situation in which the arsenic was in "coke breeze" added to the plaster used in house walls."[6]

Lead arsenate, a common insecticide spray, presents an arsenic poisoning hazard for both tree sprayers and insecticide workers. However, the danger of plumbism probably exceeds that of arsenic intoxication, except where the exposure is brief but exceptionally severe.

Watrous and McCaughey, investigating the exposures of workers manufacturing and handling arsphenamine and related arsenic compounds, found average concentrations of 0.2 mg of arsenic per cubic meter of air, and about 1.0 mg of arsenic per liter of urine of exposed persons. Thus 80 to 90% of the inhaled arsenic was found in the urine. Concentrations of arsenic in excess of 2 mg per liter of urine were considered high, and a few workers with urinary arsenic levels up to 3.7 mg per liter had symptoms suggestive of arsenic poisoning.[71]

One of the most dangerous compounds of arsenic is the hydride, arsine (arseniuretted hydrogen), a gas formed by the action of nascent hydrogen on arsenic compounds.

ARSINE, AsH_3

Molecular weight: 77.9. Boiling point: −55° C.
Harmful effects: Blood changes, liver damage.
Degree: Serious, fatal.
Maximum allowable concentration: 0.05 ppm.
0.5 mg. As/l urine.
Evaluation: Air analysis.
Urine analysis.

Typical arsine poisonings follow single accidental exposures to the gas, but Bulmer and co-workers reported a group with exposures of up to 8 months' duration.[72] These occurred among employees of a gold extraction plant. Jaundice and anemia were the outstanding medical findings. The urines of the affected men contained 0.7 to 4 mg of arsenic per liter, within a few days after exposure had ceased. Assuming 50% of the absorbed arsine is excreted in the urine, 1 mg per liter (1 day's elimination) would correspond to an intake of 2 mg, or 0.4 mg per cubic meter of air, if we take 5 cubic meters of air as the average 8-hour inhalation. This corresponds to 0.12 ppm of arsine. As many of these men were quite ill, a maximum allowable concentration of 0.05 ppm would not seem too drastic.

The writer investigated one serious, but non-fatal, case of acute or subacute arsine poisoning. Analysis of urine samples, taken shortly after the exposure which lasted only 2 or 3 days, showed about 1 mg of arsenic per liter. The urine was practically blood-red in color. Arsine was demonstrated coming off the calcium hydride and metal oxide mixture, with which the patient had worked, in concentrations averaging 0.5 ppm.

The great hazard from arsine lies in its selective volatility rather than its toxicity, extreme as that may be. Thus a dust of an inert compound containing 0.1% arsenic would be unlikely to produce poisoning; but, if the material were subjected to chemical or electrolytic reduction processes, the arsenic might be volatilized almost entirely as arsine, and a hazardous concentration could result from a relatively small amount of material.

5. Columbium

Columbium is a rare element, and no case of industrial poisoning from it has been reported.

6. Antimony, Sb

Atomic weight: 121.76. Melting point: 630° C. Boiling point: 1440° C.
Important compounds: Metallic antimony, Sb_2O_3, $SbCl_3$, Sb_2S_3, SbH_3.
Harmful effects: Dermatitis, gastrointestinal disturbances.
Degree: Usually mild.
Maximum allowable concentration: (1 mg/m³.)

Although antimony compounds are toxic and antimony finds considerable industrial application, occupational poisoning from this element is extremely rare. Dermatitis caused by contact with antimony salts has been well established, but it is not widespread. Occasionally workers exposed to the dust of antimony or its compounds exhibit symptoms of gastrointestinal upset, usually acute rather than chronic in character.

Little is known of the permissible concentration for antimony. Dernehl and others found that animals subjected to 45 mg of antimony trioxide per cubic meter of air for 3 hours daily suffered from pneumonitis and liver damage.[73]

864 Nitrogen Dioxide Poisoning Due to Metal-Cutting With Oxyacetylene Torch. W.D. Norwood, et al. J. Occ. Med. 8, 301-306 (June, 1966).

Several hours after the use of an acetylene torch for metal-cutting in a poorly ventilated water main, a worker became so short of breath that he could not sleep. He reported to the plant physician 18 hours after the exposure and an x-ray film revealed pulmonary edema. Re-enactment of the event produced a level of nitrogen dioxide of 90 ppm in 40 minutes, the total oxides of nitrogen being in excess of 300 ppm. Such a level might well be expected to produce pulmonary edema. The accident was typical of the insidious action of nitrogen dioxide, which can so easily occur under some conditions and may cause death. Recognition of the latent period between exposure and the development of pulmonary edema, timely treatment with bed rest, and, if necessary, the administration of oxygen under pressure can be life-saving. A greater awareness of the sources and toxicity of nitrogen dioxide is also needed to prevent unnecessary exposure. Eight references are listed. -- Authors' summary

865 Abnormal Trace Metals in Man: Arsenic. H.A. Schroeder and J.J. Balassa. J. Chronic Diseases 19, 85-106 (Jan. 1966).

The biological activities of pentavalent and trivalent arsenic differ markedly. Pentavalent arsenic as arsenate is nontoxic in normal concentrations, is excreted rapidly, largely through the kidneys, probably does not accumulate in human tissues, is a normal constituent of food, and may perform some unknown physiological function. Trivalent arsenic as arsenite, the principal form produced commercially, is toxic, chelating with ditiol groups and inhibiting those enzymes dependent thereon. It accumulates in the mammalian body, is excreted largely from intestine, is a contaminant of soils and foods through it use in herbicides and pesticides, and performs no known physiological function. Sea foods and a few other foods and waters consumed by man often exceed the allowable limit of arsenic concentration imposed by government agencies of the United States and Great Britain, attempting to limit residues of arsenites. The bad reputation of arsenic as a poison is due to the toxicity of the commercial trivalent form and is undeserved in the case of the natural pentavalent form. There are 66 references. -- Public Health Eng. Absts.

866 Determination of Bismuth and Tellurium in Tissues by Atomic Absorption Spectrophotometry. R.E. Kinser. Am. Ind. Hyg. Assn. J. 27, 260-265 (May-June, 1966).

An atomic absorption method for the determination of bismuth and tellurium in animal tissue is described. Samples were wet-ashed with nitric acid. A hydrochloric acid solution containing 4 mg. of tissue ash per milliliter of solution, is used for the determination of bismuth and tellurium. No chemical separations are used. Effects of total salt concentration on the determination, recovery data from spiked samples, and precision of the method are discussed. Concentrations as low as 1.5 micrograms of bismuth and 2.0 micrograms of tellurium can be determined in 1 gm. of animal tissue. There are 13 references. -- Author's abst.

867 Radiochemical Determination of Metallic Mercury Vapour in Air. L. Magos. Brit. J. Ind. Med. 23, 230-236 (July, 1966).

A radiochemical method has been developed for the estimation of atmospheric mercury. When air containing mercury is passed through a solution of Hg-203-mercuric acetate and potassium chloride, isotope exchange takes place so that the issuing air contains the same concentration of mercury, but labeled and with the same specific activity as the reagent solution. The Hg-203 is absorbed on hopcalite and estimated by gamma scintillation counting. The standard deviation of the method is 0.004 microgram Hg/liter in concentrations up to 0.2 microgram Hg/liter, and is 0.075 microgram Hg/liter in the range 0.2-1.2 micrograms Hg/liter concentration. The method is simple and can be used for snap or long-run sampling, and with continuous recording. -- Author's abst.

868 Toxicity of Triphenyltin. H.B. Stoner. Brit. J. Ind. Med. 23, 222-229 (July, 1966).

The introduction of triphenyltin into agricultural practice has led to a consideration of the hazards which might arise, particularly as the related compound triethyltin is known to be very toxic to both man and animals. In the present investigation the toxicity of triphenyltin has been determined after its acute and oral intraperitoneal administration in rats, guinea pigs, rabbits, mice, and hens, after feeding it to rats and guinea pigs, and after its application to the skin of guinea pigs. The guinea pig was the most sensitive species and its growth was inhibited by as little as 1 ppm triphenyltin acetate in the diet. With higher concentrations in the diet the relationships between the dose and the survival time and between the amount consumed and the acute

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ARSENIC

Arsenic is a common ingredient of rodenticides, insecticides, herbicides, paints, and other products. Many organic arsenic compounds are employed as therapeutic agents in clinical and veterinary medicine.

Toxicology: Arsenic was formerly used extensively as a "criminal poison" because it is odorless and nearly tasteless. Accidental poisoning is still common because arsenic compounds are widely used and readily available. The mortality in acute poisoning is high (50 to 75 per cent); death usually occurs within 48 hours. The lethal dose varies with the compound, but 0.2 to 0.3 gm. of the trioxide ("white arsenic") is usually fatal in an adult (Sollmann, 1957). Finely subdivided arsenic trioxide is significantly more toxic than coarsely powdered material, since appreciable amounts of the latter may be eliminated in feces without dissolving (Schwartze, 1923). Both, however, may show up as radio-opaque material in the gastrointestinal tract (Hilfer and Mandel, 1962).

In most cases the presenting symptoms are those of a severe gastritis or gastroenteritis. Because the lesions are due not to local corrosion but to vascular damage from absorbed arsenic (Hanna and McHugo, 1960; Sollmann, 1957), the first symptoms may be delayed several minutes or even a few hours. Eventually, a violent hemorrhagic gastroenteritis leads to profound losses of fluid and electrolytes, resulting in collapse, shock, and death. Occasionally the alimentary symptoms are mild or absent, in which case the presenting complaints are usually referable to the central nervous system: headache, vertigo, muscle spasm, stupor, delirium, and sometimes mania (Webster, 1930).

Subacute and chronic exposures may reveal themselves in these and many other ways. Among the protean manifestations of chronic poisoning are anorexia, mild gastrointestinal disturbances, low grade fever, pallor, weakness, and a catarrhal inflammation of nose, throat, conjunctivae, and larynx—simulating an infectious coryza. Stomatitis and salivation are common (Cannon, 1936; Greenberg, 1949; Sollmann, 1957). Skin afflictions are many and varied: erythema, eczema, pigmentation (arsenic melanosis), keratosis (especially of palms and soles), scaling and desquamation, brittle nails, loss of hair and nails, and localized subcutaneous edema (especially of the eyelids) (Ayres and Anderson, 1934; Carleton *et al.*, 1948; Holmquist, 1951). Signs of renal damage develop. Hepatomegaly with jaundice (and sometimes pruritus) may evolve into cirrhosis with ascites (Franklin *et al.*, 1950; Wade and Frazer, 1953). Severe blood dyscrasias result from depression of any and all cellular elements in bone marrow (Eagle and Magnuson, 1946; Kyle and Pease, 1965). These effects may be related to inhibition of folic acid metabolism (Van Tongeren *et al.*, 1965). In advanced poisoning, nervous symptoms are prominent; encephalopathies have been described (Eagle and Magnuson, 1946; Prickman and Millikan, 1953), but peripheral neuritis is more common (Heyman *et al.*, 1946). Sensation is involved first (paresthesia, hypesthesia, pain), but eventually paralysis and muscular atrophy appear, usually in the legs.

In all cases it is presumably the ion of arsenious acid, rather than the element itself, which is the toxic principle. The *in vivo* conversion to arsenite explains why all chemical forms of arsenic eventually produce the same toxic syndrome. One exception is gaseous AsH_3 or arsine, which is a potent hemolytic agent, unlike other arsenic derivatives (Kensler *et al.*, 1946; McKinstry and Hickes, 1957; Pinto *et al.*, 1950; Neuwirtova *et al.*, 1961). Survivors of arsine exposure usually experience acute renal failure secondary to hemolysis and shock (Elkins and Fahy, 1967; Neuwirtova *et al.*, 1961). As arsenite the element is an active enzyme inhibitor, presumably because of its attachment to sulfhydryl groups of essential proteins (Stocken and Thompson, 1949; Voegtlin *et al.*, 1923, 1925). Trivalent organic arsenicals such as phenylarsenoxide are more potent inhibitors of certain sulfhydryl enzymes than are inorganic arsenites (Barron *et al.*, 1947; Peters *et al.*, 1946).

Absorbed arsenic is excreted largely by the kidneys, but feces, skin and hair sometimes contain appreciable amounts (Webster, 1941). After a single dose, excretion is essentially complete within 2 weeks. About 45 per cent of arsenic inhaled in cigarette smoke is excreted in the urine and about 2.5 per cent in the feces (Holland *et al.*, 1959). Urinary excretion is markedly enhanced, without damage to the excretory organs, by the administration of BAL (dimercaprol). If prompt, this treatment