings
1	27	0.05	generalized visceral congestion small myocardial scar
2	20	1.00	generalized visceral congestion agonal aspiration of gastric contents
3	27	0.05	generalized visceral congestion

Discussion

The toxic properties of TCTFE were first evaluated in 1941 when it was introduced as a refrigerant gas and an industrial degreasing agent. It was found to be relatively nontoxic when compared to other refrigerants in use at that time [*7*]. The compound has a specific gravity of 1.5 and when discharged into closed, poorly ventilated spaces, such as shipboard compartments without windows, it displaces the air in the compartment from below upward. Imbus and Adkins studied workers chronically exposed to TCTFE at Kennedy Space Center and found no deleterious effects at levels up to 4700 ppm [*8*]. Morita et al [*9*] reported a case where four sailors asleep in lower and middle bunks were found dead following the accidental discharge of Freon®-22 into the compartment. The sailors in the upper bunks (in the same compartment) were able to escape. Two of the four victims were autopsied, and the postmortem findings were suggestive of asphyxia.

In the present case, the victims clearly did not die of asphyxia since they were able to climb an 11-m (30-ft) ladder before collapsing and developing cardiac arrest. This history is not unlike witnessed deaths following inhalational abuse of fluoroalkanes reported in the literature [*1–4*]. A typical history in such circumstances is that of up to a minute or more of manic activity followed by collapse and death [*1–5*]. Autopsy findings in such previously reported cases have been nonspecific and usually only visceral congestion has been observed, along with occasional agonal aspiration of gastric contents [*2–5*].

Experimental animal studies have shown that the halogenated hydrocarbons cause cardiac arrhythmias thought to be due to sensitization of the cardiac conducting system to the effects of endogenous epinephrine [*10–12*]. Typical arrhythmias associated with halogenated alkane exposure are profound sinus bradycardias followed by lower escape arrhythmias and asystole [*10*]. The present case is classical for halogenated alkane toxicity, that is, the victims were capable of a minute or more of volitional activity followed by collapse and cardiac arrest.

In addition, the observations during CPR support the animal experimental findings of cardiac rhythum rapidly degenerating into refractory asystole. Since no epinephrine or epinephrine-like compounds were administered during CPR, this may indicate that irreversible change occurs in the myocardium or cardiac conduction system as a result of TCTFE toxicity.

References

[*1*] Bass, M., "Sudden Sniffing Death," *Journal of the American Medical Association*, Vol. 212, No. 12, 22 June 1970, pp. 2075–2079.

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

[*3*] Kamm, R. C., "Fatal Arrhythmia Following Deodorant Inhalation," *Forensic Science*, Vol. 5, No. 1, 1975, pp. 91–93.

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

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

[6] Aviado, D. M., "Toxicity of Aerosols," *Journal of Clinical Pharmacology*, Vol. 15, No. 1, 1975, pp. 86-104.
[7] "Report on the Comparative Life, Fire and Explosion Hazards of Trifluorotrichloroethane ("Freon 113")," Underwriters Laboratories, Inc., Chicago, IL, 7 April 1941.
[8] Imbus, H. R. and Adkins, C., "Physical Examinations of Workers Exposed to Trichlorotrifluoroethane," *Archives of Environmental Health*, Vol. 24, April 1972, pp. 257-261.
[9] Morita, M., Miki, A., Kazama, H., and Sakata, M., "Case Reports of Deaths Caused by Freon Gas," *Forensic Science*, Vol. 10, No. 2, 1977, pp. 253-260.
[10] Belej, M. A., Smith, D. G., and Aviado, A. M., "Toxicity of Aerosol Propellants in the Respiratory and Circulatory Systems. IV. Cardiotoxicity in the Monkey," *Toxicology*, Vol. 2, No. 3, 1974, pp. 381-395.
[11] Aviado, D. M. and Belej, M. A., "Toxicity of Aerosol Propellants in the Respiratory and Circulatory Systems. V. Ventricular Function in the Dog," *Toxicology*, Vol. 3, No. 1, 1975, pp. 79-86.
[12] Aviado, D. M., "Toxicity of Aerosol Propellants in the Respiratory and Circulatory Systems. X. Proposed Classification," *Toxicology*, Vol. 3, No. 4, 1975, pp. 321-332.

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