Fluorocarbon Toxicity: Aerosol Deaths and Anaesthetic Reactions

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ABSTRACT

Twelve cases of death due to fluorocarbon inhalation have been seen in a four year period 1971-1975. The source of the material has been commercial spray cans. The postmortem examinations are nonspecific, but a history of excitement or sudden death is often elicited. Confirmation of the cause of death was made by demonstration of a halogenated hydrocarbon in the blood by gas chromatography.

Introduction

National concern over the extent of drug abuse by our young people reached its peak during the 1960’s. At this time, many types of drugs were used and many novel combinations were discovered. No drug or compound for producing altered mental sensations was overlooked. Approximately a decade ago, the fad of inhaling the propellants of aerosol cans began on the West Coast within its drug experimenting community. The drug users discovered what the “establishment” had overlooked: fluorooalkanes produce an altered mental state deemed by some to be pleasant.

By far the most common fluorooalkanes used in aerosol cans are Freon 11 and 12 (trichloromonofluoromethane and dichlorodifluoromethane). The chemical structure of the fluorooalkanes is similar to the anesthetic halothane, a fluorinated hydrocarbon which has been shown to have cardiovascular effects (figure 1).

The fluorooalkanes have sporadically been incriminated as the culprit in causing sudden death and, recently, they have been suspected of contributing to the destruction of the ozone layer of our atmosphere. One of the earliest reports of a death by self-administered fluorooalkane inhalation was in 1968. It was the conclusion of the author that the cause of death was anoxia by excluding oxygen from the body tissues and was not due to inherent toxic properties of Freon 11 or 12. While it is now recognized that the fluorooalkanes can cause sudden death, there seems to be some reluctance by industry to accept this possibility, especially by manufacturers of aerosol medications where the intake of fluorooalkanes is small.

In 1970, Taylor and Harris showed by experiments in mice and dogs that the
fluoroalkanes were toxic to the heart. The myocardium is apparently sensitized to asphyxia induced bradycardia with electrocardiographic evidence of A-V block and T-wave depression.

**Material and Methods**

Blood and tissues can be analyzed for fluoroalkanes using headspace gas chromatographic techniques. In our laboratory, the following method is used for qualitative identification of the halogenated hydrocarbons.

Samples of blood from the left pulmonary vein are transferred to stoppered Vacutainer® test tubes. The tubes are heated in a boiling water bath and a known amount is withdrawn from the headspace with a Hamilton microliter syringe. This is injected into a Tracor 220 gas chromatograph with a Chromosorb 101, 80/100 mesh column using helium as the carrier gas and hydrogen flame ionization detection. The retention times are compared with those of known standards added to aliquots of pooled blood specimens which are subjected to the same analysis.

Quantitative measurements have recently been instituted and will be reported subsequently. The quantitative approach requires a Coulson electrolytic conductivity detector system. Quantitative determinations of fluorocarbon content of various body tissues show the myocardium to be highest followed by the lung and liver.\(^{11}\)

**Results**

In Arkansas from May 1971 through June 1975, 12 cases of sudden death owing to inhalation of the propellant of spray cans (Freon 11 and/or 12) were observed. This has included a variety of spray substances, seven cases of PAM,\(^{®}\) one of Right Guard,\(^{®}\) one of Bactine,\(^{®}\) one of Arrid Extra Dry,\(^{®}\) one of Freon for car airconditioners and one of Pssst.\(^{®}\) It is highly significant that the victims have all been teenagers. The ages ranged from 14 to 19 years; nine were male and two female. Only one of the subjects were black, a 16-year-old female (table I). Gas chromatography of blood confirmed the presence of fluoroalkanes but quantitation was not attempted. However, it can be stated that the concentration of the hydrocarbon inhaled by the abusers was apparently quite high.

The usual method of inhalation is to discharge the can into a paper or plastic bag, then place the face in the bag and breathe in deeply. A more direct method is to place a filter over the can such as a tube stuffed with tissue paper, then insert the other end into the mouth and inhale while discharging the spray container. Analysis of blood was performed in 12 cases and qualitatively demonstrated the
presence of fluoroalkanes. No attempt was made to quantify the material in these cases.

Discussion

Scientists from the manufacturers of propellant products have taken an exception to the findings of Taylor and Harris. An effort to minimize the danger is seen in the labels on our household products. At least one aerosol can spray still lists its propellant as non-toxic, while most products only contain an inconspicuous small print warning about the inhalation hazard. However, danger warnings may not be an effective deterrent. One of our subjects carried a newspaper clipping in his wallet reporting a previous case in which death occurred as a result of the same product that took his life.

Many human fluoroalkane deaths have been associated with fright and physical excitation. In the six case histories of sniffing deaths recorded by Bass, the central theme of the immediate antemortem episode was fright and running. This behavior has been confirmed in the cases in which sufficient history to determine the episodes just preceding death has been obtained by the author. An explanation from the work of Hall, et al suggests that Freon sensitizes the heart to epinephrine-induced fibrillation. Occasionally, the fibrillatory episode may be so sudden in onset after inhalation of aerosol that no movement of the body may be seen. In such cases, instantaneous rigor may be observed.

Studies by Paterson et al would indicate that peak blood levels were attained 30 to 90 seconds after inhalation and that the blood concentrations of fluorocarbon ranged from 0.27 to 2.60 μg per l with two puffs from pressurized aerosol inhalers. However, it would appear that the inhalations by the decedents are much less controlled and probably the concentrations significantly higher, since a “symptomatic” effect is the objective of the user. At autopsy the tissue fluorocarbon levels, especially lung and myocardium, are much higher than those found in the blood, hence blood levels have only circumstantial value in establishing the cause of death but not its mechanism.

There is often a problem in recognizing the cause of sudden death in the young thrill-seeker because the true story may be hidden. Several cases of unexplained death have been referred to the author with a history indicating that the subject suddenly became ill and died. Only by careful questioning of the witnesses and by having a high index of suspicion was the real cause ascertained. In the state of Arkansas, the scene of death may be many miles from the laboratory. This has caused problems in documentation since the local law enforcement officials may not consider the possibility of an aerosol death. This has been reduced by ongoing educational programs for law enforcement personnel. Sometimes the container has been removed or “lost” by an agency; often, it has been deliberately disposed of by the co-user or friend of the decedent.

Additionally, it should be recalled that fluoroalkanes and related fluorine-containing organic substances are used in anesthesia. Moreover, defluorinases exist in many body tissues and most prominently in the liver. The presence of three fluorine atoms on one carbon, however, renders the molecule resistant to defluorinase effect. Thus, halothane and fluoroxyne (like the freons) are virtually non-defluorinated. Other fluorinated anesthetic agents are actively metabolized and the lipid solubility of some fluorinated anesthetic agents tends to make them available in the body for defluorination for extended periods of time after anesthesia. The potential for toxic fluorine effects, such as nephrotoxicity from the fluorine of these anesthetic agents, is thus significant. Their obstetrical use during labor also presents a poten-
tial transplacental fluorine hazard to the fetus.\textsuperscript{6}

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References