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DUST INHALATION IN RELATION TO PULMONARY DISEASE

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Everyone is inhaling some dust with almost every breath taken, but the results of this dust upon the lungs is considered part of the natural process of aging when it occurs outside of his occupation. The relationship of dust inhalation to pulmonary disease is therefore usually considered only from the point of view of industrial exposure. Observations on the effect of dusts in industry have consequently been limited largely to those pathological processes which can be clearly differentiated from pulmonary diseases of non-occupational origin. To these characteristic changes in the lungs produced by dust, the term pneumoconiosis has been applied.

DIAGNOSTIC FACTORS IN PNEUMOCONIOSIS

In order to establish the diagnosis of pneumoconiosis, two factors must be present: 1. pulmonary pathology which might have been produced by exposure to a particular dust, and 2. an occupational history of exposure to that dust. The chest physician is generally in a good position to evaluate the first factor, through the medium of signs, symptoms, x-rays and laboratory tests. In determining the second factor, he is often handicapped by his paucity of knowledge of the nature of his patient's work and the materials handled. Most physicians are aware that a rock miner or stone cutter may be exposed to free silica in amounts capable of causing silicosis. They may not be aware, however, that a worker in a soap factory, which includes in its products scouring powders, may be handling large amounts of silex, which is almost pure pulverized silica. In other instances, the etiology of the pulmonary pathology may not be so readily recognized because the physician is unaware of the more obscure effects upon the lungs of a material such as talc or bagasse.

Another factor which makes it difficult to recognize the importance of an industrial dust exposure in the pathogenesis of a pulmonary disease is the close resemblance which frequently exists between that disease and some other pulmonary pathology of non-industrial etiology. When the x-ray picture is characteristic, as in an obvious case of nodular silicosis, the physician is apt to inquire very carefully into the worker's occupation, and to enlist the aid of industrial hygienists or other experts in an effort to establish a dust etiology. When the clinical and sometimes even the pathological findings are almost identical with some non-occupational disease of the lungs, however, he is not so likely to seek far afield for an occupational exposure. It was not until the unusual incidence of workers dying from what was presumed to be Boeck's sarcoid in a plant manufacturing fluorescent lamps was noted, that the toxicity of one of the materials which they were handling was even suspected, and that the identity of chronic pulmonary granulomatosis due to beryllium was established.[1] Recognition of the dust etiology is even more difficult when the pathology is identical with that of a non-occupational disease, as for instance the primary lung carcinoma seen with increased frequency in chromate workers.[2]

Another way in which dust inhalation may affect pulmonary disease is by aggravation of a pre or co-existing non-occupational disease. Today, the fact that silicosis predisposes a worker to tuberculosis, and renders that tuberculosis much more severe, is well established. We are just becoming aware, however, that some of the more innocuous dusts may adversely affect the course of tuberculosis and other pulmonary infections.[3] It is no longer considered good practice for an individual with arrested tuberculosis to return to work in any dusty environment. We all too frequently, however, see workers with chronic bronchitis, pulmonary emphysema or even bronchiectasis who are permitted to work in an extremely dusty atmosphere. Even the increased coughing induced by a dust which is slightly irritant to the upper respiratory passages, or necessary to clear these passages of the accumulated particles, may be sufficient to aggravate a chronic pulmonary disease.[4]

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exaggeration of the lung markings and lymph node enlargement, which is followed by nodulation, and finally by coalescence and conglomeration. Tuberculosis may be super-imposed at any stage, and is believed by some to be present in all areas where coalescence has taken place. As a rule, there is no fever or weight loss in simple silicosis, and these signs may be indicative of a complicating tuberculosis. The patient tends to go downhill very rapidly with the development of an active tuberculosis lesion.

Many observers have been of the opinion that silicosis could be caused only by the crystalline form of silica, but evidence is accumulating to the effect that the amorphous form as well is capable of producing pulmonary damage. Observations upon the effects of amorphous silica[15] have been made largely on workers exposed to diatomaceous earth, which is a light fluffy material formed by the accumulation of skeletons or shells of diatoms. It has a wide use in industry as filters, fillers and absorbents. In contrast to crystalline silica, it produces fibrosis of the lungs without discrete nodulation. The fibrosis is much more marked when the exposure is to calcined material, during which process microcrystals of chrystobalite may be formed.

Another possible example of pulmonary damage due to non-crystalline form of silica occurs in workers fusing bauxite in electric furnaces in the manufacture of alumina abrasives.[16] The fumes given off in this operation consist[17] of 25% to 40% silicon dioxide and 40% to 60% aluminum oxide, together with small amounts of iron oxide and numerous other impurities. Under the electron microscope they are revealed as fused particles varying in size from 1/100 to ½ micron in diameter. A high percentage of the workers exposed to these fumes have developed cough with expectoration, weight loss, anorexia, tightness in the chest, and dyspnea on exertion. X-ray examinations show a lace-like or granular increase in the lung markings, beginning at the apices. These gradually extend to involve the entire lung fields, and may be accompanied by pleural adhesions causing distortion of the thoracic contents. Spontaneous pneumothorax is a common complication. While the aluminum oxide has generally been advanced as the etiological agent responsible for this disease, it seems quite possible that this may be a response to silica fume with a particle size far lower than that generally considered injurious.

Silica, when present only in combined form as a silicate, has usually been regarded as relatively inert. Considerable evidence, however, has been accumulating against this concept. Probably the best known of the silicates producing severe pulmonary damage is asbestos.[18] This is a mineral silicate occurring in the form of long fibers, which renders it capable of being carded, spun, and woven into threads and cloth, or bonded with cement into building shapes. Inhalation of these fibers in the course of mining or processing asbestos has been shown to be responsible for the development of a progresive pulmonary fibrosis with diffuse thickening of the alveolar walls. The outstanding symptoms are dyspnea and a dry cough. Emphysema, pulmonary infections, and secondary cardiovascular changes due to increased pulmonary resistance are the most common sequelae. Chest x-rays show a "ground glass" haziness, rather than the reticulation and nodulation of silicosis.

Another combined silicate which has been shown to be not completely inert is talc, a hydrated magnesium silicate used extensively in its powdered form as a dusting agent. It may contain variable percentages of free silica; and when these are high, the classical picture of silicosis has been observed in workers mining, crushing, grinding or otherwise handling it. Even when it contains under 1% free silica, however, as is usually the case, the dust, when inhaled, is capable of producing a fine diffuse pulmonary fibrosis similar to asbestosis.[19] It tends to be disabling, and is frequently accompanied by dyspnea, cough and fatigue. In addition, deposits of x-ray opaque material on the pleural surfaces in the form of plaques have been observed.

Still another silicate showing definite effects upon the lungs is mica, which is a double silicate of aluminum and either potassium or magnesium. This mineral, because it splits into thin sheets which are quite transparent, and have a high dielectric constant, has innumerable uses in industry, particularly in the manufacture of electrical equipment. For some time, it had been believed that the chest pathology so prevalent where it is mined was due to the free silica present in the rocks in which mica deposits occur. Even among workers exposed solely to pure mica dust in grinding operations, however, there has been observed[20] a high incidence of increased pulmonary fibrosis, with cough. X-rays show a fine granulation of uneven density with coalescence of the lesions in more advanced cases.

In all these exposures, the common denominator has been silica, whether free or combined. Recently, however, carborundum, which is an almost pure silicon carbide containing less than 1% free silica, has been suspected of being an agent capable of causing pulmonary changes indicating more fibrosis than usual.

Pulmonary Diseases of Non-Silicotic Origin

The pulmonary changes seen in coal miners has long been attributed to the free silica content of the shale through which they had to dig to get at the coal. Recent large scale studies in Wales have indicated,[22] however, that exposure to very heavy concentrations of coal dust with a negligible percentage of free silica, such as occurs among the coal trimmers on the docks, leads to a form of pneumoconiosis differing from the anthraco-silicosis of miners. There is a massive fibrosis of the lungs with nodular deposits of anthracotic pigment scattered throughout. The principal symptoms are due to the marked secondary emphysema, and the cardiac changes produced by pulmonary hypertension. Susceptibility to tuberculosis is increased. The x-ray picture is one of reticulation rather than nodulation, because of the lack of opacity of the nodules.

(Continued in the March, 1951 Issue.)

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