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Inhaled Mineral Dusts

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AS LONG as men have worked in stone it has been appreciated that an unusually large proportion of them suffer from disease of the lungs. It was natural to assume that such disease would be caused by the dust generated, and this belief was strengthened by the discovery of black, grey or red pigments in the lungs, sometimes accompanied by the formation of scar tissue.

The term pneumokoniosis was introduced to describe the lung pigmented in this manner. With further observation pathologists attempted to classify various forms of pneumokoniosis on a basis of the type of dust inhaled and such names as anthracosis, siderosis, chalicosis and even byssinosis and tobaccosis made their appearance. It was recognized that some forms of the disease were attended by severe symptoms and resulted in death but there was no correlation between the clinical and pathological pictures and the causative dust. Finally statistical study demonstrated that of all kinds of industrial dusts silica generally produced a definite type of disease with a characteristic pathological lesion and complex of symptoms.

Today the clinical entity known as silicosis is one form of pneumokoniosis which is quite clearly defined. Moreover, within the last three or four years asbestos dust has also been shown to produce another specific type of reaction attended by symptoms differing in some respects from those of silicosis.

The other forms of pneumokoniosis still lack complete definition. The widespread use of radiography has brought together a mass of descriptive data from individuals exposed to many kinds of occupational dusts but these findings have

not yet been correlated with pathological anatomy, chemical studies of the tissues and detailed analyses of industrial atmospheres. There is a tendency to assume that many of these pulmonary changes are due to the action of silica perhaps modified by the chemical components of the dust. We shall only be in a position to speak with assurance about them when the pure types of dust and their various combinations have been studied in human beings or in experimental animals.

For the sake of clarity no mention has been made of the infections which so frequently complicate some forms of pneumokoniosis. In the prebacteriological era of medicine the use of such terms as "grinders' consumption" and "miners' phthisis" reflects the popular association of pneumokoniosis with tuberculosis. The names were justified both by clinical and pathological observation for it has been subsequently shown that, in silicosis at least, a super-imposed tuberculosis may cause death in perhaps 75 per cent of the cases. Some observers even go so far as to state that silicosis itself does not develop unless the lungs are previously damaged by a latent tuberculous infection. This is probably an exaggeration, for typical silicosis can be produced in the experimental animal by the inhalation of silica without infection.

Asbestos Dust

The role of asbestos dust as an excitant of tuberculous infection is not as clear cut as that of pure silica. While there are numerous reports of death in asbestosis due to a terminal tuberculosis, nevertheless surveys of living workers have failed to demonstrate any excess of such infection. Simple anthracosis is said to prevent the progression of tuberculous infection. Statistics from coal mining districts do show a tuberculosis rate

much lower than that "normal" for the age group. As yet corroborated experimental proof of protective action of coal against tuberculous infection is lacking.

Silica and Tuberculosis

Not only the tubercle bacillus but other bacteria seem to develop with special facility in the tissues previously damaged by silica. For example the pneumonia rate among silicotic native laborers of South Africa is many times that in non-silicotic natives living under similar conditions. In coal miners, likewise, pneumonia is a common and often fatal complication. Case reports of fatal pneumonia in asbestosis are frequent but morbidity statistics from large groups of asbestos workers are lacking. For most other dusts the data are even more fragmentary.

What has been said of the effects of inhaled inorganic dusts would indicate that the reaction of the tissues is not merely a response to mechanical irritation by particulate matter. Today it is generally accepted that the injury is chemical in nature and that only certain of the common types of industrial dusts possess properties capable of exciting reaction. In the cases of silica and the silicate of magnesium or asbestos the slightly alkaline body fluids probably effect a slow solution of the dust particles liberating silica in colloidal form. This substance irritates the connective tissue cells which respond by multiplying. The result is an overgrowth of the supporting framework elements at the expense of the more delicate cells specialized for specific functions.

For these two types of dust the character and form of the pathological changes has been carefully worked out. In *silicosis* the essential tissue change consists of nodules of connective tissue which develop

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first in the lymphoid tissues situated in the drainage apparatus of the lungs. These nodules interfere with the physiological removal of foreign bodies and subsequently inhaled particles accumulate in the framework of the lung itself. Such reaction gradually decreases the normal elasticity of the organ and encroaches upon its functional elements. As a result the cardinal symptom of the disease, dyspnoea or shortness of breath develops. Because of the excessive amounts of scar tissue formed in the lungs the right side of the heart encounters increasing difficulty in forcing blood into the organs. In an attempt to compensate the overworked heart muscle becomes thicker and heavier and unless infection intervenes the time eventually arrives when the heart fails and death occurs from this cause.

Obscure Causes

The reasons for the prevalence of infection in the silicotic lung are not altogether clear. It is obvious that when the drainage system is so obstructed by nodules of scar tissue that more dust particles can only be removed with difficulty, the same must be true of bacteria which may be inhaled. As a consequence they remain in the lung and set up progressive infections. But this is not the whole story. The silicotic tissue apparently offers a peculiarly favorable soil for the continued multiplication of tubercle bacilli and perhaps other bacteria. Experiment has shown that even attenuated organisms of this type will produce rapidly fatal tuberculosis in a silicotic guinea pig. Furthermore, inhaled silica dust will cause a preexisting latent tuberculous focus, harboring a few attenuated bacilli, to become progressive and spread.

The cause of this effect upon tuberculous infection has not yet been ascertained but the writer believes that it is in some way associated with the death of tissue caused by the toxic silica. Associated with this factor is an increased activity of the phagocytes which are stimulated by the irritating silica so that these cells tend to migrate rapidly and carry tubercle bacilli into previously uninvolved portions of the lung.

Asbestos dust, perhaps because of its fibrous structure, is not transported very far within the lung. It tends to lodge along the walls of the

finer tubes and little of it is removed by the lymphatic drainage system. Tissue reaction, probably initiated by the solution of the fibers, occurs in their immediate vicinity. As in the case of silica this reaction consists of an overgrowth of the connective tissues which is later transformed into leather-like scars. The arrangement of the scar tissue in asbestosis is quite different from the nodular collections in silicosis because of the difference in localization of the foreign particles. In asbestosis the deposition along the walls of fine tubes radiating from larger bronchi in all planes throughout the lung gives rise to a fine linear fibrosis with little or no nodule formation.

Aluminum oxide will serve admirably as an example of a non-siliceous dust whose particles are as hard and sharp as those of crystalline silica. When a measured quantity of such particles, 1 to 3 micra in diameter, are injected into the ear vein of a rabbit the majority of them come to rest in its liver and spleen. There they remain, apparently harmless, collected in large phagocytic cells and producing no reaction of the connective tissues for at least two years. Injection of the same quantity of the same sized quartz particles excites the formation of so much scar tissue that the functional elements are almost completely destroyed.

Carborundum, the carbide of silicon, when inhaled by guinea pigs for periods as long as four years, likewise fails to excite significant overgrowth of the connective tissues. It produces only a low grade inflammatory change with no nodules.

Instances such as those cited constitute the basis for believing that the cellular response to dust particles is not due to their hardness and sharpness but to chemical substances liberated by the action of the tissues upon them. The case for solubility has not been proven directly, for the detection of soluble silica in the tissues offers technical difficulties which today are unsurmountable. Nevertheless this substance is known to be a cell poison and in weak concentrations it will provoke an overgrowth of the connective tissues.

Coal Dust

Mention should be made of the effect of inhaled coal dust. Most city dwellers breathe in sufficient quantities of this material during an average

life-time to pigment considerable areas of their lungs. Deposits of grey or black dust will be found along the course of the lymphatic drainage system but this pigmentation is associated with little or no new growth of connective tissue. The coal miner's lung is much blacker but in most instances it also develops no deforming scars. When such reaction does occur analysis usually reveals appreciable amounts of silica in the lungs. The presence of excessive quantities of carbon dust apparently influences the localization of moderate amounts of silica so that the latter is not deposited in nodules as in pure silicosis but in streaks along lymph vessels as pure coal would localize.

Coal miners' fibrosis, due to relatively small amounts of silica, is therefore different from that of the pure quartz worker. When the amount of inhaled silica is excessive, the effect of the coal is negligible and the reaction approximates that in pure silicosis. There is said to be more reaction to hard coal than to the bituminous variety but there is evidence to suggest that this is due to the greater amount of siliceous rock which must be worked in mining anthracite coal.

Finally, consider the silicosis of granite cutters. This develops somewhat more slowly than that of the pure quartz miner and it has been maintained that this is due to the relatively small amount of uncombined silica in granite (25 to 40 per cent). But it is the writer's belief that other components of the glomerate granite may neutralize or at least inhibit the effects of the silica so that only after prolonged exposures to high concentrations does significant reaction occur. While typical silicotic nodules develop in guinea pigs inhaling pure quartz for a year, not even the earliest suggestion of nodules has appeared in the lungs of animals inhaling comparable concentrations of granite (40 per cent silica) for four years.

This very brief discussion of some of the problems involved in pneumoconiosis draws attention to the limitations of our present knowledge. It indicates that the reaction to an inhaled dust is determined by the chemical and probably physical composition of that dust. It emphasizes the need to further study of different types of dust both in the pure state and in measured combinations.