Textbook of Toxicology

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Air-Borne Poisons—Dusts

MANY industrial processes cause the dispersion of fine particulate matter in the surrounding atmosphere. The concentration and the chemical and physical nature of the dusts determine whether they are merely a disagreeable contaminant of the air or whether they constitute a menace to the health of workers who may be chronically exposed. Most industrial dusts can be harmful to a few individuals on a hypersensitivity or an allergic basis, but some of them are harmful to all workers who are sufficiently exposed. Dusts may come in contact with the skin and mucous membranes to cause local reactions. They may be swallowed and cause gastrointestinal irritation or systemic poisoning. However, the most important avenue of entry is the lungs. This chapter is chiefly concerned with those dusts which remain unabsorbed in the lungs after inhalation and cause pathological reactions. Lung diseases caused in this way by dust particles are called *pneumoconioses* which means dust in the lungs. The most widely known examples of this condition are *silicosis* and *asbestosis* because of their serious nature and the irreversibility of the lung lesions produced. Some dusts produce no tissue reaction in the lungs even when inhaled in relatively large amounts. Examples of these are coal dust which produces *bituminosis* or *anthracosis*, iron dust or fumes which produce *siderosis*, and barium dust which causes *baritosis*. The roentgenologic findings in the latter conditions may be confused with those of serious lung conditions. Many dusts of organic origin also produce definite clinical syndromes which will be considered briefly in this chapter. Many of the metallic dusts

ASBESTOSIS

Occurrence. Asbestosis is produced by the inhalation of hydrated magnesium silicate (asbestos). Most of the asbestos used in the United States is mined in Canada. It has been estimated that about 10,000 persons in this country are exposed to this material in connection with the manufacture and use of fireproofing, packing, or insulating materials and of fireproof or heat-resistant clothing. The incidence of asbestosis, among those exposed, however, is low.

Toxicity and Symptoms. The maximum allowable atmospheric concentration of asbestos is 5 million particles per cubic foot of air. Higher concentrations can cause asbestosis. The onset of symptoms is slow, as in silicosis, and they usually appear before any changes are seen on the lung roentgenogram. The symptoms themselves are similar to those already described for silicosis although they may be more pronounced. Cyanosis and clubbing of the fingers associated with cardiac enlargement are more frequently seen in asbestosis. There is no evidence that this disease predisposes to tuberculosis as silicosis does, but it may add to the morbidity and mortality due to other pulmonary infections. The frequent development of lung cancers in asbestos workers is discussed later in this chapter.

Pathological Changes. A fibrous reaction in the lungs is characteristic in asbestosis, as in silicosis, but the distribution of the fibrotic lesions in the former disease is diffuse and tends to predominate in the basal portions of the lungs. Bronchiectasis and bronchiolectasis are frequent in the more fibrous portions of the lungs. If the asbestos fibers are ground to particles smaller than 2 microns in length, they lose their capacity to induce a fibrous reaction when inhaled. For this reason it is thought that a mechanical action of the longer asbestos fibers is responsible for the typical fibrous tissue response.

Roentgenologic examination of the lungs shows a diffuse distribution of shadows due to the fibrosis. Pleural involvement may cause a ground glass appearance. These findings may be either bilateral or unilateral and they are usually concentrated in the

lower half or two-thirds of the lung fields. A compensatory emphysema appears in the upper parts of the lungs.

Microscopic examination of the asbestotic lungs shows a fibroblastic proliferation of the alveolar septa. There are numerous phagocytes in the alveolar spaces which become lined with a low cuboidal epithelium. In areas where the fibrosis is advanced, the alveolar structure is absent and replaced by dense fibrous tissue. Scattered throughout the lung are dumbbell or club-shaped structures 20 to 100 microns in length which are brown in color and give a Prussian-blue reaction for iron. These structures are indicative of exposure to asbestos but they may appear before clinical asbestosis has developed.

Treatment. There is no specific chemical prophylaxis against or treatment for asbestosis. The treatment is symptomatic. Prevention depends upon dust control and the reduction of the concentration of asbestos in the atmosphere to 5 million particles per cubic foot or less.

BENIGN NONSPECIFIC PNEUMOCONIOSES

This type of pneumoconiosis causes no pathological tissue reaction and symptoms are absent or not troublesome. It causes no definite predisposition to pulmonary infections. The principal importance of the benign pneumoconioses lies in the fact that in some of them there are associated roentgenologic changes which must be considered in the differential diagnosis of pathologic lung disorders. Chronic exposure to any inert nonabsorbable dust leads to an accentuation of the normal tree-like shadows cast by the pulmonary blood vessels. Superimposed on this may be many fine reticulations thought to be caused by a thickening of the sheaths of the pulmonary arteries and of the interlobular septa.

In *baritosis*, *siderosis*, and *stannosis* induced by the dusts of barium, iron or iron oxide, and tin oxide respectively, the roentgenogram often shows a widespread dense nodulation typical of that seen in silicosis. Ultimate examinations of autopsy specimens have shown that these nodulations are due to collections of the metal with no tissue reaction or fibrosis.

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Nickel. Workers in nickel-copper refineries have an increased liability to cancer of the nasal sinuses and lungs. The active agent here is thought to be metallic nickel or nickel carbonyl. Reports from South Wales showed that 34 per cent of the cancers of the respiratory organs which occurred between 1907 and 1934 developed in copper-nickel refinery workers. The average latent period for this type of malignancy was 22 years. In view of this latency it is not surprising that experimental attempts to produce cancers in mice by the use of nickel dust have given negative results.

Asbestos. Only recently has asbestos been recognized as a cause of lung cancer in workers exposed to the dust of this material. In one study of a series of 92 autopsies on patients with asbestosis, 14 cases, or 15 per cent, had cancer of the lung. The exposure time ranged from 3 to 27 years and the ages of the patients ranged from 35 to 75 years. Similar incidence of cancer has been reported in several other studies. The experimental production of lung cancer in mice by asbestos dust has also been conclusive. Twenty per cent of the animals developed squamous cell cancer originating from the bronchial mucosa.

Other Possible Dust Carcinogens. Occupational activities other than those described above also carry an increased liability to cancer of the respiratory tract. The inhalation of *hot tar fumes* by stokers in generator plants and of *radioactive* dusts and gases by miners of radioactive ores has resulted in an excessive incidence of pulmonary cancers. Although *beryllium*-containing dusts have not yet been established as being carcinogenic to the lungs, their potentiality in this respect cannot be disregarded. Experimental malignancies have not been produced by inhalation of beryllium in animals. Intravenous injection of zinc beryllium silicate and beryllium oxide in rabbits, however, almost invariably causes highly malignant sarcomas of the bones within 5 to 7 months. In man the sarcoid granulomas which develop in the lungs of workers inhaling beryllium dusts represent a blastomatoid reaction bordering on the malignant.

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