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Analysis of Carboxyhemoglobin and Cyanide in Blood from Victims of the Dupont Plaza Hotel Fire in Puerto Rico

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ABSTRACT: Ninety-seven people died from a fire that occurred in the Dupont Plaza Hotel in Puerto Rico on 31 Dec. 1986. All, except four who died later in the hospital, were found dead at the scene. All of the fatalities at the hotel (except for eight) were burned beyond recognition. Blood from seventy-eight of the victims was screened for carboxyhemoglobin at the Institute for Forensic Sciences in Puerto Rico and was then sent to the National Institute of Standards and Technology, Gaithersburg, Maryland, for analysis of carboxyhemoglobin and cyanide concentrations. The blood data indicated that carbon monoxide and hydrogen cyanide, singly or combined, were probably not responsible for the majority of the deaths that occurred in the badly burned victims. On the other hand, the significantly higher carboxyhemoglobin in the nonburned victims indicated that carbon monoxide alone or combined with hydrogen cyanide probably played a major role in the cause of their deaths.

KEYWORDS: toxicology, carbon monoxide, carboxyhemoglobin, fires, blood analysis, Dupont Plaza Hotel, fire victims, hydrogen cyanide

A fire that occurred during the afternoon of 31 Dec. 1986 in the Dupont Plaza Hotel in San Juan, Puerto Rico, claimed 97 lives and injured more than 140 individuals [*1*]. This fire was one of the worst hotel fires to have occurred in this century, comparable to the MGM Grand Hotel fire in Las Vegas, Nevada, which claimed 85 lives in 1980,

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and the Winecoff Hotel fire in 1946, which took 119 lives [2]. Most of the articles and reports [1–4] that have been written about the Dupont Plaza Hotel fire have described the findings of the investigation by the National Fire Protection Association, who worked in cooperation with the U.S. Bureau of Alcohol, Tobacco and Firearms and Puerto Rican authorities. They found that the fire was of incendiary origin and was started in a stack of new furniture that was stored in corrugated boxes in a ballroom (Fig. 1). This furniture, which consisted of dressers (constructed of wood and particle board) and sofa beds containing foam mattresses, occupied a volume of approximately 5.5 by 9.4 by 1.8 m (18 by 31 by 6 ft). This initial fuel load plus other materials which may have become involved in the fire were sufficient to cause the ballroom to undergo flashover and produce a flame front that rapidly spread throughout the lobby and casino area. An engineering analysis of the early stages of this fire estimated that the flame front spread through the 437-m^2 (4700-ft^2) casino (where most of the casualties were found) in 20 to 30 s [5]. Both exits from the casino became blocked when the lobby area also filled with smoke.

Most of the victims were burned beyond recognition and were found in the casino area; the 8 fatalities who were not burned were discovered in various other locations (Fig. 1). The Institute of Forensic Sciences (IFS) performed a preliminary screening for carboxyhemoglobin (COHb) in blood samples from 78 victims and then sent the samples to the National Bureau of Standards (NBS), now the National Institute of Standards and Technology (NIST), where both COHb and blood cyanide were analyzed. The IFS did not analyze the blood for cyanide. The objective of this study was to determine whether exposure to these toxic gases might have been sufficient to cause the fatalities. Neither NIST nor IFS was aware of the other laboratory's results until the initial analyses were complete.

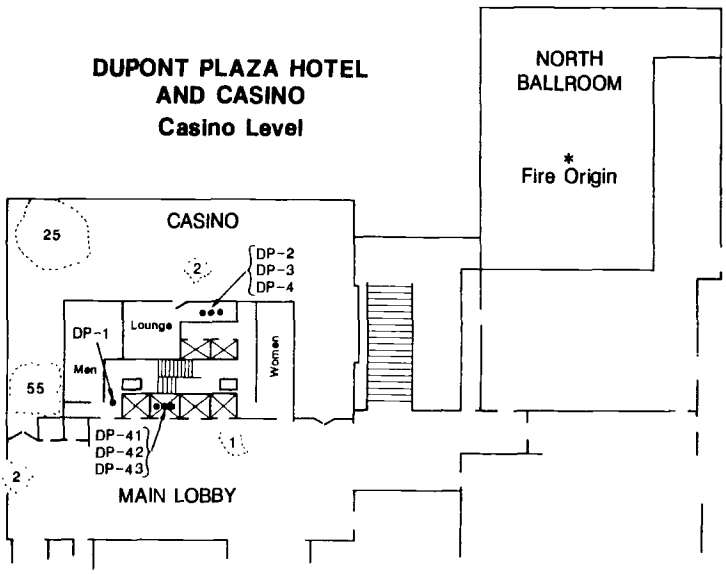

FIG. 1—*Location and number of burned and nonburned victims of Dupont Plaza Hotel fire. Numbers surrounded by dotted circles indicate number of burned victims found at that location. Solid circles designated by DP numbers indicate location of nonburned individuals. In addition, one nonburned individual (DP-19) was found in a fourth-floor bedroom.*

Methods

Methods at NIST

Blood—Seventy-eight samples of blood, packed in dry ice, were sent by Federal Express from Puerto Rico to NBS and arrived on 26 March 1987. This blood, which contained the anticoagulant sodium fluoride (NaF), had been stored in a refrigerator before being sent to NIST. The samples were in screw-top vials sealed with rubber septa. Since the blood samples were found to be frozen when the carton was opened, they were immediately placed in a freezer and stored at −20°C. On the day of analysis, the thawed blood was stored in an ice bath. In some cases, the samples were thawed and refrozen several times, as, for example, when the analyses of COHb and cyanide were not performed on the same day and when the blood was reanalyzed at a later date to determine the effect of long storage. The blood samples received at NIST were in poor condition (that is, they contained multiple clots and debris).

Except for the first few samples (see Table 1), all samples in their original sealed containers were thawed and sonicated in an ultrasonic bath at room temperature for 15 min to reduce the size of the clots before COHb analysis. Immediately before COHb analysis, aliquots of the sonicated samples were taken with a syringe and filtered with a nylon filter which attached securely to the syringe outlet. The sonication and filtering procedures were found necessary to prevent the clots and debris from entering the CO-Oximeter instrument used to measure the COHb. There was also some concern that filtration without sonication would produce aliquots that were not truly representative of the total sample. In the cyanide analysis, sonication, but not filtration, was performed since minor particulate matter did not interfere with the analysis. Measurement repeatability is indicated by the standard deviation values provided in Table 1.

Control Blood—Blood from three NIST laboratory volunteers was tested in the fresh state and then stored frozen and treated in the same manner as the experimental blood. Two of the control blood samples (Con 1 and 2) contained the anticoagulant ethylenediaminetetraacetate (EDTA), and Con 3 had the anticoagulant NaF (Table 1).

Carboxyhemoglobin Determination—Carboxyhemoglobin was measured with a CO-Oximeter IL-282[5] (Model 282, Instrumentation Laboratory Inc., Lexington, Massachusetts). The instrument was set for human blood and calibrated daily.

Blood Cyanide Determination—The measurements for blood cyanide were conducted using a gas chromatographic head-space analysis technique. These analyses were performed with a Perkin-Elmer 3920 gas chromatograph (GC) equipped with a nitrogen/phosphorus (N/P) detector, a Porapak Q packed column, and an HS-6 semiautomatic head-space sampler. Each cyanide sample was prepared by first pipetting 250 μL of blood into a glass head-space vial containing 50 μL of a sodium nitrate solution (8 mg/mL) and then sealing the vial with a polytetrafluoroethylene (PTFE)-lined septum crimp cap. The samples, acidified by injecting 50 μL of concentrated phosphoric acid through the septum top, were allowed to equilibrate in the heated (60°C) HS-6 head-space sampler for at least 30 min. Samples of the head-space were then automatically taken and injected into the GC. Controls containing varying concentrations of sodium cyanide (NaCN) in 0.1*N* sodium hydroxide (NaOH) were analyzed between experimental samples.

[5]Certain commercial equipment, instruments, or materials are identified in this paper to specify adequately the experimental procedure. In no case does such identification imply recommendation or endorsement by the National Institute of Standards and Technology, nor does it imply that the equipment or material identified is necessarily the best available for the purpose.

TABLE 1—*Dupont Plaza Hotel fire blood data.*

	IFS Results		National Institute of Standards and Technology Results								Time Tested	
Sample No.	COHb, %	Mean COHb, %	n^a	Standard Deviation, %	Mean Cyanide, μg/mL	n	Standard Deviation, μg/mL	Fresh or Frozen	Sonicated	Filter	COHb	CN
Con[b]1	. . .	1.6	2	. . .	. . .	. . .	. . .	fresh	no	no	90 min	. . .
Con 2	. . .	1.4	2	. . .	0.7	1	. . .	fresh	no	no	90 min	. . .
Con 1	. . .	1.4	2	. . .	0.5	3	0.04	frozen	no	no	24 h	3/27
Con 2	. . .	1.1	2	. . .	0.5	3	0.09	frozen	no	no	24 h	3/27
Con 3	. . .	2.8	2	. . .	0.4	3	0.05	fresh	no	yes	NR	3/30
DP-1[c]	80	80.2	3	0.2	1.0	2	. . .	frozen	no	no	3/27	3/31
DP-2[c]	90	40.3	3	1.2	0.5	2	. . .	frozen	no	no	3/27	3/31
DP-3[c]	80	67.2	3	2.1	2.0	3	0.35	frozen	no	yes	3/27	3/31
DP-4[c]	80	31.5	3	0.2	2.0	3	0.58	frozen	no	yes	3/31	3/31
DP-5	15	22.2	3	0.8	0.7	3	0.36	frozen	no	yes	3/31	3/31
DP-6	60	13.4	3	1.6	2.9	3	0.14	frozen	no	yes	3/31	3/31
DP-7	45	13.2	3	1.1	0.6	3	0.14	frozen	no	yes	3/31	3/31
DP-9	30	13.3	3	0.6	2.5	3	0.55	frozen	no	yes	3/31	4/7
DP-10	30	8.9	3	0.4	0.7	3	0.05	frozen	no	yes	3/31	4/7
DP-11	25	2.6	3	0.3	3.9	3	0.12	frozen	no	yes	3/31	4/7
DP-12	15	1.9	3	0.6	0.5	3	0.03	frozen	yes	yes	4/8	4/8
DP-13	50	21.0	3	2.4	1.8	3	0.08	frozen	yes	yes	4/8	4/8
DP-14	25	11.6	3	0.2	1.1	3	0.13	frozen	yes	yes	4/8	4/8
DP-15	60	8.9	3	0.1	1.0	3	0.05	frozen	yes	yes	4/8	4/8
DP-16	20	0.1	2	. . .	2.5	3	0.13	frozen	yes	yes	4/8	4/8
DP-17	35	14.0	3	0.5	0.6	3	0.13	frozen	yes	yes	4/8	4/8
DP-18	30	18.9	3	0.6	0.9	3	0.08	frozen	yes	yes	4/9	4/15
DP-19[c]	60	71.5	3	0.2	0.8	3	0.05	frozen	yes	yes	4/9	4/15
DP-21	20	16.6	3	0.4	1.0	3	0.24	frozen	yes	yes	4/9	4/15
DP-22	15	0.6	4	0.7	2.6	3	0.38	frozen	yes	yes	4/9	4/15
DP-23	40	5.3	3	0.3	2.1	3	0.03	frozen	yes	yes	4/9	4/15
DP-24	40	9.2	3	0.1	0.6	3	0.02	frozen	yes	yes	4/9	4/15
DP-25	30	19.4	3	0.1	1.0	3	0.15	frozen	yes	yes	4/10	4/15
DP-26	40	20.2	3	0.5	1.1	3	0.01	frozen	yes	yes	4/10	4/29
DP-27	35	13.3	3	0.1	1.1	3	0.07	frozen	yes	yes	4/10	4/29
DP-28	40	22.6	3	2.1	0.5	3	0.05	frozen	yes	yes	4/10	4/29

DP-29[d]	30	13.0	3	0.2	11.4	3	1.72	frozen	yes	yes	4/10	4/29
DP/30[e]	35	18.1	3	0.3	1.0	3	0.06	frozen	yes	yes	4/10	4/29
DP-31	35	13.5	3	0.8	2.3	3	0.05	frozen	yes	yes	4/10	4/29
DP-32	10	15.2	3	0.1	1.0	3	0.02	frozen	yes	yes	4/10	4/30
DP-33	20	40.4	3	0.9	0.5	3	0.06	frozen	yes	yes	4/10	4/30
DP-34	40	57.4	3	0.2	0.4	3	0.02	frozen	yes	yes	4/13	4/30
DP-35	40	53.4	3	0.4	0.7	3	0.01	frozen	yes	yes	4/13	4/30
DP-36	40	39.5	3	0.2	1.8	3	0.04	frozen	yes	yes	4/13	4/30
DP-37	10	30.4	3	3.8	0.8	3	0.01	frozen	yes	yes	4/13	4/30
DP-38	40	54.9	4	1.6	0.9	3	0.09	frozen	yes	yes	4/13	4/30
DP-41[c]	90	65.3	3	0.4	1.2	3	0.24	frozen	yes	yes	4/13	4/30
DP-42[c]	. . .	56.8	3	0.6	0.3	3	0.09	frozen	yes	yes	4/14	5/4
DP-43[c]	. . .	49.0	3	0.3	0.8	3	0.05	frozen	yes	yes	4/14	5/4
DP-44	40	24.6	3	0.8	0.9	3	0.06	frozen	yes	yes	4/14	5/4
DP-45	35	23.4	3	0.3	0.7	3	0.06	frozen	yes	yes	4/14	5/4
DP-46	40	22.1	3	1.1	0.4	3	0.04	frozen	yes	yes	4/14	5/5
DP-47	30	16.6	3	1.4	1.4	3	0.21	frozen	yes	yes	4/14	5/5
DP-49	40	23.4	4	0.4	2.3	2	. . .	frozen	yes	yes	4/14	5/5
DP-50[f]	35	. . .	. . .	. . .	1.3	3	0.18	frozen	yes	yes	4/14	5/5
DP-51	40	46.5	4	0.4	1.9	3	0.15	frozen	yes	yes	4/14	5/5
DP-52[g]	15	. . .	. . .	. . .	. . .	. . .	. . .	frozen	yes	yes	4/15	. . .
DP-53	30	34.6	3	0.1	0.8	3	0.07	frozen	yes	yes	4/15	5/5
DP-55	15	24.6	3	0.4	2.2	3	0.21	frozen	yes	yes	4/15	5/5
DP-56[h]	20	10.6	3	0.3	2.4	3	0.16	frozen	yes	yes	4/15	5/11
DP-57[h]	30	16.5	3	0.1	0.8	3	0.08	frozen	yes	yes	4/15	5/11
DP-58[h]	30	26.7	3	0.3	1.7	3	0.07	frozen	yes	yes	4/15	5/11
DP-59[i]	30	. . .	. . .	. . .	1.3	1	—	frozen	yes	yes	4/15	5/11
DP-60	50	59.7	3	0.6	1.4	3	0.02	frozen	yes	yes	4/15	5/11
DP-61	30	42.4	3	0.1	0.6	3	0.10	frozen	yes	yes	4/16	5/11
DP-62	50	38.5	3	0.3	0.4	3	0.04	frozen	yes	yes	4/15	5/13
DP-63	30	17.1	3	0.4	1.3	3	0.21	frozen	yes	yes	4/1	5/13
DP-64	20	19.9	3	0.2	1.2	3	0.10	frozen	yes	yes	4/16	5/13
DP-66	40	19.2	3	0.2	0.5	3	0.05	frozen	yes	yes	4/16	5/14
DP-67[h]	45	18.9	3	1.1	1.7	3	0.08	frozen	yes	yes	4/16	5/14
DP-68[j]	60	. . .	. . .	. . .	1.8	2	. . .	frozen	yes	yes	4/16	5/14
DP-69	40	17.4	3	1.0	0.7	3	0.04	frozen	yes	yes	4/16	5/14
DP-70	35	18.9	3	0.5	0.7	3	0.06	frozen	yes	yes	4/17	5/15
DP-73	80	27.9	3	0.1	1.2	3	0.09	frozen	yes	yes	4/17	5/15
DP-74	45	9.1	4	2.4	1.9	3	0.16	frozen	yes	yes	4/17	5/15

TABLE 1—*Continued.*

Sample No.	IFS Results	National Institute of Standards and Technology Results									Time Tested	
	COHb, %	Mean COHb, %	n^a	Standard Deviation, %	Mean Cyanide, μg/mL	n	Standard Deviation, μg/mL	Fresh or Frozen	Sonicated	Filter	COHb	CN
DP-82	35	14.0	3	0.7	0.8	3	0.10	frozen	yes	yes	4/17	5/15
DP-83[k]	40	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
DP-85	30	13.4	3	0.1	0.4	3	0.04	frozen	yes	yes	4/17	5/15
DP-86	30	14.0	3	0.3	1.6	3	0.39	frozen	yes	yes	4/17	5/15
DP-91	40	17.7	4	3.0	0.8	3	0.15	frozen	yes	yes	4/20	5/15
DP-92	40	15.7	3	0.1	1.1	3	0.10	frozen	yes	yes	4/20	5/15
DP-93[k]	35	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
DP-94	15	5.7	3	1.6	0.7	3	0.02	frozen	yes	yes	4/20	5/18
DP-95	35	25.0	3	0.6	1.4	3	0.21	frozen	yes	yes	4/20	5/18
DP-201	40	44.1	3	0.7	1.4	3	0.23	frozen	yes	yes	4/20	5/18
DP-203	40	67.2	3	1.1	2.5	3	0.21	frozen	yes	yes	4/20	5/18
DP-204	40	31.4	3	0.2	0.8	3	0.20	frozen	yes	yes	4/21	5/18
DP-205	41	12.2	3	0.1	0.8	3	0.07	frozen	yes	yes	4/21	5/18
DP-206[l]	15	. . .	. . .	. . .	1.0	3	0.17	frozen	yes	yes	4/21	5/18
DP-207	50	66.1	3	0.4	2.2	2	. . .	frozen	yes	yes	4/21	5/18
Con 1		−0.5	3	0.7	1.1	2	. . .	frozen	yes	yes	26 day	9/11
Con 2		−0.4	3	0.2	1.0	3	0.2	frozen	yes	yes	26 day	9/11
Con 3		3.1	4	0.1	0.7	3	0.1	frozen	yes	yes	22 day	9/11

[a]Number of samples in mean.
[b]Control blood.
[c]Nonburned victims.
[d]Hairline crack in vial. Thawed and transferred without contamination.
[e]DP-30 had a cracked vial bottom, possible contamination with water.
[f]Bottom of vial broken; very viscous sample transferred to new vial. NIST unable to obtain consistent results for COHb.
[g]Sample very pale pink. NIST unable to get reasonable values.
[h]Broken vial. Thawed and transferred contents to new vial without problem.
[i]Green tinted sample. NIST unable to obtain reasonable COHb value.
[j]Highly clotted sample. NIST unable to obtain consistent COHb results.
[k]This sample was not received by NIST.
[l]NIST unable to obtain reliable COHb results.

Carboxyhemoglobin Determination at IFS

Carboxyhemoglobin was screened in blood that had been stored less than one month. A microdiffusion technique [*6*], in which 10% silver nitrate ($AgNO_3$) is added to the blood sample to release the carbon monoxide (CO) from the carboxyhemoglobin, was used. In this technique, the released CO diffuses into the center well (containing palladium chloride, $PdCl_2$) of a Conway cell and reduces the $PdCl_2$ to metallic palladium. The gray to black color of the reduced metallic palladium (determined by visual comparison with standards) gives a fairly good estimate of the released CO up to about 40%. For values above 40%, the sample size has to be reduced to half. The sensitivity of this method is about 5%. To convert to percent COHb saturation, a knowledge of the total hemoglobin content of the blood is required. This technique is restricted to COHb concentrations above 10% and is much more reliable when the blood is in good condition. Confirmation of the values by the more specific CO-Oximeter at IFS was not possible because of equipment failure.

Results

Carboxyhemoglobin

The results on the COHb determined by the IFS screening and those determined at NIST are presented in Table 1. This table also gives information on the treatment of the blood at NIST before the blood measurements and on the times and dates of the NIST analyses. In general, the results of both laboratories showed that substantive concentrations of COHb were detected in only a few cases. For example, by comparison of the results with 50% COHb (quoted frequently as the usual human lethal level), NIST and the IFS investigators found only 11/73 (15%) and 14/78 (18%) victims, respectively, with this level or greater (Table 1 and Fig. 2). All the other victims had lower than 50% COHb in their blood.

According to the autopsy findings, there were eight people who were found dead at the hotel without burns. In general, the range of COHb values were higher in the

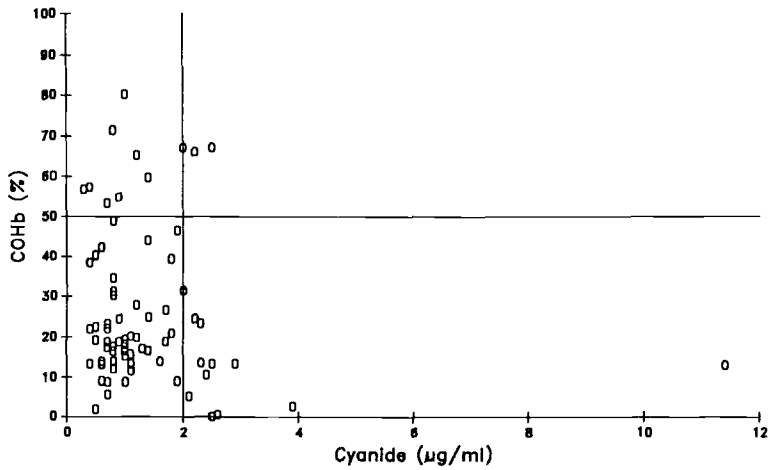

FIG. 2—*NIST carboxyhemoglobin and blood cyanide results from victims of Dupont Plaza Hotel fire.*

nonburned fatalities than in the victims who endured substantial burns. The IFS results showed that the nonburned victims had blood COHb concentrations ranging from 60 to 90% (n = 6) and the NIST results showed COHb levels ranging from 31.5 to 80.2% (n = 8). In the burned victims, the COHb values found by IFS ranged from 10 to 80 (n = 72, only 8 were ≥50%) and those found by NIST ranged from 0.1 to 67.2 (n = 65, only 6 were ≥50%). These results indicate that, while the majority of all the victims had less than lethal concentrations of COHb, the nonburned victims did have COHb levels much higher than the burned victims and generally above 50%.

The individual COHb concentrations determined by the IFS investigators differed from those found by NIST. These differences were not consistent: in 17 cases, the NIST values were higher, whereas in 52 cases, the IFS values were higher. The blood COHb analyses on a few samples, where sufficient blood was available, were repeated by NIST approximately 1 month and 4 months after the initial NIST analysis. In all cases, the values dropped. For example, shortly after receiving the blood, NIST determined the percent COHb values for DP-19 and DP-55 to be 71.5 ± 0.2 and 24.6 ± 0.4, respectively. Approximately 4 months later, these values were 59.6 ± 1.4% and 19.9 ± 0.9%, 17 and 19% decreases, respectively. Consistent with these findings, repeat analysis after one month showed that the NIST control values decreased from an average of 1.7% COHb to essentially 0 (see Con samples 1, 2, and 3 at beginning and end of Table 1). Therefore, the blood COHb concentrations detected in this study by NIST are probably lower than the values at the time of death.

Blood Cyanide

Table 1 also gives the mean blood cyanide levels as determined by NIST; the IFS investigators did not examine the blood for cyanide. Examination of the nonburned victims (n = 8) showed a range of blood cyanide levels from 0.3 to 2.0 μg/mL, while that in the burned victims ranged from 0.4 to 3.9 μg/mL. (One extremely high concentration of blood cyanide of 11.4 μg/mL found in a burned victim was not included in this calculation.) These results indicate no significant difference in blood cyanide between the burned and nonburned victims.

Since some information indicated that the blood cyanide concentrations may increase upon prolonged storage [7], the cyanide concentrations in a number of blood samples were reexamined after varying lengths of time (Table 2). For the control samples, a period of time at least as long as the time between the fire and the receipt of the samples by NIST was allowed between measurements. In 18 out of 25 control and experimental cases, the change that occurred was an increase in the mean cyanide level. In 7 out of 20 cases where there was enough data to determine the standard deviation, the change in blood cyanide as a result of storage exceeded 2 standard deviations of the mean.

A statistical analysis of the results indicated that under these storage conditions, there was a significant increase in blood cyanide over time. Since the victims' blood samples were frozen for various lengths of time before the first cyanide analysis, the detected blood cyanide values are probably higher than the concentrations in the blood at the time of death.

There were insufficient data to model the trend in blood cyanide concentrations over time.

Discussion

Effect of CO Exposure

There were two distinct sets of fatalities in the Dupont Plaza Hotel fire—the majority who were very badly burned and eight others who suffered no burns. The burned victims

were discovered primarily in the casino, whereas the nonburned victims were found in various other locations: one (DP-1) was found in a bathroom, three (DP-2, 3, and 4) were found in a lounge connected to the casino, three (DP-41, 42, and 43) were in an elevator, and one (DP-19) was found in a fourth-floor bedroom. The burned fatalities had lower concentrations of COHb than the nonburned victims. The mean IFS COHb value for the burned victims was 34% ± 13 (n = 72) and that found by NIST was 23% ± 16 (n = 65). With regard to the nonburned victims, the mean IFS COHb value was 80% ± a standard deviation of 11% (n = 6) and the mean NIST COHb value was 58% ± a standard deviation of 17% (n = 8). Therefore, the finding by both NIST and IFS that the nonburned victims had higher COHb values (generally above 50%) than the burned victims indicates that inhalation of toxic gases (especially CO) played a greater role in the deaths of those without burns than those with burns.

As noted earlier, the COHb data from NIST and IFS differ (that is, the largest number of people were found by NIST in the 10 to 19% COHb group, whereas the IFS investigators found the largest number of people in the 30 to 49% category) (Table 3). The differences between these two sets of data may be due to the aging or poor condition of the blood samples, the use of different sample preparation or analytical techniques. The aging problem is discussed below.

Effect of HCN Exposure

Previous studies on the lethal concentrations of cyanide in rats at NIST found that, except in 1-min exposures, blood cyanide concentrations of 2 μg/mL were sufficient to kill the animals [*8*]. If one assumes that this level is also the lethal level of cyanide in humans, then 82% of the victims of the Dupont Plaza Hotel fire had insufficient levels of cyanide to be lethal (Table 4 and Fig. 2). Some very limited data indicate that the lethal blood level in humans is about 5 μg/mL [*6,9*]. In this case, only one victim from this fire would have had more than the lethal blood concentration. These results are similar to the blood cyanide results found in the MGM Grand Hotel fire in which 97% of the victims had less than 2 μg/mL of cyanide in their blood (Table 4) [*10*].

The blood cyanide data did not show the same distinction between the burned and nonburned victims as the COHb values. For example, the mean and standard deviation of the nonburned victims was 1.1 ± 0.6 μg/mL (n = 8), whereas that of the burned victims was 1.3 ± 0.7 μg/mL (n = 68). (One extremely high concentration of blood cyanide of 11.4 μg/mL found in a burned victim was not included in the calculation of the mean.)

Effect of Combined CO and HCN Exposures

Although in the majority of cases, the individual concentrations of CO or HCN appear to be below the lethal levels, the possibility exists that sublethal concentrations of these gases may in combination be lethal. Since both CO and HCN greatly reduce the available oxygen at the cellular level, it is reasonable to expect that they may be acting in concert. The brain and heart with a high oxygen demand are especially vulnerable.

Prior work at NIST [*11*] has indicated that acute inhalation exposures to combinations of atmospheric CO and HCN resulted in an additive toxicity, such that some percentage (not zero or 100%) of the test rats would die if

$$\frac{[\mathrm{CO}]}{\mathrm{LC}_{50}\mathrm{CO}} + \frac{[\mathrm{HCN}]}{\mathrm{LC}_{50}\mathrm{HCN}} \approx 1(0.88 - 1.26) \tag{1}$$

where the numbers in brackets are the atmospheric concentrations of the gases, LC_{50} is the calculated atmospheric concentration which causes half the subjects to die in a spec-

TABLE 2—*Repeatability of cyanide results.*

Sample	Days[a]	Mean[b] CN Value (μg/mL) Sample 1[c]	Standard Deviation of Mean	Sample 2[c]	Standard Deviation of Mean	Percent Change	Significant[d]
Control 1	168	0.5	0	1.1	. . .	+120	U[f]
Control 2	1	0.7[e]	. . .	0.5	0.1	− 29	U
Control 2	168	0.5	0.1	1.0	0.2	+100	yes
Control 3	165	0.4[e]	0.1	0.7	0.1	+ 75	yes
DP-1	1	1.0	—	1.0	0.6	0	U
DP-3	52	2.0	0.4	2.7	—	+ 35	U
DP-4	1	2.0	0.6	2.0	0.1	0	no
DP-5	1	0.7	0.4	0.7	0.1	0	no
DP-7	52	0.6	0.1	0.7	0.0	+ 17	no
DP-9	50	2.5	0.6	2.5	0.2	0	no
DP-10	5	0.6	0.2	0.7	0.1	+ 17	no
DP-11	5	3.2	0.4	3.9	0.1	+ 22	no
DP-17	44	0.6	0.1	1.1	0.2	+ 83	yes

DP-21	44	1.0	0.2	2.1	0.2	+110	yes
DP-25	14	1.0	0.2	1.0	0.2	0	no
DP-29	36	11.4	1.7	12.9	1.8	+ 13	no
DP-41	29	1.2	0.2	1.3	0.1	+ 8	no
DP-61	18	0.6	0.1	0.9	. . .	+ 50	U
DP-63	16	1.3	0.2	1.9	0.2	+ 46	yes
DP-86	14	1.6	0.4	1.5	0.1	− 6	no
DP-91	15	0.8	0.2	0.9	0.0	+ 13	no
DP-95	15	1.4	0.2	1.5	0.3	+ 7	no
DP-201	15	1.4	0.2	1.7	0.1	+ 21	no
DP-204	15	0.8	0.2	1.6	0.2	+100	yes
DP-206	15	1.0	0.2	1.6	0.0	+ 60	yes

[a]Number of days between sample analysis.
[b]Mean of three sample analyses.
[c]All blood stored frozen except where noted.
[d]Based on two standard deviations of the mean.
[e]Fresh blood (never frozen).
[f]U = Undetermined because of unavailability of standard deviation for one set.

TABLE 3—*Comparison of distribution of COHb values in various fires.*

COHb, %	Number of Victims				Percent of Victims			
	NIST[a] Data	IFS[b] Data	MGM[c] Fire	MD[d] Study	NIST[a] Data	IFS[b] Data	MGM[c] Fire	MD[d] Study
0–9	10	0	1	48	13.7	0	1.3	9.1
10–19	28	9	3	42	38.4	11.5	3.8	7.9
20–29	12	7	4	37	16.4	9.0	5.0	7.0
30–39	6	24	12	38	8.2	30.1	15.0	7.2
40–49	6	24	19	43	8.2	30.1	23.8	8.1
50–59	5	4	24	58	6.8	5.1	30.0	10.9
60–69	4	4	11	79	5.4	7.1	13.8	14.9
70–79	1	0	6	111	1.4	0	7.5	20.9
80+	1	6	0	74	1.4	7.7	0	14.0
TOTALS	73	78	80	530	99.9	100.6	100.2	100.0

[a]NIST data from Dupont Plaza Hotel fire.
[b]IFS data from Dupont Plaza Hotel fire.
[c]NIST data from MGM Grand Hotel Fire [*15*].
[d]Data from Fire Fatality Study of State of Maryland [*16*].

TABLE 4—*Comparison of blood cyanide values found in Dupont Plaza Hotel fire and MGM Grand Hotel fire.*

Blood CN, μg/mL	Number of Victims		Percent of Victims	
	DP[a]	MGM[b]	DP[a]	MGM[b]
0–1	36	80	47	95
1–2	27	2	35	2
2–3	12	1	16	1
3–4	1	0	1	0
> 4	1	1	1	1
TOTALS	77	84	100	99

[a]NIST data from Dupont Plaza Hotel Fire.
[b]Data from MGM Grand Hotel Fire [*10*].

ified exposure time, and the numbers in parentheses are the 95% confidence limits around 1. These confidence limits are those determined during 30-min animal exposures to constant concentrations. The thermal decomposition of real materials or products would produce increasing exposure concentrations, which would broaden the 95% confidence limits of Eq 1. The extent of this effect is not known at this time.

To see if additive effects could have had an impact in the Dupont Plaza Hotel fire, a number of uncertainties must be overcome. First, we do not know the atmospheric concentrations of the gases nor do we know the exposure times, although we can probably assume the concentrations were high and the exposure times were low. All we do know are the COHb and blood cyanide values in the blood at the time of testing. Second, the problem is compounded because the detected COHb and cyanide values may be lower and higher, respectively, than those in the blood at the time of death (see discussion on Effect of Storage). Third, the lethal concentrations of these blood compounds in humans are not known precisely and can vary according to individual circumstances. Animal studies indicate, however, that the lethal blood levels of COHb depend upon the exposure time. More CO and, therefore, higher COHb is necessary to kill in shorter periods of time. For example, in previous NIST studies, rats died with approximately 83% COHb from 30-min exposures, whereas, in 1-min exposures, COHb could reach 99% before death occurred. The lethal level of blood cyanide appears to be more stable. In experiments ranging from 1 to 60 min, all rats (except those exposed for only 1 min) with greater than 2 μg/mL of blood cyanide died either during the exposures or the following 24 h [*8*]. Finally, based on the atmospheric CO and HCN concentrations, we have evidence from studies on rats that the presence of cyanide causes the equilibrium levels of COHb to be lower than expected. Therefore, it is not clear that the blood concentrations of COHb and cyanide will exactly reflect the additive toxicity of CO and HCN in the atmosphere. Despite all these difficulties, it is possible that combinations of less than lethal concentrations of COHb and blood cyanide may cause death.

Therefore, to provide further insight for the forensic scientist in the consideration of the combined effects of the COHb and blood cyanide, we made the following *assumptions*:

1. The toxicities of COHb and blood cyanide are additive.
2. The human lethal level of COHb is 50%. This value is probably low, but since the blood values of COHb may have dropped with storage, it is a conservative choice.
3. The human lethal level of blood cyanide is 5 μg/mL [*6,9*].

Based on these assumptions, Eq 1 may be rewritten in terms of the blood parameters as

$$\frac{[\text{COHb}]}{50\%} + \frac{[\text{HCN}]}{5\mu\text{g/mL}} = X \qquad (2)$$

where the numbers in brackets are the NIST blood concentrations given in Table 1. A calculated value of $X \geq 0.8$ was used to predict death as a result of the combined gases. The results of this examination of the data appear in Table 5. The range of values among the eight nonburned victims was 0.8 to 1.8, with the mean and standard deviation being 1.3 ± 0.4 ($n = 8$). The range of values for the burned victims was 0.1 to 1.8 (48 victims or 67% were below 0.8) with the mean and standard deviation being 0.7 ± 0.3 ($n = 64$). (DP-29, who had a very high blood cyanide value and therefore a very high value for Eq 2, was not included in this calculation.) Thus, the results of this examination of the possible additive effects of CO and HCN helps to support the earlier data interpretation that these toxic gases probably contributed to the deaths of the nonburned victims, whereas other causes are probably responsible for the deaths of the majority (48/64) of the burned victims. The most obvious other cause is that they were burned so rapidly that little CO was inhaled.

Another possibility to consider is that the combined concentrations of the gases (although insufficient to cause death) were sufficient to cause incapacitation. If we assume that when $X \geq$ approximately 0.6 [*12–14*], the people were physically unable to execute their escape, then 63% of the burned victims would have been affected by the combined gases before the flames. The time between incapacitation and death would have had to been quite brief since the blood levels of toxic gases would have increased during a prolonged exposure.

Effect of Storage of Blood Samples

A decrease in COHb values has been noted in a previous study when blood samples have been stored for extensive times [*15*]. In that study, control blood from a living volunteer (that is, not postmortem blood) was mixed with carbon monoxide and stored in a refrigerator for four months [*15*]. The COHb decreased from 62.3 to 57.9%, or approximately 7%, during this time period. In the current study, NIST investigators found decreases as high as 19% when the blood was stored frozen for three months. The history of the Dupont Plaza Hotel fire victims' blood indicates that it was refrigerated for approximately three months and then sent as frozen samples to NIST, where it was stored at −20°C. The NIST values given in Table 1 were obtained within the first month after the blood was received. Therefore, the aging of the blood samples may be responsible for the lower COHb values found by NIST when compared to those found by IFS, and a good possibility exists that the COHb values found in this study are lower than the original blood levels. The exact amount of the decrease is undeterminable, since the blood was stored under different conditions by IFS and NIST and was analyzed at different times. However, it appears that under the worst-case conditions investigated here, the decrease was 20%. If one assumes that all the values dropped by 20% and projects higher initial values, only four additional people would fall into the greater than 50% COHb group.

As in the case of the blood COHb values, the long storage time and the overall poor condition of the blood (multiple clots, debris, and hemolysis) raises concern about the significance of the exact blood cyanide values determined. The results in Table 2 indicate that long-term storage could have caused an increase in the blood cyanide values; and, if so, the number of people with lower than 2 μg/mL of cyanide (that is, lower than the HCN lethal concentration as determined in rats) at the time of death would increase.

Cause of Fire Deaths—Toxic Gases or Burns

Examinations of major fires (ones that produce multiple deaths) and small residential fires (where one or two people die, but where the majority of U.S. fire deaths occur)

have led many investigators to conclude that most people die in fires from the inhalation of toxic gases and not from burns. For example, as shown in Table 3, the majority of the victims in the MGM Grand Hotel fire [*15*] and in the Maryland fire fatality study (in which over 500 victims of residential fires were examined over a 5-year period) [*16*] had COHb values above 50% (51.3 and 60.7%, respectively). The Dupont Plaza Hotel fire, however, appears to be different. The overall results of NIST and IFS indicate that 80 to 85% of the victims had COHb values below 50%. The dissimilarities in COHb blood values between the Dupont Plaza Hotel fire and either the MGM Hotel fire or the Maryland Fire Fatality study reflect the different exposures experienced by the victims. Similar to the Dupont Plaza Hotel fire, the fireball was estimated to have traversed the 136.6-m (448-ft) casino of the MGM Grand Hotel in a very short time (approximately 25 s). However, in the MGM Grand Hotel fire, most of the victims were found many floors away from the fire, which originated in a restaurant on the same level with the casino [*10*]. Only 18 persons died on the casino level; the others were on the 16th floor or above (6 were at unknown locations). In the Maryland fire fatality study, 80% of the deaths were attributed to smoke inhalation. Therefore, both the MGM and Maryland study are examples where the majority of people died from the inhalation of toxic smoke [*15,16*].

In contrast to those of the other fires, most of the victims (89/97) of the Dupont Plaza Hotel fire were very badly burned and found in the area of the fire. If the engineering analysis of the fire is correct and the fireball rolled across the casino in 20 to 30 s [*5*], it is understandable how the fire and heat, not the toxic gases, could have been responsible for the majority of the deaths. A more detailed review of the forensic pathology aspects of the Dupont Plaza Hotel fire has been prepared by the forensic pathologists of IFS.

Conclusions

1. In the Dupont Plaza Hotel fire in Puerto Rico, most of the fatalities whose blood was analyzed had lower than lethal levels of COHb indicating that CO inhalation alone was not the likely cause of most of these deaths.
2. In general, those victims with significant burns had lower COHb values (generally less than 50%) than those without burns (usually greater than 50%) indicating that CO more likely contributed to the deaths of the nonburned victims.
3. Blood cyanide levels were also below lethal concentrations in most of the fatalities, regardless of whether or not they had suffered burns.
4. Estimation of the combined toxicological effect of COHb and blood cyanide substantiated the findings for the individual gases; that is, toxic gases played a greater role in the deaths of the nonburned victims than the burned victims.
5. Most of the badly burned victims appear to have died from causes other than exposure to CO or HCN, either singly or combined. The data do not rule out the possibility that the combined levels of CO and HCN could have produced an incapacitating effect which prevented escape and led to the deaths from other causes. If this were the case, however, the time between incapacitation and death would have had to be quite brief or the toxic gases in the blood would have been higher.
6. Aging of blood samples, methods of storage, and difficulties in true sampling can affect the accuracy of the analytical results. This study showed that, under the storage conditions used here, the COHb decreased and the blood cyanide increased with time.
7. Consideration of the potential blood changes due to aging and storage does not significantly change the previous six conclusions.
8. More work is needed to provide guidelines on speed of analysis, storage conditions, or corrective factors to assure more accurate results.
9. The results from the Dupont Plaza Hotel fire differ from those of the MGM Grand

TABLE 5—*Evaluation of toxicity of combined COHb and blood cyanide.*

Sample	Burned (B) or Nonburned (NB)	Equation[a] Results, X	Sample	Burned (B) or Nonburned (NB)	Equation[a] Results, X
DP-1	NB	1.8	DP-41	NB	1.5
DP-2	NB	0.8	DP-42	NB	1.2
DP-3	NB	1.7	DP-43	NB	1.1
DP-4	NB	1.0	DP-44	B	0.7
DP-5	B	0.6	DP-45	B	0.6
DP-6	B	0.8	DP-46	B	0.5
DP-7	B	0.4	DP-47	B	0.6
DP-9	B	0.8	DP-49	B	0.9
DP-10	B	0.3	DP-51	B	1.3
DP-11	B	0.8	DP-53	B	0.9
DP-12	B	0.1	DP-55	B	0.9
DP-13	B	0.8	DP-56	B	0.7
DP-14	B	0.5	DP-57	B	0.5
DP-15	B	0.4	DP-58	B	0.9
DP-16	B	0.5	DP-60	B	1.5
DP-17	B	0.4	DP-61	B	1.0
DP-18	B	0.6	DP-62	B	0.9
DP-19	NB	1.6	DP-63	B	0.6
DP-21	B	0.5	DP-64	B	0.6

DP-22	B	0.5	DP-66	B	0.5
DP-23	B	0.5	DP-67	B	0.7
DP-24	B	0.3	DP-69	B	0.5
DP-25	B	0.6	DP-70	B	0.5
DP-26	B	0.6	DP-73	B	0.8
DP-27	B	0.5	DP-74	B	0.6
DP-28	B	0.6	DP-82	B	0.4
DP-29	B	2.5	DP-85	B	0.3
DP-30	B	0.6	DP-86	B	0.6
DP-31	B	0.7	DP-91	B	0.5
DP-32	B	0.5	DP-92	B	0.5
DP-33	B	0.9	DP-94	B	0.3
DP-34	B	1.2	DP-95	B	0.8
DP-35	B	1.2	DP-201	B	1.2
DP-36	B	1.2	DP-203	B	1.8
DP-37	B	0.8	DP-204	B	0.8
DP-38	B	1.3	DP-205	B	0.4
			DP-207	B	1.8

[a] $\frac{[\text{COHb}]}{50\%} + \frac{[\text{HCN}]}{5\ \mu\text{g/mL}} = X$

where [COHb] and [HCN] results come from Table 1. When $X \geq 1$, death from the combined gases is likely.

Hotel fire [*10,15*] and the residential fires studied and reported in the Maryland Fire Fatality Study [*16*]. In the latter two cases, the majority of victims were not burned and their deaths are most likely attributable to the inhalation of toxic gases.

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