Asbestos haemolysis

Chrysotile (but not crocidolite, amosite or anthophyllite) haemolyses human erythrocytes *in vitro* (*Cited in F.C.T.* 1970, **8**, 210). In a study of the mechanism of this phenomenon, Schnitzer & Pundsack (*Envir. Res.* 1970, **3**, 1) inhibited chrysotile-induced haemolysis of sheep erythrocytes *in vitro* with ethylenediaminetetraacetic acid, carboxymethylcellulose and certain polystyrene sulphonate polymers, but polyvinylpyridine-*N*-oxide proved less effective. This pattern of inhibition is the opposite of that obtained with silica-induced haemolysis. Chrysotile is considered to owe its haemolytic activity to the surface area or degree of opening of the fibres. The exposed surface of chrysotile is essentially magnesium hydroxide, while the surface of the other forms is more like that of silica. Further work showed that chrysotile heated to 1000°C still retained haemolytic activity although the pattern of its inhibition was more like that of silica haemolysis than of haemolysis by unheated chrysotile. The significance of this haemolysic action to the *in vivo* situation in man is, like several other aspects of the asbestos problem, still questionable.

A COLUMN OF MERCURY: PART II

In our last issue we considered various ways in which man and domestic animals may be exposed to inorganic or organic mercury (Hg). It now remains for us to look further into the fate of this element within the animal organism and to try to relate this to its known effects.

Brain levels of Hg

While contamination of food has been shown to be a formidable hazard under certain conditions (*Cited in F.C.T.* 1971, **9**, 140) inhalation is probably still the route which offers the greatest likelihood of intoxication. The preferential uptake of Hg by the brain following parenteral administration especially via the lungs is well known (*ibid* 1967, **5**, 101), but further demonstrations of this effect have recently been reported. Cassano *et al.* (*J. Neuropath. exp. Neurol.* 1969, **28**, 308) have studied the distribution of Hg in the brain tissues of mice and rats exposed to ^{203}Hg-labelled vapour at a concentration of about 8 mg/m^3 for 6 hr daily for 10 days. Whole-body autoradiography of mice was carried out at the end of the experiment and at intervals up to 60 days afterwards. Micro-autoradiography of mouse and rat tissues was carried out immediately, and at intervals up to 50 days after the last exposure, brain extracts being measured for ^{203}Hg activity for up to 120 days. The highest Hg concentrations were found in kidney, brain and myocardium. In brain tissues, the highest concentrations were seen in certain neurons of the cerebellum (particularly in the nucleus dentatus neurons and Purkinje cells) and in the spinal cord, medulla, pons and midbrain. The general indication was that after penetrating to the brain, inhaled Hg was preferentially retained in nerve cells in two forms, probably as free Hg and bound to proteins. These two fractions appeared to be in equilibrium, since the decay of radioactivity in them showed parallel courses with time.

Nordberg & Serenius (*Acta pharmac. tox.* 1969, **27**, 269) have reported that the Hg concentration in the brains of guinea-pigs exposed for 5 hr to radioactive Hg vapour at a concentration of 7 mg/m^3 was 3–9 times higher than that resulting from 0·4 mg Hg/kg given intravenously as labelled mercuric nitrate, a finding in close agreement with the results of an earlier study in mice (*Cited in F.C.T.* 1967, 5, 101) and of another by the same