J Forensic Sci, Jan. 2003, Vol. 48, No. 1
Paper ID JFS2002227_481
Available online at: www.astm.org

James R. Gill,[1] M.D.; Lara B. Goldfeder,[1] M.D.; and Marina Stajic,[2] Ph.D.

The Happy Land Homicides: 87 Deaths Due to Smoke Inhalation

ABSTRACT: We reviewed all 87 deaths from the Happy Land Social Club fire. All deaths were due to smoke inhalation. The carboxyhemoglobin (COHb) concentrations ranged from 37 to 93% with a mean of 76.5%. The vast majority (97%) of the decedents had a COHb concentration over 50%. Cyanide blood concentrations ranged from 0 to 5.5 mg/L with a mean of 2.2 mg/L. Nine decedents had no cyanide detected, and seven had cyanide concentrations of less than 1 mg/L. Fewer than one third of the decedents had thermal injuries, and most were partial thickness burns involving less than 20% body surface area. Ethanol was detected in 72% of decedents with a range of 0.01 to 0.29 g% and a mean blood concentration of 0.11 g%. Cocaine or cannabinoid use was identified in 9% of the decedents. All decedents were visually identified, and all had soot in the airway extending to the major bronchi. Carboxyhemoglobin concentrations corresponded well with deaths from smoke inhalation. Cyanide concentrations did not correspond with the extent of smoke inhalation, and the role of cyanide in contributing to these deaths is doubtful. Hydrogen chloride inhalation, as evidenced by comparison of the pH of tracheal mucosa to controls, was not a factor.

KEYWORDS: forensic science, carbon monoxide, smoke inhalation, fatality, homicide

On March 25, 1990, 87 people died of smoke inhalation at the "Happy Land Social Club" in New York City. The fire was started by a 36-year-old man who earlier had been ejected from the unlicensed club after a verbal altercation with his former girlfriend, who worked at the club entrance. He went to a nearby service station, filled a plastic container with a dollar's worth of gasoline, and returned to the club. He threw the gasoline and lit matches into the only entrance of the two-story social club. Smoke quickly filled the first floor of the club, where 18 people were found dead. Smoke billowed up the narrow front and back staircases to a windowless dance floor on the second story. The first fire truck arrived minutes later, and the blaze was quickly extinguished. There were 69 fatalities on the second floor. Many were found around tables, and several had collapsed on the stairs. All of the decedents died in the building, and none were resuscitated. Five people, including the disc jockey from the second floor, escaped and survived. The club had no accessible second exit, fire alarms, exit signs, emergency lighting, or sprinklers.

In fewer than 36 h, the New York City Office of Chief Medical Examiner had identified 86 of 87 victims, performed 87 autopsies, prepared necessary documents, and was ready to release bodies to funeral directors. Since this was a group of healthy, young people, all of whom died in a matter of minutes from smoke inhalation from a common fire source, we reviewed these deaths to better understand the interpretation of the toxicologic parameters of fatal smoke inhalation.

The man who set the fire was found guilty of murder, arson, and assault (a total of 176 counts) and sentenced to 174 concurrent sentences of 25 years to life. Prior to September 11, 2001, this was the largest mass murder in New York City.

Materials and Methods

The Office of Chief Medical Examiner (OCME) investigates all unexpected, violent, and suspicious deaths in New York City. Toxicologic testing is performed as a part of all autopsies. All deaths from the Happy Land fire underwent autopsy.

Autopsy blood specimens were collected with the addition of sodium fluoride and stored at 4°C. All toxicologic testing was performed by the Forensic Toxicology Laboratory at the Office of Chief Medical Examiner. Carboxyhemoglobin (COHb) concentrations were determined by differential spectrophotometry using a CO oximeter. Cyanide concentrations were determined using spectrophotometry with a limit of detection of 0.2 mg/L. Ethanol concentrations were determined in blood using head space gas chromatography (limit of quantitation = 0.1 g%). Brain ethanol concentrations were determined in all cases with a positive blood ethanol finding. Urine specimens were tested for the presence of opiates, barbiturates, benzoylecgonine (BE), methadone, and phencyclidine by enzyme immunoassay. In instances where urine was not available, blood was tested for the presence of opiates, benzoylecgonine, and barbiturates using radioimmunoassay. Urine and/or blood were also screened for the presence of basic drugs (including cocaine) by gas chromatography with a nitrogen phosphorous detector (GC/NPD).

The acidity of the tracheal mucosa was tested by pH paper.

Results

All 87 people died of smoke inhalation. Carboxyhemoglobin (COHb) concentrations ranged from 37 to 93% with a mean of 76.5%. The vast majority (92%) of the decedents had COHb concentrations over 60% (Fig. 1, Table 1). Only 30% of the decedents

[1] New York City Office of Chief Medical Examiner and Department of Forensic Medicine, New York University School of Medicine, New York, NY.
[2] Forensic Toxicology Laboratory, New York City Office of Chief Medical Examiner and Department of Forensic Medicine, New York University School of Medicine, New York, NY.

Received 29 June 2002; and in revised form 17 Aug. 2002; accepted 17 Aug. 2002; published 11 Dec. 2002.

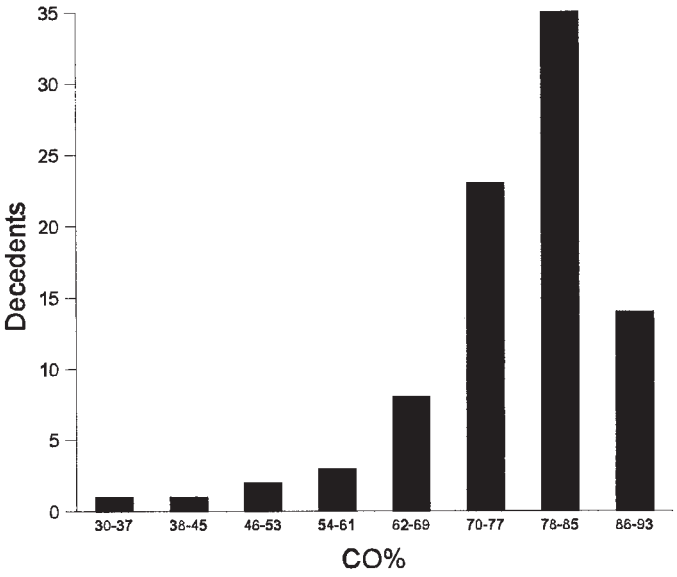

FIG. 1—*Carboxyhemoglobin concentrations.*

TABLE 1—*Postmortem blood toxicology results.*

	Detected	Concentration Range (mean)
Carboxyhemoglobin	87/87	37 to 93% (77%)
Cyanide, mg/L	78/87*	0.2 to 5.5 (2.4)
Ethanol, g%	63/87	0.01 to 0.29 (0.11)
Cocaine, mg/L	3/87	0.1 to 0.6 (0.3)
Benzoylecgonine, mg/L	7/87	0.2 to 0.8 (0.4)

* Nine decedents had no cyanide detected.

TABLE 2—*Thermal injuries*.

	n
Thermal burns	26/87 (30%)
<10% BSA*	9
10 to 20% BSA	14
20 to 50% BSA	3
>50% BSA	0

* BSA = Body surface area.

had thermal injuries, and most were partial thickness burns involving less than 20% body surface area (Table 2).

The three decedents with COHb concentrations of under 50% (37, 39, 48%) had blood ethanol concentrations of 0.14, 0.05, and 0.17 g% and no other detected drugs of abuse. The cyanide concentrations of these three decedents were 1.5, 2.5, and 0.2, respectively. The decedent with a COHb concentration of 48% was 58 years old, the eldest of the 87 people, and had no heart or lung disease at autopsy. The other two were healthy women, a 29-year-old and a 32-year-old.

Ethanol was detected in 72% of decedents with a range of 0.01 to 0.29 g% and a mean blood concentration of 0.11 g%. Of the 24 decedents with no ethanol and the 4 with blood ethanol concentrations of <0.02 g%, the average COHb concentration was 77%. The 15 decedents with blood ethanol concentrations of 0.15 g% or higher had an average COHb concentration of 73%. Drugs of abuse were detected in eight (9%) of the decedents: cocaine and benzoylecgonine—3; benzoylecgonine without cocaine—4; or cannabinoids—1.

Cyanide concentrations ranged from 0 to 5.5 mg/L with a mean of 2.2 mg/L. Nine decedents had no cyanide detected, and seven had cyanide concentrations of less than 1 mg/L. The range of cyanide concentrations tested on Day 1 and Day 2 were similar.

The age of the decedents ranged from 14 to 58 years with a mean of 25.9 years. The decedents were: 66% male, 85% Hispanic, 8% Black, and 7% White. All decedents were visually identified, and all had soot in the airway extending to the major bronchi. Pink lividity was described in 50 decedents. Although lividity was not appreciated in 34 deaths, the vast majority of the internal examinations described similar pink discoloration of the muscle, blood, and/or internal organs. The tracheal pH ranged from 5 to 7 (mean 6.4).

Discussion

Combustion produces numerous toxins (1,2). In fire deaths, carbon monoxide and cyanide are two intoxicants commonly used to assist in determining if the death was due to smoke inhalation. Several studies have examined cyanide and carbon monoxide concentrations in smoke inhalation deaths (2–6). These studies were limited by numerous confounding factors. Attempted resuscitation, duration of exposure, underlying natural disease, and the type of fire may affect the concentrations of toxins detected at the hospital or autopsy. Studies did not differentiate the toxicologic results of those who did and did not undergo resuscitative efforts (2,4). Underlying natural disease or fire types (e.g., automobile versus residence) also were not always considered (3,5,6).

These limitations are inevitable when attempts are made to examine a large number of decedents with disparate circumstances and histories. The Happy Land fatalities provide the unique opportunity to examine the effects of these toxins on a homogeneous healthy young adult population that was exposed to the same smoke-filled environment. All of the decedents died of smoke inhalation within minutes without resuscitative attempts.

The cause of death was determined by three major findings. First, all 87 of the decedents had copious soot deposits from the nasopharynx to the major bronchi. Second, all had markedly elevated COHb concentrations. Third, none had another disease or injury incompatible with life. Thermal burns were described in 30% of the decedents; however, most involved less than 20% of the body surface area and were partial-thickness.

The COHb concentrations demonstrate the range that may be detected in smoke inhalation deaths in healthy young adults. With the exception of three people, all the decedents had COHb concentrations over 50%. This confirms that most healthy people who succumb to smoke inhalation will have a COHb concentration of greater than 50%. A COHb concentration of 50% is the physiologic equivalent of quickly removing half of the oxygen-carrying capacity of the blood (i.e., decreasing the hematocrit by 50%). If this loss of blood were to occur chronically, the body may be able to adjust, but the rapid loss of this oxygen-carrying capacity, particularly at a time where there may be strenuous demands on the body, results in grave consequences.

Smoke typically contains from 0.1% (1000 ppm) to 10% (100,000 ppm) carbon monoxide. Under strenuous exertion, the increase in carbon monoxide saturation has been calculated at 15% carboxyhemoglobin per minute in a 1% environment (10,000 ppm) to 75% carboxyhemoglobin per minute in a 5% environment. In a 10% carbon monoxide environment, a lethal concentration will occur in under 30 s (7). Some have suggested that due to the rapidity of some smoke inhalation deaths, cyanide "may play a significant role" in fire deaths (8). High concentrations of carbon monoxide, however, are quite capable of causing a very rapid death. The carbon monoxide

and cyanide concentrations of an individual fire are, however, usually unknown because it is not feasible to measure it at the time. In the Happy Land fire, some decedents attempted to escape as evidenced by the group of bodies on the stairs. Others appeared to have simply succumbed at the table at which they had been sitting.

Interpretations of COHb concentrations in fire deaths may be complicated by three factors. The first is resuscitation attempts. Resuscitation with supplemental oxygen will decrease the COHb concentration more quickly than if the person continued breathing normal room air. Therefore the COHb concentration obtained at autopsy or at admission to the hospital will not be an accurate measurement of what the maximum extent of smoke inhalation (and carbon monoxide poisoning) had been before resuscitation commenced. For example, if a person has a 30% COHb concentration in the hospital after receiving 100% oxygen for about an hour, his/her starting COHb concentration was probably about 60%. Various formulas may be used to extrapolate back to the "original" COHb concentration, which may have prognostic as well as cause of death implications (9–11).

Secondly, people who die quickly in flash fires may not have an elevated COHb concentration. The rapidity of death, efficiency of the combustion, and/or amount of ventilation may explain how fatalities occur from flash fires without concomitant COHb concentration elevations (12,13).

Thirdly, underlying disease (e.g., coronary artery atherosclerosis) may make a person more susceptible to smoke inhalation either by earlier incapacitation (2) or by direct cardiac ischemia. A person with pulmonary or cardiac disease may die with a carboxyhemoglobin concentration that an otherwise healthy person may survive. The fact that none of these three confounding factors occurred in the Happy Land deaths makes this a unique and valuable population for analysis (5,14).

Acute alcohol intoxication is a common finding in fire victims (3,15). Those impaired by alcohol have an increased risk of death in fires (15). Acute alcohol intoxication plays a role in the circumstances of starting fatal fires (15), but does it make a pathophysiologic contribution to the death? Birky and Clarke concluded that alcohol intoxication does not have any effect on the concentration of COHb associated with death (2). Our findings support this conclusion with similar mean COHb concentrations in the intoxicated (BAC $>$ 0.14 g%) and sober groups (73 versus 77%).

Postmortem concentrations of cyanide in fire victims have been previously studied, and the interpretations of these concentrations are varied (2,3,5,8). Cyanide may be detected at autopsy in people who die of natural deaths unrelated to fires or intoxications. These "normal" postmortem cyanide concentrations have ranged from 0.2 to 0.43 mg/L in New York City. Concentrations up to 2.2 mg/L have been reported in chronically exposed factory workers in the electroplating industry (16). The key questions regarding cyanide are: is cyanide a reliable indicator of smoke inhalation and, if so, does it also contribute to death? Since all decedents in this instance died from smoke inhalation in the same smoke-filled environment, if cyanide were a reliable indicator of smoke inhalation, all the decedents would have had detectable cyanide concentrations. The fact that nine of the decedents had no cyanide detected and another seven had less than 1 mg/L demonstrate that cyanide did not play any role in several of these deaths. Since all of the blood samples were collected and tested in the same manner, the lack of cyanide is unlikely to be secondary to postmortem artifacts. In addition, although some autopsies were done up to 36 h after death, there was no difference in the range of cyanide concentrations detected from samples collected on the day of death compared to the following day.

The cause of all deaths was smoke inhalation, and the manner was homicide. We do not certify smoke inhalation deaths with a cause of death of carbon monoxide or cyanide poisoning because there are many other toxins in smoke (1,2). It would be arbitrary to ignore these other toxins and to certify the deaths as solely due to carbon monoxide poisoning simply because that is the toxin for which we test. In smoke inhalation deaths, soot deposition in the bronchi and carbon monoxide testing are helpful and reliable tests as long as the various complicating factors are considered.

Carbon monoxide testing is an easy, fast, and reliable method for determining the extent of smoke inhalation. Pathologically, as clinically, carbon monoxide concentrations correspond well with morbidity and mortality results. Cyanide concentrations, however, do not correspond with the extent of smoke inhalation, and the role of cyanide in contributing to these deaths is doubtful. By certifying the death as due to smoke inhalation, as opposed to carbon monoxide or cyanide poisoning, the cyanide issue becomes moot. In fire deaths, cyanide concentrations do not provide helpful, interpretable information and need not be performed on suspected smoke inhalation deaths. Hydrogen chloride inhalation, as judged by comparison of the pH of tracheal mucosa to controls, was not a factor.

References

1. Terrill JB, Montgomery RR, Reinhardt CF. Toxic gases from fires. Science 1978;200(4348):1343–7.
2. Birky MM, Clarke FB. Inhalation of toxic products from fires. Bull NY Acad Med 1981;57(10):997–1013.
3. Wetherall HR. The occurrence of cyanide in the blood of fire victims. J of Forensic Sci 1966;11:167–73.
4. Pane GA, Mohler SR, Hamilton GC. The Cincinnati DC-9 experience: lessons in aircraft and airport safety. Aviat Space Environ Med 1985;56 (5):457–61.
5. Lundquist P, Rammer L, Sorbo B. The role of hydrogen cyanide and carbon monoxide in fire casualties: a prospective study. Forensic Sci Int 1989;43(1):9–14.
6. Birky M, Malek D, Paabo M. Study of biological samples obtained from victims of MGM Grand Hotel fire. J Anal Toxicol 1983;7(6):265–71.
7. Stewart RD, Stewart RS, Stamm W, Seelen RP. Rapid estimation of carboxyhemoglobin level in fire fighters. JAMA 1976;235(4):390–2.
8. Noguchi TT, Eng JJ, Klatt EC. Significance of cyanide in medicolegal investigations involving fires. Am J Forensic Med Pathol 1988;9(4): 304–9.
9. Saffle JR. The 1942 fire at Boston's Cocoanut Grove nightclub. Am J Surg. 1993;166(6):581–91.
10. Ryan CM, Schoenfeld DA, Thorpe WP, Sheridan RL, Cassem EH, Tompkins RG. Objective estimates of the probability of death from fire burns. NEJM 1998;338:362–6.
11. Marshall S, Runyan C, Bangdiwala S. Fatal residential fires: who dies and who survives? JAMA 1998;279:1633–37.
12. Hirsch CS, Adelson L. Absence of carboxyhemoglobin in flash fire victims. JAMA 1969;210:2279–80.
13. Hirsch CS, Bost RO, Gerber SR, Cowan ME, Adelson L, Sunshine I. Carboxyhemoglobin concentrations in flash fire victims. AJCP 1977;68: 317–20.
14. Ruszkiewicz A, de Boer B, Robertson S. Unusual presentation of death due to carbon monoxide poisoning. A report of two cases. Am J Forensic Med Pathol 1997;18(2):181–4.
15. Marshall SW, Runyan CW, Bangdiwala SI, Linzer MA, Sacks JJ, Butts JD. Fatal residential fires: who dies and who survives? JAMA 1998;279 (20):1633–7.
16. Chandra H, Gupta BN, Bhargava SK, Clerk SH, Mahendra PN. Chronic cyanide exposure—A biochemical and industrial hygiene study. J Anal Toxicol 1980;4(4):161–5.

Additional information and reprint requests:
James R. Gill, M.D.
Deputy Chief Medical Examiner, Bronx County
Office of Chief Medical Examiner
520 First Avenue
New York, NY 10016