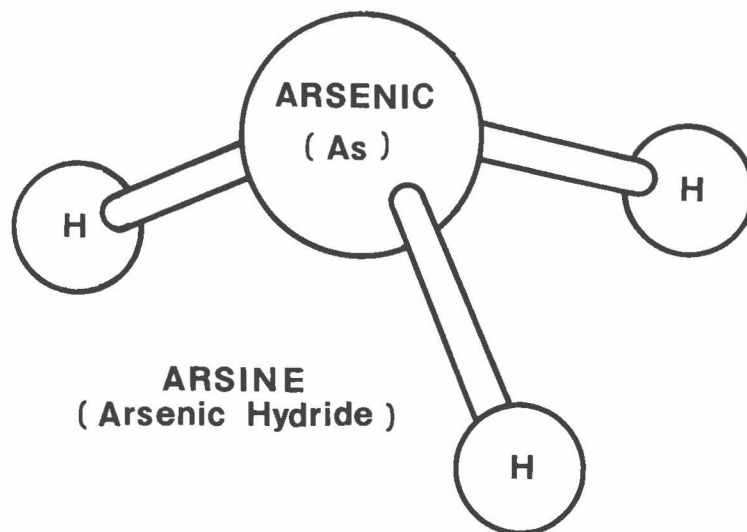


NIOSH

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AUGUST 3, 1979

ARSINE (Arsenic Hydride) POISONING IN THE WORKPLACE



U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health



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PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL

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The NIOSH Current Intelligence Bulletin is the primary product of the Current Intelligence System. The purpose of the Current Intelligence System is to promptly review, evaluate, and supplement new information received by NIOSH on occupational hazards that are either unrecognized or are greater than generally known. The staff of the NIOSH Technical Evaluation and Review Branch, Office of Extramural Coordination and Special Projects was responsible for the preparation of this Bulletin.

As warranted by this evaluation, the information is capsulized and disseminated to NIOSH staff, other government agencies, and the occupational health community, including labor, industry, academia, and public interest groups. With respect to currently known hazard information this system also serves to advise appropriate members of the above groups of recently acquired specific knowledge which may have an impact on their programs or perception of the hazard. Above all, the Current Intelligence System is designed to protect the health of American workers and to allow them to work in the safest possible environment.

IDENTIFIERS AND SYNONYMS FOR ARSINE

Chemical Abstracts Service Registry Number: 7784-42-1

NIOSH RTECS Number: CG6475000

Chemical Formula: AsH₃

Arsine

Arsenic hydride

Arsenic trihydride

Arseniuretted hydrogen

Arsenous hydride

Hydrogen arsenide

The above information was obtained from the National Institute for Occupational Safety and Health's computerized Registry of Toxic Effects of Chemical Substances (RTECS), and from the National Library of Medicine's computerized chemical dictionary file CHEMLINE. Registered trademark information is not included in these files. Therefore, some of the above synonyms and identifiers may be trademarked but are not so indicated above.

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CURRENT INTELLIGENCE BULLETIN

ARSINE (Arsenic Hydride) POISONING IN THE WORKPLACE

The National Institute for Occupational Safety and Health (NIOSH) recommends that appropriate workpractices be implemented to reduce the risk of worker exposure to arsine (AsH_3) gas. There is a high potential for the generation of arsine gas when inorganic arsenic is exposed to nascent (freshly formed) hydrogen. This recommendation is based on several reports of worker exposure to arsine resulting in severe toxic effects or death. Most of the reported cases occurred when arsine was accidentally generated during an industrial process. NIOSH would like to inform the occupational health community of some of the circumstances in which workers have been poisoned by arsine, with particular emphasis on the underlying mechanisms of generating the gas. We request that producers and distributors of arsenic and materials containing arsenic transmit information to their customers and employees, and that professional associations and unions inform their members.

Stibine (SbH_3), another toxic gas, is formed when antimony is exposed to nascent hydrogen. In most situations where arsine can be formed, stibine can also be formed if antimony is present. Therefore, similar workpractices should be implemented to reduce the risk of worker exposure to stibine.

BACKGROUND

Identified in 1775, arsine is a highly poisonous, colorless, nonirritating gas with a mild garlic odor. It is soluble in water, and slightly soluble in alcohol and alkalis. When nascent hydrogen is generated in the presence of arsenic, or when water reacts with a metallic arsenide, arsine evolves. Most cases of arsine poisoning have been associated with the use of acids and crude metals, one or both of which contained arsenic as an impurity. Ores contaminated with arsenic can liberate arsine when treated with acid (1). Arsine is commercially produced for use in organic synthesis, and the processing of solid state electronic components.

In industrial settings arsine poisoning generally results from the accidental formation of arsine gas. Most reported cases of exposure to arsine have occurred during the smelting and refining of metals. However, there are many other situations where exposures to lethal concentrations of the gas have been reported, including galvanizing, soldering, etching and lead plating operations. Arsine can be produced by fungi (especially in sewage) in the presence of arsenic. The renewed interest in coal as a source of energy causes concern for a possible increase in the number of exposures to arsine, because coal contains considerable quantities of arsenic. The processes for converting coal to gas and other by-products should include preventative measures aimed at reducing the chance of transformation of the arsenic impurities into arsine (1).

OCCUPATIONAL STANDARDS AND EXPOSURES

The current Department of Labor, Occupational Safety and Health Administration (OSHA) standard for occupational exposure to arsine is 0.05 ppm (0.2 mg/cu m of air) as a time-weighted average in any 8-hour work shift of a 40-hour work week. The present OSHA standard for occupational exposure to stibine is 0.1 ppm (0.5 mg/cu m of air) in any 8-hour work shift of a 40-hour work week (2). The 1975 NIOSH Criteria Document on inorganic arsenic recommended that worker exposure to inorganic arsenic and to arsine be limited to 0.002 mg (2.0 µg) of arsenic/cu m of air as determined by a 15-minute sampling period. The document states that the short-term limit is intended to achieve the greatest practicable reduction in worker exposure while avoiding spurious sampling results which can be produced by natural background concentrations of inorganic arsenic (3). The 1978 NIOSH criteria document on antimony recommended the retention of the specific Federal limit for occupational exposure to antimony, without recommending a limit for stibine (4).

The NIOSH National Occupational Hazard Survey (NOHS) estimates that approximately 900,000 workers are occupationally exposed to identified sources of arsenic for varying periods of time during the workday. This estimate is not, however, based on actual workplace environmental exposure measurements. However, arsenic is a widespread element, and therefore unidentified exposures can occur in unsuspected work situations. The NOHS estimate for occupational exposure to antimony is approximately 1,700,000 workers (5).

TOXICITY

The first case of arsine poisoning was reported in 1815 after a German chemist died from an exposure to arsine in his laboratory. From 1815 to 1928, 247 cases of arsine poisoning were reported. From 1928 to 1974 an additional 207 cases were reported, of which 51 (25%) were fatal (1).

Acute Arsine Toxicity - Arsine is the most acutely toxic form of arsenic and one of the major industrial causes of sudden extensive hemolysis (destruction of red blood cells). It has the ability to combine with hemoglobin within the red blood cell, causing destruction or severe swelling of the cell, rendering it nonfunctional (1). Inhalation of 250 ppm (800 mg/cu m) of arsine gas is instantly lethal. Exposures of 25-50 ppm (80-160 mg/cu m) for one-half hour are lethal, and 10 ppm (32 mg/cu m) is lethal after longer exposures. The Mean Lethal Dose (MLD) is unknown for man, but in small mammals it is about 0.5 mg/kg body weight (6).

The characteristic features of acute arsine poisoning are abdominal pain, bloody urine, and jaundice (yellow discoloration of the skin). Initial symptoms of arsine

poisoning are headache, malaise, weakness, dizziness, difficult breathing, abdominal pain, nausea, and vomiting, which are usually first noticed 2 to 24 hours after exposure. Bloody urine, light to dark red, is frequently noticed 4-6 hours after exposure to arsine and is often followed by jaundice 12-48 hours later. An unusual bronze discoloration of the skin can often be observed accompanying the jaundice. If the arsine exposure is severe, the products resulting from the breakdown of red blood cells and hemoglobin will clog the kidneys, causing a reduction in the amount of urine formed, sometimes to the point of complete blockage of urine formation. Other toxic effects of arsine include damage to the liver and heart, either by direct actions of arsine in the cells or due to the formation of arsenic (1,7).

Chronic Arsine Toxicity - Most reported cases of arsine poisoning have been acute or sub-acute in nature, usually resulting from a single short exposure or from breathing the gas for a few hours. In one report of chronic arsine poisoning, it was noted that arsine in very small concentrations appeared to exert a cumulative, damaging effect. This was manifested by a progressive drop in the number of red blood cells and in the hemoglobin level. The exposed victims experienced shortness of breath on exertion, and a general feeling of weakness. However, in relation to the degree of blood destruction, the degree of known disability experienced by the victims of chronic arsine poisoning was less than expected (8).

Chronic Arsenic Toxicity - Since inorganic arsenic is needed to generate arsine, prolonged exposures to low levels of arsine are likely to occur under conditions where workers are also exposed to inorganic arsenic. Once arsine is inhaled, it breaks down, releasing inorganic arsenic into the blood stream. The worker's risk of arsenic poisoning is therefore increased by the combination of inorganic arsenic exposure and the breakdown of arsine. A number of signs and symptoms are associated with arsenic poisoning. When ingested, arsenic compounds can cause nausea, vomiting and diarrhea within a few hours. Dermatitis may be observed after chronic ingestion, but the typical signs include increased pigmentation, and thickening of the skin on the palms and soles of the feet. Changes in the heart's performance, as measured by the electrocardiogram (ECG) have been reported after chronic arsenic intoxication. Observed ECG changes regressed after arsenic exposure ceased. Decreased numbers of red and white blood cells were reported in cases of chronic intoxication, but these changes also regressed after arsenic ingestion ended. Skin cancer has long been considered a consequence of arsenic exposure, however multiple cancers of the internal organs have also been reported (3).

Case Reports - Most cases of arsine poisoning occur after the accidental generation of the gas in the workplace. During recent years many incidents have involved a reaction between arsenic and aluminum, with the subsequent release of hydrogen in the presence of water to permit the formation of arsine gas (9). Tables 1, 2, and 3 list examples of accidental arsine poisoning reported in the literature.

TABLE 1. Examples of workers poisoned by arsine in smelting and refining operations.

-
- o Three workers were poisoned while using a jackhammer to remove slag from ladles. To reduce dust, water was sprayed on the slag. The water reacted with the arsenide of the alkali metal, generating arsine (9).
 - o A worker was poisoned while cleaning an obstructed industrial drain which contained acid liquors with arsenic impurities. A galvanized shovel and bucket were used to carry the sludge from the drain. The acidic arsenic liquor reacted with the zinc coating of the bucket and produced arsine (10).
 - o Thirteen workers were poisoned (3 died) during the purification of lead alloys. Arsine fumes were generated by moisture reacting with aluminum arsenide in metal dross (11).
 - o A worker was poisoned during reclamation of metal from flue dust obtained from blast furnaces. Zinc, water, and sulphuric acid had been added to the dust. Upon heating with steam, arsine was formed because arsenic impurities and nascent hydrogen were both present (12).
-

TABLE 2. Examples of workers poisoned by arsine in enclosed spaces.

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- o Two workers were poisoned while cleaning an aluminum trailer tank with a phosphoric acid solution. The tank had been used 6 months previously for the transport of a 42% solution of sodium arsenite (weed killer). Since that time it had been cleaned with steam and detergent, and used for storage and transportation of alcohol and other industrial solvents. Before assigning the trailer to another client, a thorough cleaning job was ordered, which required the hand application of the acid cleaner. Subsequently, arsine was generated by a reaction between the acid cleaner and the aluminum which had arsenic impurities (13).
 - o Three workers were poisoned while using an aluminum ladder to descend into a chemical evaporation tank containing a few inches of sodium arsenite. The aluminum ladder reacted with the sodium arsenite, liberating arsine (14).
 - o Eight sailors were poisoned when a cylinder containing arsine developed a leak, emitting the gas into the airspace of a cargo hold on board a freighter (15).
-

TABLE 3. Examples of workers poisoned by arsine in miscellaneous occupational settings.

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- o A worker was poisoned while pouring freshly diluted commercial hydrochloric acid through the pipes of a water jacket. The manufacturer had added sodium arsenite and aniline hydrochloride to the acid to act as inhibitors to the corrosive action of the acid. Subsequently the mixture of water and sodium arsenite under these conditions led to the generation of arsine (16).
 - o Five workers were poisoned while washing aluminum slag to dissolve out soluble constituents. Apparently, the copper in this mixture was contaminated with arsenic (17).
 - o Eight children and one adult were poisoned on a farm while cleaning a dipping vat. Two years before, arsenic had been used in the vat as an insecticide. Later, with another insecticide, superphosphate was added to create an acid medium, thereby leading to arsine production (18).
 - o Two workers were poisoned after a commercial drain cleaner was added to a drain which contained water and arsenic residues. The drain cleaner contained sodium hydroxide, sodium nitrate, and aluminum chips. These chemicals reacted to produce nascent hydrogen, which bonded with the arsenic, leading to generation of arsine (19).
-

Although the accidents illustrated in the above tables differ with respect to the surrounding circumstances, the basic reactions leading to arsine formation are similar. Arsine usually evolved when nascent hydrogen was generated in the presence of arsenical compounds, or by the hydrolysis of a metallic arsenide in contact with water. Invariably, there was an acid medium where metal was present (e.g., dross residues, galvanized implements, aluminum tanks or implements) thereby creating the key ingredients necessary for arsine formation.

A more recent area of concern involves the recycling of batteries. Lead alloys in car batteries contain antimony as a hardener, with arsenic and silver added to inhibit corrosion. In the production of "maintenance-free" batteries, calcium is added to the lead alloys as a hardening agent. During recycling, arsine can be released if scrap containing arsenic is melted down with the "maintenance-free" batteries containing calcium. When the scrap mixture is in the molten state, calcium arsenide is formed. As cooling occurs, the calcium arsenide floats to the surface as part of the dross, and in the presence of water, arsine evolves (20).

OTHER CONSIDERATIONS

Stibine (SbH₃; hydrogen antimonide; antimony hydride; antimony trihydride)-Antimony (Sb) can be converted to stibine by a similar series of reactions required to convert arsenic to arsine. Stibine equals or surpasses arsine in toxicity, and causes a specific toxic action which closely resembles that of arsine (21). Although stibine is chemically similar to arsine, it is less stable. Probably because of this instability fewer cases of stibine poisoning have been reported (22). Stibine can evolve when certain alloys containing antimony (Sb) are treated with acid and subjected to electrolytic action, when certain antimony compounds are treated with steam, or whenever nascent hydrogen comes in contact with metallic antimony or with a soluble antimony compound.

When stibine enters the bloodstream, it reacts with the hemoglobin of red blood cells, leading to destruction of the cells (21). Stibine exerts a direct effect on the brain tissue cells, leading to various degrees of degeneration (23). Victims of stibine poisoning have experienced marked weakness, headache, nausea, severe abdominal and lower back pain, and blood in the urine. These symptoms are similar to those caused by arsine toxicity (24).

NIOSH RECOMMENDATION

In light of the serious and accidental nature of exposure to arsine and/or stibine, NIOSH recommends that steps be taken to prevent exposure to these gases. Whenever the possibility exists for either gas being generated, such as when working with metals (crude, drosses, or implements made of metal) care should be taken to assure that arsenic and antimony do not react with any sources of fresh hydrogen. Similarly, when working with arsenical compounds, care should always be taken to prevent the inadvertant generation of hydrogen gas in the presence of arsenicals. In all occupational settings where there is arsenic, workers should be informed of the possibility of arsine formation when there is nascent (freshly formed) hydrogen present. Likewise, workers exposed to antimony compounds should be informed of the possibility of exposure to stibine when freshly formed hydrogen is present.

Further research on the chronic and acute effects of exposure to arsine and stibine is needed. Although the acute toxicity of arsine in humans is fairly well defined, very little information is available on long term effects of exposure to arsine with simultaneous exposure to other arsenic compounds. In addition, more research into methods of sampling for the presence of arsine and stibine in air is needed, for both monitoring and documentation purposes.

In the event arsine and/or stibine is generated, immediate steps should be taken to remove workers from the contaminated environment. In cases of exposure, or when any symptoms are first observed, prompt medical attention is imperative. Treatment of arsine poisoning should include: (a) immediate blood exchange transfusion to replace the destroyed red blood cells, and also to remove arsenic

and the hemoglobin-arsine complex; (b) the administration of therapeutic amounts of dimercaprol (BAL); and (c) dialysis should be started if the patient has suffered kidney damage. Exchange transfusions lower blood arsenic levels, but dialysis, though it may be life-saving, does not remove arsenic from the patient. Therefore, efforts should be made to remove arsenic from the victim's body (25). Other medical support measures should be utilized as indicated.

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to **Anthony Robbins, M.D.**
Director

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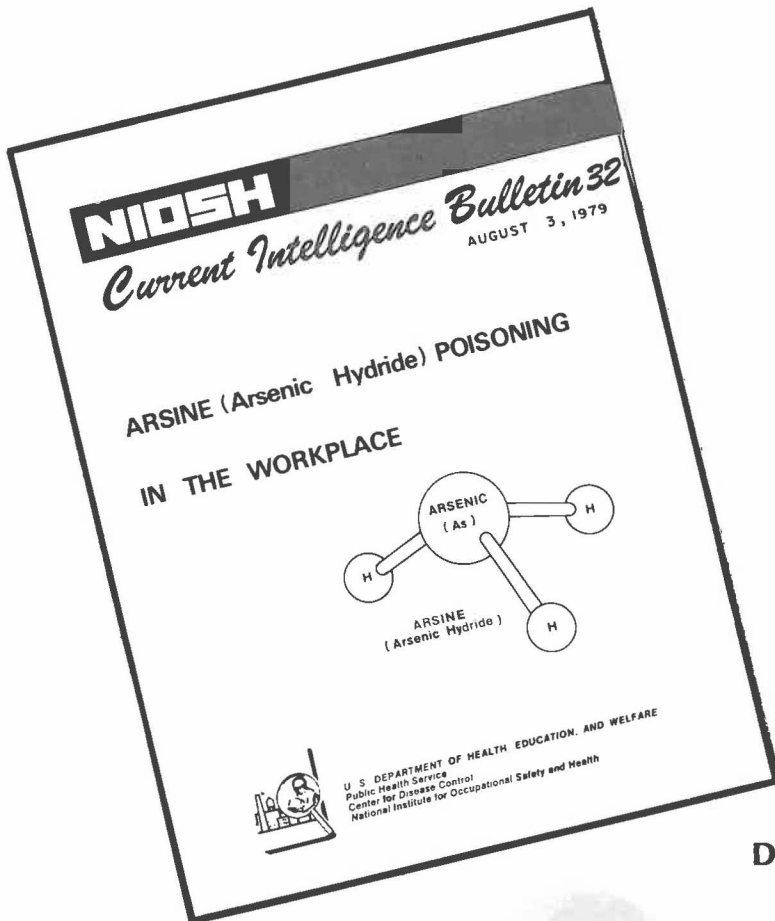
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