CASE REPORT

Marcus B. Nashelsky,[1] M.D.; Jay D. Dix,[1] M.D.; and Eddie H. Adelstein,[1] M.D.

Homicide Facilitated by Inhalation of Chloroform

REFERENCE: Nashelsky, M. B., Dix, J. D., and Adelstein, E. H., "**Homicide Facilitated by Inhalation of Chloroform,**" *Journal of Forensic Sciences,* JFSCA, Vol. 40, No. 1, January 1995, pp. 134–138.

ABSTRACT: Three related homicides in which each decedent had significant concentrations of chloroform in blood, fat, brain and/or liver are described. The tissue concentrations of chloroform in one of three decedents were within reported lethal ranges. The concentrations in the remaining two decedents were less than lethal but were well above blood levels in nonoccupationally exposed, healthy subjects. The cause of death in one decedent with sublethal chloroform concentrations was suffocation; the cause of death in the other decedent could not be determined with certainty. The manner of death in each case was homicide. Through a review of the literature the authors discuss the history of chloroform as an inhalation anesthetic and the history of chloroform as an agent of abuse, suicide, assault, and homicide. Blood and/or tissue concentrations of chloroform in nonoccupationally exposed, healthy subjects and victims of suicide or homicide from previous reports are compared and contrasted with the amounts in blood and/or tissue in the three subjects described in this study. The authors conclude that, in addition to a direct lethal effect, chloroform may be used to incapacitate a victim of assault who then dies by another cause.

KEYWORDS: pathology and biology, chloroform, homicide

Chloroform has contemporary application in industry and the laboratory as a solvent and chemical intermediate but is historically well known as a nineteenth and twentieth-century inhalation anesthetic. The central nervous system depressant effect has allowed for the nontherapeutic but intentional use of chloroform for recreation, suicide, assault, and homicide. In this report we describe three related homicides facilitated or directly caused by chloroform and the literature is reviewed.

Case Reports

A 28-year-old white male independent insurance salesman living in Columbia, Missouri, had elderly clients in numerous counties throughout mid- and eastern Missouri. He did not have a permanent office and sold insurance by traveling to the homes of potential customers. He was on probation for burglary and had a history of petty theft in California, check fraud in Missouri and cocaine and weapons offenses in Illinois.

Received for publication 7 April 1994; revised manuscript received 30 May 1994; accepted for publication 31 May 1994.

[1]Department of Pathology, University of Missouri - Columbia Hospital and Clinics, Columbia, MO.

Case #1

On December 17, 1992, the salesman attempted to cash a $5,000 check allegedly written to him by an 85-year-old white female resident of a nearby small college town. The local bank refused to cash the check because the signature on the check did not match the signature of record. The bank notified the local police on December 18 and officers dispatched to the house found the woman dead on the kitchen floor. The front door was ajar and there was a partially eaten plate of food on a tray in the living room. The home was otherwise entirely in order. In light of the check-cashing attempt of the previous day, an autopsy was ordered and performed on the same day. The police elected to delay further investigation until the autopsy results were reported.

On January 7, 1993, the same salesman attempted to sell an annuities plan to an 84-year-old white female in her home in a small eastern Missouri town. She refused his pitch. Later that day the salesman returned to the woman's home, brandished a gun and threatened to kill her. He then displayed a small, liquid-filled bottle, moistened a handkerchief with the contents of the bottle and forcefully placed it over the mouth and nose of the elderly woman. She awoke approximately one-half hour later and called the police. A warrant was issued for the arrest of the insurance salesman.

Case #2

On the evening of January 8, 1993, a 72-year-old white female living independently in a small northeastern Missouri town was on the telephone with a friend. She told her friend that someone was at the door and then put down the phone to go to the front door. She then returned to the phone and told her friend that she would call back later. The call was not made and the friend on the telephone later reported no suggestion of stress or other abnormality in the other woman's voice. On January 9 the woman failed to appear for volunteer work at the local senior center. The police were called and search of the woman's home revealed the front door ajar, house lights on, two canes in the house (recent history of hip replacement) and previously frozen meat and orange juice left to thaw on the kitchen table. The woman (dead or alive) was not present.

The salesman was arrested at his home in a Columbia apartment complex on January 9. In his car the police discovered a list of several hundred names, a paint pellet gun resembling a semiautomatic pistol, a small, empty bottle and a purse belonging to the woman described in Case #2.

Case #3

Law enforcement officers began to review the lengthy list of clients or potential clients. In addition to the names of the three previously described clients, they discovered the name of a 78-year-old white male from a small mid-Missouri town who had been found dead and extensively decomposed at home on the previous August 18, 1992. There was a week's accumulation of mail in the mailbox. The county coroner had ruled the manner of death as natural without investigating the scene or performance of an autopsy. The decedent had written a total of $15 000 in checks to the insurance salesman but no insurance policy had ever been issued. The body was exhumed and an autopsy was performed on January 15, 1993.

Also on January 15 the frozen body of the woman described in Case #2 was discovered at the edge of the ice-packed Missouri River in central Missouri, approximately ten miles from Columbia and approximately 125 miles from her home in northeastern Missouri. An autopsy was performed on January 17 after the body had thawed.

On January 21 law enforcement officers searched a storage locker used exclusively by the insurance agent at his apartment complex and discovered a one-gallon bottle of chloroform. On January 25 the insurance agent was charged with first degree murder for the death of the woman described in Case #2.

Autopsy Results

Autopsies on the decedents in Cases #2 and #3 (performed on January 17 and 15, respectively) included toxicologic studies for the usual drugs of abuse. Chloroform and ether were empirically considered by police and pathologists as chemicals warranting specific assay given the history of assault with a liquid-soaked handkerchief. Laboratory investigation began with assays for chloroform on blood, fat, and brain from Case #2 and fat, brain, and liver from Case #3. Only blood was available for analysis from the December 18 autopsy of the decedent in Case #1. Studies for ether were not pursued after discovery of a large supply of chloroform among the insurance salesman's possessions.

Final anatomic diagnoses and toxicology results are listed in Table 1. Chloroform levels were determined by a reference laboratory utilizing head space gas chromatography [*1,2*]. Blood specimens from Cases #1 and #2 were free of the usual drugs of abuse. Blood was not available for analysis in Case #3.

The cause of death in Cases #1 and #3 was undetermined. The cause of death in Case #2 was suffocation by compression of the chest or occlusion of the airway and blunt impact injuries to the head and chest were contributing factors. The manner of death in each case was homicide.

Discussion

Anesthetics (volatile and nonvolatile) are drugs that interfere with nervous system function to eliminate reflex responses to external stimuli (especially painful stimuli) and reduce muscle tone. Volatile anesthetics provide analgesia while nonvolatile agents may or may not provide analgesia. Dose-dependent changes in central nervous system function are classically related to four stages of anesthesia. Analgesia is attained in stage I (with volatile drugs) and consciousness is lost in stage II. Stage III provides a level of anesthesia appropriate for surgery and is divided into four planes. These range from a state with preservation of muscle tone (plane 1) to a state with abolition of reflexes and complete muscle relaxation with absent intercostal muscle activity and reduced diaphragmatic excursion (plane 4). The medulla is inactive in stage IV of anesthesia. The respiratory center is depressed and spontaneous respiratory activity is absent [*3*].

Chloroform ($CHCl_3$, trichloromethane, methenyl trichloride) is a clear, colorless, sweet-smelling, strongly lipophilic, volatile liquid with no endogenous production. It was first synthesized in 1831 independently by Guthrie in the United States, Souberain in France and von Liebig in Germany. In 1847 James Y. Simpson, M.D., an obstetrician of Edinburgh, Scotland, first used chloroform as an inhalation anesthetic during childbirth [*4*]. Soon thereafter chloroform became a popular and widely used anesthetic agent.

Over the ensuing decades it became clinically evident that although chloroform produced excellent anesthesia, its use was accompanied by a significant risk of hepatic toxicity, myocardial depression, and cardiac arrythmia. Cardiac arrhythmias were secondary to cardiac sensitization to catecholamines [*5*]. In 1912 a committee of the American Medical Association recommended that the use of chloroform as an anesthetic for both major and minor operations cease. The committee cited the unpredictable occurrence of hepatic necrosis and ventricular fibrillation [*6*], however, the use of chloroform as a surgical anesthetic continued until 1957 [*7*].

Currently, the presence of chloroform in blood is not restricted to people with known exposure in the laboratory or industrial workplace. It has been detected in the exhaled breath or blood of subjects without known occupational exposure and has been related to the presence of chloroform and other volatile organic compounds in air, chlorinated tap water (especially hot water and swimming pools), other beverages (especially soft drinks) and food [*8–12*]. We are aware of four studies using gas chromatography that document blood levels of chloroform in subjects without known occupational exposure [*8,9,12,13*]. There was a total of 241 subjects and a blood chloroform range of <0.0000955 to 4.0 mcg/mL. If one excludes three outlying values from one study (0.205 mcg/mL, 2.9 mcg/mL and 4.0 mcg/mL) [*8*] the blood chloroform range in 238 subjects was <0.0000955 to 0.085 mcg/mL (Pfaffenberger and Peoples [*8*] attribute the three elevated blood chloroform levels to acute exposure of unknown etiology and there is no mention of altered mental status.) The results of each study are listed in Table 2. The range of very low blood chloroform levels listed in Table 2 could be considered "physiologic" in the industrialized twentieth century.

Fry et al. reported the blood concentrations of chloroform after intentional oral administration of chloroform in 12 willing subjects [*14*]. The peak concentrations ranged from 1 mcg/mL–5 mcg/mL (gas chromatography). Alteration in mental status was not mentioned for any of the subjects.

Nonphysiologic levels of chloroform from the medical or nonlaboratory, nonindustrial use of chloroform are due to the drug's ability to rapidly produce dose-dependent narcosis, unconsciousness, surgical anesthesia or death when inhaled, ingested or injected. There are reports in the medical literature from as early as 1848 describing intentional inhalation, ingestion or injection of chloroform for alteration of mood or suicide [*15–26*]. Articles describing homicide related to chloroform are less common [*16,17,19,27–30*]. Also, there are a few nineteenth-century reports of chloroform used to incapacitate a victim of theft [*31,32*].

An "Attempted Daring Robbery by the Aid of Chloroform" was recounted in the "Medical Facts, News, Items, Etc." column of *The Lancet*, 1850 [*31*]. An elderly clergyman, lodged at an hotel in Kendal, was "awakened by the attempt of a person to render

TABLE 1—*Anatomic diagnoses and blood chloroform levels on cases 1, 2, 3.*

Anatomic diagnoses	Toxicology (chloroform, mcg/mL) (gas chromatography)			
	BLOOD	FAT	BRAIN	LIVER
Case #1, 85 WF, Dec. 18				
1×1×1 cm focus of soft tissue hemorrhage over left carotid artery atherosclerotic coronary artery disease, moderate focal atherosclerotic occlusion of small branch of LAD angiodysplasia of the colon	2.0	nt	nt	nt
Case #2, 72 WF, Jan. 17				
bilateral conjunctival petechiae facial abrasions and lip laceration subscalpular hemorrhage focal subarachnoid hemorrhage and focal brain contusion fractures of sternum and right and left ribs multiple contusions of right and left upper extremities and right shoulder atherosclerotic coronary artery disease, moderate	3.0	10.0	3.0	nt
Case #3, 78 WM, Jan. 15				
atherosclerotic coronary artery disease, moderate chronic pulmonary emphysema marked decomposition	nt	42.0	46.0	24.0

W = white; F = female; M = male; LAD = left anterior descending coronary artery; nt = not tested.

TABLE 2—*Previous studies of blood chloroform in subjects without known exposure (gas chromatography).*

Study	*n*	Range(mcg/mL)
Peoples 1979	10	0.013–0.049
Pfaffenberger 1982	22	0.004–0.085*
Hajimiragha 1986	39	<0.0001–0.0017
Aggazzotti 1990	40	<0.000 095 5–0.003

* = Elevated chloroform levels are discussed in text and not included in table.

him powerless by placing a cloth over his mouth; and at the time of his rescue by those whom his cries brought to the apartment, a strong smell of chloroform is reported to have been perceptible, and two bottles of that substance are said to have been discovered.... It is not the first time that an agent of good has been converted into an agent of evil."

An editorial in *The Lancet*, 1865, lamented attention given in the popular literature of the day to the "common highwayman" and "the fact that a highwayman can, by shaking a handkerchief impregnated with chloroform under the nose of a victim, produce instantaneous insensibility" [*32*]. The editorial contended that "it is within the experience of medical men that anaesthesia by chloroform is not very quickly or very easily effected upon a nonconsenting person, and that with the utmost resignation and good-will some five minutes or more are requisite to produce anaesthesia." Each point of view was probably founded in some truth.

In 1954, a review of volatile compounds briefly mentioned chloroform-related deaths recorded by the Office of the Chief Medical Examiner of New York City from 1918 to 1951 [*17*]. The manner of death was homicide in five cases, accident in eight cases and suicide in 53 cases. Other details were not provided.

Forced inhalation of chloroform was implicated in two separate homicidal deaths of 18- and 24-year-old women by Bonnichsen and Maehly in 1966 [*19*], in a double homicide of a 29-year-old man and a 23-year-old woman by McGee et al. in 1987 [*29*] and in the homicide of a pregnant woman (age not given) by McIntyre in 1988 [*30*].

Several of the previously cited studies document chloroform levels in blood and other organs in persons who died secondary to recreational, accidental, suicidal, or homicidal inhalation or ingestion of chloroform. The values from the four studies utilizing gas chromatography are listed in Table 3. Blood and tissue concentrations of chloroform from six subjects range from 30 to 180 mcg/mL.

We are aware of a single study of blood chloroform levels in anesthetized human subjects [*33*]. In 1951 Morris et al. reported a study of 58 patients in planes 1 to 4 of stage III anesthesia. The method of quantitation was the Fujiwara colorimetric technique, which preceded development and use of gas chromatography. Mean blood chloroform levels were as follows: plane 1:71 mcg/mL, plane 2:106 μg/mL, plane 3:122 μg/mL, and plane 4:165 mcg/mL. These values appear to be incompatible with the lethal levels listed in Table 3. The use of different quantitation techniques (Fujiwara and gas chromatography) may not allow for meaningful comparison of blood and tissue chloroform levels.

Using the Fujiwara technique Gettler et al. in 1931 described another variable which complicates comparison between studies

TABLE 3—*Previous studies of chloroform in blood and other organs in fatalities (gas chromatography).*

Study	Manner of Death	Toxicology (chloroform)			
		Blood (mcg/mL)	Fat	Brain (mcg/g)	Liver (mcg/g)
Giusti 1981	suicide	40	nt	nt	nt
	suicide	30	nt	37[a]	nt
McGee 1987	homicide	130.96	nt	54.46	63.65
	homicide	nt	nt	133.27	123.08
Allan 1988	accident	120	nt	nt	nt
Kohr 1990	suicide	180	nt	nt	298

nt = not tested; [a] = units in original report = parts per million (ppm).

[16]. They demonstrated progressive reduction in tissue chloroform with duration of putrefaction. There was a non-linear change with 82% recovery of chloroform at one day, 65% at three days, 49% at six days, 31% at ten days and 18% at 42 days. The authors suggested that the increasing alkalinity of decomposing tissue promotes the breakdown of chloroform.

The chloroform levels from two of the three fatalities described in this report present a contrast with the previously discussed "physiologic" levels and lethal levels. The blood chloroform in Cases #1 and #2 were 2.0 and 3.0, respectively, and therefore intermediate between levels known to be physiologic and levels known to be lethal. Extensive postmortem chloroform degradation was probably unlikely in either instance. In Case #1 the postmortem interval was less than one day. In Case #2 the postmortem interval was ten days but the decedent was frozen for most of that period and there were no signs of decomposition at autopsy. The blood levels suggest that chloroform was used with the intent of incapacitating the victim. The decedent in Case #2 demonstrated autopsy findings of suffocation. The decedent in Case #1 demonstrated only a soft tissue contusion of the neck over the left carotid artery but death by suffocation cannot be ruled out. Another consideration is a lethal arrhythmia secondary to cardiac sensitization to catecholamines during the assault with chloroform. Additionally, the vagal inhibitory effect of chloroform could have been exacerbated by pressure or trauma to the area of the carotid sinus.

The brain and liver levels of chloroform in Case #3 approach the lethal values from other cases in Table 3. We suggest that blood and tissue chloroform concentrations were higher at the time of death given one week of decomposition before discovery of the body and interment for five months without embalming. Anatomic evidence of injury was not seen at autopsy but the examination was compromised by decomposition. In Case #3 chloroform levels may have been high enough to cause death without the contribution of a physical assault.

Conclusion

We have described one non-fatal assault and three deaths linked to a single individual and the use of chloroform, a volatile anesthetic. The manner of death in each case was homicide although a cause of death was identified in only one case (suffocation). This small series confirms evidence presented in previous reports and demonstrates that chloroform may be an effective and unsuspected agent of assault or homicide, especially in the elderly.

On March 8, 1994, the insurance agent was found guilty of first degree murder in Case #2.

References

[1] Dubowski, K. M., "Organic Volatile Substances," in *Methodology for Analytical Toxicology,* I. Sunshine, Ed., CRC Press, Cleveland, 1975, pp. 407–411.

[2] Reddrop, C. J., Riess, W., and Slater, T. F., "Two Rapid Methods for the Simultaneous Gas-Liquid Chromatographic Determination of Carbon Tetrachloride and Chloroform in Biological Material and Expired Air," *Journal of Chromatography,* Vol. 193, 1980, pp. 71–82.

[3] Adriani, J., "General Anesthetics," in *Clinical Management of Poisoning and Drug Overdose,* L. M. Haddad and J. F. Winchester, Eds., W. B. Saunders Co., Philadelphia, 1983, pp. 753–771.

[4] Simpson, J. Y., "A New and Anesthetic Agent, More Efficient Than Sulfuric Ether," *The Lancet,* Vol. 7, No. 1, New York, Jan. 1848, pp. 34–35.

[5] Meredith, T. J. and Vale, J. A., "The Halogenated Hydrocarbons," in *Clinical Management of Poisoning and Drug Overdose,* L. M. Haddad and J. F. Winchester, Eds., W. B. Saunders Co., Philadelphia, 1983, pp. 779–780.

[6] Henderson, Y., Cullen, T. S., Martin, E. D., Huntington, T. W., and Murphy, F. T., "Report of Committee on Anesthesia," presented at the American Medical Association House of Delegates Meeting, June 5, 1912, *Journal of the American Medical Association,* Vol. 58, No. 24, 1912, pp. 1908–1910.

[7] Miller, R. D., *Anesthesia,* Churchill Livingston, New York, 1981, p. 399.

[8] Pfaffenberger, C. D. and Peoples, A. J., "Long-Term Variation Study of Blood Plasma Levels of Chloroform and Related Purgeable Compounds," *Journal of Chromatography,* Vol. 239, 1982, pp. 217–226.

[9] Hajimiragha, H., Ewers, U., Jansen-Rosseck, R., and Brockhaus, A., "Human Exposure to Volatile Halogenated Hydrocarbons from the General Environment," *International Archives of Occupational and Environmental Health,* Vol. 58, 1986, pp. 141–150.

[10] Wallace, L. A., Pellizzari, E. D., Hartwell, T. D., Davis, V., Michael, L. C., and Whitmore, R. W., "The Influence of Personal Activities on Exposure to Volatile Organic Compounds," *Environmental Research,* Vol. 50, 1989, pp. 37–55.

[11] Skolnick, A., "Even Air in the Home Is Not Entirely Free of Potential Pollutants," *Journal of the American Medical Association,* Vol. 262, No. 22, 1989, pp. 3102–3107.

[12] Aggazzotti, G., Fantuzzi, G., Tartoni, P. L., and Predieri, G., "Plasma Chloroform Concentrations in Swimmers Using Indoor Swimming Pools," *Archives of Environmental Health,* Vol. 45, No. 3, 1990, pp. 175–179.

[13] Peoples, A. J., Pfaffenberger, C. D., Shafik, T. M., and Enos H. F., "Determination of Volatile Purgeable Halogenated Hydrocarbons in Human Adipose Tissue and Blood Serum," *Bulletin of Environmental and Contaminant Toxicology,* Vol. 23, 1979, pp. 244–249.

[14] Fry, B. J., Taylor, T., and Hathway, D. E., "Pulmonary Elimination of Chloroform and Its Metabolite in Man," *Archives Internationales de Pharmacodynamie et de Therapie,* Vol. 196, 1972, pp. 98–111.

[15] Anonymous, "Death from Chloroform," *The Lancet,* Vol. 7, No. 4, New York, April 1848, p. 354.

[16] Gettler, A. O. and Blume, H., "Chloroform in the Brain, Lungs and Liver, Quantitative Recovery and Determination," *Archives of Pathology,* Vol. 11, 1931, pp. 554–559.

[17] Gonzales, T. A., Vance, M., Helpern, M., and Umberger, C. J., *Legal Medicine, Pathology and Toxicology,* 2nd ed., Appleton-Century-Crofts, Inc., New York, 1954, pp. 795–796.

[18] Schroeder, H. G., "Acute and Delayed Chloroform Poisoning," *British Journal of Anaesthesia,* Vol. 37, 1965, pp. 972–975.

[19] Bonnichsen, R. and Maehly, A. C., "Poisoning by Volatile Compounds II. Chlorinated Aliphatic Hydrocarbons," *Journal of Forensic Sciences,* Vol. 11, No. 3, 1966, pp. 414–427.

[20] Storms, W. W., "Chloroform Parties," *Journal of the American Medical Association,* Vol. 225, No. 2, July 9, 1973, p. 160.

[21] Timms, R. M. and Moser, K. M., "Toxicity Secondary to Intravenously Administered Chloroform in Humans," *Archives of Internal Medicine,* Vol. 135, Dec. 1975, pp. 1601–1603.

[22] Giusti, G. V. and Chiarotti, M., "Double 'Suicide' by Chloroform in a Pair of Twins," *Medicine Science and Law,* Vol. 21, No. 1, 1981, pp. 2–3.

[23] Hutchens, K. S. and Kung, M., "Experimentation with Chloroform," *American Journal of Medicine,* Vol. 78, April 1985, pp. 715–718.

[24] Allan, A. R., Blackmore, R. C., and Toseland, P. A., "A Chloroform Inhalation Fatality—An Unusual Asphyxiation," *Medicine Science and Law,* Vol. 28, No. 2, 1988, pp. 120–122.

[25] Kohr, R. M., "Suicide by Chloroform Ingestion Following Self-Mutilation," *The American Journal of Forensic Medicine and Pathology,* Vol. 11, No. 4, 1990, pp. 324–328.

[26] Rao, K. N., Virji, M. A., Moraca, M. A., Diven, W. F., Martin, T. G., and Schneider, S. M., "Role of Serum Markers for Liver Function and Liver Regeneration in the Management of Chloroform Poisoning," *Journal of Analytical Toxicology,* Vol. 17, No. 2, 1993, pp. 99–102.

[27] Maltby, J. R., "Criminal Poisoning with Anaesthetic Drugs: Murder, Manslaughter, or Not Guilty," *Forensic Science,* Vol. 6, 1975, pp. 91–108.

[28] Kaempe, B. and Dalgaard, J. B., "Chloroform As a Tool for Homicide," presented at the IXth International Congress of the European Association of Poison Control Centers and the European Meeting of the International Association of Forensic Toxicologists, Thessaloniki, Greece, August 24–27, 1980, pp. 422–430.

[29] McGee, M. B., Jejurikar, S. G., and VanBerkom, L. C., "A Double Homicide As a Result of Chloroform Poisoning," *Journal of Forensic Sciences,* Vol. 32, No. 5, Sept. 1987, pp. 1453–1459.

[30] McIntyre, J. W. R., "The Criminal Use of Chloroform Administered by Inhalation," *Medicine and Law,* Vol. 7, 1988, pp. 195–202.

[31] Anonymous, "Attempted Daring Robbery by the Aid of Chloroform," *The Lancet,* Vol. 2, No. 6, New York, Dec. 1850, p. 592.

[32] Anonymous, "Chloroform Amongst Thieves," *The Lancet,* Oct. 28, 1865, pp. 490–491.

[33] Morris, L. E., Frederickson, E. L., and Orth, O. S., "Differences in the Concentration of Chloroform in the Blood of Man and Dog During Anesthesia," *Journal of Pharmacology and Experimental Therapeutics,* Vol. 101, 1951, pp. 56–62.

Address requests for reprints or additional information to
Jay D. Dix, M.D.
Dept. of Pathology, Room M263
Univ. of Missouri - Columbia Hospital and Clinics
One Hospital Drive
Columbia, MO 65212