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to show the different effects individual dusts have on the lung tissues. The rabbits were placed in a special inhalation chamber, into which the dust was introduced from below. The dust was thoroughly mixed with air by passing an intermittent air current through a series of three glass cylinders, the dust being put in the first. The dust concentration was generally from 60 to 80 mg. per cubic meter of air. Inhalations lasting from one-half to one hour a day were continued for three and one-half to five months; then the animals were killed and the lungs examined both macroscopically and microscopically. The results are arranged in three series: (1) the effects of dust from steel grinding, and from porcelain, coal, chalk, and soot; (2) the effects from cement, tobacco, and schist dusts; and (3) the effects from limestone, quartz, basic slag, white lead, and cotton textile dusts.

The greatest development of pulmonary tuberculosis was caused by the dust from grinding steel, and the next from porcelain and schist dusts, all of which contain much free silica. In spite of its high content of silica, quartz was found essentially harmless. Basic slag, coal, soot, and limestone were only slightly injurious. Cement, textile, and tobacco dusts were almost noninjurious, and the same may be said for white lead dust and chalk dust ($Ca(OH)_2$), the latter of which might almost be regarded as a therapeutic agent. These results agree generally with statistics of diseases, and where variations occur they can be explained by special circumstances, such as careful vocational selection, the employment of many juveniles, and home work under bad housing conditions. A high rate of pulmonary tuberculosis had been expected from the quartz dust, owing to the high content of free silica and therefore its harmlessness came as a surprise. It seems, however, that Smith and Collis have previously shown that this harmlessness may be due to a considerable admixture with aluminium oxide, and Heffernan has shown the same among quartz workers in Derbyshire. The failure of white lead dust to produce pulmonary tuberculosis was due to the great solubility of this dust in the lung tissues.—A. J. C.

PULMONARY ASBESTOSIS. *E. Beinlker. Abstr. as follows from Arch. f. Gewerbepath. u. Gewerbehyg., 1931, vol. 2, pp. 345-358, in Bull. Hyg., Dec., 1931, vol. 6, pp. 862-863.*

The author records two cases, with postmortem examination, of pulmonary asbestosis occurring in a factory concerned with the crushing, cleansing, and spinning of asbestos and the manufacture of insulation materials.

The first had been exposed to asbestos dust from 1902 to 1922, and from 1922, when symptoms first appeared, until his death in 1929 as a porter. On X-ray examination the heart shadow was increased in diameter and the lung roots showed enlargement. The lung fields showed diffuse shadowing with small nodules about the size of a millet seed. These appearances were on the whole most marked in the left lower lobe. The patient died of heart failure. Histologic examination of the lung showed increase in connective tissue and extensive inflammation of the lung, together with asbestosis bodies.

The exposure in the second case began in 1900. There was a long period of dyspnea and the patient finally died of heart failure. The X-ray appearances were chiefly confined to the lower lobes.

In discussing the formation of the asbestosis bodies, the author is in

agreement with the general opinion that they are formed from the asbestos fiber *in situ* in the body tissues, probably as the result of a biochemical action with silica contained in the raw material. He attributes the diffuse character of the X-ray shadowing to the fact that the asbestos fibers penetrate impartially throughout the lung tissue and are not aggregated together as they are in the other forms of silicosis. He believes that the asbestos loses its magnesium portion in the tissues and that pure silica remains, which stimulates the formation of connective tissue.

In conclusion, the author notes that asbestosis of the lung differs from silicosis in its radiologic picture as well as in anatomic findings, in that the connective tissue increase is very diffusely distributed. It is arranged in nodules in silicosis, whereas in asbestosis it is distributed diffusely throughout the lung. The actual morbid anatomy picture appears to depend upon the distribution of the asbestos fibers in the lung tissue, and it is assumed that the asbestos fibers which gain access to the tissues probably do not begin their development into bodies until they reach the tissues. They are apparently the accumulation of the silica containing material. The author believes that the X-ray picture in all types of silicosis, including asbestosis, depends not so much on the quantity of the thickened tissues as on its distribution in the lung. Diffuse distribution does not give the same intensity of shadow to the radiologic picture as does the nodular distribution. He suggests that the fibrosis of the lung tissue develops, in asbestosis, through the breaking down of the material of the asbestos fibers into silica.—S. R. G.

OCCUPATIONAL INFECTIOUS DISEASES: OCCURRENCE, TREATMENT, AND PREVENTION

Discussion on the Present-Day Position of Pneumonia on the Mines. *A. Smith, F. Daubenton, P. A. Peall, L. I. Coleman, and H. Q. F. Thompson. Abstr. as follows from Jour. Med. Assn. So. Africa, 1931, vol. 5, pp. 389-396, in Bull. Hyg., Nov., 1931, vol. 6, p. 807.*

This is a report of a meeting held at Johannesburg in May, 1931. Dr. A. Smith, who read the first paper, said that since 1923 pneumonia had steadily increased despite the use of Lister's pneumonia vaccine. Since 1928 the prophylactic vaccination of new recruits had been given up at most of the mines, as it was considered that a new type of pneumococcus had developed, and its discontinuance did not seem to have any ill effect. In 1930 the number of cases increased owing, it was believed, to the increase in the new laborers taken on that year, and to the occurrence of influenza in overcrowded quarters. While the mortality from other causes had decreased of recent years, mortality from pneumonia had increased.

Dr. Daubenton called attention to the incidence rate of 3 per cent. for the Rand and 1 per cent. in other countries, and the case mortality of 11 per cent. on the Rand compared with over 20 per cent. in Britain and between 30 and 40 per cent. in the United States. There were a number of cases resembling lobar pneumonia which cleared up in from two to three days. These he