

to assume that the fibrosis is simply the result of the action of free silica which is present in sufficient concentration to induce proliferative cell reaction. Because it occurs chiefly in hard coal miners, it has been known for many years as "miner's asthma."

The cardinal symptom is shortness of breath, frequently associated with "productive cough. More advanced cases complain of weakness, chest pain, gastric disturbances and hemoptysis. Sayers⁹ states that in a U. S. Public Health Survey of anthracite miners in Pennsylvania in 1933, fever and night sweats were seldom mentioned. (This is true of silicosis cases in iron ore miners.) He notes such physical signs as dyspnea, prolonged expiration, change in contour of the chest, decreased chest expansion, clubbing of the fingers, change in breath sounds, altered fremitus and impaired resonance. Where infection complicated the picture, the symptoms were more marked and included cyanosis and loss of weight and strength.

The pathology of anthraco-silicosis is characterized by accumulations of coal dust in the lungs associated with varying degrees of pulmonary fibrosis. The lungs are gray and firm and may show dark colored markings on the pleural surfaces. On section, areas of black pigmentation appear scattered throughout the lung fields. Coalescence of the nodules produces larger areas, particularly in the hilar regions, but many large discrete nodules appear throughout the parenchyma.

Fibrous hyperplasia can be seen along the lymphatics. Microscopic section reveals deeply pigmented areas of fibrous connective tissue with dust laden macrophages around the outer border of the nodule.

The earliest x-ray evidence of anthraco-silicosis consists of exaggeration of the linear markings which increases with continued exposure, until the general pattern is obscured and replaced by definite nodulation. Further progress of the disease will be evidenced by more massive conglomerate shadows which may be complicated by the presence of infection.

Asbestosis as an occupational disease has been recognized only in recent years. Forty-one deaths were reported from this cause in England up to 1934. In this country it has been estimated by Lanza that approximately 10,000 persons are exposed to asbestos dust. In a study by Dressen, Edwards and Miller¹⁰ of the U. S. Public Health Service, 541 asbestos workers were examined. No cases of asbestosis were found among workers exposed to dust concentrations below 2,500,000 particles per cubic foot of air.

THE greatest occupational hazard exists in mining, handling and crushing crude asbestos, making insulation and the carding and weaving of asbestos. In other industries such as the compounding of materials for automobile brake linings, the hazard is recognized but the disease is uncommon.

Asbestos is a hydrated magnesium silicate. It is the one silicate which does produce a fibrosis, but this differs pathologically and roentgenologically from the nodular reaction of silica. Industrial asbestos dust is comprised of very small fibres. Only the larger ones, over 2 microns in length, are thought to be capable of producing fibrosis. Gardner¹¹ is of the opinion that inhaled asbestos fibres are irritating not because they are silicates, but because they are stiff fibres which mechanically irritate the lungs. When the fibres used in animal experiments were finely ground (under 2 microns), the irritating property of asbestos practically disappeared. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body other than the lungs. While the action of free silica is chemical, that of asbestos is mechanical. The

typical lesions, observed microscopically, show large macrophages in the distal portions of the bronchial tree, the formation of the asbestos giant cell, and the generalized fibrosis surrounding the bronchioles, alveoli, air sacs and blood vessels. This results in obliteration of the lung structure. The alveoli have literally become plugged, and the function of the lung impaired mechanically.

Merewether and Price¹² examined 363 workers exposed to practically pure asbestos dust in factories in Great Britain. The following table indicates their findings as regards exposure and fibrosis.

TABLE III.

Years at Work	Cases Examined	Showing Fibrosis	Per Cent
0 to 4	89	0	0.0
5 to 9	141	36	25.5
10 to 14	84	27	32.1
15 to 19	28	16	53.6
20 and over	21	17	80.9

As in cases of fibrosis produced by other dusts, there is no typical symptom of asbestosis. Dyspnea is the most striking feature of the disease. The onset is gradual, and the symptoms increase as the condition advances. Cough, expectoration, cyanosis, loss of weight, and emaciation are late occurrences probably associated with infection.

A feature of asbestosis is the occurrence of so-called asbestos bodies. Cook¹³ states that "the 'curious bodies' so characteristic of pulmonary asbestosis are found in the alveoli and bronchioles and in the fibrous and necrotic areas. They measure 20 to 100 microns in length. One or both ends are bulbous, giving a clubbed or dumb-bell appearance. The shafts are either homogeneous or segmented crosswise. They are golden yellow to brownish in color. They do not stain but give a Prussian blue reaction to iron. From a diagnostic standpoint, it is generally agreed that the 'curious bodies' signify exposure to asbestos dust but cannot be depended upon for a diagnosis of asbestosis." Apparently the asbestos body is formed from the original fibre by a tissue reaction, the nature of which is still obscure. The fact that segmented figures are not found in crude asbestos or asbestos dust indicates that they are formed only after the fibres have come in contact with living tissue.

Another interesting skin lesion found in this disease is the "asbestos corn" due to the penetration of fibres into the superficial layers of the skin.

To establish a diagnosis, a history of exposure to asbestos dust is essential. The length of this exposure is important and should be correlated with the chest roentgenogram in which the "ground glass appearance" is characteristic. The lesions are limited to the lower halves of the lungs and there is hyperventilation in the upper portions. The cardiac outline becomes blurred and the domes of the diaphragm obliterated. The appearance of the heart and lower chest may suggest cardiac disease which should be definitely ruled out in a worker in an asbestos plant before diagnosing his condition as asbestosis. The diagnosis of asbestosis from the x-ray is not an easy matter, but the following features are fairly characteristic:

1. Lesions basal in character.
2. Hyperventilation in upper lobes.
3. Blurred cardiac outline.
4. Obliteration of the diaphragm.
5. No nodulation.
6. "Ground glass" appearance.
7. May be unilateral.

The occupational hazard of asbestos is not particularly significant. Its recognition as a clinical entity