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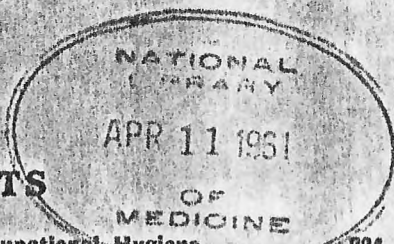
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of these cases showed the features of mesothelioma. In the ensuing 2 years 8 additional cases occurred in the same neighbourhood and 5 elsewhere in the Union. The possibility that exposure to asbestos might be a causal factor was suggested by the fact that the first case was accompanied by asbestosis and 10 of the other cases were referred to hospital from a large asbestos mining area. These observations stimulated the present inquiry. 33 cases (22 males and 11 females, ages 31-68) of diffuse pleural mesothelioma were collected and with 1 exception all had been exposed to the inhalation of asbestos dust (crocidolite). The series included workers at the mines, transport men and individuals living in the vicinity of the mines and mills. Details of the individual cases are tabulated; the items include year of birth, age at diagnosis, race, sex, asbestos exposure, diagnosis (biopsy or necropsy or both), histology and period of survival in months after diagnosis. 6 patients are still alive and in the 27 fatal cases the survival period varied between 5 and 48 months, mean 14.6 months. According to an addendum 2 months after the submission of manuscript the number of cases observed had increased from 33 to 47. In 45 of this total there was a possible exposure to crocidolite. In 1 case a mesothelioma of the peritoneum was present.

8 of the cases are described in detail and the article is excellently illustrated by pathological and histological specimens and radiographs.

The authors, quite reasonably, regard the findings as highly suggestive of an aetiological relationship between mesothelioma of the pleura and exposure to crocidolite asbestos. Nevertheless they emphasize that the evidence is not conclusive and so wish this paper to be read as a preliminary communication.

[The authors are to be commended for this original and continuing study. At the Pneumoconiosis Conference (see *Proceedings of the Pneumoconiosis Conference*, SOUTH AFRICAN COUNCIL FOR SCI. AND INDUST. RES., London, 1960, J. and A. Churchill Ltd.) held at the University of Witwatersrand in 1959, Dr. J. C. Wagner gave a preliminary account of this work. Among the final recommendations adopted by the Conference was the following (No. 12):

"That the relationship between exposure to asbestos and mesothelioma of the pleura, which appears more prevalent in South Africa, should be further investigated.

"This investigation would be facilitated by the establishment of a central registry of cases under the South African Council for Scientific and Industrial Research." (Address P.O. Box 4788, Johannesburg, South Africa.)

Between 1930 and 1946 I attended many necropsies on cases of asbestosis. These derived from a factory engaged in the processing and fabrication of asbestos textiles from crocidolite (Cape blue) asbestos. An outstanding feature of many of these cases was the gross thickening of the pleura. The appearance was peculiar in that the whole lung seemed to be uniformly ensheathed in a cuirass up to $\frac{1}{2}$ inch thick. Professor J. GOUGH, Cardiff, has produced beautiful large sections of such cases, in which this cuirass is displayed by differential staining. Only once do I recollect that Dr. S. R. GROYNE reported the pleural condition as malignant. A search of the original reports and sections at the London Chest Hospital might now produce some interesting observation.] A. Meiklejohn

BÖHME, A. Asbestose und Lungencarcinom. I. Mitteilung. [Asbestosis and Cancer of the Lung. 1st Part] *Arch. f. Gewerbepath. u. Gewerbehyg.* 1959, v. 17, No. 4, 384-95, 2 figs. [16 refs.]

The first communication deals with the results of the periodical examinations of 250 workers in an asbestos factory. Examinations carried out at intervals of 1 or 2 years revealed 92 cases of asbestosis. Investigations of a similar type had already been carried out in Dresden by BOHME and JACOB [this *Bulletin*, 1956, v. 31, 648] and by BRAUN and TRUAN [*ibid.*, 1958, v. 33, 1142]. The 92 cases comprised 32 in stage 0-1, 26 in stage 1, 14 in stage 1-II or II, and 20 in stage II-III or III. 6 patients had died of asbestosis and cancer of the lung. Autopsies were performed in all 6 and details of each are given in full. The reports of the autopsies confirm the known fact that carcinoma in asbestosis cases is generally localized to the parts most affected by the asbestosis namely, the lower lobes. In contrast to the usual experience only 1 was a squamous carcinoma, the others being either of an adenomatous or of the undifferentiated type. The carcinoma appears to arise from several centres; in 4 of the subjects metastases were present. In one patient, the cancer was not recognized during life; the appearance of a tumour may be masked by the radiological changes produced by the asbestosis. Even at post-mortem examination, the diagnosis of neoplasm may depend on the histological examination.

The age at death was on the average 51 years and the average duration of exposure was 29 years. Cancer appears in patients with asbestosis in later life and after the asbestosis has existed for many years. The author agrees that his figures are too small to allow a definite conclusion.

Charles L. Sutherland

BÖHME, A. Asbestose und Lungencarcinom. II. Mitteilung. [Asbestosis and Cancer of the Lung. 2nd Part] *Arch. f. Gewerbepath. u. Gewerbehyg.* 1959, v. 17, No. 5, 457-62.

In the first communication, 6 cases of carcinoma were described as occurring in 92 cases of asbestosis, but if the very mild cases are omitted the figures are 6 cases in 60 (10%). The author now extends his study by using data supplied by the Textile and Clothing Trade Company, Augsburg. This Company covers the insurance of the asbestos factories of north Germany with the exception of the Frankfurt area. Subjects compensated under the Fifth Regulation, No. 28 (asbestosis) and No. 28b (asbestosis in combination with carcinoma of the lung), numbered 125 during the years 1952-58. There were 31 deaths during that period and post-mortem examinations were performed on 20. In 11 of these the diagnosis of carcinoma was verified. In 7, other causes of death were found and 2 remained uncertain. In one case of the 11 not submitted to post-mortem the cause of death was given as carcinoma of the lung. In half of the verified cases of carcinoma, the condition was diagnosed or suspected during life. In a further case where the patient was still alive the diagnosis was made by resection of the lung.

Of the 125 subjects with asbestosis 63 were women and 62 men. Carcinoma was verified in only 2 of the

14 women who died, but it was found in 12 of the 17 men who died. Heavier smoking and more intense exposure to dust in men may account for this difference in incidence.

Carcinoma might have developed in a few of these persons even if there had been no asbestosis, and this might have been the case where there was a new growth of the pleura with only mild asbestosis. Moreover, in a few cases the tumour was in the unaffected upper lobes. But in general the results point to a causal connexion between the presence of asbestosis and the appearance of a carcinoma. This is admitted in the wording of No. 28b of the Fifth Regulation.

Charles L. Sutherland

KIPLING, M. D. & BECH, A. O. Talc Pneumoconiosis. *Trans. Ass. Indust. Med. Officers.* 1960, Oct., v. 10, No. 3, 85-93, 11 figs.

Talc is a hydrated magnesium silicate ($Mg_3Si_2O_5(OH)_4$ or $3MgO \cdot 4SiO_2 \cdot H_2O$). Talc is related to both asbestos and mica, the commercial form contains no free silica. Investigations in America in the 1930s left little doubt that talc dust could give rise to a pneumoconiosis on its own account. MEREWETHER [this *Bulletin*, 1934, v. 9, 230] and McLAUGHLIN [*ibid.*, 1949, v. 24, 975] investigated the disease in the rubber industry. It may be said that at present anyone working with rubber is liable to be exposed to talc dust but cases only occur when evolution of dust is high; the proportion affected is small. Cases have occurred in the pottery industry in the manufacture of the insulation of pylons, in the paper industry and the manufacture of lead accumulators. Talc produces characteristic bodies in the lesions similar to those found in asbestosis, but the author is not satisfied that every talc pneumoconiosis should be included with asbestosis in the category of fibrous silicatoses.

Details are given of 7 cases comprising 4 lead casters, 1 tyre extruder and 2 who had dusted or sprayed aluminium castings. It is unfortunate that the results of physiological tests, particularly those indicating the possible presence of alveolar capillary block, are not included.

Charles L. Sutherland

AGNOLETTI, A. & FABBIOCCI, P. A. Febbre da inalazione di polvere di madreperla. [Fever caused by the inhalation of Mother-of-Pearl Dust] *Med. d. Lavoro.* 1960, Oct., v. 51, No. 10, 612-23. [12 refs.] English summary.

This is a long and detailed report on one patient who after having worked at making buttons of mother-of-pearl for 5 years began to have febrile attacks in the afternoons, these attacks occurring one or more hours after he had restarted work after the midday break. The attacks, which at first were occasional, gradually became daily and consisted of fever up to 39° - 40° C. with shivering and headache. No other signs or symptoms were found and there were no organic changes. The attacks stopped when he was transferred to other work, only to start again when he returned to button-making. The condition was therefore regarded as definitely occupational but in

spite of the most intensive clinical and especially laboratory investigations, including inhalation of aerosols of mother-of-pearl dust, patch testing of the intact and scarified skin, the use of antihistaminics etc., no allergic condition was found, though *inter alia* the investigations showed that the concentration of Zn in the dust of the particular shell used (*Trochus macassar*) was very much higher than has been reported in the literature hitherto, while again 50% of the dust particles were less than 1μ in size, also not previously reported.

No final conclusion was reached as to the aetiology except that it was definitely occupational, and no similar case has been found in the literature.

W. K. Dunscombe

GROSS, P., WESTRICK, Marian L. & MCNERNEY, J. M. Glass Dust: a Study of its Biologic Effects. *Arch. Indust. Health.* Chicago. 1960, Jan., v. 21, No. 1, 10-23, 9 figs. [10 refs.]

The authors made a comprehensive investigation of the effects of glass dust on the conjunctiva, gastro-intestinal tract and lungs of guineapigs and rats.

In the study of possible eye irritation the conjunctiva was dusted with finely milled glass flakes of average thickness 5μ and mostly less than 50μ in diameter. No ill effects were demonstrated.

In the ingestion studies, similar milled glass flakes to the amount of 10% in one group of young rats and 50% in another were added to the normal diet for one year. There were no significant changes in health. All of the 10 rats which ingested such massive doses of glass as the equivalent weight of their daily food survived the year's experiment. Post-mortem examinations in both groups showed no abnormalities.

The pulmonary studies consisted of intratracheal injections of coarse glass suspended in corn oil, of corn oil itself, and of fine glass (mean diameter 1.1μ), quartz and kaolin, each suspended in water. [Corn oil had to be used as the suspending medium for the coarse glass to counteract the rapid settling which occurred in water.] 15 rats were used for each type of injection, 3 being killed from each group at the end of 6 months and survivors at the end of 12 months. Inhalation studies were also conducted with fine glass, quartz and kaolin on 3 groups of 40 rats and 3 groups of 15 guineapigs, exposures continuing for one year and examinations being made at 6 months, one year and two years after starting exposure.

The pulmonary reactions to both coarse and fine glass were minimal and consisted of small focal alveolar collections of macrophages with a little proliferation of alveolar lining causing slight thickening of alveolar walls. The lesions are described as non-fibrogenic although in some a few collagen fibres were noted. Glass dust deposits in lymph glands were completely inert. Quartz produced less marked lesions than expected, silicosis being not apparent or very slight on naked eye examination, while microscopically the consolidations were more cellular, irregular and contained less collagen. The lesions produced by kaolin were similar to but slightly more severe than those produced by glass dust. The possible reason for the unexpected inhibition of silicotic fibrosis