progressively. All the results obtained suggested that the biochemical disturbances were the result of an increasing impairment of liver function. The results of this study confirm and amplify those of other workers and are significant because they show that beryllium salts are suitable agents for the experimental production of liver necrosis. The same dosage produced remarkably consistent effects in a variety of laboratory animals, namely, a rapidly spreading liver necrosis with few changes in other vital organs.

J. A. M. A.

LEAD ENCEPHALOPATHY IN A COOPERAGE. WILLIAM HAY, Brit. J. Indust. Med. **7**:177-186 (Oct.) 1950.

A cooperage is described in which a severe lead hazard caused lead poisoning in two persons; in one patient encephalopathy developed.

The syndrome of lead encephalopathy in adults is reviewed. The difficulties in diagnosis, the possible causes of apparent susceptibility to lead poisoning, the absence of hypertension in this and other cases, and the treatment of encephalopathy are discussed. Some of the essential differences between this form of encephalopathy and that due to tetraethyl lead are outlined.

FROM AUTHOR'S SUMMARY.

THE SIZE AND NATURE OF DUST PARTICLES FOUND IN LUNG TISSUE. THOMAS BEDFORD and C. G. WARNER, Brit. J. Indust. Med. **7**:187-194 (Oct.) 1950.

A shot-firer died of silicosis after many years' work in an anthracite mine. Specimens of lung tissue were examined for size distribution of dust particles. The size distributions of particles of coal and of minerals other than coal are given separately. Of all the particles measured, 0.3 to 0.4 per cent were larger than 5 microns, and particles as large as 8 microns were found in both nodules and reticulation areas. As in the dust clouds in mine air, the larger particles were of coal. Of the noncoal particles, 86 or 87 per cent were less than 0.8 microns in diameter.

The size distributions of the dust in the lung sections were similar in type to those of the air-borne dust, after shot-firing, at the colliery where the man had worked, although the proportion of very small particles was somewhat smaller in the lung sections than in the air-borne dust. It is shown that when allowance is made for the effects of particle size on alveolar retention, the observed size distributions of the air-borne dust are very close to what would be expected to account for the size distributions of the dust particles found in the lung tissues.

In nodules and in reticulation areas the particle size distributions were very similar. Rough calculations of the effects of partial solution of the dust on particle size suggest that the similarity of the size distributions should not be taken to indicate that no solution occurs after particles are deposited in the lung tissue.

ADAPTATION OF AUTHORS' SUMMARY.

FATAL CASE OF POISONING DUE TO INHALATION OF METHYL BROMIDE. A. C. MACDONALD, V. C. MUNRO, and G. I. SCOTT, Brit. M. J. **2**:441-442 (Aug. 19) 1950.

Fatal methyl bromide poisoning is reported in a young man who worked at emptying and cleaning aircraft fire extinguishers. Because of cold weather the work was done in a shed, where ventilation was inadequate. When admitted to the hospital the patient was having rapidly recurring convulsive seizures in the nature of clonic spasms. Thiopental sodium controlled the spasms for short periods. A later intravenous administration of paraldehyde with atropine immediately controlled the seizures, but the general condition deteriorated and death occurred within less than 15 hours after treatment began. At necropsy, the appearance of the organs accorded with the absorption of an acute irritant poison which had caused submucous hemorrhages of the stomach and duodenum and had acted as a convulsant, causing congestion and hemorrhage of the cerebral cortex and acute congestion of the kidneys in the process of elimination. The mode of action of the poison depends largely on the concentration. No curative treatment is known when convulsions have begun.

INDUST. HYG. DIGEST.