Its toxicology (table 2) has been studied experimentally and practically by Lehman [10] (1892), Haggard [11] (1925) and Sayers and associates [8] (1925). It has an irritant action on the mucous membranes somewhat similar to, but less intense than, that of chlorine and it acts on the nervous system.

Even in the concentration of about 0.005 per cent it exerts a marked irritant action on the conjunctivas, the epithelium of the corneas being eroded presumably by the alkali sulfide formed. It also acts as an irritant on the mucous membrane of the respiratory tract, causing a hoarseness in the throat, coughing, nasal secretion and pulmonary edema as the most serious consequences.

According to Haggard [11] (1925), part of the alkali sulfide formed is absorbed through the membranes of the respiratory tract and enters the blood stream, where it is hydrolyzed with liberation of hydrogen sulfide. This is rapidly oxidized to the harmless sulfate, and the systemic action is occasioned by the unoxidized hydrogen sulfide temporarily held in the blood. No sulfhemoglobin is thus formed, and generally the concentration of hydrogen sulfide cannot be measured in the blood. Because of its rapid oxidation it is a noncumulative poison.

In small amounts (about 0.02 per cent in the respiratory air) it depresses the nervous system, in larger amounts it stimulates, and in very large amounts (0.1 per cent) it paralyzes. Death in acute poisoning—if not caused by edema of the lungs—results from failure of respiration and the consequent asphyxia. According to Haggard, this respiratory failure is occasioned through two separate processes depending on the concentrations of the gas inhaled. A moderately high concentration may cause hyperpnea by stimulating the respiratory center, and the excessive breathing lowers the carbon dioxide content of the blood, and apnea results. A very high concentration, however, causes respiratory paralysis. Consequently, spontaneous breathing does not return after the paralytic form of failure unless artificial respiration is applied, but after the overstimulated form, breathing returns spontaneously.

Several deaths from acute hydrogen sulfide intoxication have been described in the literature. The course is extremely rapid, with dyspnea, disturbed coordination and unconsciousness. Autopsy findings are negative when pulmonary edema has not developed. In acute intoxications without lethal course, restitution occurs very rapidly and systemic sequelae are said to be rare.

Tolerance of the effect of hydrogen sulfide is not acquired, as with sulfur dioxide; instead, animal experiments and experience of humans indicate that hypersusceptibility may result from being exposed to the gas.

Poisonings that result only or predominantly in damage to the mucous membrane of the eyes or the respiratory tract are usually called subacute, and have been described in detail by Larsen [12] (1944), among others. The subjective troubles are intense itching, photophobia and lacrimation. Examination reveals keratoconjunctivitis and small blisters, usually peripherally, in the corneas, which heal rapidly under treatment, leaving no defect of the sight. Other lesions of the mucous membrane are usually trifling.

10. Lehman, K. B.: Arch. f. Hyg. **14**:135, 1892.
11. Haggard, H. W.: J. Indust. Hyg. **7**:113, 1925.
12. Larsen, V.: Acta ophth. **21**:271, 1944.