

- 981 Studies on the Visual Toxicity of Methanol. II. The Effect of Parenterally Administered Substances on the Systemic Toxicity of Methyl Alcohol.  
Anita P. Gilger, A. M. Potts, and L. V. Johnson. *Am. J. Ophthalmol.*  
35, 113-126 (Part 2, May 1952).

The acute toxicity of methanol and formaldehyde for white mice has been determined. The LD50 for single intraperitoneal injections of methanol was between 10.5 and 11.0 g. per kg. The LD50 for single intraperitoneal injections of formaldehyde was around 0.07 g. per kg. Antabuse increased the toxicity of methanol by about two times. Glycine protected against methanol poisoning, significantly but slightly. Cysteine did not counteract methanol poisoning. In addition, it apparently formed a highly toxic compound with formaldehyde. Under our experimental conditions, cortisone had no effect on methanol poisoning. Ethyl alcohol significantly increased the toxicity of methanol and formaldehyde and significantly lessened the time of death following methanol poisoning. Under our experimental conditions and with white mice, ethyl alcohol, a substance which has been recommended and used as an antidote for methanol poisoning, is definitely harmful from the point of view of life itself. For this reason, use of ethyl alcohol in the therapy of human methanol poisoning should be discontinued pending further investigation.

-- Authors' Summary

- 982 Acrylonitrile: Spectrophotometric Determination, Acute Toxicity, and Mechanism of Action. H. Brieger, F. Rieders, and W. A. Hodes.  
*Arch. Ind. Hyg. & Occ. Med.* 6, 128-140 (Aug. 1952).

A new simple and accurate spectrophotometric method for determining acrylonitrile in air and body fluids is presented. By means of this method the air concentrations of acrylonitrile in an exposure chamber were exactly determined and maintained. Rats, dogs, and rhesus monkeys were exposed for seven hours to concentrations of 100, 75, 50, or 25 ppm. The toxic signs were those of anoxia; death was due to respiratory paralysis. The blood color was, in general dark, but hyperoxygenation was evident soon after exposure to higher concentrations. Histologic changes characteristic of anoxia were found in the brains. Differences in susceptibility between species were confirmed. Cyanide and cyanmethemoglobin were demonstrated in the blood. This proves that the toxic action of acrylonitrile is based on the formation of cyanide in the body. The amounts found were in proportion to the intensity and length of exposure. In rats and dogs the increase of thiocyanate in plasma showed an inverse relationship to the amount of cyanide found.

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