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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 elminated 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, 1963), 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