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as were the rest of the pulmonary tissues. No tubercle bacilli could be found anywhere, but throughout the lungs there were "curious bodies" which are so distinctive of asbestos particles lying in living tissues. These bodies were seen in fibrous zones, in the alveoli, and even included within giant phagocytes. This man, aged 49, had been totally incapacitated with pronounced dyspnoea on exertion and cardiac failure. Particles of asbestos were found in 10 out of 12 samples of sputum and "curious bodies" in 3 samples; such findings, however, were not associated with X-ray shadows indicative of pulmonary fibrosis. The evidence is against much danger arising from the manufacture of fibro-cement.

E. L. Collis

Bucs-Hansen, V., Frost, J. & Georg, J. **Talcosis pulmonum.** *Ugeskr. f. Laeger* 1950, Dec. 7, v. 112, No. 49, 1691-7, 2 skiagrams. [26 refs.] English summary.

In 1940, the Danish public health authorities investigated the possibility of talc poisoning in two rubber factories, and in one of them ten workers who had been employed in it from 15 to 30 years were examined with special reference to deposits in the lungs. All the skiagrams were normal, and there were no complaints of symptoms. In 1949, one of these factories was again studied with reference to talc poisoning, and five workers with the longest time of employment (from 19 to 29 years) were examined. A worker who had been employed for 29 years showed diffuse radiographic changes in the lungs. The present publication gives a detailed account of five cases in which there were radiographic pulmonary changes after prolonged exposure to talc dust during the manufacture of rubber inner tubes. Three of these workers had shown no radiographic evidence of fibrosis after respectively 6, 15 and 15 years of exposure to the dust. But after a further period of 10 years there were marked radiographic changes. None of the five workers presented any symptoms of talc poisoning, but three of them showed a slight reduction of pulmonary function. The authors recommend measures for the reduction of talc dust in the industries concerned, and they suggest that when such pulmonary changes as they found are demonstrable, it is advisable to move the worker to a less dusty employment.

Claude Lillingston

de Comnène, J. S. & Besson, J. La pneumokomose à poussières de céréales. **Pneumoconiosis due to the Dusts of Cereals.** *Arch. Malad. Professionnelles* Paris 1950, v. 11, No. 5, 459-70, 9 figs. [15 refs.]

The claim is advanced that dust from such cereals as barley, wheat and oats can cause pneumocomosis, and published work is quoted in support. Experiments are described in which guineapigs were exposed to cereal dust for ½ to 1 hour for 3 or 4 sessions per week during 100 to 245 days. The dust contained sharp fragments of vegetable hairs and such were found in the lungs of the animals. The lungs showed changes which are divided into three stages, passing from bronchiolitis with abrasion or perhaps destruction of the epithelium of the bronchi, the bronchioles containing polynuclear cells and dust particles, to a stage in which the bronchioles become either obliterated or emphysematous with thickened and sclerotic walls. These changes cause X-ray shadows passing from reticulation to nodulation. They have been ascribed to trauma from the sharp dust, or to histamines in the dust, or to bacteria introduced with the dust and gaining access through the traumatic action of the dust, or to moulds belonging to these vegetable dusts. Dusts from different cereals differ in toxicity. Search, however, for either bacteria or moulds has failed to detect the noxious agent, and the authors are compelled to blame the dust as such. No evidence of susceptibility to tuberculosis was found. The dust is generated when grain is being transhipped to land, often by mechanical elevators and chutes. Eighty-four men employed in the port of Marseilles were examined and 21 of them exhibited signs of pneumoconiosis. On first starting on the work, the men experience "cereal fever" somewhat reminiscent of "mill fever" in cotton mills, which after a few days passes off; but as years go by, from 5 to 20, bronchial attacks become more and more frequent and associated with asthmatic attacks, while the men become dyspnoeic and cyanotic. Of the 21 cases detected, 9 were in the first stage, 11 in the second, and only one in the third. Prevention lies in controlling the dust as far as possible by engineering appliances, such as closed-in machinery and exhaust draughts; and in medical examination before employment and every six months thereafter. In contrast to silicotic fibrosis with its inevitable advance, rest and freedom from exposure would seem to permit the lungs to recover slowly.

E. L. Collis

Morgis, G. G. & Forbes, J. J. **Review of Literature on Health Hazards of Beryllium and its Compounds.** *U.S. Bur. Min. Inform. Circ. 7574.* Wash. 1950, July, 23 pp. 38 refs.

This circular presents a review of literature pertaining to health hazards arising from beryllium poisoning. Beryllium occurs in nature as a minor constituent of pegmatite ores, especially of beryl, a complex aluminium beryllium silicate; it is the basis of emerald gems. It is found in many countries in small amounts and is coming into many industrial uses chiefly in alloys with copper. Such alloys have high fatigue strength, high tensile strength and electrical conductivity, hardness and resistance to corrosion. One particular use is in the production of fluorescent lamps, although for this purpose the less toxic cadmium is being substituted. Many cases of beryllosis have been reported, due to direct contact with the skin, or to the action of its dust on the lungs, or to general toxaemia. Contact with the skin will set up erythema of exposed parts and conjunctivitis; if the skin be abraded, particles may get in and cause indolent ulcers which heal