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Toxicologic Findings in the USS Iowa Disaster

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ABSTRACT: The toxicologic results from the 47 victims of the explosion on the USS Iowa are presented. Good correlation between carboxyhemoglobin saturations and cause of death was found. There were no correlations between blood cyanide concentrations and causes of death. Volatile analysis suggested postmortem ethanol production rather than antemortem ethanol ingestion. No drugs except nicotine were detected in any of the victims.

KEYWORDS: toxicology, explosion, USS Iowa, disaster

On the morning of April 19, 1989, the USS Iowa was undergoing a gunnery exercise approximately 330 miles northeast of Puerto Rico. At 9:55 A.M., an explosion occurred in the Number 2 turret and killed 47 crewmen. To understand the cause of the explosion, it is necessary to review the operating principles of the naval guns involved. The guns are mounted in three triple gun turrets. The turret contains the three guns on the top deck. Below this turret deck are a machinery deck, electrical deck, upper and lower projectile decks and a powder-handling platform. The shells fired by these guns are over 4 feet long and weigh 2700 pounds. The shells are discharged from the gun by a propelling charge that consists of silk bags filled with cordite, a mixture of nitrocellulose and nitroglycerin. Attached to one end of the silk bags was a bag of black powder, which served as the igniter. Black powder contains potassium nitrate, charcoal, and sulfur and is easily ignited by heat.

Once the bodies were recovered, they were transported to Dover Air Force Base in Delaware for identification, autopsy to determine the causes of death and collection of specimens.

Toxicological Analysis

Carbon Monoxide (CO)

All cases were examined for CO using the IL 482 CO-Oximeter. Blood was tested in 41 cases; tissue fluid was analyzed in the remaining cases. All specimens with carboxy-

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hemoglobin (CO-Hb) greater than 10% saturation were confirmed by gas chromatography—thermal conductivity detection.

Cyanide (CN)

All cases with appropriate specimens were screened for CN by microdiffusion with colorimetry [*1*] with a limit of detection of 0.5 mg/L. All specimens screening positive were confirmed and quantitated by headspace gas chromatography with nitrogen—phosphorus detection [*2*]. Blood was tested in 40 cases while tissue fluid was used in 5 cases; insufficient specimens were available for CN analysis in two cases.

Volatiles

For all cases, two specimens were examined for the presence of acetaldehyde, ethanol, acetone, 1-propanol, 2-propanol, 1-butanol, 2-butanol, isobutanol and t-butanol by headspace gas chromatography with flame ionization detection.

Drug Testing

The following drugs or drug classes were analyzed by a combination of gas chromatography, thin layer chromatography, or immunoassay: amphetamine, antihistamines, barbiturates, benzodiazepines, cannabinoids, cocaine and metabolite, lidocaine, methaqualone, narcotic analgesics, nicotine, opiates, phenytoin, sympathomimetic amines, and tricyclic antidepressants. Urine was tested if received; otherwise, blood or tissue was analyzed. All positive results were identified by two independent methods.

Results

Autopsies were begun within 48 h after death and completed within 48 h. Due to the flooding of the turret, many of the bodies were significantly decomposed. All 47 victims were identified by dental comparisons, 45 positively and 2 classified as "consistent with." Fingerprints identified 33 victims, including the two classified as "consistent with" by dental examination. All bodies were photographed, radiographed, and autopsied. Correlations between injuries and body position were made.

The causes of death, body position, and the results of the CO, CN, and ethanol analyses on the 47 victims are summarized in Table 1 and Table 2.

Discussion

The most crucial component in the toxicologic investigation of this incident was the analysis of CO-Hb in the blood or tissue fluid. Ten of the 47 who died had CO-Hb saturation values equal to or less than 10%, which is within normal limits. The low amount of CO-Hb measured suggests that these individuals died rapidly after the explosion. A study of body location of these victims revealed that nine were found in the turret region and the other body position was unknown. Furthermore, seven were in the vicinity of the center gun where the explosion occurred. Assuming that the explosion was directed rearward, these victims would have been closest to the explosion and thus would have died most rapidly. The cause of death in the 10 individuals with normal CO-Hb saturation were blunt force trauma injuries (BFI) (8) or a combination of the BFI and thermal injuries (2). Eleven other victims were recovered from the turret region; the CO-Hb in these cases ranged from 33 to 64% saturation. The cause of death in 10 of these individuals was attributed to the resulting fire while the eleventh death resulted

TABLE 1—*Causes of death and CO and CN results on the 47 fatalities.*

Victim no.	Body[a] position	Cause[b] of death	CO-Hb (% saturation)	CN (mg/L)
1	Turret	Thermal	64	8.9
2	Turret	Thermal	33	neg
3	Outside	Thermal	58	neg
4	Turret	Thermal	64	0.7
5	Outside	Thermal	56	neg
6	Turret	Thermal	43	8.7
7	Outside	Thermal	54	23
8	Turret	Mix	9	12
9	Turret	Thermal	51	5.2
10	Outside	Thermal	38	neg
11	Turret	Thermal	60	8.2
12	Unknown	BFI	45	5.4
13	Turret	BFI	1	neg
14	Outside	Thermal	55	6.6
15	Outside	Thermal	50	8.0
16	Unknown	Mix	2	neg
17	Turret	BFI	1	neg
18	Outside	Thermal	53	27
19	Turret	BFI	5	neg
20	Turret	Thermal	52	6.0
21	Outside	Mix	49	1.1
22	Turret	Mix	54	36
23	Outside	Thermal	60	neg
24	Outside	BFI	28	neg
25	Outside	Thermal	62	7.3
26	Outside	Thermal	48	19
27	Outside	Thermal	62	54
28	Outside	Thermal	56	36
29	Outside	Mix	56	12
30	Turret	Thermal	47	neg
31	Outside	Thermal	60	11
32	Outside	Thermal	64	24
33	Unknown	Thermal	64	neg
34	Outside	Thermal	65	3.8
35	Unknown	Thermal	37	8.3
36	Turret	BFI	10	6.1
37	Outside	Thermal	54	neg
38	Outside	Thermal	36	2.9
39	Turret	Thermal	59	0.3
40	Turret	BFI	1	—
41	Outside	Thermal	52	3.9
42	Outside	BFI	44	neg
43	Turret	Thermal	51	17
44	Turret	BFI	8	—
45	Outside	BFI	60	3.7
46	Turret	BFI	<1	neg
47	Turret	BFI	8	15

[a]Outside = outside turret deck.
Unknown = position unknown.
[b]BFI = blunt force injuries.
Thermal = smoke and soot inhalation/burn.
Mix = combination of BFI and thermal.

from a mixture of BFI and thermal injuries. There were 23 victims who were found outside of the turret region; 19 had CO-Hb saturation greater than 48% and 4 had CO-Hb between 28 and 48%. The cause of death in these 23 cases were thermal injuries (18), BFI (3), and combination (2). There were three victims with elevated CO-Hb whose

TABLE 2—*Ethanol concentration in the 47 fatalities.*

Victim no.	Blood	Ethanol concentrations (mg/dL) other (specimen)
1	Neg	—
2	0.19	neg (urine)
3	0.11	neg (bile)
4	0.02	neg (vitreous fluid)
5	0.08	—
6	0.05	neg (urine)
7	—	neg (vitreous fluid)
8	—	neg (vitreous fluid)
9	0.01	0.02 (bile)
10	0.02	neg (urine)
11	0.02	0.01 (urine)
12	0.01	neg (urine)
13	Neg	—
14	Neg	—
15	0.03	0.06 (bile)
16	Neg	—
17	0.02	neg (urine)
18	Neg	—
19	0.04	neg (vitreous fluid)
20	Neg	—
21	0.03	neg (urine)
22	Neg	—
23	0.02	0.02 (bile)
24	0.09	—
25	Neg	—
26	0.02	—
27	Neg	—
28	Neg	—
29	Neg	—
30	0.07	neg (urine)
31	Neg	—
32	Neg	—
33	0.03	neg (bile)
34	0.01	—
35	Neg	—
36	—	neg (brain)
37	0.09	0.02 (urine)
38	Neg	—
39	0.04	0.03 (bile)
40	Neg	—
41	Neg	—
42	0.05	0.04 (bile)
43	Neg	—
44	Neg	—
45	0.02	—
46	—	neg (kidney)
47	—	neg (kidney)

body position was unknown. The CO-Hb saturation in these cases were 37, 45, and 64%. Two of these deaths were attributed to the fire and the third death was caused by BFI. In total, there were 37 victims with elevated CO-Hb. The fire caused death in 30 of these individuals. This indicates a good correlation between CO-Hb saturation, body location, and cause of death.

CN concentrations in blood or tissue fluid were measured in 45 individuals. However, no correlation between CO-Hb and CN was readily apparent. In the 10 victims with low CO-Hb, five had no CN, three had CN greater than 6 mg/L and two were not analyzed for CN. The remaining 37 were divided as follows: 10 were negative for CN, 3 had CN

concentrations between 0.1 and 2 mg/L and 24 had CN concentrations greater than 2 mg/L. A complicating component in interpreting postmortem CN concentrations is that CN may disappear from the blood as pH drops, or may be produced in great quantities by bacterial action.

As with CN, the interpretation of postmortem ethanol analysis can be complicated by putrefaction and microorganism contamination. Of the 47 victims, 23 tested positive for ethanol in blood, fluid, or tissue. There was no evidence to suggest that any of the victims had been drinking alcoholic beverages prior to the accident. Thirteen had ethanol concentrations less than or equal to 0.03 g/dL and can rather easily be attributed to postmortem ethanol formation. For cases with higher ethanol concentrations, multifluid analysis enabled the presence of ethanol to be attributed most likely to postmortem formation. For example, victim 2 had a blood ethanol concentration of 0.19 g/dL; however, the bile ethanol and urine ethanol concentrations were less than 0.01 g/dL. Much higher fluid concentrations would be expected to be associated with a blood concentration of 0.19 g/dL resulting from ethanol consumption. Of the 10 individuals with a blood ethanol concentration greater than 0.03 g/dL, 5 had associated urine, bile or vitreous fluid ethanol concentrations which were negative. In two other cases, only a blood specimen was analyzed.

Although there was no suspicion that the use or abuse of drugs played a role in the tragedy, comprehensive drug testing was requested on each case. Except for the presence of nicotine in 11 individuals, no drugs were detected in the 47 victims.

Although the circumstances of this tragedy are highly unusual, the results of the pathologic and toxicologic investigation were similar to those found in other fire fatalities. All deaths were accounted for by BFI, thermal injuries, or a combination of the two. The cause of death in this incident were assigned as follows: BFI—12; thermal injuries—30; combined causes—5. In one study of residential fires in Maryland [*3*] 80% of the deaths were caused by CO inhalation with or without underlying heart disease, 11% were caused by thermal injuries and 9% were unexplained. In the Dupont Plaza Hotel fire in Puerto Rico [*4*], 89 of the 97 people died of thermal injuries; the measured—CO-Hb concentrations ranged from 10 to 80%, with only eight having CO-Hb greater than 50%. On the other hand, in the nonburned individuals, measured CO-Hb concentrations ranged from 60 to 90%.

One interesting toxicologic finding in these cases was the presence of high concentrations of CN in some of the victims. In the Maryland study, about 90% of the CN concentrations were less than 2 mg/L. Wetherell [*5*] reported CN concentrations up to 2.2 mg/L in 53 fire victims. CN was detected in only 6 of 36 fire fatalities studied by Okae et al. [*6*]. It can be suggested that the nature of the fire produced in this case may have generated much higher HCN concentrations in the air. However, it would be expected that individuals found in the same environment would all have similarly elevated CN concentrations as was observed with CO; this was not found. For example, 12 bodies were recovered from the powder room. Although the CO-Hb in these individuals ranged from 36 to 64% saturation, the CN concentrations ranged from 0 to 54 mg/L. Six of the 12 had CN concentrations less than 1 mg/L. On the other hand, postmortem production of high concentrations of CN has been reported by numerous authors [*7,8*] and may provide an explanation for the incongruity of the CN data, especially when the time lag of more than a day between death and autopsy is considered.

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