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- 886 Cancer of the Lungs and Other Diseases After Exposure to Asbestos Dust. K. F. W. Hinson. Brit. J. Diseases Chest 59, 121-129 (July, 1965).

That exposure to the fibers of asbestos may cause a disabling and fatal fibrosis of the lungs has been recognized for many years. That patients with an established fibrosis (asbestosis) have high incidence of bronchial carcinoma is also known. During the last few years there has been an accumulation of evidence to suggest that certain pleural or peritoneal tumors may be associated with a slight, and therefore possibly unrecognized, exposure to these minerals. The world production of asbestos has been increasing rapidly since 1959 and its industrial uses are becoming more varied. Three varieties of asbestos are most often used industrially: chrysotile (white asbestos) crocidolite (blue asbestos), and amosite. In Great Britain, exposure to asbestos dust might be to any or all three of the above kinds of asbestos; on the North American Continent it would probably be to chrysotile because of the huge native deposits found in Canada. The author discusses briefly the pathogenesis of asbestos, but dwells much more on the possible relationship of asbestos exposure and pleural tumors and tumors of the peritoneum. He also reviews the literature concerning the probable association between asbestosis and carcinoma of the lungs. Mentioned are the findings of J. G. Thomson and his co-workers (Industrial Hygiene Digest, Abst. 663, July 1963) of the high incidence (26.4%) of asbestos bodies found in the lung fluid of 500 consecutive autopsy subjects in South Africa. One of the authors of the paper on those findings is said to consider that the incidence in Miami, Florida, would be even higher.

-- APCA Absts.

- 887 Asbestos Related Disease. H. L. Hardy. Am. J. Med. Sci. 250, 381-389 (Oct. 1965).

Four cases of asbestos related disease have been briefly described. Together with past and presently accumulating evidence, epidemiologic and experimental, these cases illustrate the potential hazard posed by asbestos exposure to both worker and neighbor. The purpose of this report is to interest clinicians in the harmful biologic effects of asbestos already documented. Evidence is impressive that asbestos exposure, often delayed in effect, will in the future be responsible for an important amount of pulmonary disease, especially chest tumors. In addition new evidence points to asbestos exposure as being significantly associated with abdominal tumors as well.

-- APCA Absts.

- 888 Chemical Studies of Asbestos. J. S. Harington. Ann. N.Y. Acad. Sci. 132, 31-47 (Dec. 31, 1965).

Further chemical studies of the three main types of asbestos, crocidolite, amosite, and chrysotile have been made. The origin of the primary oils associated with crocidolite and amosite has been discussed. By suitable methods 0.32% of extractable material can be removed from crocidolite and 0.28% from amosite. A sample of commercial amosite known to be carcinogenic yielded 0.76% of extractable material. Chemical investigations have been made of the oils from crocidolite and amosite by various techniques. Low levels of polycyclic aromatic hydrocarbons were detected. In addition to the primary oils, asbestos may contain secondary oils derived from contamination or from the intentional application of oil during processing in industry. Up to 80% of the jute oil in the jute bags used for storing asbestos may be absorbed by the asbestos fiber. This could explain why chrysotile, which apparently has no primary oils, may contain, after milling and other industrial treatment, yields of from 0.1 to 0.4%. Insulation materials also contain significant amounts of extractable material. Finally, studies have been made to assess wherever possible, the part which metals and metal complexes may play in the carcinogenic action. --APCA Absts.

- 889 Some Observations of the Dust Content and Composition in Lungs With Asbestosis, Made During Work on Coal Miner's Pneumoconiosis. G. Nagelschmidt. Ann. N.Y. Acad. Sci. 132, 64-76 (Dec. 31, 1965).

In the conclusion of this paper it is said that the most interesting result of this study of lung tissue from asbestosis sufferers, was that in over half the lung tissue samples from Great Britain and South Africa practically no asbestos at all was found irrespective of the grade of fibrosis. It is unlikely that this was due to faulty technique although further study of the technique of recovery of asbestos, especially of chrysotile, from lungs is undoubtedly required. It is likely that this effect—the disappearance of asbestos from the lungs—is real and needs to be explained. The most likely explanation is that asbestos does get dissolved in the lungs. The process of dissolution is likely to start with a removal of the cations, magnesium, sodium, iron, and aluminum, which would leave a skeleton of hydrated silica or polysilicic acid. On physicochemical and structural reasoning one would expect chrysotile to be most easily attacked, crocidolite to be intermediate, and amosite to be the most resistant form. The finding of amosite only in some of the lung residues would be in agreement with such reasoning. The most prevalent type of fibrosis seen in asbestos is a diffuse interstitial fibrosis, in contrast to focal or nodular lesions

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found initially in most other forms of pneumoconiosis. At a later stage massive lesions may develop. A theory linking this contrast with lung dust studies has been advanced (Nagelschmidt, 1960). According to this theory the dust remaining in the lungs causes the fibrosis in coal workers and similar forms of pneumoconiosis, and a positive correlation between amount of dust and severity of fibrosis or x-ray category of simple pneumoconiosis is found. The amounts of dust in the lungs are large and may average 50gm. for advanced stages of coal workers pneumoconiosis. In diffuse interstitial fibrosis, as seen mainly in aluminosis and asbestosis, this does not seem to be the case; the amounts of asbestos found in the lung are very small and are not clearly related to grade of fibrosis; dissolution of the dust is the most likely explanation. Similar views with regard to the action of chrysotile have already been expressed. Further research is obviously required before the mechanism by which asbestosis is produced can be said to be known.

-- APCA Absts.

- 890 Asbestos Dust Deposition and Retention in Rats. J. C. Wagner and J. W. Skidmore. Ann. N.Y. Acad. Sci. 132, 77-86 (Dec. 31, 1965).

A method of producing asbestos dust clouds has been devised and an animal inhalation experiment carried out to test it. Observations on the histological distribution of the dusts in the lungs of the rats have shown that the dusts tend to accumulate in the alveoli arising directly from the respiratory bronchioles. The weights of the asbestos dusts found in the lungs of the rats at the end of exposure were found to differ considerably, but the subsequently determined elimination rates indicated that the weights of dust deposited were in a constant ratio with the weights of dust collected by the size-selective sampler. To deposit an equal amount of a nonfibrous dust, allowance must be made for the reduced efficiency of the upper respiratory tract in preventing the penetration of the compact dust particles to the alveolar regions. The elimination rate of Rhodesian chrysotile has been found to be three times greater than that of amosite and crocidolite, which suggests an explanation for the previously observed reduced fibrogenicity of this dust. The reason for the difference in the elimination rate remains to be determined.

-- APCA Absts.

- 891 Experimental Asbestosis With Four Types of Fibers. Importance of Small Particles. P. F. Holt, J. Mills, and D. K. Young. Ann. N.Y. Acad. Sci. 132, 87-97 (Dec. 31, 1965).

The guinea pig lung reacts immediately to inhalation of asbestos dust. There is a bronchiolitis with extension of the inflammatory reaction to the adjacent alveoli. At a later stage there is a widespread and progressive fibrosis of the lung, adenoid proliferation of the bronchiolar epithelium, and reticulosis and fibrosis of the tracheal lymph glands. The inhaled dust early becomes coated with an iron-containing protein to form asbestos bodies. These have been recognized within seven days of exposure to the dust. Fine dust particles, too small to be seen under the light microscope, will produce asbestosis in the guinea pig. The difficulty of controlling the spread of dust has been demonstrated.

-- APCA Absts.

- 892 Quantitative Determination of Chrysotile, Amosite and Crocidolite by X-ray Diffraction. J. V. Crable. Am. Ind. Hyg. Assn. J. 27, 293-298 (May-June, 1966).

The determination of asbestos by x-ray diffraction in environmental samples is described. Evenly distributed mats of asbestos samples on molecular membrane filters are prepared for x-ray diffraction examination. Each asbestos mineral has its characteristic x-ray diffraction pattern. A qualitative scan of a mounted filtered sample identifies the crystalline substances present including the asbestos minerals. A quantitative determination of a given type of asbestos is made by measuring the area under its major diffraction peak and comparing this area with that of a known quantity of an external standard.

-- Author's abst.

RADIOACTIVITY AND X-RADIATION

- 893 Radiation—A Look at the Past and the Present. I. L. Beauchamp. J. Occ. Med. 8, 329-337 (June, 1966).

The 300 years prior to the discovery of x-rays and natural radioactivity were punctuated with scientific breakthroughs that brought the state of scientific knowledge to the point where x-rays were observed by an alert scientist. Following Roentgen's discovery of x-rays and the discovery of radioactivity by Becquerel, the use of radiation expanded rapidly in research and medicine. Many scientists, physicians, and patients were injured because the need for protection and dosimetry was unrecognized. No satisfactory theory had been advanced at the time to explain the nature of x-rays and radioactivity. Several writers surveyed the literature in that early