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OCCUPATIONAL DISEASES

A Guide to Their Recognition

Revised Edition

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PERSONAL PROTECTIVE METHODS

Workers in electrolysis manufacturing plants should be provided with respirators for protection from fluoride fumes. Dust masks are recommended in areas exceeding the nuisance levels. Aluminum workers generally should receive training in the proper use of personal protective equipment. Workers involved with salts of aluminum may require protective clothing, barrier creams, and where heavy concentrations exist, fullface air supplied respirators may be indicated.

BIBLIOGRAPHY

ARSENIC

DESCRIPTION

As, elemental arsenic, occurs to a limited extent in nature as a steel gray metal that is insoluble in water. Arsenic in this discussion includes the element and any of its inorganic compounds excluding arsine. Arsenic trioxide (As$_2$O$_3$), the principal form in which the element is used, is frequently designated as arsenic, white arsenic, or arsenous oxide. Arsenic is present as an impurity in many other metal ores and is generally produced as arsenic trioxide as a by-product in the smelting of these ores, particularly copper. Most other arsenic compounds are produced from the trioxide.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Arsenic compounds have a variety of uses. Arsenates and arsenites are used in agriculture as insecticides, herbicides, larvicides, and pesticides. Arsenic trichloride is used primarily in the manufacture of pharmaceuticals. Other arsenic compounds are used in pigment production, the manufacture of glass as a bronzing or decolorizing agent, the manufacture of opal glass and enamels, textile printing, tanning, taxidermy, and antifouling paints. They are also used to control sludge formation in lubricating oils. Metallic arsenic is used as an alloying agent to harden lead shot and in lead-base bearing materials. It is also alloyed with copper to improve its toughness and corrosion resistance.
A partial list of occupations in which exposure may occur includes:

**Alloy makers**  Lead shot makers
**Aniline color makers**  Lead smelters
**Arsenic workers**  Leather workers
**Babbitt metal workers**  Painters
**Brass makers**  Paint makers
**Bronze makers**  Petroleum refinery workers
**Ceramic enamel makers**  Pigment makers
**Ceramic makers**  Printing ink workers
**Copper smelters**  Rodenticide makers
**Drug makers**  Semiconductor compound makers
**Dye makers**  Silver refiners
**Enamelers**  Taxidermists
**Fireworks makers**  Textile printers
**Gold refiners**  Tree sprayers
**Herbicide makers**  Type metal workers
**Hide preservers**  Water weed controllers
**Insecticide makers**  Weed sprayers

**PERMISSIBLE EXPOSURE LIMITS**

The Federal standard for arsenic and its compounds is 0.5 mg/m³ of air as As. NIOSH has recommended 0.002 mg/m³ of air as As based on its carcinogenic effects.

**ROUTES OF ENTRY**

Inhalation and ingestion of dust and fumes.

**HARMFUL EFFECTS**

**Local**

Trivalent arsenic compounds are corrosive to the skin. Brief contact has no effect, but prolonged contact results in local hyperemia and later vesicular or pustular eruption. The moist mucous membranes are most sensitive to the irritant action. Conjunctiva, moist and macerated areas of skin, the eyelids, the angles of the ears, nose, mouth, and respiratory mucosa are also vulnerable to the irritant effects. The wrists are common sites of dermatitis, as are the genitalia if personal hygiene is poor. Perforations of the nasal septum may occur. Arsenic trioxide and pentoxide are capable of producing skin sensitization and contact dermatitis. Arsenic is also capable of producing keratoses, especially of the palms and soles. Arsenic has been cited as a cause of skin cancer, but the incidence is low.

**Systemic**

The acute toxic effects of arsenic are generally seen following ingestion of inorganic arsenical compounds. This rarely occurs in an industrial setting. Symptoms develop within ½ to 4 hours following ingestion and are usually characterized by constriction of the throat followed by dysphagia, epigastric pain, vomiting, and watery diarrhea. Blood may appear in vomitus and stools. If the amount ingested is suf-
ficiently high, shock may develop due to severe fluid loss, and death may ensue in 24 hours. If the acute effects are survived, exfoliative dermatitis and peripheral neuritis may develop.

Cases of acute arsenical poisoning due to inhalation are exceedingly rare in industry. When it does occur, respiratory tract symptoms—cough, chest pain, dyspnea—giddiness, headache, and extreme general weakness precede gastrointestinal symptoms. The acute toxic symptoms of trivalent arsenical poisoning are due to severe inflammation of the mucous membranes and greatly increased permeability of the blood capillaries.

Chronic arsenical poisoning due to ingestion is rare and generally confined to patients taking prescribed medications. However, it can be a concomitant of inhaled inorganic arsenic from swallowed sputum and improper eating habits. Symptoms are weight loss, nausea and diarrhea alternating with constipation, pigmentation and eruption of the skin, loss of hair, and peripheral neuritis. Chronic hepatitis and cirrhosis have been described. Polyneuritis may be the salient feature, but more frequently there are numbness and paraesthesias of “glove and stocking” distribution. The skin lesions are usually melanotic and keratotic and may occasionally take the form of an intradermal cancer of the squamous cell type, but without infiltrative properties. Horizontal white lines (striations) on the fingernails and toenails are commonly seen in chronic arsenical poisoning and are considered to be a diagnostic accompaniment of arsenical polyneuritis.

Inhalation of inorganic arsenic compounds is the most common cause of chronic poisoning in the industrial situation. This condition is divided into three phases based on signs and symptoms.

First Phase: The worker complains of weakness, loss of appetite, some nausea, occasional vomiting, a sense of heaviness in the stomach, and some diarrhea.

Second Phase: The worker complains of conjunctivitis, a catarrhal state of the mucous membranes of the nose, larynx, and respiratory passage. Coryza, hoarseness, and mild tracheobronchitis may occur. Perforation of the nasal septum is common, and is probably the most typical lesion of the upper respiratory tract in occupational exposure to arsenical dust. Skin lesions, eczematoid and allergic in type, are common.

Third Phase: The worker complains of symptoms of peripheral neuritis, initially of hands and feet, which is essentially sensory. In more severe cases, motor paralyses occur; the first muscles affected are usually the toe extensors and the peronci. In only the most severe cases will paralysis of flexor muscles of the feet or of the extensor muscles of hands occur.

Liver damage from chronic arsenical poisoning is still debated, and as yet the question is unanswered. In cases of chronic and acute arsenical poisoning, toxic effects to the myocardium have been reported based on EKG changes. These findings, however, are now largely discounted and the EKG changes are ascribed to electrolyte disturbances concomi-
Itant with arsenicalism. Inhalation of arsenic trioxide and other inorganic arsenical dusts does not give rise to radiological evidence of pneumoconiosis. Arsenic does have a depressant effect upon the bone marrow, with disturbances of both erythropoiesis and myelopoiesis. Evidence is now available incriminating arsenic compounds as a cause of lung cancer as well as skin cancer.

**MEDICAL SURVEILLANCE**

In preemployment physical examinations, particular attention should be given to allergic and chronic skin lesions, eye disease, psoriasis, chronic eczematous dermatitis, hyperpigmentation of skin, keratosis and warts, baseline weight, baseline blood and hemoglobin count, and baseline urinary arsenic determinations. In annual examinations, the worker's general health, weight, and skin condition should be checked, and the worker observed for any evidence of excessive exposure or absorption of arsenic.

**SPECIAL TESTS**

Chest X-rays and lung function should be evaluated; analysis of urine, hair, or nails for arsenic should be made every 60 days as long as exposure continues.

**PERSONAL PROTECTIVE METHODS**

Workers should be trained in personal hygiene and sanitation, the use of personal protective equipment, and early recognition of symptoms of absorption, skin contact irritation, and sensitivity. With the exception of arsine and arsenic trichloride, the compounds of arsenic do not have odor or warning qualities. In case of emergency or areas of high dust or spray mist, workers should wear respirators that are supplied-air or self-contained positive-pressure type with fullface mask. Where concentrations are less than 100 x standard, workers may be able to use halfmask respirators with replaceable dust or fume filters. Protective clothing, gloves and goggles, a hood for head and neck should be provided. When liquids are processed, impervious clothing should be supplied, clean work clothes should be supplied daily and the workers should shower prior to changing to street clothes.

**BIBLIOGRAPHY**


ARSINE

DESCRIPTION

$\text{AsH}_3$, arsine, is a colorless gas with a slight garlic-like odor which cannot be considered a suitable warning property in concentrations below 1 ppm. Arsine's solubility is 20 ml. in 100 ml. of water at 20°C.

SYNONYMS

Hydrogen arsenide, arseniuretted hydrogen.

POTENTIAL OCCUPATIONAL EXPOSURES

Arsine is not used in any industrial process but this gas is generated by side reactions or unexpectedly; e.g., it may be generated in metal pickling operations, metal dressing operations, or when inorganic arsenic compounds contact sources of nascent hydrogen. It has been known to occur as an impurity in acetylene. Most occupational exposure occurs in chemical, smelting, and refining industry. Cases of exposure have come from workers dealing with zinc, tin, cadmium, galvanized coated aluminum, and silicon steel metals.

A partial list of occupations in which exposure may occur includes:

- Acid dippers
- Aniline workers
- Bronzers
- Dye makers
- Ethers
- Fertilizer makers
- Galvanizers
- Jewelers
- Lead burners
- Paper makers
- Plumbers
- Solderers
- Submarine workers
- Tinners

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for arsine is 0.05 ppm. NIOSH has recommended that arsine be controlled to the same concentration as other forms of inorganic arsenic ($0.002 \text{ mg/m}^3$).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

High concentrations of arsine gas will cause damage to the eyes. Most experts agree, however, that before this occurs systemic effects can be expected.

Systemic—

Arsine is an extremely toxic gas that can be fatal if inhaled in sufficient quantities. Acute poisoning is marked by a triad of main effects caused by massive intravascular hemolysis of the circulating red cells. Early effects may occur within an hour or two and are commonly characterized by general malaise, apprehension, giddiness, headache, shivering, thirst, and abdominal pain with vomiting. In severe acute cases
the vomitus may be blood stained and diarrhea ensues as with inorganic arsenical poisoning. Pulmonary edema has occurred in severe acute poisoning.

Invariably, the first sign observed in arsine poisoning is hemoglobinuria, appearing with discoloration of the urine up to port wine hue (first of the triad). Jaundice (second of triad) sets in on the second or third day and may be intense, coloring the entire body surface a deep bronze hue. Coincident with these effects is a severe haemolytic-type anemia. Severe renal damage may occur with oliguria or complete suppression of urinary function (third of triad), leading to uremia and death. Severe hepatic damage may also occur, along with cardiac damage and EKG changes. Where death does not occur, recovery is prolonged.

In cases where the amount of inhaled arsine is insufficient to produce acute effects, or where small quantities are inhaled over prolonged periods, the hemoglobin liberated by the destruction of red cells may be degraded by the reticuloendothelial system and the iron moiety taken up by the liver, without producing permanent damage. Some hemoglobin may be excreted unchanged by the kidneys. The only symptoms noted may be general tiredness, pallor, breathlessness on exertion, and palpitations as would be expected with severe secondary anemia.

MEDICAL SURVEILLANCE

In preemployment physical examinations, special attention should be given to past or present kidney disease, liver disease, and anemia. Periodic physical examinations should include tests to determine arsenic levels in the blood and urine. The general condition of the blood and the renal and liver functions should also be evaluated. Since arsine gas is a by-product of certain production processes, workers should be trained to recognize the symptoms of exposure and to use appropriate personal protective equipment.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

In most cases, arsine poisoning cannot be anticipated except through knowledge of the production processes. Where arsine is suspected in concentrations above the acceptable standard, the worker should be supplied with a supplied air fullface respirator or a self-contained positive pressure respirator with full facepiece.

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ANTIMONY AND COMPOUNDS

DESCRIPTION

Sb, antimony, is a silvery-white, soft metal insoluble in water and organic solvents. The ores most often found are stibnite, valentinite, kermesite, and senarmontite.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Exposure to antimony may occur during mining, smelting or refining, alloy and abrasive manufacture, and typesetting in printing. Antimony is widely used in the production of alloys, imparting increased hardness, mechanical strength, corrosion resistance, and a low coefficient of friction. Some of the important alloys are babbitt, pewter, white metal, Britannia metal and bearing metal (which are used in bearing shells), printing-type metal, storage battery plates, cable sheathing, solder, ornamental castings, and ammunition. Pure antimony compounds are used as abrasives, pigments, flameproofing compounds, plasticizers, and catalysts in organic synthesis; they are also used in the manufacture of tartar emetic, paints, lacquers, glass, pottery, enamels, glazes, pharmaceuticals, pyrotechnics, matches, explosives. In addition they are used in dyeing, for blueing steel, and in coloring aluminum, pewter, and zinc. A highly toxic gas, stibine, may be released from the metal under certain conditions.

A partial list of occupations in which exposure may occur includes:

- Bronzers
- Ceramic makers
- Drug makers
- Fireworks makers
- Leather mordanters
- Miners
- Paint makers
- Pewter workers
- Rubber makers
- Textile workers
- Typesetters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for antimony and its compounds is 0.5 mg/m³, expressed as Sb (see also Stibine).

ROUTE OF ENTRY

Ingestion or inhalation of dust or fume; percutaneous absorption.