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Modern Occupational Medicine

Editors

A. J. FLEMING, M.Sc., M.D., F.A.C.P.
Medical Director, E. I. du Pont de Nemours & Company

AND

C. A. D'ALONZO, M.D., F.A.C.P.
Assistant Medical Director, Medical Division, E. I. du Pont de Nemours & Company

Associate Editor

J. A. ZAPP, Ph.D.
Director, Haskell Laboratory for Toxicology and Industrial Medicine
E. I. du Pont de Nemours & Company

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Chapter 33

Occupational Chest Diseases

C. W. H. Schepers, D.Sc., M.D.

Pulmonary disability resulting from occupational factors always has been and still remains an important problem in industrial medicine. In the United States alone more than 4 million industrial workers are exposed to potentially hazardous substances. The current bibliography on the subject exceeds 8,000 references, and it is obviously possible to review here only those occupational chest ailments which are of greatest topical importance. At the same time, emphasis will be placed on basic principles and pragmatic issues.

I. SILICOSIS

Because of the predominance of silica in the earth's crust, it is natural that silicosis should constitute an important occupational chest disease. Of the more than 3,000 known minerals, more than 500 are compounds of silica. It is indeed fortunate that many of these naturally occurring varieties of silica, as well as the element silica, are biologically inert. It should, however, be cautioned that epidemiologic surveys and experimental inquiry have concerned themselves with fewer than 10 per cent of these substances. In recent years, numerous synthetic siliceous substances have been introduced.

Many theories of the biological action of SiO_2 have been suggested, *e.g.*, *(a)* the mechanical injury concept, which presumed that angular quartz particles can lacerate vital cell components; *(b)* the solubility theory, silicic acid being presumed to be the pathogen; *(c)* the polymerization theory, the slow formation of polysilicic acid being invoked to explain the retarded development of silicotic nodules; *(d)* the piezoelectric theory, based on the assumption that the well-known piezoelectric forces characteristic of quartz may cause local tissue injury; *(e)* the unsaturated valency theory, which postulates the existence of a layer of highly reactive SiO molecules at the fracture angles of quartz particles; *(f)* the colloidal silica theory, which is based on the concept that the crystalline quartz particle is covered with a film of toxic colloidal silica; *(g)* the protein denaturing theory, which postulates that the physical field at the surface of the quartz particle induces organic chemical

Asbestosis is provoked wherever exposures to asbestos dust occurs. However, in textile industries there appears to be a greater prevalence of severe disease. In contrast with silicosis, the asbestotic reaction in the lung is excited not by minute particles but by relatively long fibers. Thus, if asbestos be pulverized to particles no longer than 3 microns, very little disease develops and the process advances much more slowly than when the aerosol contains a predominance of fibers of 10 to 50 microns. Again, if the fibers are introduced into the lung in a high caliber form (*e.g.* conjoined multiple fibrils), less disease results than when the individual fibers are split into their component fibrils of submicron caliber.

Asbestosis is essentially an interstitial pulmonary lesion in which all component tissues are involved with focal emphasis of the process. The lesions range in severity from mere alveolar mural cellular infiltration of a microscopic nature, not detectable by radiography or even on gross anatomical examination, to massive consolidation with associated vascular occlusion, bronchiectasis, and carcinomatosis.

The interstitial lesions result after fine short fibers have been ingested by phagocytes and transferred into the lymphatic channels of the alveolar septa. Here fibrocytes proliferate, new capillaries form and reticulin, collagen and new elastic fibers are laid down. This type of lesion cannot readily be distinguished from other interstitial pneumonitides unless the asbestos fibrils are detected by oil immersion or electron microscopy. Rarely, some of these fibrils will be rendered obvious by conversion into an asbestos body through proteinaceous encapsulation and ferrous pigmentation. If the disease remains limited to this stage, disability may remain limited being merely due to increased work of breathing.

With extension of the process three major complications arise. There may be progressive invasion of venous adventitia with perivascular fibrosis, intimal hyperplasia, and ultimately occlusion of the vascular lumen. Relatively large venous channels may become involved but arteries tend to escape early damage. This obstruction to the venous channels leads to progressive elevation of pulmonary tension and marked cor pulmonale. Heart failure is the usual cause of death in these cases. The asbestotic origin of the venous obstruction is readily proven by demonstrating the asbestos fibers in the peri- and endovascular granulation tissue. Perl's stain often is sufficient for this purpose as it will demonstrate the presence of iron deposits in the protein sheaths around the asbestos fibers. Another tell-tale feature consists of the deposition of abundant perivascular elastic laminae and fibers.

While the stage of the alveolar mural invasion is not radiographically detectable, except for blurring of lesser pulmonary markings, the stage of perivenous fibrosis is characterized on the *x*-ray by the development of a coarse web and the effacement of the normal vascular pattern. The

extent of the latter damage is often underestimated until tomography or angiography are resorted to.

Massive fibrosis tends to develop mainly in the parts of the lung which are in constant agitation, such as the lung apex, the supradiaphragmatic zone of the basal lobes, and the para-cardiac components. The consolidation is effected by progressive interstitial invasion, atelectasis, trapping of fibers within alveolar spaces where phagocytes surround them and organization takes place, and by abundant pleural thickening on both sides of the lamina elastica. In these zones carnification is often so complete that all semblance of pulmonary architectonics is completely lost. Major blood vessels can only be identified by elastic stains which reveal their elastic skeletons.

Radiographically, these consolidated zones present as diffuse and spreading opacities along the upper mediastinum, above the diaphragm and on either side of the heart, whose silhouette becomes blurred: the so-called "shaggy heart" feature. Calcification may occur in these dense zones.

Symptomatically, these patients usually are merely dyspnoeic on exertion unless there is marked accompanying vascular obstruction. In such cases cor pulmonale will be the predominating lesion. The thorax is usually small and immobile while breath sounds, vocal fremitus and resonance are markedly impaired over the affected zones. Bronchitis and bronchiolitis are not common in asbestosis, though some of these air passages may become involved in the fibro-granulomatous reaction. Advanced cystic bronchiectasis may develop as a result of the fibrotic distortion of the lung. At first there is mere bronchial distension and some fluid or mucus may become trapped in the dilated ducts. Later infection supervenes with all the attendant inflammatory processes leading to chronic suppurative bronchiectasis. The symptoms in these cases do not differ much from conventional varieties of bronchiectasis except that hemoptysis is less common and cor pulmonale develops early.

Tuberculosis may occur in asbestotic subjects, but is not more prevalent among these than it is in the general population. The lesions are not any more severe than those usually seen and often are limited by the fibrous barriers caused by the asbestosis.

Pulmonary carcinoma has been observed with such high frequency in employees of the asbestos industry that a causal relationship has been accepted by most authorities. Indeed, pulmonary carcinoma is compensable as an occupational disease in England, Germany and South Africa. Most of the carcinomata are squamous cell epitheliomata. Pleural mesothelioma is also quite prevalent, especially in crocidolite industries.

Since the lymphatic system is not completely destroyed in asbestosis, a neoplasm may spread diffusely throughout the lung in spite of extensive interstitial fibrosis. This does not readily occur in silicosis.