PLAINTIFF'S EXHIBIT AB-77
PENGAD-Bayonne, N.J.

# Chest Conditions Simulating Silicosis

L. E. HAMLIN, M.D., F.A.C.S.,
*Medical Director,*
American Brake Shoe Company,
Chicago

IN RECENT years the recognition of true occupational fibrosis has become more difficult because of an ever increasing number of conditions which produce patterns in the roentgenogram resembling silicosis. These are by no means confined to diseases caused by the inhalation of dust but range all the way from certain heart afflictions to the exaggerated linear markings representing vascular changes in the lungs associated with advancing age. Their careful appraisal as a health consideration as well as an industrial complication therefore becomes more essential as new complexities appear.

The following classification of diseases whose x-ray pattern may be confused with that of silicosis is offered not as a complete listing but as one which includes those derived from a search of the available literature. Mycotic infections have been placed in the non-industrial group because the majority of them occur without any relationship to specific occupations. However, they do result, in some instances, from occupational exposure.

**Chest Conditions Whose X-rays Simulate Silicosis**

| | | |
|---|---|---|
| A. Industrial | | |
| I. Organic | (a) | Byssinosis |
| | (b) | Bagassosis |
| | (c) | Tabacosis |
| II. Inorganic | (a) | Anthracosis |
| | (b) | Radiopaque Deposits— |
| | | Barium (Baritosis) |
| (Inert) | | Iron (Siderosis) |
| | | Tin |
| (Toxic) | (c) | Beryllium |
| (Proliferative) | (d) | Asbestosis |
| B. Non-Industrial | | |
| I. Mycotic | (a) | Aspergillosis |
| | (b) | Moniliasis |
| (Yeasts | (c) | Wood dust spores |
| and | (d) | Coccidioidomycosis |
| Molds) | (e) | Blastomycosis |
| | (f) | Actinomycosis |
| II. Miscellaneous | (a) | Miliary tuberculosis |
| | (b) | Sarcoidosis |
| | (c) | Mitral Stenosis |
| | (d) | Cancer (Metastases) |
| | (e) | Vascular changes |
| | (f) | Miliary calcicosis (Wheatena) |
| | (g) | Polycythaemia Vera |

Since the year 1818 certain pulmonary conditions are believed to have developed as a result of exposure to dust in the cotton industry. Reports from Europe as well as from this country indicate that prolonged exposure to high concentrations of cotton dust have caused definite symptoms and permanent disability. In England the Byssinosis Act of 1940 awards compensation for total disability following periods of exposure in cotton mills up to 20 years. In the United States the existence of byssinosis as an occupational disease is questionable. A few cases have been reported from Eastern textile mills but after a very thorough investigation of the cotton industry in Mississippi in 1944, Ritter and Nussbaum were unable to demonstrate any x-ray findings characteristic of the disease, either in employees in the cotton mills or in the files of private roentgenologists or the State Tuberculosis Sanatorium. They did find certian allergic individuals who showed very definite asthmatic reaction to high concentrations of cotton dust, but otherwise clinical evidence was lacking. Postmortem and histological examinations revealed nothing of a specific nature. They concluded that "no evidence was found to clarify or even support the existence of byssinosis as a clinical entity arising among employees exposed to Upland cotton in a cross section of that industry in Mississippi."

Bagassosis is the name given to an industrial disease observed in a few workers in the sugar-cane industry. The disease is described by Castleden and Hamilton-Patterson and also Gillison and Taylor in the *British Medical Journal* in 1942. They state that "bagasse," a waste product after the sugar has been extracted, is used in the manufacture of board. The material is crushed or shredded into small fragments, the fibres of which contain about 1% protein and 5 to 7% silica. Symptoms after one to two months' exposure, consisted of dyspnoea, cyanosis, violent cough, scanty sputum, occasionally blood streaked, and mild elevation of temperature. The x-ray suggested a bilateral pneumonia of the influenzal type which either resolved completely after several weeks or left the patient with some degree of residual fibrosis. As in the case of byssinosis an allergic response in the lungs to this dust was a definite possibility.

Tabacosis has been described as far back as 1865 as producing disease in the lungs. However, very few cases seem to have been reported. In Argentina and Brazil the condition is considered an occupational disease and is compensable. C. F. Long, after a period of 10 years experience among 4,000 workers in the tobacco industry, made a special investigation of over 1,247 who had been subjected to tobacco dust and compared them to 1,007 who had never been exposed. He concluded that "Tabacosis does not develop in the lungs of those who inhale tobacco dust."

Some inorganic dusts produce alterations in the lungs which are represented by a mild accentuation of the linear markings in the x-ray. Included in this group are dusts from such materials as limestone, marble, talc, chalk, calcined magnesium for insulation, furnace linings, carbon and cement. Since they are relatively inert and non-productive of symptoms, their presence is only detectable by x-ray and the shadows observed may resemble the exaggerated lineal pattern of pre-silicotic stages.

Dusts from substances like barium, iron and tin, can result in radiopaque deposits in the perivascular lymphatics and interalveolar septa which cast shadows in the roentgenogram closely simulating those of discrete, nodular silicosis. The deposits have real sig-

Presented at the Annual Meeting of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS at Chicago, April 11, 1946.

nificance from the standpoint of litigation but are of no apparent consequence as far as health or disability is concerned. In 1938 Sander and Enzer described findings in welders who had no known exposure to silica but whose chest x-rays showed marked nodulations almost indistinguishable from silicosis which were demonstrated to be the result of inhalation of iron oxide fumes. The borders of these shadows were generally more sharply defined than those of silicotic nodules and the hilium shadows less prominent. Postmortem findings on one case, who died from accident, gave no indication of fibrosis either grossly or histologically. On microscopical section, deposits observed about the blood vessels were simple pigmentations which gave the typical Prussian Blue reaction for iron. Development of clinical symptoms and susceptibility to complicating infection were lacking.

In recent years our own experience with foundry grinders and burners seems to indicate that much of the nodulation observed in films of men working at these occupations does not represent actual silicosis but is the result of excessive exposure to iron oxide, similar to that described by Sander. Clinical findings and occupational exposure are not consistent with the present conception of silicosis. The analysis of representative samples of air borne dust by x-ray diffraction, petrographic and chemical examination (over a three year period) indicates that there is not enough free silica in the atmosphere to constitute a definite hazard but enough iron oxide in finely divided form does exist to produce the pattern noted in the x-ray. Experimental work is now being carried on in an effort to determine the effects of this particular dust on laboratory animals.

A comparatively new disease entity caused by beryllium and its compounds has recently been established. Beryllium is a strategic metal which was of considerable importance during the war in the production of precision instruments. The exact effect of this metal on the tissues of the body has not been determined but its action is thought to be a toxic one, producing dermatitis, nasal irritation, skin ulcer and chemical pneumonitis. Van Ordstrand and his associates have reported their observations and findings in 170 cases of poisoning among workers in the beryllium industry. They noted that the disease was not limited to men in one type of work or in one phase of production and the incidence and severity were proportional to the degree of exposure and chemical irritation of dusts and fumes. The chemical pneumonitis which developed in 88 workers represented the severest form of the disease and progressed without exacerbation or remission, either to complete recovery or to death. Roentgenologic changes in the lung fields did not appear until two to three weeks after the onset of symptoms and physical signs. Changes were bilateral and diffuse in all cases and varied with the severity of the disease. In order of appearance the changes were (1) diffuse haziness of both lungs, (2) development of soft irregular areas of infiltration with prominence of peribronchial markings, (3) absorption of soft infiltration and appearance of discrete large or small conglomerate nodules scattered throughout both lung fields and (4) clearing of the lung fields after one to four months. Necropsies on five patients showed a typical pneumonitis. Fibroblasts with evidence of organization were present although in patients who recovered, no fibrosis was evident on roentgenologic examination of the chest.

The so-called fibrosis due to asbestos fibres in the lungs has long been recognized as an occupational disease. It does not seem to occur frequently [illegible] publicity it has received would indicate. A hydrated magnesium silicate, it is believed to produce its effects through mechanical plugging of the alveoli by long fibres (over two microns) which have an irritating effect on the lung tissue. There is some associated fibrosis which is responsible for the characteristic ground glass appearance observed in the roentgenogram. The greatest occupational hazard exists in mining, handling and crushing crude asbestos.

Fungi are filamentous plants of simple structure which include a variety of yeasts and molds capable of producing disease in the lungs as well as in other structures of the body. A common fungus, aspergillus, consists of many species of molds one of which, aspergillus fumigatus, attacks the lungs and is responsible for the disease known as aspergillosis. Monilia albicans, a yeast-like fungus, may produce primary lesions in the lung or sometimes gain access as a secondary invader. Infection with any type of this mold produces "Moniliasis," a disease which may affect the skin as well as the pulmonary structures. Coccidioidomycosis, Sporotrichosis and Blastomycosis are due to yeast-like organisms which generally cause a progressive infection affecting the mucous, cutaneous, subcutaneous, osseous and visceral structures. Involvement of the lungs does occur in some cases directly through inhalation of spores, and sometimes fungi may be superimposed on existing diseased area.

In 1931 Towey, Sweany and Huron described a previously unrecognized disease which occurred in 35 workers who came in close contact with dust from maple logs. The logs had been cut for more than a year and were not disturbed throughout a hot, moist summer, a circumstance favoring fungus growth. The dust, which was of the consistency of lamp black, accumulated beneath the cork layer of the bark and was liberated during peeling or sawing operations. On examination it was found to be pure spores of a fungus tentatively classified as nummularia or coniosporium corticola.

Symptoms produced by inhalation of the spores were acute and asthmatic in character. They included dyspnea, cough, loss of weight and expectoration. The condition was considered an allergic response to sensitization by the fungus combined with a possible toxic element in the spores.

X-ray findings were proportionate to the degree of dyspnea observed and consisted of definite mottling throughout the lower halves of the lungs with increased hilar, basal and trunk shadows. Peribronchial markings were increased throughout both lungs, except in the apices. Physical improvement with clearing of the x-ray occurred in all cases within two to three months after removal from exposure.

The general characteristics of mycotic infections of the lungs are sufficiently alike to warrant their consideration as a group. The x-ray findings may show some variation, as they do in silicosis, but usually they range from accentuated linear markings to miliary-sized densities which may coalesce. Advanced cases may show discrete nodules scattered throughout the parenchyma of the lungs. The differentiation from silicosis, tuberculosis, metastatic cancer, sarcoidosis, etc., can be difficult and diagnosis in individual cases should be based on correlation of clinical evidence and exposure with the roentgenogram.

Diseases such as coccidioidomycosis are endemic in some localities and while no region is exempt, a higher incidence occurs in certain areas. There does not appear to be any occupational prevalence, but cases of mycotic infection do occur in certain industries. Recently a case of primary bronchopulmonary aspergil-