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DISEASES of the CHEST

With Emphasis on X-ray Diagnosis

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The Principles of Surgical Treatment

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355 ILLUSTRATIONS

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Fig. 178.—Silicotuberculosis showing grayish-black appearance of lung due to associated anthracosis; ulcerative tuberculosis in the upper lobe. Roentgenogram reveals nodular silicosis and increased hilar densities. *A*, Silicotic nodules consisting of dense cores of fibrous tissue surrounded by laminated whorls of hyaline fibers. *B*, Spindle-shaped structures (asbestos bodies) which give a prussian blue reaction for iron. (Copyright, Ciba Pharmaceutical Products, Inc., Summit, N. J.)

ized extension into adjacent lung fields often directs the physician's attention to the possible existence of a dust disease. Pendergrass has suggested the term "modified silicosis" to cover such instances where abnormal shadows are noted in the roentgenogram in the absence of a definite history of exposure to a silicious atmosphere or where exposure has been to a mixture of dusts. A presumptive diagnosis of "modified silicosis" is justified on the basis of the roentgen findings, to be confirmed or disproved by additional studies.

Several physicians have drawn attention to peculiar eggshell calcifications occasionally encountered in the roentgenogram of silicotic lungs. These nodules are spheroid in shape, having an apparently calcified casing around them. They may occur within the parenchyma or in the hilar glands. A number of explanations have been offered to explain their peculiar configuration. It has been postulated that they represent calcified areas of tuberculosis infection or silicotic nodules or calcified degeneration in or around silicotic nodules. Riemer suggests that the shadows result from direct inhalation of calcium along with silica particles. As additional evidence for his belief he cites studies on calcium dust inhalation showing analagous roentgen shadows.

Clinical Features.—A diagnosis of silicosis cannot be made on the basis of symptoms and physical findings alone, although the presence of the condition may be suspected in communities where silicosis is prevalent. A detailed occupational history and a roentgenogram are indispensable. What may seem to an individual a trivial occupation, particularly if he was engaged in it many years before, may prove to be important as a possible source of lung injury. On the other hand, a man who has spent many years in a mining industry may not have contracted a dust disease. A recent experience was quite illuminating. A man of twenty-nine came to me for a routine chest examination because his wife was found to have advanced pulmonary tuberculosis. The roentgenogram revealed moderate accentuation of the hilar markings and linear, in places nodular, densities in the midportions of both lungs, chiefly the right (Fig. 185). The appearance of the roentgenogram did not suggest tuberculosis. The patient's occupation at the moment was that of a fruit merchant. On closer questioning, however, it was found that for almost three years, during the war, he had worked on ships as an electric arc welder of steel plates. Part of the time he used galvanized steel with a coating of zinc and the rest of the time black iron without a coating. Although he had some protection against the fumes, it seemed likely that the roentgen changes were due to inhalation of metal fumes, a recognized source of pulmonary injury.

The past history may reveal increased susceptibility to infections of the upper respiratory tract, pleurisy, recurring pneumonias or "asthma." The initial stages of simple silicosis seldom cause significant symptoms except for unaccountable dyspnea which is out of proportion to the extent of the disease. With greater lung involvement, the dyspnea becomes increasingly severe and is soon associated with cough, at first dry, later productive, the sputum often mixed with blood. The individual may have a feeling of tightness in the chest, at times of a painful nature. Increasing weakness and vague abdominal complaints are frequent. Silicosis with infection is characterized, in addition, by abundant expectoration, at times frank hemoptysis, fever, sweating and marked loss of weight. Although dyspnea and cyanosis may be evident even when the individual is at rest, orthopnea is not seen unless there is also cardiac insufficiency caused by increasing interference with the lesser circulation. Engorgement of the veins in the neck, enlargement of the liver and spreading edema appear later. The few who escape tuberculosis or cardiac failure are apt to succumb to intercurrent pneumonia.

Several observers have found a greater incidence of carcinoma of the lung in silicotics than among nonsilicotics. The role played by silicosis in carcinogenesis is obscure. Many doubt that a relationship exists between the two, their presence in the same lung being ascribed to age, sex and other factors which favor the development of carcinoma.

The physical findings are not illuminating. Silicosis of advanced degree may be present in an individual who appears well nourished and in the best of health. Examination of the chest reveals evidence of emphysema. There is diminished costal and diaphragmatic excursion, the expiratory phase especially being prolonged. The finger tips may show clubbing. There is usually hyperresonance on percussion

roentgen examinations facilitate the detection of incipient stages of the disease.

ASBESTOSIS

Asbestos is a hydrated silicate of magnesium in combination with traces of iron, nickel, calcium and aluminum. The substance is mined in many parts of the world, including Canada, where most of the American supply (chrysotile) is obtained. Mined asbestos comes in long, thin, fibrous strands which can be spun or woven. Its pliable texture and high resistance to heat and chemicals make asbestos an important industrial product in the manufacture of mattresses, brake-lining, fire-proofing material, electrical insulation, in jacketing boilers and steam pipes, and in a variety of building fixtures. Although the dust hazard associated with the asbestos industry does not nearly compare in prevalence with that of the silica industry, the rapid growth of the former puts asbestosis among the important forms of dust diseases.

Inhaled asbestos fibers range in size from 10 to 200 microns or more. Their action on the lungs is mechanical rather than chemical. The large particles, unable to enter the alveoli, lodge in the lumen and obstruct the respiratory bronchioles. Atelectasis of the distal alveoli is followed by a fibrotic reaction in the collapsed tissue. Nonobstructed alveoli undergo compensatory emphysema. Contrary to that seen in silicosis, there is practically no nodulation unless silica is mixed with the asbestos dust. The hilar lymph nodes are not much enlarged for the reason that the lymphatics are not actively engaged in the pathologic process.

The gross appearance of the lung is characterized by scattered areas of diffuse fibrosis, affecting chiefly the lower lobes, emphysema of the uninvolved parts and intense pleuritis. Depending on the extent of coexisting anthracosis and silicosis, there are associated changes and pigmentation of the lungs and lymph nodes. Infection with pyogenic organisms and tubercle bacilli modify the pathology. Tuberculosis has been found in about one-third of the autopsied cases, but there is some doubt as to whether asbestosis per se favors the development of tuberculosis, or whether vulnerability to tuberculosis is primarily due to poor working conditions and incidental factors.

A striking feature of the pathology is the presence of "asbestos bodies" seen on histologic examination of lung tissue. These golden yellow or brown bodies have been shown by Gloyne to be composed of a central core of asbestos fibers, covered by a layer of iron-containing material which is believed to be derived from blood pigment of the tissues. Asbestos bodies may be found in the sputum of asbestos workers, but their presence does not necessarily indicate lung disease.

The roentgen appearance of early asbestosis is not revealing. Advanced disease often shows distinguishing characteristics. The fine pulmonary fibrosis, patchy areas of interspersed emphysema and overlying pleuritis are reflected roentgenologically in a "ground-glass" appearance of a uniform quality, in places showing denser opacities which, however, seldom assume the nodulation of silicosis (Figs. 183, 184). The lower portions of the lungs are chiefly involved. A marked pleural reaction manifests itself in obliteration of the costophrenic sinuses, an unevenness of the diaphragm and a felted or "porcupine" appearance at the periphery of the cardiac silhouette, the last caused by pleuropericardial adhesions.

The onset of the disease is insidious with gradual increase in cough, expectoration, dyspnea, loss of weight and, in time, inability to work. The physical examination is not revealing. Wood and Gloyne draw attention to a peculiar earthy complexion of the face and a slight, violet tinge in the cheeks and lips of some individuals. Occasionally, asbestos corns occur in the skin of the hands caused by the penetration of asbestos fibers into the superficial epidermis. It takes, on the average, between five to ten years for asbestosis to develop. Whether the disease can progress after contact is broken with asbestos dust is still unsettled. Death results from tuberculous or nontuberculous infection, congestive heart disease or other intercurrent diseases. The measures listed for the prevention of silicosis apply also to asbestosis.

Several other silicates have been reported to cause pneumonoconiosis. Fullers' earth, which is used extensively in bleaching fats and oils, has been found to cause pathologic changes in the lungs with symptoms similar to those seen in silicosis. Beryllium dust, a silicate compound, used in the manufacture of

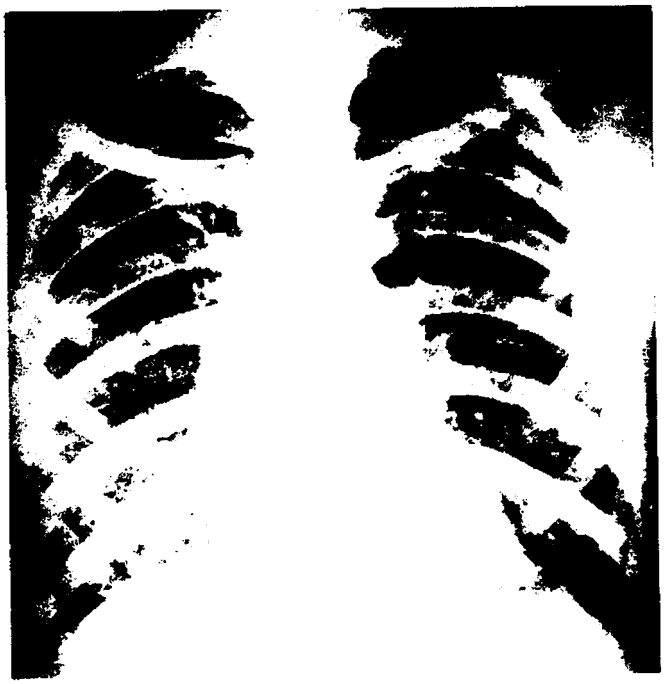

Fig. 183.—Asbestosis in a man of 31 exposed to asbestos dust for five years. Roentgenogram reveals fine reticular infiltrations affecting chiefly the lower portions of the lungs. (Courtesy, Dr. E. P. Pendergrass, Hospital of the University of Pennsylvania, Philadelphia, Pa.)

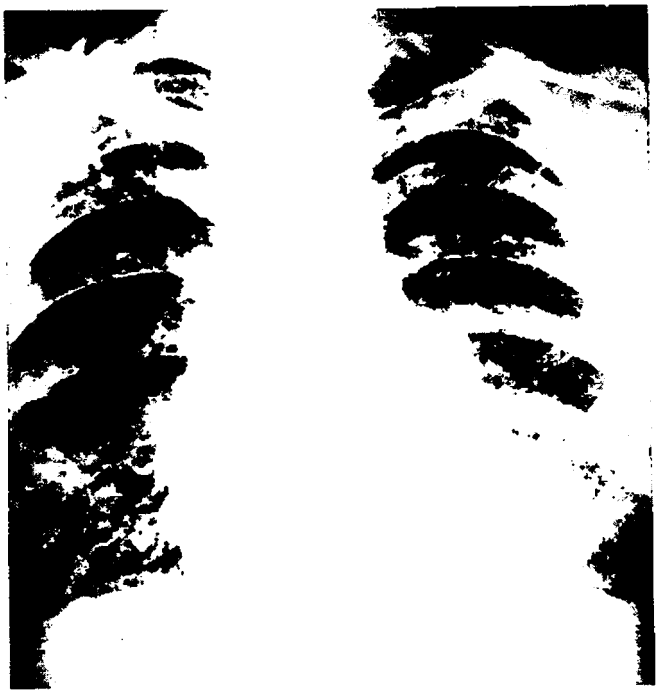

Fig. 184.—Asbestosis in a man of 53 who worked for an asbestos company part time for eight years and full time for thirty years. Fourteen fellow workers died of asbestosis. Roentgenogram reveals nodular and dense, diffuse linear infiltrations involving both lungs with intervening emphysema. (Courtesy, Dr. E. P. Pendergrass, Hospital of the University of Pennsylvania, Philadelphia, Pa.)