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# **A Fatality Caused by Hydrogen Sulfide Produced From an Accidental Transfer of Sodium Hydrogen Sulfide Into a Tank Containing Dilute Sulfuric Acid**

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## N O T I C E

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16. Abstract  The National Transportation Safety Board has an agreement with the Federal Aviation Administration (FAA) that the FAA's Civil Aeromedical Institute (CAMI) provide toxicological services for selected surface transportation accidents. Under this agreement, postmortem biosamples from a hazardous chemical accident fatality were submitted to CAMI for toxicological evaluation. The victim succumbed from breathing the hydrogen sulfide (H <sub>2</sub> S) gas produced by an accidental transfer of sodium hydrogen sulfide (NaHS) from a tanker truck to a tank containing 4% sulfuric acid (H <sub>2</sub> SO <sub>4</sub> ) and iron(II) sulfate (FeSO <sub>4</sub> ). After inhaling the gas, the 55-year old male Caucasian truck driver was dead at the scene. Autopsy examination of the decedent's body revealed pulmonary edema and passive congestion in lungs, spleen, kidneys, and adrenal glands. The submitted samples were analyzed for carbon monoxide, cyanide, alcohols, and drugs. Since a potential exposure to H <sub>2</sub> S was involved, blood was also analyzed for sulfide (S <sup>2-</sup> ). The analysis entailed isolating S <sup>2-</sup> from blood as H <sub>2</sub> S using 0.5 M H <sub>3</sub> PO <sub>4</sub> , trapping the gas in 0.1 M NaOH, and determining the electromotive force using a sulfide ion specific electrode. Carbon monoxide, cyanide, or ethanol was not detected in blood, but acetaminophen at a therapeutic concentration of 14.3 µg/mL of blood was found, and metoprolol was detected in the blood, liver, and kidney samples. Analysis further revealed the presence of S <sup>2-</sup> in blood at the level of 1.68 µg/mL. This S <sup>2-</sup> concentration is approximately 2 times higher than that reported in the blood of 2 separate fatalities associated with accidental exposures to H <sub>2</sub> S. The blood S <sup>2-</sup> value in the present case was about 34 times higher than the blood S <sup>2-</sup> concentration (< 0.05 µg/mL) in normal subjects. The observed pulmonary edema and the passive congestion in various organs were also in agreement with the pathological characteristics of H <sub>2</sub> S poisoning. Since H <sub>2</sub> S toxicity manifests rapidly by inhibiting the cytochrome oxidase system, causing histotoxic cellular hypoxia, death occurs quickly. Based on the case history, pathological findings, and blood S <sup>2-</sup> concentration, it is concluded that the cause of death was H <sub>2</sub> S poisoning associated with a hazardous material accident in an industrial situation.			
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# A FATALITY CAUSED BY HYDROGEN SULFIDE PRODUCED FROM AN ACCIDENTAL TRANSFER OF SODIUM HYDROGEN SULFIDE INTO A TANK CONTAINING DILUTE SULFURIC ACID

## INTRODUCTION

The National Transportation Safety Board has an agreement with the Federal Aviation Administration (FAA) that the FAA's Civil Aeromedical Institute (CAMI) provide toxicological services for selected surface transportation accidents. Under this agreement, biological samples collected at autopsy from an industrial fatality caused by an accidental inhalation of hydrogen sulfide were submitted to CAMI for toxicological evaluation.

Hydrogen sulfide, a powerful rapidly acting poisonous gas (1,2), is produced and used in industry for the manufacturing of chemicals (3). This gas is also naturally generated during the putrefaction of organic substances (4,5). The presence of this gas can be identified by the characteristic odor of rotten eggs (5), but its detection by the odor is not always possible because this gas in higher concentrations paralyzes the olfactory nerves (4).

Hydrogen sulfide has been the primary source of sulfide ( $S^{2-}$ ) in several human poisonings, and numerous acute poisonings and fatalities have been reported from exposure to hydrogen sulfide in industrial settings, sewers, wells, and septic tanks (4-7). However, blood sulfide levels in the literature are reported in only a limited number of hydrogen sulfide fatalities—one paper describing a fatality with 0.92  $\mu\text{g/mL}$  of sulfide in blood was published in 1968 by Winek et al. (6); another paper describing 5 fatalities with blood sulfide concentrations of 1.70-3.75  $\mu\text{g/mL}$  was published in 1979 by McAnalley et al. (7). Additionally, 3 fatalities with blood sulfide levels of 0.8, 3.6, and 5.0  $\mu\text{g/mL}$  were reported in 1981 by Osborn and Crapo (2,8). Sulfide, similar to cyanide ( $CN^-$ ), produces acute systemic toxicity by inhibiting the cytochrome oxidase system (1,2). In the present study, we report toxicological findings, along with the blood sulfide value, of the unique industrial fatality caused by an accidental production of, and exposure to, hydrogen sulfide.

## Case History

A 55-year-old, healthy Caucasian male truck driver, 1.83 m in height and 88.5 kg in weight, was found dead at the scene where he had accidentally transferred sodium hydrogen sulfide ( $\text{NaHS}$ ) from his tanker truck into a tank containing 4% sulfuric acid ( $\text{H}_2\text{SO}_4$ ) and iron(II) sulfate ( $\text{FeSO}_4$ ). He had been quickly overpowered by the chemically produced hydrogen sulfide gas and had collapsed. This incident was not discovered for several minutes, when another employee entering the area smelled the gas, immediately evacuated the area, and called for help. The death had occurred at 4:30 a.m., and the autopsy was conducted about 8 hr later. The body was well preserved, unembalmed, and refrigerated.

## Pathology

External examination of the body revealed normal features. Internal examination of organs disclosed no significant anatomic abnormalities, except prevalence of pulmonary edema and congestion in lungs and kidneys. Microscopically, passive congestion was evident in lungs, spleen, kidneys, and adrenal glands. The cause of death was determined to be hydrogen sulfide poisoning. Blood, brain, kidney, liver, urine, and vitreous humor samples collected during necropsy were submitted for toxicological evaluation.

## MATERIALS AND METHODS

### Materials

All reagents and solvents were of analytical grade and were of the highest available purity. These chemicals, drug standards, and other agents were obtained from commercial sources. All calibrators, open controls, and negative controls were prepared in human blood obtained from the local blood bank (Oklahoma Blood Institute, Oklahoma City, OK).

## Toxicological Evaluation

Carbon monoxide in blood was analyzed spectrophotometrically as carboxyhemoglobin (9) and hydrogen cyanide colorimetrically as  $\text{CN}^-$  (10). Alcohols and drugs were analyzed according to the laboratory's standard analytical procedures.

## Hydrogen Sulfide Determination

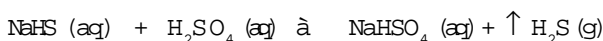
Hydrogen sulfide in blood was determined as sulfide ( $\text{S}^{2-}$ ) by adopting a modified literature procedure (11), using a sulfide ion specific electrode (Model 9616; Orion Research, Inc., Beverly, MA). Sulfide from blood was isolated as the hydrogen sulfide gas by mixing 2.0 mL of blood with 2.0 mL of 0.5M  $\text{H}_3\text{PO}_4$  in the outer ring of a Conway microdiffusion dish. The evolved gas was absorbed in 1.5 mL of 1.0 M NaOH in the center well of the dish. After incubating the covered dish for 4 hr at ambient temperature, a 1.5-mL portion of Sulfide Antioxidant Buffer (Orion Research, Inc., Beverly, MA) was added into the central well. The chemical solutions in the well were mixed, and the entire mixture was transferred into a 5-mL beaker containing a magnetic stirrer. To this 3-mL mixture, 100  $\mu\text{L}$  of a standard sulfide solution (1  $\mu\text{g}/\text{mL}$ ) was added. The addition of the standard sulfide solution to each sample was necessary to minimize the fluctuation in the electromotive force (EMF; -mV) readings, particularly at low sulfide concentrations. The EMF reading of each sample, including calibrators, open controls, and negative controls, was determined using the ion specific electrode and a digital pH/mV meter (Model 801; Orion Research Inc., Beverly, MA). Calibrators were prepared in human blood by serial dilution of a stock sulfide solution (1 mg/mL), which was prepared in deionized water using  $\text{Na}_2\text{S}\cdot 9\text{H}_2\text{O}$ . All samples were analyzed sequentially, and the EMF readings were taken within an hour. A linear blood calibration curve was obtained by using 0.0, 1.0, 2.0, 4.0, and 8.0  $\mu\text{g}/\text{mL}$  sulfide (Figure 1). Although the added amount of sulfide (100 ng) is excluded from the calibrator concentrations (see the figure), the EMF values represented the readings from the added sulfide and the sulfide originally present in the respective samples.

## RESULTS AND DISCUSSION

Toxicological evaluation of the blood sample failed to disclose the presence of carbon monoxide, cyanide, or ethanol in a detectable amount. However, 14.3  $\mu\text{g}/\text{mL}$  of acetaminophen was present in the blood sample. Metoprolol was also detected in the blood, liver, and kidney samples. Acetaminophen is an over-the-counter analgesic/antipyretic medication (12,13) and metoprolol is a  $\beta$ -adrenergic blocking prescription drug (14,15). The level of acetaminophen found in the decedent's blood was within the therapeutic range (10-20  $\mu\text{g}/\text{mL}$ ) (13,16). The presence of metoprolol suggests that the victim was being treated for hypertension.

Further analytical toxicology of the blood sample revealed the presence of sulfide at the concentration of 1.68  $\mu\text{g}/\text{mL}$  (Table 1). The sulfide concentration found in the blood of the victim was approximately 2 times higher than that reported in 2 separate fatal cases (2,4,6-8) and falls well within the range of 0.8-5.0  $\mu\text{g}/\text{mL}$  in the postmortem blood of the individuals who have died in hydrogen sulfide related accidents (6-8). The observed sulfide value in the present case was about 34 times higher than the sulfide concentration ( $< 0.05 \mu\text{g}/\text{mL}$ ) expected in blood from normal subjects (7), which is consistent with the sulfide values determined in the normal control (Table 1). Since the victim's body was well preserved and refrigerated, the autopsy was performed within about 8 hr, and the blood sample storage temperature was  $6^\circ\text{C}$  or less, the postmortem production of sulfide was presumably suppressed (4,17,18).

It is evident from the case history that the victim was exposed to a lethal concentration of hydrogen sulfide generated by the chemical reaction between sodium hydrogen sulfide ( $\text{NaHS}$ ) and sulfuric acid ( $\text{H}_2\text{SO}_4$ ), as represented by the chemical equation:



The potential for other chemical reactions in the mixture is negligible. Therefore, the gaseous material generated during the accidental transfer of sodium hydrogen sulfide was the hydrogen sulfide gas. Since

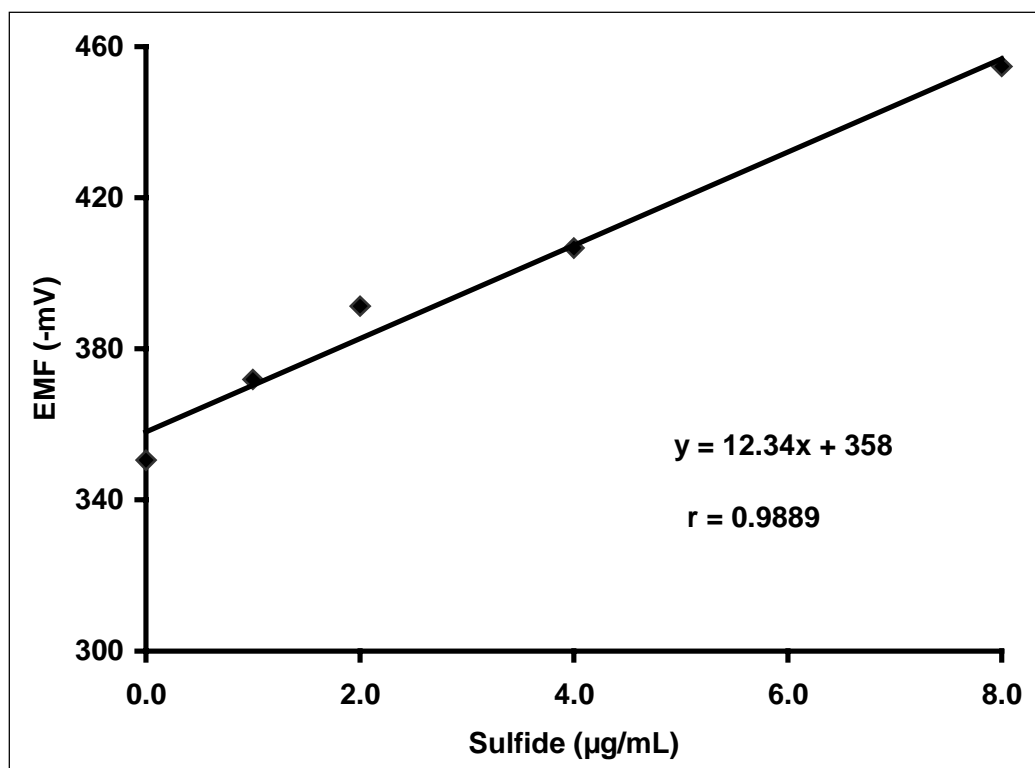


Figure 1. The calibration curve of sulfide ( $S^{2-}$ ) in blood. Details are given in the text of the Materials and Methods section.

**Table 1.** The Blood Sulfide ( $S^{2-}$ ) Concentration in the Fatality Resulting from Exposure to Hydrogen Sulfide

Sample Description	Mean (SD <sub>n</sub> ) Sulfide ( $S^{2-}$ ) Concentration (µg/mL; n = 4)
Normal Subject Blood	0.03 (0.015)
Decedent Blood	1.68 (0.206)

hydrogen sulfide toxicity manifests rapidly similar to hydrogen cyanide poisoning by inhibiting the cytochrome oxidase system, causing histotoxic cellular hypoxia (1,2), death occurs quickly. The pulmonary edema and the passive congestion in various organs observed during the pathological examination of the

victim's body were consistent with the pathological characteristics of hydrogen sulfide poisoning (1,2,6). In view of the findings, it is concluded that the cause of death was hydrogen sulfide poisoning associated with a hazardous material accident in an industrial situation.

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