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A survey of five industries, covering 15,587 persons and including 1,357 cases of silicosis was made by the Saranac Laboratory. Only one case of pulmonary carcinoma was found in the silicotics (0.074 per cent), and two in the nonsilicotics (0.014 per cent). Autopsy observations made on native and European miners in South Africa, many of whom had silicosis, showed similar negative results. Furthermore, animal experiments also have given no evidence that lung tumors result from exposure to silica dust. Thus, there is no evidence that lung cancer is related in any way to silicosis.

SILICOSIS AND PNEUMONIA. There is no evidence, at the present time, to indicate that exposure to silica dust increases susceptibility to lobar pneumonia. Although high mortality and morbidity rates for pneumonia have been reported in some silica industries, such as in foundries and mines, factors other than silica dust appear to be responsible for this. Furthermore, abnormally high pneumonia rates have not been found in other industries and mines where exposure to silica 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.

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. Opinions differ regarding the value of aluminum in the therapy of silicosis. 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.

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

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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. Aluminium, which is effective in preventing the action of silica dust is not effective in preventing 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 few cases of lung cancer associated with asbestosis have been found in various autopsy studies. Whether these cases are only chance occurrences or whether they are related to the asbestos exposure has not been proven as yet.

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 findings in cases where death resulted from other causes, and animal experimentation have proven that these shadows are not due to fibrosis but to the radio-opaque iron oxide which had accumulated in foci throughout the lungs. Studies on animals indicate that iron oxide dust does not cause fibrosis or affect the course of experimentally induced tuberculosis.

The inhalation and deposition of tin oxide in the lungs may also produce similar pseudonodulation in the roentgenograms.

Anthracosis. Coal dust, in the absence of free silica, has been considered to produce only a benign type of pneumoconiosis. Recent observations have indicated that some lung changes may result from exposure to certain types of coal dust. The significance of these findings cannot be evaluated at present.

DUSTS OF PLANT ORIGIN

General Effects. Public health officials are often asked if grain, wood, cotton and other dusts of plant origin produce any harmful effects on the lungs or other tissues. In general, it can be said that these natural organic dusts may cause allergic reactions in susceptible persons leading to a variety of conditions in the respiratory tract, skin or other tissues, and that many of these are irritating to the respiratory tract and skin even when no allergic reactions are visible. In some cases, the effects are due to the smuts, mites, fungi or other parasites which contaminate these dusts. In other cases, the irritation is due to the physical rather than to the chemical properties of the dust. Many natural organic dusts present an additional hazard due to their high degree of inflammability. Dust clouds of flour, wood or tobacco may be explosive.

There is no evidence that any of these dusts produce fibrosis of the lungs, such as is found in cases of silicosis or asbestosis. In spite of statements in the literature that a high rate of tuberculosis exists among workers exposed to cotton, tobacco, and grain dusts, and that exposure to these dusts is partly responsible for this rate, there is no sound evidence to support this belief. Tuberculosis rates are not consistently high throughout the occupations which involve exposure to these natural dusts, and a low standard of living appears to be the major factor in those instances where a high tuberculosis rate does occur. There is also no evidence at the present

