

for their irritant action on the gastrointestinal system. In industry, these compounds can produce lesions of the eye, causing lights to appear to have a halo.

Antimony and its compounds are irritating to the skin and mucous membranes and are systemic poisons. It resembles the action of lead in causing a metallic taste in the mouth, vomiting, colic, loss of appetite and weight, but diarrhea is much more common. Effects of antimony resemble those of arsenic primarily in the dermatitis, which may start as an inflammation of the hair follicles and progress through pus formation and sloughing to leave a contracted scar.

Chronic inhalation of antimony trioxide produces a reduction in white blood cells and damage to the liver. Chronic exposure can produce much damage even when the total amount absorbed is less than the LD_{50} for a single acute dose.

Arsenic, in the form of arsenic trioxide, produces inflammation of the stomach and intestines and causes nervous collapse. However, cases of severe industrial poisoning are rare. There is some contradiction in the literature as to the acute toxicity of arsenic trioxide, and it is possible that this contradiction is caused by variations in impurities within the compound.

Men who handle the solid compounds of arsenic may suffer from a husky voice and cough, perforation of the nasal septum, ulceration of the skin, loss of hair and nails, warts, and sometimes skin cancers which tend to follow a slow and not very malignant course. These troubles are all severe nuisances, but they are practically never fatal and seldom disabling.

Arsenic is a highly cumulative poison, being deposited in all the tissues of the body, with the largest quantities in the liver and kidneys. There are also considerable deposits in the hair and nails where arsenic may be detected about two weeks after the beginning of exposure and where it persists for months and sometimes for years after the exposure is terminated. Inhaled arsenic is excreted to a considerable extent through the kidneys and can be detected in the urine, within a few hours of the start of the exposure and for a

long time after.

The compound BAL (2,3-dimercapto-1-propanol) was developed as an antidote for arsenical gas poisoning. It is effective in prevention of poisoning from otherwise harmful doses of the arsenic compounds in general, and in the treatment of existing cases of poisoning, although cases have been reported in which BAL was not effective.

Arsine (arsenic hydride). Severe and acute industrial poisonings with arsenic are likely to be the result of inhalation of arsine, often produced accidentally when hydrogen is generated in the presence of a material which contains arsenic. The gas has been reported in the acid cleaning of filter plates, in the handling of wet dross from a light metals plant, in the cleaning of tank cars which have held sulfuric and hydrochloric acids, in the cleaning of pipes with an acid contaminated with arsenic, in the precipitation of cadmium with metallic zinc, and in other operations.

Arsine gas causes a characteristic syndrome of massive hemolysis leading to renal failure. It is associated with nausea, vomiting, diarrhea, change in skin color, disturbance of vascular tone, electrocardiographic abnormality, and liver dysfunction. If the patient survives long enough, manifestations of chronic arsenic poisoning may appear. After exposure, a latent period of 1 to 36 hours may ensue. Inhalation of 250 ppm is instantly lethal. Exposures of 25–50 ppm for half an hour are lethal and 10 ppm are lethal after longer exposure. The mean lethal dose (LD_{50}) is unknown in man, but in small mammals is about 0.5 mg/kg. Arsine gas can be fixed by tissues other than blood. It binds to the kidneys and liver and inhibits respiration of slices of these tissues *in vitro*. In low concentration exposures, clearance of arsine from plasma, where it dissolves into the erythrocyte, is rapid and efficient. When high concentrations of arsine gas are suddenly dissolved in plasma, in large acute exposures, the amount of circulating arsine may exceed the binding capacity of the erythrocytes, and the gas may damage vital organs directly. This would explain death due to arsine poisoning before hemolysis occurs.

The possibility of arsine formation should always be considered when there is freshly