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dust occurs. Laboratory experiments on animals, carried out in the author's laboratory, have shown that exposure of rats to high concentrations of silica dust does not lower their resistance to pneumococci Type I, injected intrabronchially. In fact, under some conditions, the rats were more resistant to the lobar pneumonia following prolonged exposure to silica dust. The relation of silicosis to bronchopneumonia is not clear.

TREATMENT AND PREVENTION. There is no known treatment for silicosis which will reduce the fibrosis, although relief of symptoms to some degree may be obtained in some cases. Because aluminum dust produces a coating on silica particles, the inhalation of aluminum dust has been tried as a therapeutic measure. The Council on Industrial Health and the Council on Pharmacy and Chemistry of the American Medical Association summarized the available data in 1949. Their conclusions were as follows: "Studies on the therapy of silicosis thus far have been inadequately controlled. The majority of subjects have reported subjective improvement, apparently of psychic origin. No convincing evidence of objective improvement either of pulmonary function or by roentgen ray has been forthcoming. Certain cases have shown eventual progression by roentgen ray subsequent to aluminum therapy under present conditions of dosage." (Brown and Van Winkle, 1949.)

Prevention is the only positive means of attack on silicosis. With modern engineering technics, there is no reason why silica exposures cannot be reduced to safe concentrations and silicosis completely eliminated in the course of a few years. Until that time comes, all persons working in such atmospheres should be examined clinically and radiologically at frequent intervals by a competent physician. Those with silicosis should be protected from further exposure to silica dust. Persons with tuberculous lesions should not be employed where silica exposures exist.

Measures for the prevention of exposure to dusts and fumes, including silica dust, are discussed under prevention of occupational diseases due to chemical substances, on pages 1086 to 1091.

ASBESTOSIS

Asbestosis is a pneumoconiosis due to the inhalation of asbestos dust.

Nature of Asbestos. Asbestos is a general term which is applied to minerals of fibrous form. These minerals are silicates of varying composition which fall into two distinct groups, serpentine and amphibole. Chrysotile, a hydrous magnesium silicate of the serpentine type, is by far the most common type of asbestos. The amphibole group includes various silicates of iron, calcium, magnesium, and, in some cases, sodium; examples are crocidolite and tremolite. Asbestos fibers are usually from 20 to 500 microns in length, and from 0.5 to 50 microns in diameter. The fibers of crocidolite are much stiffer and straighter than those of chrysotile.

Reaction of Tissues to Asbestos. Pulmonary fibrosis results in man and in some animals from the inhalation of chrysotile, the serpentine type of asbestos. This fibrosis differs from that which occurs in silicosis, being a fine, interstitial, diffuse fibrosis around the terminal bronchioles, rather than nodulation. It is believed that fibers up to 200 microns in length can penetrate the respiratory tract if the maximum diameter does not exceed 5 microns. Fibers which are inhaled accumulate in the lumen of the respiratory bronchioles and later in the alveolar ducts, but do not seem to penetrate to the alveoli.

It is generally believed that the fibrosis is due to the mechanical irritation of the long fibers (between 20 and 50 microns in length). Presumably the motion of the lung tissue in breathing, combined with the filamented structure and the flexibility of the fibers are responsible for this irritating effect. This theory is supported by the fact that asbestos fibers, unlike silica dust, are inactive when in contact with other tissues, such as the liver, where there is no mobility, and by the fact that they do not cause pulmonary fibrosis if the fibers are short or are ground to small particle size. The stiff, straight fibers of crocidolite do not stay in the walls of the bronchioles, but pass on into the alveoli, and do not appear to cause fibrosis. Fibrosis occurs only where there is mechanical rubbing of the tissue against the fibers. Commercial talc or its component materials, such as tremolite, after prolonged exposure appears to cause a fibrosis resembling asbestosis.

Clinical Aspects. The clinical symptoms are due to the fibrosis and the concomitant pathological changes, chiefly emphysema and bronchiectasis. Progressive dyspnea, which can lead to disablement, is the chief symptom. Cough and loss of weight also occur. Death is due to secondary respiratory infection or to cardiac involvement. It is generally believed that the fibrosis will not progress after cessation of exposure.

Diagnosis of asbestosis must rest on a history of exposure to asbestos dust, on the clinical symptoms, and on the radiological examination. The presence of asbestosis bodies in the sputum is not of any diagnostic value. Asbestosis bodies are fibers which are surrounded by an iron-containing coating and which have fractured to give peculiar structures. They may be formed from any fibrous material, including asbestos, but are neither indicative of the degree of exposure nor associated with the fibrosis.

Although some cases of tuberculosis have been reported in asbestos workers, there is no convincing evidence, either clinical or experimental, that asbestosis increases susceptibility to tuberculous infection. A number of cases of lung cancer associated with asbestosis have been found in various autopsy studies especially in Great Britain. Authorities differ in their opinion regarding the possible role of asbestos in the etiology of lung cancer.

There is no known treatment for asbestosis; prevention of exposure to asbestos dust is the only effective weapon against this condition.

Incidence of Asbestosis. The incidence of asbestosis in persons working in environments containing asbestos dust varies with the extent and duration of exposure. The disease does not usually appear until after 5 to 10 years of exposure. The incidence of asbestosis increases progressively with the duration of exposure. More than half of the workers with 20 years of exposure have the disease. Studies in asbestos industries indicated that 5,000,000 particles per cubic foot of air is the maximum safe concentration, but recent work suggests that the standards may vary with the percentage of long particles in the dust.

OTHER HARD ROCK AND MINERAL DUSTS

Siderosis. Exposure to iron oxide produces a benign pneumoconiosis called siderosis. "By itself this mineral provokes no significant fibrosis but merely pigmentation, which has no influence on pulmonary function" (Gardner, 1940). However, roentgenograms of workers exposed to the iron oxide dust show nodular shadows simulating those of silicosis. Clinical studies of these workers, autopsy